1.0 Reference: CSTS IR1 20.1

FortisBC response says:

"Safety Code 6 is a legally binding standard in Canada. The basis and provisions of Safety Code 6 are similar to standards developed by many other national and international scientific, health, and governmental agencies."

1.1 Please confirm that Safety Code 6 refers to the Health Canada Safety Code 6, Limits of Human Exposure to Radiofrequency Electromagnetic Fields in the Frequency Range from 3 kHz to 300 GHz, issued by Health Canada in 1999, and updated by Health Canada in 2009.

Response:

Confirmed.

1.2 Please confirm that the Preface section of Safety Code 6 (2009) indicates that the code establishes safety limits for human exposure to RF in the frequency range from 3 kHz to 300 GHz, that these safety limits apply to all individuals working at or visiting federally regulated sites, and that the code has been adopted as the scientific basis for the equipment certification specifications outlined in Industry Canada's regulatory compliance documents that govern the use of wireless devices in Canada, such as cell phones, cell towers (base stations) and broadcast antennae.

Response:

Confirmed.

1.3 Please confirm that the federal government sets technical requirements and standards for radio apparatus, interference-causing equipment and radio-sensitive equipment in Canada under s. 5(1)(d) of the Radiocommunication Act, and that s. 2.1 of the Radiocommunication Regulation specifies that the standards are set out in the Category I Equipment Standards List and the

Category II Equipment Standards List, both as amended from time to time, and both published by the Department of Industry.

Response:

Confirmed.

1.4 Please confirm that the Category I Equipment Standards List and the Category II Equipment Standards List both include Radio Standards Specifications (RSS) 102, Radio Frequency (RF) Exposure Compliance of Radiocommunication Apparatus (All Frequency Bands).

Response:

Confirmed.

1.4.1 Please confirm that RSS 102 sets out the requirements used to evaluate RF exposure compliance of radiocommunication apparatus designed to be used within the vicinity of the human body, including mobile, portable and fixed transmitters having an integral antenna, systems requiring licensing with detachable antennas sold with the transmitters, or licence-exempt transmitters with detachable antennas as defined in RSS-Gen.

Response:

Confirmed.

1.4.2 Please confirm that RSS 102 states that proponents and operators of antenna system installations are responsible for ensuring that all radiocommunication and broadcasting installations comply at all times with Safety Code 6; and that section 4 of RSS-102 states that “for the purpose of this standard [RSS-102], Industry Canada has adopted the

SAR and RF field strength limits established in Health Canada's RF exposure guideline, Safety Code 6.

Response:

Confirmed.

1.5 Please confirm that section 7.23 of B.C.'s Occupational Health and Safety Regulation requires that equipment producing radiofrequency must be installed, operated and maintained in accordance with Health Canada Safety Code 6, Limits of Human Exposure to Radiofrequency Electromagnetic Fields in the Frequency Range from 3 kHz to 300 GHz, 1999, as amended from time to time.

Response:

Confirmed.

1.6 Please confirm whether FortisBC's AMI program will comply with Safety Code 6.

Response:

FortisBC confirms that its AMI program will comply with Safety Code 6.

1.7 Please confirm that the radio frequency exposure from a smart meter, measured as specified by Safety Code 6, should take duty cycle into account.

Response:

FortisBC confirms that the radio frequency exposure from an advanced meter, measured as specified by Safety Code 6, should take duty cycle into account.

1.8 Please provide the maximum radio frequency exposure from a FortisBC smart meter based on Safety Code 6 measurement standards that properly account for the duty cycle.

Response:

Please see Table 8.4.2.a from the Application (Exhibit B-1) which sets out the radio frequency exposure Safety Code 6 limits at 0.5 meters, and the related exposure from a FortisBC AMI meter at the mean duty cycle, the maximum typical duty cycle, and the maximum supported duty cycle.

FortisBC notes that BC Hydro has commissioned reports to further quantify the duty cycle and number of transmissions for the same Itron meters that FortisBC has selected for use in the proposed AMI project. The real-world measured data collected during these studies suggests that the total duty cycle and the number of transmissions for the max, min and average meter will be reduced from the numbers FortisBC has provided in the Application and in response to CEC IR2 Q34.1. Please refer to Appendix CEC IR2 34.1 for these studies and their conclusions.

2.0 Reference: CSTS IR1 34.4 and BCRUCA IR1 7.1

FortisBC response says:

In matters related to health, FortisBC relies on the expertise of the Provincial Health Officer, Health Canada, and World Health Organization, who have all confirmed that wireless meters pose no known health risk or reason for concern.

In situations requiring accommodation, FortisBC will assess extenuating circumstances for individual customers on a case-by-case basis.

2.1 Please provide the Statement of the Chief Medical Health Officer of British Columbia, dated June 2011, regarding health concerns about cellular phone transmission antennae and base stations, located at:

http://www.vch.ca/media/CMHO_CellPhones-June2011.pdf

1 **Response:**

2 The requested document is provided as Attachment BCH 2.1.

Vancouver CoastalHealth
Promoting wellness. Ensuring care.

Statement of the Chief Medical Health Officer

June, 2011

Health Concerns About Cellular Phone Transmission Antennae and Base Stations

In 2005, in response to community concerns and after reviewing the evidence, the Vancouver Coastal Health Chief Medical Health Officer concluded that the installation of cellular antennae in the community did not create health risks for the public, and that Health Canada's Safety Code 6 provided an appropriate level of protection. At that time, the Chief Medical Health Officer also committed to undertake periodic reviews of the evidence and to provide public updates as necessary. The Chief Medical Health Officer provides the following updated evidence review and associated conclusions:

Background on Cellular Transmission Technology

The original cellular (analog) technology uses the radiofrequency part of the electromagnetic spectrum between 800-900 MHz (near the FM/TV, AM Radio bands and cordless telephone frequencies). The newer digital technology uses the frequency bands of 800-900 MHz and 1800-2200 MHz and relies on antennae of significantly less power than the analog system, emitting significantly lower radiofrequency (RF) radiation. Cellular communication operates through a network of base stations that transmits and receives signals. The area covered by a base station is called a cell – giving rise to the name cell phone. The number of base stations (cells) in an area varies, depending on the concentration of cell phone users. For example, compared to smaller communities, the number of base stations is greater in populated urban centres, with many cell phone users. Each base station consists of signal processing equipment, power supply, and one or more antennae. The antennae are the most visible parts of base stations. However, a network of many lower powered based stations may result in lower levels of RF radiation exposure to the public compared to a network that uses a few higher powered base stations covering the same area. This is because the power required to communicate between a cell phone and base station increases as the distance between the cell phone and the base station increases.

Conclusions At A Glance

1. The international scientific consensus remains unchanged: radiation from cellular base stations is far too low to cause adverse health effects in the community.

2. There is no public health benefit from prudent avoidance regarding base stations.

3. Telecommunication regulators and the industry need to be explicitly transparent in engaging communities and providing access to monitoring data to show compliance with expected standards.

To meet the demand for service, increasing numbers of cellular base stations have been installed across the country. However, it is not easy for the public to access information on the number, types, and locations of cellular base stations in their community. This difficulty has contributed to public concerns regarding potential harm from these installations.

Health Risks

The study of RF radiation and its possible effect on health is growing steadily. Since the last report in 2005, reviews from recognized scientific organizations include the International Commission on Non-Ionizing Radiation Protection (ICNIRP) 2009 Review, the European Commission Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) 2009 Review, the Swedish Radiation Safety Authority, SSM, Independent Expert Group on Electromagnetic Fields 2009 Report, and the Health Canada Safety Code 6 revised in 2009. The scientific consensus remains unchanged: radiation from cellular base stations is far too low to cause adverse health effects in the community. The current Canadian (Safety Code 6 revised 2009) and international standards such as ICNIRP provide significant safety margins for public exposure to RF.

In Safety Code 6 (2009), Health Canada states:

> *"The scientific literature with respect to possible biological effects of RF energy has been monitored by Health Canada scientists on an ongoing basis since the last version of Safety Code 6 was published in 1999. During this time, a significant number of new studies have evaluated the potential for acute and chronic RF energy exposures to elicit possible effects on a wide range of biological endpoints including: human cancers (epidemiology); rodent lifetime mortality; tumor initiation, promotion and co-promotion; mutagenicity and DNA damage; EEG activity; memory, behaviour and cognitive functions; gene and protein expression; cardiovascular function; immune response; reproductive outcomes; and perceived electromagnetic hypersensitivity (EHS) among others. Numerous authoritative reviews have summarized this literature.*
>
> *Despite the advent of thousands of additional research studies on RF energy and health, the predominant adverse health effects associated with RF energy exposures in the frequency range from 3 kHz to 300 GHz still relate to the occurrence of tissue heating and excitable tissue stimulation from short-term (acute) exposures. At present, there is no scientific basis for the premise of chronic and/or cumulative health risks from RF energy at levels below the limits outlined in Safety Code 6. Proposed effects from RF energy exposures in the frequency range between 100 kHz and 300 GHz, at levels below the threshold to produce thermal effects, have been reviewed. At present, these effects have not been scientifically established, nor are their implications for human health sufficiently well understood. Additionally, a lack of evidence of causality, biological plausibility and reproducibility greatly weaken the support for the hypothesis for such effects. Thus, these proposed outcomes do not provide a credible foundation for making science-based recommendations for limiting human exposures to low-intensity RF energy."*

Critics of Safety Code 6 have challenged the adequacy of the Canadian standard to protect the public from effects other than those resulting from the thermal heating of cells in the body. However, when scientifically sound methods are used to assess the evidence, Health Canada's conclusions are consistent with the conclusions reached by other credible scientific bodies. In its review of evidence in 2009, the ICNIRP states:

> *"It is the opinion of ICNIRP that the scientific literature published since the 1998 guidelines has provided no evidence of any adverse effects below the basic restrictions and does not necessitate an immediate revision of its guidance on limiting exposure to high frequency electromagnetic fields. The biological basis of such guidance remains the avoidance of adverse effects such as "work stoppage" caused by mild wholebody heat stress and/or tissue damage caused by excessive localized heating (D'Andrea et al. 2007). With regard to non-thermal interactions, it is in principle impossible to disprove their possible existence but the plausibility of the various non-thermal mechanisms that have been proposed is very low. In addition, the recent in vitro and animal genotoxicity and carcinogenicity studies are rather consistent overall and indicate that such effects are unlikely at low levels of exposure. Therefore, ICNIRP reconfirms the 1998 basic restrictions in the frequency range 100 kHz–300 GHz until further notice."*

Similarly, SCENIHR of the European Commission in its 2009 review states:

> *"It is concluded from three independent lines of evidence (epidemiological, animal and in vitro studies) that exposure to RF fields is unlikely to lead to an increase in cancer in humans. However, as the widespread duration of exposure of humans to RF fields from mobile phones is shorter than the induction time of some cancers, further studies are required to identify whether considerably longer-term (well beyond ten years) human exposure to such phones might pose some cancer risk.*
>
> *Regarding non-carcinogenic outcomes, several studies were performed on subjects reporting subjective symptoms. In the previous opinion, it was concluded that scientific studies had failed to provide support for a relationship between RF exposure and self reported symptoms. Although an association between RF exposure and single symptoms was indicated in some new studies, taken together, there is a lack of consistency in the findings. Therefore, the conclusion that scientific studies have failed to provide support for an effect of RF fields on self-reported symptoms still holds. Scientific studies have indicated that a nocebo effect (an adverse non-specific effect that is caused by expectation or belief that something is harmful) may play a role in symptom formation. As in the previous opinion, there is no evidence supporting that individuals, including those attributing symptoms to RF exposure, are able to detect RF fields. There is some evidence that RF fields can influence EEG patterns and sleep in humans. However, the health relevance is uncertain and mechanistic explanation is lacking. Further investigation of these effects is needed. Other studies on functions/aspects of the nervous system, such as cognitive functions, sensory functions, structural stability, and cellular responses show no or no consistent effects. Recent studies have not shown effects from RF fields on human or animal reproduction and development. No new data have appeared that indicate any other effects on human health."*

In its 2009 Report, the Independent Expert Group of the Swedish Radiation Safety Authority SSM concludes regarding cancer and transmitters:

> *"The majority of studies on cancer among people who are exposed to RF from radio- or TV-transmitters or from mobile phone base stations have relied on too crude proxies for exposure to provide meaningful results. Indeed, only two studies, both on childhood leukaemia, have used models to assess individual exposure and both of those provide evidence against an association. One cannot conclusively exclude the possibility of an increased cancer risk in people exposed to RF from transmitters based on these results. However, these results in combination with the negative animal data and very low exposure from transmitters make it highly unlikely that living in the vicinity of a transmitter implicates an increased risk of cancer."*
>
> Regarding electromagnetic hypersensitivity, the SSM expert group writes:
>
> *"While the symptoms experienced by patients with perceived electromagnetic hypersensitivity are very real and some subjects suffer severely, there is no evidence that RF exposure is a causal factor. In a number of experimental provocation studies, persons who consider themselves electrically hypersensitive and healthy volunteers have been exposed to either sham or real RF fields, but symptoms have not been more prevalent during RF exposure than during sham in any of the experimental groups. Several studies have indicated a nocebo effect, i.e. an adverse effect caused by an expectation that something is harmful. Associations have been found between self-reported exposure and the outcomes, whereas no associations were seen with measured RF exposure."*

Canadian Exposure Assessments

In 1997, Health Canada conducted a survey of radiofrequency radiation from cellular base stations in and around 5 schools in Vancouver, in response to the concerns raised by nearby residents earlier that year. The measurements revealed that:

- The highest level of electromagnetic radiation from a PCS antenna (across the street) was more than 6,000 times below the Safety Code 6 levels.
- In three of the schools the levels of radiation from all PCS digital antenna were actually lower than the normal AM and FM radio signals that have been in the area for decades.

In 2003, Health Canada released the results of comprehensive ground level RF measurements representative of human exposures near base stations within the Regional Municipality of Ottawa. The highest power density measured was 3000 times below Safety Code 6. Health Canada considers these measurements as likely representative of levels in other Canadian urban areas.

In 2010, the Public Health Department of the Health and Social Services Agency of Montreal was asked to assess two cell phone base station sites located near schools in Outremont, an urban residential neighbourhood. One location has 12 antennae (130 m to 145 m away respectively from two primary schools) and the other has three (50 m from a high school). The investigation team estimated that the level of exposure to students would be over 5000 times below Safety Code 6 inside the school and over 1000 times below Safety Code 6 on school playgrounds and adjacent streets. The team also reviewed the scientific literature on the subject and concluded that:

> *"The results of numerous scientific studies conducted to date do not argue in favour for a causal relation between RF exposure and health impact at exposure commonly encountered, whether cancer or more general symptoms. Moreover, no mechanism of action of RF on cells or human and animal tissues has been shown. However, due to uncertainties still present in this area of research, health agencies recommend further studies in some promising avenues (e.g. for cell phone users). As for cellular antennae, given the very low exposure levels and research results to date, most experts believe it is unlikely that this exposure, well below the limits allowed, can cause effects on the health of the population."*

In May 2011, the International Agency for Research on Cancer (IARC) placed radio frequency electromagnetic fields in its group 2B classification – possibly carcinogenic to humans. IARC defines group 2B as a category used

> *"for agents for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals. It may also be used when there is inadequate evidence of carcinogenicity in humans but there is sufficient evidence of carcinogenicity in experimental animals. In some instances, an agent for which there is inadequate evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals together with supporting evidence from mechanistic and other relevant data may be placed in this group. An agent may be classified in this category solely on the basis of strong evidence from mechanistic and other relevant data."*

Agents in Group 2B are not proven carcinogens. Details of the IARC review is expected to be published in July 2011. In the meantime, the IARC does make it clear that the primary reason for the Group 2B classification relates to uncertainty regarding long term heavy cell phone use and certain rare brain cancer. The type of radio frequency exposure of concern is associated with using the cell phone close to the ear. As stated above, the energy of radio frequency field from cell phone base stations experienced by the general public is thousands of times lower than from a cell phone near the head. The IARC conclusion therefore does not alter the assessment for radio frequency exposure due to cell phone base stations.

"Prudent Avoidance"

The practice of "prudent avoidance" has been advocated by some in their opposition to specific location of cellular base stations in the vicinity of schools, child care centres or residential buildings. "Prudent avoidance" in these situations does not result in any increased level of protection. It would be difficult, if not impossible, to "prudently avoid" some level of exposure to RF fields in an urban setting, whether it be from AM, FM, TV or cellular phones. The Medical Health Officer concludes that scientific evidence provides no basis for recommending prudent avoidance with respect to cellular base stations. There is no public health benefit. In fact, prudent avoidance ignores the reality that the area immediately below an antenna has the lowest RF levels.

Community Consultation and Public Access to Information

Despite reassuring evidence, some members of the public remain concerned about the presence of cell phone antennae and base stations. Telecommunications regulators and industry can do a better job in providing information (particularly about base station types and locations), as well as providing meaningful opportunities for public consultation when planning base stations. Industry Canada in 2009 established public and local government consultation guidelines for permit applications for mobile phone base stations. The requirement for consultation unfortunately applies only to antennae 15 metres or higher. There are a number of practices the telecommunications regulators and industry can implement to mitigate public concerns. These include:

- Meaningful discussion with communities.
- Clear and publicly accessible supporting documents when deploying base stations.
- Greater consideration for site sharing, where possible.
- Greater consideration for sensitive location and design.
- Improved public access to information on network compliance with Safety Code 6.
- Prompt response to community enquiries about base stations.
- Periodic but systematic and comprehensive measurements of population level exposure to RF to monitor trends.

Conclusion

As has Health Canada, the Chief Medical Health Officer concludes that, in light of the current scientific understanding of the risks of RF exposures to the public, the installation of base stations and cellular antennae in the community do not pose an adverse health risk and Safety Code 6 provides an appropriate level of protection. However, public engagement by telecommunication regulators and industry concerning the installation of base stations and antennae needs improvement.

The Chief Medical Health Officer will continue to monitor new scientific knowledge in this area and will provide updates when necessary.

Chief Medical Health Officer

2.1.1 Please confirm that the conclusion of this Statement found on page 5 states:

"As has Health Canada, the Chief Medical Health Officer concludes that, in light of the current scientific understanding of the risks of RF exposures to the public, the installation of base stations and cellular antennae in the community do not pose an adverse health risk and Safety Code 6 provides an appropriate level of protection."

Response:

Confirmed.

2.2 Please provide the test results from the BC Centre for Disease Control titled "Measurement of Radiofrequency (RF) Emissions from BC Hydro's Itron Smart Meters" located at:

http://www.bccdc.ca/NR/rdonlyres/43EF885D-8211-4BCF-8FA9-0B34076CE364/0/452012AmendedReportonBCHydroSmartMeterMeasurements.pdf

Response:

The requested document is provided as Attachment BCH 2.2.

BC Centre for Disease Control
An agency of the Provincial Health Services Authority

Measurement of Radiofrequency (RF) Emissions from BC Hydro's Itron Smart Meters

Date of test: January 10, 2012

1. Radiofrequency exposure metric measured:

RF **Peak Power Density S** in **$\mu W/cm^2$** from BC Hydro's Itron Smart Meters

2. Testing Set-Up

2.1. Smart Meters Tested

- Brand name: Itron Smart Meters
- Frequency: Within **902 MHz - 928 MHz**
- Nominal Power: **30dBm (1 Watt)**
- Where tested: BC Hydro test facility, Surrey, BC

2.2. RF Survey Meter used: Narda Broadband NBM-520 with Probe EC5091

*2.3. Characteristics of the **NBM-520 Meter** and the **EC 5091 Probe*** (Ref. Narda flyer)

2.3.1. **NBM-520 meter**

- Frequency Range: 300 MHz to 50 GHz
- Isotropic Response (response is independent of probe orientation)
- Display Range: 0.0001% to 9999% of standard
- Calibration: Last calibration on July 26, 2011 (considered valid until July 22, 2013)

2.3.2. **EC 5091 Probe**, E field, shaped SC 6 Canada

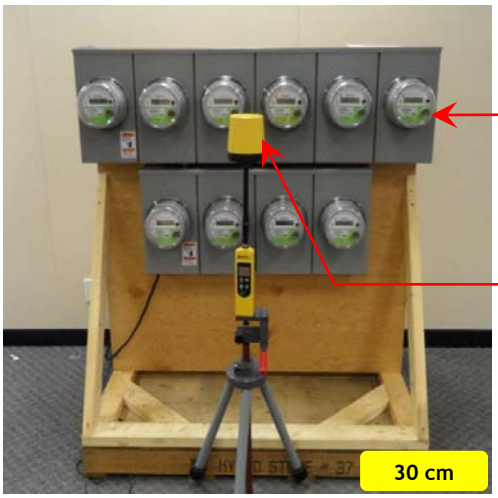
- Measurement range: **0.5% to 600% of Safety Code 6 Limits for RF/Microwave workers**.

2.4. Tripod (survey meter support)

A non-conducting plastic tripod was used to hold the survey meter & probe assembly vertically above the ground.

Figure 1 below shows the testing set-up used to measure the peak RF power density from a bank of 10 of BC Hydro's Itron Smart Meters.

Figure 1. Smart meter measurement set-up



Probe at a distance of 30 cm from the smart meter bank

Probe at 1 meter from the smart meter bank

Probe at 3 meters from the smart meter bank

3. Testing Procedure

3.1. RF emission mode

- Smart meters are designed to emit pulses that last approximately 100 to 150 milliseconds (0.1 to 0.15 seconds) at irregular intervals.
- In a cluster of smart meters, the meters do not emit simultaneously but are programmed to communicate with a single controller in a random sequence. This mode of operation eliminates the potential for exposure to more than one signal at the same time (**Ref.1**).
- In this trial, smart meters were scheduled to emit brief pulses every 5 minutes (5 minute interval interrogation).
- The continuous monitoring of RF levels was maintained for a time period of 10 minutes at each position of the probe to ensure that the highest possible RF exposure level was detected and recorded.

3.2. The RF Measurements

At 30 cm from the smart meters and beyond, RF measurements are made in the far field region where plane wave conditions apply. Hence, the selected RF exposure metric is the power density (in units of W/m^2).

Electric field strength **E** (V/m) at each position can also be determined from power density ***S*** (W/m^2) readings using the following formula (plane wave conditions):

$$E = \sqrt{377\,S}$$

Where the value **377** represents the characteristic impedance of free space in units of Ohms (Ω).

The peak power density readings were taken at 3 different distances from the RF sources: 0.3 m, 1 m, and 3 m.

The peak readings were recorded using the "peak hold" option of the survey meter.

3.3. RF Exposure Situations

The first series of tests were carried out with one operating smart meter and the second series with a bank of 10 operating smart meters.

4. Testing Results

The instantaneous **Peak Power Density** recorded from BC Hydro smart meter emissions during the tests are shown in **Table 1** below.

Table 1 - Instantaneous Peak Power Density S (μW/cm^2) from BC Hydro Smart Meters

Distance RF source - RF Probe	Peak Power Density S* 1 operating smart meter (μW/cm^2)	Peak Power Density S* Bank of 10 smart meters (μW/cm^2)
30 cm	3.204	4.035
1 meter	2.016	2.610
3 meter	1.170	1.779

Note: These readings incorporate the existing background generated by uncontrolled external RF sources.

5. Remarks

5.1. Radiofrequency Background:

- During the test, RF sources within the testing facility, namely cell phones carried by testers and other smart meters in the facility were turned off to minimize the internal background.
- The RF background was monitored at each position (30 cm, 1 meter, 3 meters) over30 minutes while the smart meters were idle.
- The RF background level due to uncontrolled external RF sources (power lines, external wireless systems,…) varied from less than 01 μW/cm^2 to occasional peaks at 2 to 3 μW/cm^2.

5.2. Instantaneous peak power density readings:

- RF signals were taken over a period of 10 minutes. The instantaneous peak power density reading at each position (30 cm, 1 meter, 3 meters) was saved using the "peak hold" option of the meter.
- The recorded levels correspond to the highest outdoor exposure levels that would be expected to be generated during smart meter pulses.

5.3. Comparison of smart meter exposure to other household RF devices:

To put RF exposure to smart meters in context, levels generated by other RF household devices under similar exposure conditions are compared to those for smart meters.

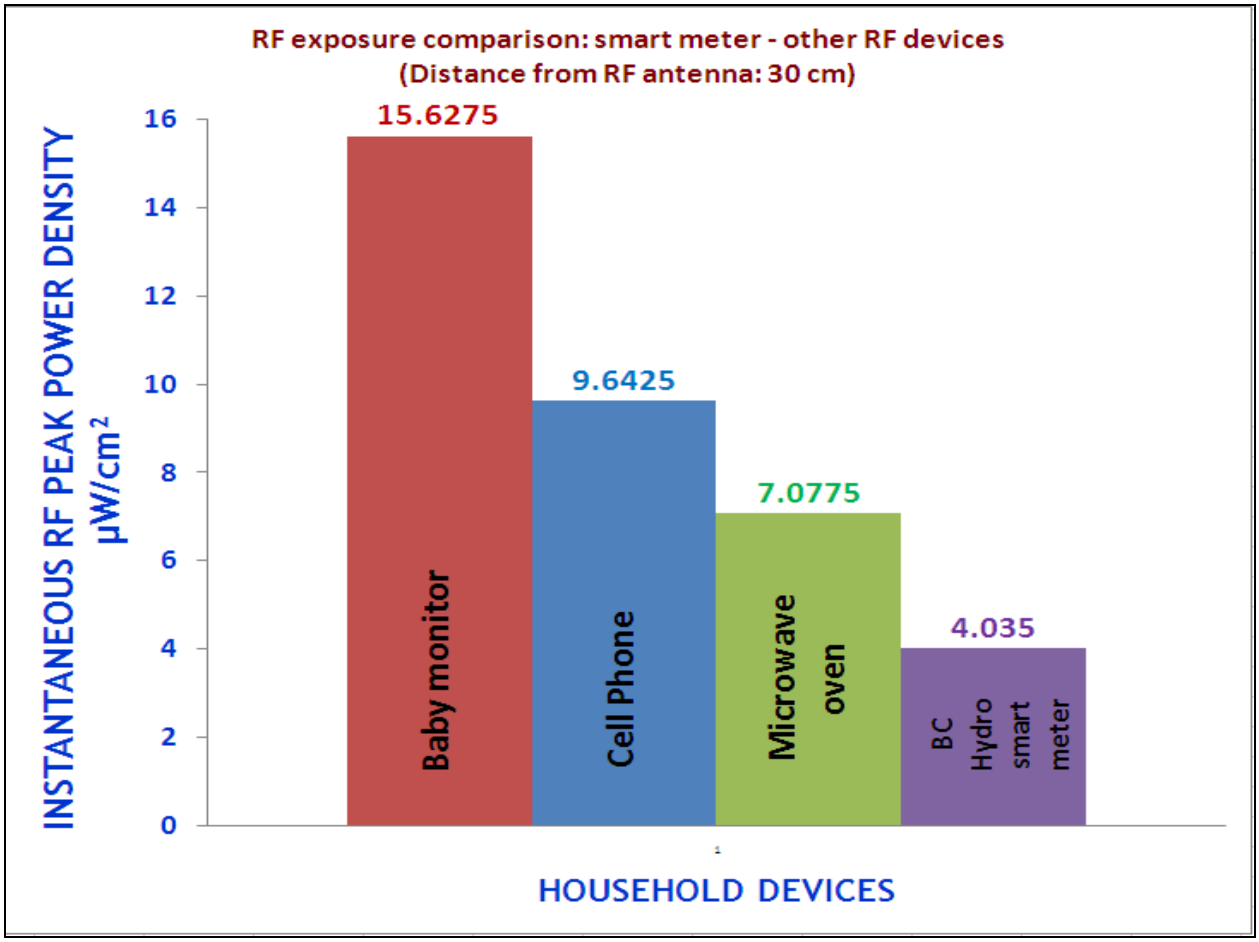
Typical values of RF emissions from mobile phones, microwave ovens, baby monitors, and cordless phones at a distance of 30 cm are listed below in **Table 2** (Source: **Ref. 2**)

Table 2 – Radiofrequency emissions from some household devices

DEVICE	Instantaneous Peak Power Density at 30 cm (Source: Ref. 2) In units of μW/cm^2
RF Baby Monitor	15.6275
Cell Phone	9.6425
Microwave Oven	7.0775

Figure 2 shows the instantaneous power density levels for cell phones, microwave ovens, baby monitors, and BC Hydro smart meters (outdoor).

Figure 2 – Comparison of RF smart meter exposure to other RF devices



(Source: Ref. 2)

As shown in Figure 2, BC Hydro smart meter RF emissions outside homes are lower than the RF exposure associated with some household devices, e.g. baby monitors, cell phones, and microwave ovens.

5.4. Duty cycle of BC Hydro smart meters and time-averaged power density

Since smart meters release brief radiofrequency pulses at irregular intervals, to better understand the effective exposure of persons near smart meters, the **duty cycle** (**fraction of time a smart meter is transmitting for a given period**) of the meters should be taken into account.

BC Hydro smart meters have a duty cycle of 0.07% corresponding to a cumulative emission duration of 1 minute per day on the average (**Ref.3**).

Table 3 gives the values of the **time-averaged power density S** at 30 cm, 1meter, and 3 meters for a duty cycle of 0.07%.

The ratios of the **S** values to Health Canada Safety Code 6 (**SC.6**) Limit for the public are shown **in brackets**.

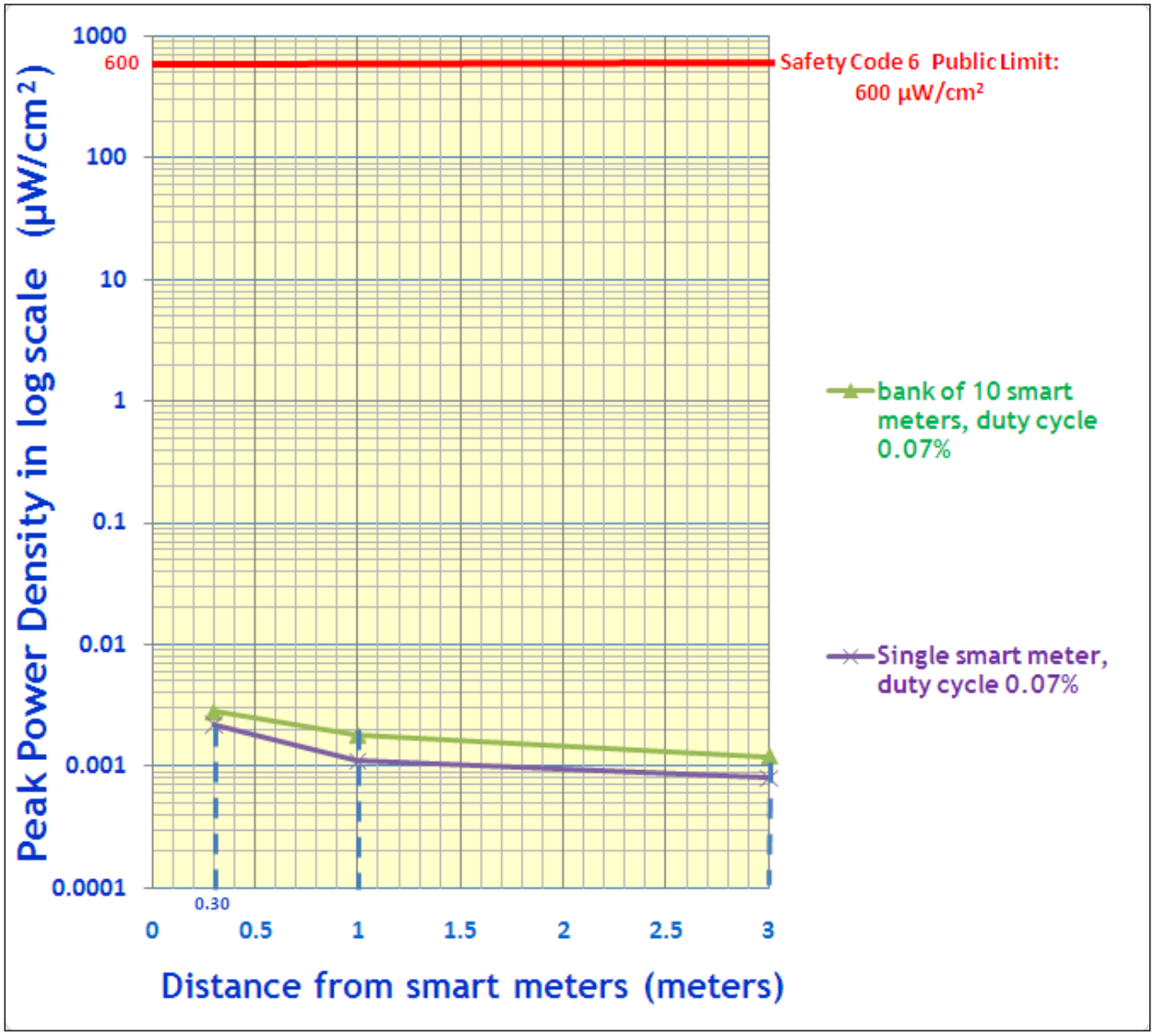
Table 3 – Comparison of smart meter emissions to Health Canada Safety Code 6 Limits for public environment

Distance from smart meter	Time-averaged Power Density S 1 operating smart meter ($\mu W/cm^2$)	Time-averaged Power Density S 10 operating smart meter ($\mu W/cm^2$)
30 cm	0.0022 (0.00037 % of SC 6 Limit)	0.0028 (0.00047 % of SC 6 Limit)
1 meter	0.0011 (0.00018 % of SC 6 Limit)	0.0018 (0.00030 % of SC 6 Limit)
3 meters	0.0008 (0.00013 % of SC 6 Limit)	0.0012 (0.00021 % of SC 6 Limit)

Figure 3 below shows the levels of smart meter emissions in comparison to Health Canada's Safety Code 6 Limit of 600 $\mu W/cm^2$ of continuous exposure for general public areas.

As illustrated in **Figure 3**, the time-averaged RF exposure levels from smart meters is low (Less than 0.001 % of Health Canada's Safety Code 6 limit), even at a short distance from the RF antenna.

Figure 3. Comparison of Smart Meter Emissions to HC safety Code 6 Limits



The RF power density results obtained during this test are comparable to values reported by investigators referenced below (**Ref 1 thru 6**).

6. References

Ref. 1 - A Discussion of Smart Meters and RF Exposure Issues - An EEI-AEIC-UTC White Paper – A Joint Project of the EEI and AEIC Meter Committees - March 2011 Published by: Edison Electric Institute; 701 Pennsylvania Avenue NW, Washington, DC 20004-2696;

http://www.aeic.org/meter_service/smartmetersandrf031511.pdf

Ref. 2 - AMI Meter Electromagnetic Field Survey Final Report Prepared For Department of Primary Industries - Document Number: M110736 – Prepared by: EMC Technologies Pty Ltd 176 Harrick Road Keilor Park, Victoria 3042 (Australia), 20 October 2011.

http://www.dpi.vic.gov.au/smart-meters/publications/reports-and-consultations/ami-meter-em-field-survey-repor

Ref. 3 - Planetworks Consulting – Safety Code 6 Report –Single Smart Meter and a bank of 10 smart meter (October 2011).

http://www.bchydro.com/etc/medialib/internet/documents/smi/SMI_MeterBank.Par.0001.File.SMI-MeterBank-2011-Oct-11.pdf

Ref. 4 An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter, EPRI, Palo Alto, CA: 2010. 1021126

http://my.epri.com/portal/server.pt?space=CommunityPage&cached=true&parentname=ObjMgr&parentid=2&control=SetCommunity&CommunityID=405

Ref. 5 Federal Communications Commission /USA - ACS Report: 11-0093.W06

http://www.fcc.gov/search/results/smart%20meters

Ref. 6 - Richard A. Tell, RICHARD TELL ASSOCIATES, INC. An Analysis of Radiofrequency Fields Associated with Operation of the Hydro One Smart Meter System – Revised October 13, 2010 - Prepared for Hydro One Networks Inc. 483 Bay Street, North Tower, Toronto, Ontario M5G 2P5

http://www.hydroone.com/MyHome/MyAccount/MyMeter/Documents/Smart_Meters_Report_on_RFE.pdf

2.3 Please provide letter of advice to Richmond Council on Smart Meters from the Vancouver Coastal Health dated December 20, 2011 and located at:

http://www.health.gov.bc.ca/pho/pdf/vch-response-to-richmond-city-council-re-investigation-into-smart-meters.pdf

Response:

The requested document is provided as Attachment BCH 2.3.

vancouver coastalHealth
Promoting wellness. Ensuring care.

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Richmond, BC V6Y 3T6

T: 604-233-3150
F: 604-233-3221

December 20, 2011

Mayor and Council, City of Richmond
c/o David Weber,
Director, City Clerk's Office
City of Richmond
6911 No. # Road
Richmond, BC V6Y 2C1

Dear Mayor and Council:

This letter is in response to Council's resolution made on November 14, 2011, requesting the Medical Health Officer to "conduct an investigation as to whether smart meters pose a health hazard". We wish to direct Council also to an earlier response made by the Medical Health Officer following Council resolution R11/17-7 regarding the same issue.

The BC Public Health Act defines a health hazard as:

a) a condition, a thing or an activity that
   (i) endangers, or is likely to endanger, public health, or
   (ii) interferes, or is likely to interfere, with the suppression of infectious agents or hazardous agents, or

(b) a prescribed condition, thing or activity, including a prescribed condition, thing or activity that
   (i) is associated with injury or illness, or
   (ii) fails to meet a prescribed standard in relation to health, injury or illness

The "Smart Meters" installed and used by BC Hydro are not health hazards as defined by the BC Public Health Act. The Provincial Health Officer has stated that given the current scientific evidence, exposure to the radio-frequency electromagnetic fields from Smart Meters "do not constitute a threat to the health of the public" (http://www.health.gov.bc.ca/pho/issues.html). As well, the transmitters in Smart Meters produce electromagnetic fields at levels significantly lower than the maximum allowed for the Canadian public under Health Canada's Safety Code 6.

The transmitters used in these meters are similar to a cell phone in power **but** they are active only for a very short duration at a time. BC Hydro posted on its website that total transmission time would average 1 minute a day for a Smart Meter. In fact, the independent engineering firm recently engaged by BC Hydro reports that a single meter is actively transmitting for a total of less than 2 seconds a day under conditions simulating normal use (0.904 sec on test day 1, and 1.83 sec on test day 2). For a bank of 10 meters under the same conditions, the total active transmission time is around 10 seconds a day (10.150 sec on test day 1 and 9.606 sec on test day 2).

In terms of power density, Safety Code 6 requires it to be less than 600 microwatts per square centimeter for publicly accessible areas, at the radio-frequency used by the Smart Meters. The independent consultant found the average power density to be 0.3795% of Safety Code 6 (or 2.3 microwatts per square centimeter) for a single Smart Meter, with a range between 0.034% and 0.916% of Safety Code 6. For a bank of 10 Smart Meters, the average power density was found to be 0.4507% of Safety Code 6 (or 2.7 microwatts per square centimeter), with a range from 0.0015% to 1.6835% of Safety Code 6. Even the highest power density value recorded from a bank of 10 meters is more than 50 times less than the Safety Code 6 limit, while the average power density for both single and a 10 meter bank are more than 200 times less than Safety Code 6. Moreover, the power density recorded for a 10 meter bank is not 10 times that of a single meter. The average power density for the 10 meter bank is about 1.2 times that of a single meter, while the maximum value from the 10 meter bank is slightly less than twice the maximum value recorded from the single meter set up. These measurements support the information provided on BC Hydro's website that BC Hydro expects the power density from meter banks to be about twice compared to a single meter.

Information obtained from BC Hydro by the Medical Health Officer indicates that there are two types of transmissions from Smart Meters. The first type is for network coordination (for example, power outage detection) – this occurs every 30 minutes. The second type of transmission is to convey consumption data – this occurs three times a day. The independent consultant notes that the actual incremental contribution by Smart Meters, whether singly or in a bank, above the radio-frequency background in everyday environment is barely measureable, and that in calculating the power densities the consultant assumed that everything measured were from the Smart Meters. Therefore, in reality, the radio-frequency fields generated by Smart Meters are very likely to be less in power density than what the consultant reported. The independent consultant's reports have been posted recently by BC Hydro on its website:

http://www.bchydro.com/etc/medialib/internet/documents/smi/SMI_SingleSmartMeter.Par.0001.File.SMI-SingleSmartMeter-2011-Oct-11.pdf

http://www.bchydro.com/etc/medialib/internet/documents/smi/SMI_MeterBank.Par.0001.File.SMI-MeterBank-2011-Oct-11.pdf

Delegations to Council have suggested that measurements by the BC Centre for Disease Control contradict BC Hydro's public statements. Most of the measurements performed by BCCDC are reported as below the measurement limit of the instrument used. Measurable levels are found by BCCDC only when the instrument probe was ***in actual contact*** with certain areas of the Smart Meter casing, or in close proximity to the collector antenna, equivalent to holding a cell phone close to the head. The levels that are measurable by the BCCDC instrument even in these circumstances were at the lower end of the range of radio-frequency field strengths typical of what people would experience from cell phones when cell phones are held to the ear. In addition, it is highly unlikely for the public to be close to the collector antennae since they are located on top of utility poles, 18 – 24 feet above ground. Moreover, because it was performed with the Smart Meter and the collector on a continuous transmitting mode, which is not how the Smart Meters and collectors will function in real life, the BCCDC report provides no information on exposure when radio transmission is intermittent. As well, the instrument used by BCCDC was not very sensitive and had a high detection limit. Therefore, the BCCDC measurements do not contradict the information provided by BC Hydro.

Delegations also were concerned about the discrepancies in measurements between the independent consultant and other available reports such as from the Electrical Power Research Institute (EPRI). A comparison of the model numbers shows that the Smart Meters used in the EPRI report and the ones used by BC Hydro are different. Information from BC Hydro indicates that the Smart Meters being introduced by BC Hydro is a newer generation meter. Newer technology has allowed transmission time to be shortened and therefore less overall power density. It should also be noted that the measurements in the EPRI report, referred to on the BC

Hydro website, are from meters transmitting continuously, which is not how the Smart Meters function in real life. In contrast, the independent consultant performed his measurements in a testing environment that "has been constructed to be as realistic a representation of usage environment as possible".

Regarding cancer risk, the recent decision by the WHO to classify radio frequency electromagnetic field as possibly carcinogenic (Class 2B) is based on epidemiological uncertainties surrounding the long term and heavy use of cell phones held to the ear. This is clearly not the case with respect to exposure from Smart Meters or the collectors. Information regarding the WHO decision is available online:

http://www.iarc.fr/en/media-centre/pr/2011/pdfs/pr208_E.pdf

With regard to the many other health concerns raised by public delegations to Council at the October 14, November 7 and November 14 Council meetings, they are not new. Indeed, these concerns with respect to radio frequency radiation have been raised and studied for many years. Scientific evidence weighed all together has not been able to substantiate the concerns. A public delegation on October 14 provided a quote from the WHO regarding Electromagnetic Hypersensitivity (EHS). Because the quote is only partial, the reader may conclude that the WHO believes EHS is caused by radio-frequency electromagnetic fields. The following is found in the conclusion section of the WHO fact sheet on EHS: "EHS is characterized by a variety of non - specific symptoms that differ from individual to individual. The symptoms are certainly real and can vary widely in their severity. Whatever its cause, EHS can be a disabling problem for the affected individual. EHS has no clear diagnostic criteria and there is no scientific basis to link EHS symptoms to EMF exposure. Further, EHS is not a medical diagnosis, nor is it clear that it represents a single medical problem".

http://www.who.int/mediacentre/factsheets/fs296/en/index.html .

In conclusion, the public may be opposed to the BC Hydro Smart Meter Program for a number of reasons. That these Smart Meters are health hazards should not be one of them. These devices are active only for an extremely short amount of time each day. They add so little to the existing background radio-frequency fields that it is very difficult to separate them apart from our everyday environment. We recognize that some may disagree with our assessment. We respectfully differ. We are confident however that our assessment is in agreement with the overall scientific understanding regarding radio frequency electromagnetic fields.

Respectfully,

Dr. James Lu
Medical Health Officer – Richmond
Vancouver Coastal Health

Dr. Patricia Daly
Chief Medical Health Officer
Vancouver Coastal Health

P.R.W. Kendall
OBC, MBBS, MHSc, FRCPC
Provincial Health Officer

2.3.1 Please confirm that this letter concludes with the following statement:

"In conclusion, the public may be opposed to the BC Hydro Smart Meter Program for a number of reasons. That these Smart Meters are health hazards should not be one of them. These devices are active only for an extremely short amount of time each day. They add so little to the existing background radio-frequency fields that it is very difficult to separate them apart from our everyday environment. We recognize that some may disagree with our assessment. We respectfully differ. We are confident however that our assessment is in agreement with the overall scientific understanding regarding radio frequency electromagnetic fields."

Response:

Confirmed.

2.4 Please provide Health Canada's Smart Meters Update dated December 2011 and located at:

http://www.hc-sc.gc.ca/hl-vs/alt_formats/pdf/iyh-vsv/prod/meters-compteurs-eng.pdf

Response:

The requested document is provided as Attachment BCH 2.4.

Health Canada Santé Canada

Your health and safety… our priority. *Votre santé et votre sécurité… notre priorité.*

Smart Meters

Updated:

December 2011

IT'S YOUR HEALTH

Smart Meters

THE ISSUE

In recent years, utility companies in several provinces have started installing wireless smart meters in Canadian businesses and residences. Some people have expressed concern about the possibility of health effects from exposure to the radiofrequency (RF) energy that these devices emit.

SMART METERS

Smart meters are relatively new wireless devices that transmit information using RF signals to let utility companies know how much water, gas or electricity a household or business is using. The signals emitted by smart meters are of relatively low power, similar to cell phones and wireless internet routers. The maximum amount of power that a smart meter device can transmit must comply with Industry Canada regulations.

HEALTH RISKS

As with any wireless device, some of the RF energy emitted by smart meters will be absorbed by anyone who is nearby. The amount of energy absorbed depends largely on how close your body is to a smart meter. Unlike cellular phones, where the transmitter is held close to the head and much of the RF energy that is absorbed is localised to one specific area, RF energy from smart meters is typically transmitted at a much greater distance from the human body. This results in very low RF exposure levels across the entire body, much like exposure to AM or FM radio broadcast signals.

Survey results have shown that smart meters transmit data in short bursts, and when not transmitting data, the smart meter does not emit RF energy. Furthermore, indoor and outdoor survey measurements of RF energy from smart meters during transmission bursts were found to be far below the human exposure limits specified in Health Canada's Safety Code 6.

Based on this information, Health Canada has concluded that exposure to RF energy from smart meters does not pose a public health risk.

Canada

REDUCE YOUR RISK

Since RF energy exposure levels are far below Canadian and international safety limits, Health Canada does not consider that any precautionary measures are needed to reduce RF energy exposure from smart meters.

In cases where multiple smart meters are installed together, as in some townhouses or high-rise buildings, the total exposure levels from multiple smart meters will still be far below Health Canada's RF energy exposure limits, due to the infrequent nature of transmissions.

THE GOVERNMENT OF CANADA'S ROLE

Health Canada has developed guidelines for safe human exposure to RF energy. The current version of these exposure guidelines is specified in a document called *Limits of Human Exposure to Radiofrequency Electromagnetic Energy in the Frequency Range from 3 kHz to 300 GHz* – Safety Code 6 (2009).

The limits specified in these guidelines are based on an ongoing review of published scientific studies on the health impacts of RF energy. Using data from these studies, Health Canada set the general exposure limits far below the threshold for potentially adverse health effects.

Health Canada continues to monitor the science regarding RF exposure and will take action if future research establishes that RF energy exposure poses a health risk to Canadians.

FOR MORE INFORMATION

- World Health Organization, Electromagnetic fields and public health: base stations and wireless technologies at : www.who.int/media centre/factsheets/fs304/en/index.html
- International Agency for Research on Cancer electromagnetic fields news release at: www.iarc.fr/en/media-centre/pr/2011/pdfs/pr208_E.pdf
- World Health Organization, Electromagnetic Fields at: www.who.int/peh-emf/en/

FOR INDUSTRY AND PROFESSIONALS

- Health Canada's Consumer and Clinical Radiation Protection Bureau at: www.hc-sc.gc.ca/ahc-asc/branch-dirgen/hecs-dgsesc/psp-psp/ccrpb-bpcrpcc-eng.php
- Health Canada's RF exposure guidelines (Safety Code 6) at: www.hc-sc.gc.ca/ewh-semt/pubs/radiation/radio_guide-lignes_direct-eng.php
- Industry Canada's Radio Standards Specification 102 at: www.ic.gc.ca/eic/site/smt-gst.nsf/eng/sf01904.html
- Industry Canada's Client Procedures Circular CPC-2-0-03 at: www.ic.gc.ca/eic/site/smt-gst.nsf/eng/sf08777.html
- Industry Canada's Guidelines for the Protection of the General Public in Compliance with Safety Code 6 at: www.ic.gc.ca/eic/site/smt-gst.nsf/eng/sf05990.html

RELATED RESOURCES

- For safety information about food, health and consumer products, visit the Healthy Canadians website at: www.healthycanadians.gc.ca
- For more articles on health and safety issues go to the *It's Your Health* web section at: www.health.gc.ca/iyh

You can also call toll free
at 1-866-225-0709
or TTY at 1-800-267-1245

Original: December 2011

Cat.: H13-7/117-2011E-PDF
ISBN: 978-1-100-19838-5

2.4.1 Please confirm this Update states on page 2:

"Since RF energy exposure levels are far below Canadian and international safety limits, Health Canada does not consider that any precautionary measures are needed to reduce RF energy exposure from smart meters."

Response:

Confirmed.

2.5 Please provide the Smart Meter and Cancer Risk Statement of the British Columbia Ministry of Health, dated December 23, 2011, located at:

http://www.health.gov.bc.ca/pho/issues.html

Response:

The requested document is provided as Attachment BCH 2.5.

- **Smart Meter and Cancer Risk Statement**

The following statement was prepared at the request of the Provincial Health Officer by Mary McBride, Distinguished Scientist, Department of Cancer Control, of the BC Cancer Agency, Vancouver, BC. It has been approved by Dr David McLean, Head of cancer Prevention BCCA.

Date: 23 December, 2011

Personal exposure to radiofrequency radiation (RF), through the use of cell phones, has increased dramatically, since their introduction in the mid- to late-1980s. The expanding use of this technology has been accompanied by concerns about public health. Therefore, since that time, there has been extensive research into the health effects, including cancer effects, of exposure to RF. These studies included (1) laboratory studies of biological effects on cells, which provide information on possible mechanisms by which RF could cause cancer; (2) studies of effects in animals, which provide information on whether biological changes lead to health-related change; and (3) direct observation and assessment of cancer risk in humans.

Smart Meters emit RF radiation, but only intermittently, and at a level several times below that of the highest level of personal exposures from cell phones, and well below existing limits for RF exposure to the public. Smart Meters generate an RF signal an average of only one minute per day. In addition, Smart Meters emit very low power – about one watt. This is less than 2 microwatts per square centimetre (μW/cm2) when standing adjacent to the meter. A microwatt is one millionth of a watt. And, exposure to RF drops quickly with distance from the device. Three meters (10 feet) from the smart meter, the radio frequency signal drops to less than 0.001 per cent (0.005 μW/cm2) of the Health Canada exposure limits. This exposure level is much less than exposure to RF from cell phone use.

However, since both cell phones and Smart Meters emit RF, the research into RF exposures for cell phones is relevant to an understanding of cancer risk from Smart Meters.

First, it is important to note that brain tumour rates (the kind of cancer of most concern) in adults and children have *not* increased with increasing cell phone use. Major recent studies of cell phone use and brain cancer include the *Interphone study*, the largest case-control study ever undertaken, that compared the RF exposures, as determined by cell phone usage, of those who developed a brain tumour and those who did not; and a *Danish study* of 365,000 cell phone subscribers in the country,

whose subscriber records were linked to the population cancer records to determine cancer rates by extent of cell phone use.

The Interphone study (2010) reported that, although estimates of risk of brain tumour were the same or lower for most groups of users, there was a statistically higher risk of glioma, a malignant type of brain tumour, observed among the few cases who had used a cell phone 20 years or more. The authors concluded that "because of biases and errors inherent in the study design, the results could not be interpreted as meaning that RF exposure actually was the cause of the excess risk of brain tumours".

Subsequent to publication of the Interphone study, a multidisciplinary working group of 31 experts convened by the International Agency for Research on Cancer (IARC), the cancer research arm of the World Health Organization, reviewed all the published research to date. They concluded that there was *limited evidence of carcinogenicity* (IARC Classification 2B): that is,
"a positive association (had) been observed between exposure to (RF) and cancer, for which a causal interpretation is considered … to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence".

Following the publication of the Interphone study, a report summarizing the IARC Working Group review (The Lancet Oncology, July 2011) concluded that the Interphone results, "along with those from other epidemiological, biological, and animal studies, and brain tumour incidence trends, suggest that within the first 10-15 years of exposure to RF radiation from cell phones, the period of use examined in Interphone, there is unlikely to be a material increase in brain tumours in adults".
The report also concluded that, "although there remains some uncertainty, the trend in the accumulating evidence was increasingly against the hypothesis that (RF radiation from) mobile phone use can cause brain tumours in adults."

Subsequently, the large Danish study, whose study design is not subject to the same bias and error as the Interphone study, did not find any excess risk of brain tumours among any group of cell phone users, even those with heavy or long-term use.

The evidence, therefore, does not support a conclusion that RF fields, whether from cell phones or Smart Meters, can cause brain tumours in adults. There is so far little direct human data on those with more than 20 years' cell phone use, and limited information on risk of other cancers, but the limited information we have is generally negative. There is also no direct information on children. More studies, to address these gaps in our understanding of RF and cancer risk, are

underway. However, the extensive laboratory research to date has not identified any mechanisms that could operate in either adults or children that would lead to an excess risk of tumours in general.

Advice to Richmond Council on Smart Meters

VCH Response to Richmond City Council Reinvestigation into Smart Meters (PDF 76K)

BC Cancer Agency Recommended Websites - Electromagnetics (& radiofrequency) and Radiation

http://www.bccancer.bc.ca/PPI/RecommendedLinks/causesprevention/electromagnetics.htm
http://www.bccancer.bc.ca/PPI/RecommendedLinks/causesprevention/radiation.htm

BC Centre for Disease Control - Smart meters

http://www.bccdc.ca/healthenv/Radiation/ElectromagRadiation/SmartMeters.htm

Measurement of Radiofrequency (RF) Emissions from BC Hydro's Itron Smart Meters

http://www.bccdc.ca/NR/rdonlyres/43EF885D-8211-4BCF-8FA9-0B34076CE364/0/452012AmendedReportonBCHydroSmartMeterMeasurements.pdf

BC Provincial Health Officer - cell phones, radiofrequency waves

http://www.health.gov.bc.ca/pho/issues.html

Health Canada - Smart meters

http://www.hc-sc.gc.ca/hl-vs/iyh-vsv/prod/meters-compteurs-eng.php

World Health Organization – International Agency for Research on Cancer (IARC) – radiofrequency electromagnetic field

IARC Recent Meetings - Recently Evaluated - Vol. 102 [not yet published]
Non-Ionizing radiation, Part II: Radiofrequency Electromagnetic Fields [includes mobile telephones]
Links to this article: Carcinogenicity of radiofrequency electromagnetic fields

US National Institutes of Health

U.S. National Cancer Institute statement on IARC classification of radiofrequency electromagnetic fields
U. S. National Institute of Environmental Health Sciences - Cell Phones

BC Hydro's information and links on radiofrequency and smart meters

http://www.bchydro.com/energy_in_bc/projects/smart_metering_infrastructure_program/faqs/radio_frequency.html?WT.mc_id=rd_metersafety

2.5.1 Please confirm that this Statement of the British Columbia Ministry of Health states:

"Smart Meters emit RF radiation, but only intermittently, and at a level several times below that of the highest level of personal exposures from cell phones, and well below existing limits for RF exposure to the public. Smart Meters generate an RF signal an average of only one minute per day. In addition, Smart Meters emit very low power – about one watt. This is less than 2 microwatts per square centimetre (μW/cm2) when standing adjacent to the meter. A microwatt is one millionth of a watt. And, exposure to RF drops quickly with distance from the device. Three meters (10 feet) from the smart meter, the radio frequency signal drops to less than 0.001 per cent (0.005 μW/cm2) of the Health Canada exposure limits. This exposure level is much less than exposure to RF from cell phone use."

Response:

Confirmed.

2.5.2 Please confirm that this Statement of the British Columbia Ministry of Health states:

"The evidence, therefore, does not support a conclusion that RF fields, whether from cell phones or Smart Meters, can cause brain tumours in adults."

Response:

Confirmed.

2.6 Please provide the World Health Organization backgrounder dated December 2005 and found at:

http://www.who.int/peh-emf/publications/facts/fs296/en/

Response:

The requested document is provided as Attachment BCH 2.6.

Electromagnetic fields (EMF)

Electromagnetic fields and public health

Share Print

Electromagnetic hypersensitivity

Backgrounder
December 2005

For more information contact:

WHO Media centre
Telephone: +41 22 791 2222
E-mail: mediainquiries@who.int

As societies industrialize and the technological revolution continues, there has been an unprecedented increase in the number and diversity of electromagnetic field (EMF) sources. These sources include video display units (VDUs) associated with computers, mobile phones and their base stations. While these devices have made our life richer, safer and easier, they have been accompanied by concerns about possible health risks due to their EMF emissions.

For some time a number of individuals have reported a variety of health problems that they relate to exposure to EMF. While some individuals report mild symptoms and react by avoiding the fields as best they can, others are so severely affected that they cease work and change their entire lifestyle. This reputed sensitivity to EMF has been generally termed “electromagnetic hypersensitivity” or EHS.

This fact sheet describes what is known about the condition and provides information for helping people with such symptoms. Information provided is based on a WHO Workshop on Electrical Hypersensitivity (Prague, Czech Republic, 2004), an international conference on EMF and non-specific health symptoms (COST244bis, 1998), a European Commission report (Bergqvist and Vogel, 1997) and recent reviews of the literature.

What is EHS?

EHS is characterized by a variety of non-specific symptoms, which afflicted individuals attribute to exposure to EMF. The symptoms most commonly experienced include dermatological symptoms (redness, tingling, and burning sensations) as well as neurasthenic and vegetative symptoms (fatigue, tiredness, concentration difficulties, dizziness, nausea, heart palpitation, and digestive disturbances). The collection of symptoms is not part of any recognized syndrome.

EHS resembles multiple chemical sensitivities (MCS), another disorder associated with low-level environmental exposures to chemicals. Both EHS

and MCS are characterized by a range of non-specific symptoms that lack apparent toxicological or physiological basis or independent verification. A more general term for sensitivity to environmental factors is Idiopathic Environmental Intolerance (IEI), which originated from a workshop convened by the International Program on Chemical Safety (IPCS) of the WHO in 1996 in Berlin. IEI is a descriptor without any implication of chemical etiology, immunological sensitivity or EMF susceptibility. IEI incorporates a number of disorders sharing similar non-specific medically unexplained symptoms that adversely affect people. However since the term EHS is in common usage it will continue to be used here.

Prevalence

There is a very wide range of estimates of the prevalence of EHS in the general population. A survey of occupational medical centres estimated the prevalence of EHS to be a few individuals per million in the population. However, a survey of self-help groups yielded much higher estimates. Approximately 10% of reported cases of EHS were considered severe.

There is also considerable geographical variability in prevalence of EHS and in the reported symptoms. The reported incidence of EHS has been higher in Sweden, Germany, and Denmark, than in the United Kingdom, Austria, and France. VDU-related symptoms were more prevalent in Scandinavian countries, and they were more commonly related to skin disorders than elsewhere in Europe. Symptoms similar to those reported by EHS individuals are common in the general population.

Studies on EHS individuals

A number of studies have been conducted where EHS individuals were exposed to EMF similar to those that they attributed to the cause of their symptoms. The aim was to elicit symptoms under controlled laboratory conditions.

The majority of studies indicate that EHS individuals cannot detect EMF exposure any more accurately than non-EHS individuals. Well controlled and conducted double-blind studies have shown that symptoms were not correlated with EMF exposure.

It has been suggested that symptoms experienced by some EHS individuals might arise from environmental factors unrelated to EMF. Examples may include “flicker” from fluorescent lights, glare and other visual problems with VDUs, and poor ergonomic design of computer workstations. Other factors that may play a role include poor indoor air quality or stress in the workplace or living environment.

There are also some indications that these symptoms may be due to pre-existing psychiatric conditions as well as stress reactions as a result of worrying about EMF health effects, rather than the EMF exposure itself.

Conclusions

EHS is characterized by a variety of non-specific symptoms that differ from individual to individual. The symptoms are certainly real and can vary widely in their severity. Whatever its cause, EHS can be a disabling problem for the affected individual. EHS has no clear diagnostic criteria and there is no scientific basis to link EHS symptoms to EMF exposure. Further, EHS is not a medical diagnosis, nor is it clear that it represents a single medical problem.

Physicians: Treatment of affected individuals should focus on the health symptoms and the clinical picture, and not on the person's perceived need for reducing or eliminating EMF in the workplace or home. This requires:

- a medical evaluation to identify and treat any specific conditions that may be responsible for the symptoms,
- a psychological evaluation to identify alternative psychiatric/psychological conditions that may be responsible for the symptoms,
- an assessment of the workplace and home for factors that might contribute to the presented symptoms. These could include indoor air pollution, excessive noise, poor lighting (flickering light) or ergonomic factors. A reduction of stress and other improvements in the work situation might be appropriate.

For EHS individuals with long lasting symptoms and severe handicaps, therapy should be directed principally at reducing symptoms and functional handicaps. This should be done in close co-operation with a qualified medical specialist (to address the medical and psychological aspects of the symptoms) and a hygienist (to identify and, if necessary, control factors in the environment that are known to have adverse health effects of relevance to the patient).

Treatment should aim to establish an effective physician-patient relationship, help develop strategies for coping with the situation and encourage patients to return to work and lead a normal social life.

EHS individuals: Apart from treatment by professionals, self help groups can be a valuable resource for the EHS individual.

Governments: Governments should provide appropriately targeted and balanced information about potential health hazards of EMF to EHS individuals, health-care professionals and employers. The information should include a clear statement that no scientific basis currently exists for a connection between EHS and exposure to EMF.

Researchers: Some studies suggest that certain physiological responses of EHS individuals tend to be outside the normal range. In particular, hyper reactivity in the central nervous system and imbalance in the autonomic nervous system need to be followed up in clinical investigations and the results for the individuals taken as input for possible treatment.

What WHO is doing

WHO, through its International EMF Project, is identifying research needs and co-ordinating a world-wide program of EMF studies to allow a better understanding of any health risk associated with EMF exposure. Particular emphasis is placed on possible health consequences of low-level EMF. Information about the EMF Project and EMF effects is provided in a series of fact sheets in several languages www.who.int/emf/.

FURTHER READING

WHO workshop on electromagnetic hypersensitivity (2004), October 25 - 27, Prague, Czech Republic, www.who.int/peh-emf/meetings/hypersensitivity_prague2004/en/index.html

COST244bis (1998) Proceedings from Cost 244bis International Workshop on Electromagnetic Fields and Non-Specific Health Symptoms. Sept 19-20, 1998, Graz, Austria

Bergqvist U and Vogel E (1997) Possible health implications of subjective symptoms and electromagnetic field. A report prepared by a European group of experts for the European Commission, DGV. Arbete och Hälsa, 1997:19. Swedish National Institute for Working Life, Stockholm, Sweden. ISBN 91-7045-438-8.

Rubin GJ, Das Munshi J, Wessely S. (2005) Electromagnetic hypersensitivity: a systematic review of provocation studies. Psychosom Med. 2005 Mar-Apr;67(2):224-32

Seitz H, Stinner D, Eikmann Th, Herr C, Roosli M. (2005) Electromagnetic hypersensitivity (EHS) and subjective health complaints associated with electromagnetic fields of mobile phone communication---a literature review published between 2000 and 2004. Science of the Total Environment, June 20 (Epub ahead of print).

Staudenmayer H. (1999) Environmental Illness, Lewis Publishers, Washington D.C. 1999, ISBN 1-56670-305-0.

2.6.1 Please confirm that this World Health Organization backgrounder states:

"EHS [Electromagnetic hypersensitivity] is characterized by a variety of non-specific symptoms that differ from individual to individual. The symptoms are certainly real and can vary widely in their severity. Whatever its cause, EHS can be a disabling problem for the affected individual. EHS has no clear diagnostic criteria and there is no scientific basis to link EHS symptoms to EMF exposure. Further, EHS is not a medical diagnosis, nor is it clear that it represents a single medical problem."

Response:

Confirmed.

2.7 Please provide the January 10, 2011 article from the New York Times titled "Health Concerns Over 'Smart' Electric Meters Gain Traction in Calif." located at:

http://www.nytimes.com/gwire/2011/01/10/10greenwire-health-concerns-over-smart-electric-meters-gai-81496.html

Response:

The requested document is provided as Attachment BCH 2.7.

The New York Times

January 10, 2011

Health Concerns Over 'Smart' Electric Meters Gain Traction in Calif.

By DEBRA KAHN of

The newest opposition to "smart" electric meters is gaining traction -- even if its validity is questionable.

Amid claims of malfunctioning meters, privacy issues and dubious economic value, health issues stemming from electromagnetic waves are the latest objection that smart meter opponents have seized upon to block California's multibillion-dollar rollout.

Northern California residents and lawmakers have been sounding the alarm for the past year, saying that the meters, when layered on top of microwaves, cell phones, wireless routers and other emitters, are the final straw.

Things came to a head last week when the Marin County Board of Supervisors passed an **ordinance** (pdf) that deems the installation of smart meters a misdemeanor in some areas of the county (*Greenwire*, Jan. 5).

"What we're trying to say is, it's not just endangered species we need to watch out for," said Katharina Sandizell, a co-director of the nonprofit West Marin Community Coalition for Public Health who was arrested last month for blocking smart meter installers' trucks. "Humans are also the canaries in the closet."

Sandizell believes that her two children could suffer developmental problems, brain tumors or other disorders as a result of electromagnetic radiation.

State Rep. Jared Huffman (D), who represents Marin, introduced a bill last month that would require the California Public Utilities Commission to suspend smart meter installation until there is a provision to allow residents to opt out of the program with a wired meter. He has also requested a report on the potential health effects of smart meters from the state Council on Science and Technology, an advisory group established by the state Legislature, that is due out this month.

"This bill is about giving consumers reasonable choices," Huffman said. "Whether or not you believe RF [radio frequency] exposures from smart meters are harmful, it's only fair that consumers who are concerned about health effects be given complete technical information and the choice of another technology for devices that are installed at their homes."

But are they actually dangerous? A **study** released last week by environmental consulting firm Sage Associates contends that they are. The report claims violations of federal emissions standards at a variety of distances from the meters and argues that the devices are more dangerous when grouped together and when coupled with other wireless technologies.

"Indiscriminate exposure to environmentally ubiquitous pulsed RF from the rollout of millions of new RF sources (smart meters) will mean far greater general population exposures, and potential health consequences," the study says.

The Federal Communications Commission has classified the meters as devices that will be used more than 20 centimeters away from the body and thus can meet an emissions standard that is averaged over time, taking into account the maximum emissions during each transmission. Determining the specific absorption rate (SAR) by the body is not necessary, as it is for cell phones, FCC says.

Additionally, the dangers of several smart meters clustered together is a nonissue because they all use the same transmitter. "The general issue of cumulative exposure from an arbitrary group of transmitter installations or from all transmitters distributed in the environment can appear to be complex, but as discussed, the need for orderly communications requires that a few sources normally dominate," Julius Knapp, FCC's head of engineering and technology, wrote in an August 2010 letter to Sage.

Utility Consumers' Action Network, a watchdog group in San Diego, cites a November 2010 **study** by the Maine Center for Disease Control and Prevention that found no dangers associated with smart meters. "Smart meters appear to be similar to having a wireless router on the side of a house that operates only 10 percent of the time," UCAN says.

The World Health Organization is concerned about cell phones, but not smart meters. "It sort of sounds, I wouldn't say specious, but far-fetched, really," said Daniel Epstein, spokesman for the WHO's Americas region. "If the U.S. or some other country wanted help with that, we'd provide some expertise, but this is not really an issue on our radar at all, with all the major public health problems that we have."

A review of the literature on cell phone use is due out sometime this year, he said, and a 2007 WHO review found that low-frequency electromagnetic fields in general are not

associated with cancer, depression, suicide, cardiovascular disorders, reproductive dysfunction, developmental disorders, immunological modifications, neurobehavioral effects or neurodegenerative disease. In the case of childhood leukemia, which has been found to have a positive association with low-frequency fields, WHO did not find a causal relationship.

The CPUC itself has been reluctant to enter the debate, but its consumer watchdog arm, the Division of Ratepayer Advocates, has been an avid observer. The sheer amount of concern compels the agency to act, said Joe Como, DRA's acting director and general counsel.

"We're not saying there is a health-related problem from exposure to smart meters, but we're saying there is enough public concern and enough studies out there that indicate there may be a health threat in terms of EMF exposure generally that the commission needs to take it seriously and be aboveboard -- having the right kind of experts addressing the problem, making reports available -- so there's an open dialogue about what should be done," Como said.

But Sandizell said she did not have much faith in the CPUC.

"The CPUC is neutered, really; it's regulated by [Pacific Gas & Electric Co.], so if you're saying CPUC has jurisdiction, it's like saying PG&E is the law," she said. Her group is planning on sending a letter airing its concerns to Gov. Jerry Brown (D).

For more news on energy and the environment, visit www.greenwire.com.
Greenwire is published by Environment & Energy Publishing. Read More »

2.7.1 Please confirm the following quote from Daniel Epstein, spokesperson for World Health Organization in Americas region is contained in this New York Times article:

"The World Health Organization is concerned about cell phones, but not smart meters. "It sort of sounds, I wouldn't say specious, but far-fetched, really," said Daniel Epstein, spokesman for the WHO's Americas region. "If the U.S. or some other country wanted help with that, we'd provide some expertise, but this is not really an issue on our radar at all, with all the major public health problems that we have."

A review of the literature on cell phone use is due out sometime this year, he said, and a 2007 WHO review found that low-frequency electromagnetic fields in general are not associated with cancer, depression, suicide, cardiovascular disorders, reproductive dysfunction, developmental disorders, immunological modifications, neurobehavioral effects or neurodegenerative disease. In the case of childhood leukemia, which has been found to have a positive association with low-frequency fields, WHO did not find a causal relationship."

Response:

Confirmed.

2.8 Please provide the report titled "U-17000 Report to the Commission" prepared by staff of the Michigan Public Service Commission dated June 29, 2012, Case No. U-17000 found at:

http://efile.mpsc.state.mi.us/efile/docs/17000/0455.pdf

Response:

The requested document is provided as Appendix BCH IR2 2.8.

2.8.1 Please confirm the Executive Summary (on page 2) of this report states:

"After careful review of the available literature and studies, the Staff has determined that the health risk from the installation and operation of metering systems using radio transmitters is insignificant. In addition, the appropriate federal health and safety regulations provide assurance that smart meters represent a safe technology."

Response:

Confirmed.

2.9 Please provide the April 2011 study on the health impacts of RF affects from Smart Meters titled "Health Impacts of Radio Frequency Exposure from Smart Meters" by the California Council on Science and Technology, located at:

http://ccst.us/publications/2011/2011smart-final.pdf

Response:

The requested document is provided as Appendix BCH IR2 2.9.

2.9.1 Please confirm that the key report findings, found on page 4, are:

"1. Wireless smart meters, when installed and properly maintained, result in much smaller levels of radio frequency (RF) exposure than many existing common household electronic devices, particularly cell phones and microwave ovens.

2. The current FCC standard provides an adequate factor of safety against known thermally induced health impacts of existing common household electronic devices and smart meters.

3. To date, scientific studies have not identified or confirmed negative health effects from potential non-thermal impacts of RF emissions such as those

produced by existing common household electronic devices and smart meters.

4. Not enough is currently known about potential non-thermal impacts of radio frequency emissions to identify or recommend additional standards for such impacts"

Response:

Confirmed.

2.10 Please provide the March 2011 report titled "A Discussion of Smart Meters and RF Exposure Issues" publish by the Edison Electric Institute, located at:

http://www.aeic.org/meter_service/smartmetersandrf031511.pdf

Response:

The requested document is provided as Appendix BCH 2.10.

2.10.1 Please confirm the report stated in the Executive Summary (page 3):

"• All smart meter radio devices must be certified to the FCC's rules.

• Tests simulating multi-family metering locations containing several meters in close proximity have shown RF exposure levels dramatically less than FCC limits.

• The FCC limits on maximum permissible exposure (MPE) for application to the general public were set using safety factors fifty times lower than the levels of known effects.

• Exposure levels drop significantly (1) with the distance from the transmitter, (2) with spatial averaging, and (3) in living spaces due to the attenuation effects of building materials.

• Due to shielding of the meter enclosure and signal patterns, RF exposure from the rear of a metering location is nominally 10 times less than in

front of the meter and dramatically below FCC limits, not including the spatial averaging and building material attenuation reductions.2

- For measurement and calculation purposes some studies use a 100% duty cycle parameters. However, the maximum operational Duty Cycle for Smart meter systems is less than 50% to prevent message traffic congestion and collisions. The typical Duty Cycles for Smart Meter Systems is between 1% and 5%.
- An RF exposure comparison of a person talking on a cell phone and a person 3 and 10 feet from a continuously operating Smart Meter would result in Smart Meter RF exposure 125 to 1250 times less than the cell phone.3
- In test environments simulating operational conditions, for power (250 mWatt - 2 Watt), duty cycle (2%-5%) at close distance (1 foot) from in front of the transmitter, Smart Meters produce very low RF exposure to the consumer, typically well under 10 % of the FCC exposure regulations."

Response:

Confirmed.

2.11 Please provide the Electric Power Research Institute (EPRI) 2010 technical report titled "An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter" located at:

http://my.epri.com/portal/server.pt?space=CommunityPage&cached=true&parentname=ObjMgr&parentid=2&control=SetCommunity&CommunityID=221&PageIDqueryComId=0

If this link does not work, the report may be obtained by searching by the report title at EPRI's home page found at:

http://my.epri.com/portal/server.pt?

Response:

The requested document is provided as Appendix BCH IR2 2.11.

2.11.1 Please confirm that on page 1-5 this report states:

"A detailed evaluation of possible RF fields produced by the Itron meters included in this study shows that regardless of duty cycle values for end point and cell relay meters, typical exposures that result from the operation of Smart Meters are very low and comply with scientifically based human exposure limits by a wide margin."

Response:

Confirmed.

2.12 Please provide C95.1-2005 - IEEE Standard for Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz located at:

http://standards.ieee.org/findstds/standard/C95.1-2005.html

Response:

The requested document is provided as Appendix BCH IR2 2.12.

2.13 Please provide the report from International Commission on Non-Ionizing Radiation Protection (ICNIRP) titled "Exposure to high frequency electromagnetic fields, biological effects and health consequences (100 kHz-300 GHz)" located at:

http://www.icnirp.de/documents/RFReview.pdf

Response:

The requested document is provided as Appendix BCH IR2 2.13.

3.0 Reference: Appendix of CSTS IR1 59.4, BCSEA IR1 10.5

3.1 Please provide the report from IEEE 802.15 Working Group dated September 2010 titled "Coexistence analysis of IEEE Std802.15.4 with other IEEE standards and proposed standards." found at:

http://grouper.ieee.org/groups/802/19/pub/CA/15-10-0808-00-0000-802-15-4-2011-coexistence-analysis.pdf

Response:

The requested document is provided as Appendix BCH IR2 3.1.

3.2 Please confirm that the IEEE 802.15 working group states on page 2:

"While not required by this standard, IEEE 802.15.4 devices can be reasonably expected to "coexist," that is, to operate in proximity to other wireless devices."

Response:

Confirmed.

4.0 Reference: CSTS IR1 21.4

The position of a limited number of scientists who have been most vociferous in advocating a contrary position is summarized in the 2007 Bioinitiative report (http://www.bioinitiative.org/).

4.1 Please provide a copy of the report from Health Council of Netherlands dated September 2008 that analyzed the scientific value of the BioInitiative report, found at:

http://www.gezondheidsraad.nl/en/publications/bioinitiative-report-0

Response:

The requested document is provided as Attachment BCH 4.1.

Health Council of the Netherlands

The Minister of Housing, Spatial Planning
and the Environment (VROM)

Subject	: BioInitiative report
Your ref.	: -
Our ref.	: U-5601/EvR/iv/673-L1 Publication nr 2008/17E
Annexes	: -
Date	: 2 September 2008

Dear Minister,

A report published on 31 August 2007 is playing an increasingly prominent role in the debate on electromagnetic fields and health: the *BioInitiative Report: A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF)*[1]. The report contains recommendations on establishing limits for exposure to electromagnetic fields that are much lower than the limits that are currently applied in the Netherlands and in many other countries, and is receiving increasing attention from society.

Your Ministry has expressed interest in a judgement of the Health Council on the BioInitiative report. In this advisory letter therefore, the Council's Electromagnetic Fields Committee, after consultation of the Standing Committee on Radiation and Health, gives its opinion as to the scientific value of this report.

Method used to compile the BioInitiative report

Scientific advisory reports are usually the result of a process in which a group of experts, using the current state of science, extensively discusses a topic until a consensus is reached. The group is made up of independent experts from the various areas of expertise relevant to the topic. In the case of electromagnetic fields, for example, this would be biologists, epidemiologists, technical experts, physicians and in some cases also psychologists and risk experts. This procedure is followed by bodies such as the World Health Organisation (WHO) and the Health Council, as well as organisations involved in drafting proposals for exposure limits, such as the International Commission on Non-ionizing Radiation Protection (ICNIRP) and the International Commission

[1] See www.bioinitiative.org.

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for Electromagnetic Safety (ICES) of the Institute of Electrical and Electronics Engineers (IEEE). The various experts and the interactions between them, combined with a review of all relevant scientific information, ensure that a balanced judgement on the latest scientific knowledge can be reached. It is of importance that this process is transparent. This multidisciplinary weight-of-evidence method leads to a scientifically sound judgement that is as objective as possible.

The BioInitiative report did not follow this procedure. The report is a collection of a number of chapters, called 'sections', written by individual authors. Seemingly no consultation or discussion on these sections took place between the authors. The report also does not indicate what, if any, brief was given to the authors. In any event, the sections were not written in a standardised way. Notably, not all authors are scientists. The methods used to collect literature are not defined. In many cases a selection of the available scientific material has been made, but the selection criterion is not stated. The Committee points for example to Section 12, in which the authors refer, among other things, to epidemiological studies into the association between exposure to 50 Hz magnetic fields and the prevalence of breast cancer. The authors dismiss a number of studies carried out in the home environment because exposure could not be determined with sufficient accuracy. However, this also applies to all studies into the association between living close to power lines and the prevalence of childhood leukaemia, which are discussed at length in another section of the report. The authors have also excluded various studies that did not find an association between breast cancer and exposure to magnetic fields from their analysis. It can be concluded that the scientific quality of the review sections is extremely varied.

The first section, written by one of the main initiators of the BioInitiative report, contains the summary and conclusions, which in many cases go further than the conclusions reached by the authors of the review sections. It is unclear if or how this has been discussed with them, whether they support the phrasing of conclusions in the Summary and on what basis the author reached different conclusions.

Why was the BioInitiative report written?

In Sections 2, 3 and 4, the same author presents exhaustive arguments in support of her belief why the current exposure limits are inadequate. In Section 2, the reason for writing the report is given:

The Report has been written to document the reasons why current public exposure standards for non-ionizing electromagnetic radiation are no longer good enough to protect public health.

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Upfront, therefore, the reason for writing the report was not to give an objective analysis of the current state of science, that would subsequently lead to recommendations. Instead, the aim was to present information to demonstrate why current standards are inadequate.

Shortcomings

In addition to the objections of principle and methodology outlined above, several sections also contain a number of factual errors. The Committee gives two examples. On page 6 of Section 1 the author states:

It appears it is the INFORMATION[2] *conveyed by electromagnetic radiation (rather than heat) that causes biological changes - some of these biological changes may lead to loss of wellbeing, disease and even death.*

This statement lacks a scientific basis and is, according to the Committee, incorrect. First of all no information is being transferred by low frequency fields and heating does not occur. With radiofrequency fields, information is being transferred by modulation. Some experimental studies found indications that certain biological effects may occur upon exposure to a modulated signal, but not, or to a lesser extent, with exposure to an unmodulated signal. As yet, there is no sufficient scientific evidence to confirm this. It is not known whether such effects may lead to health effects. The suggestion that some of the observed biological effects may lead to reduced wellbeing, disease, or even death lacks scientific basis.

On page 15 of Section 1 the author states:

For example, the roll-out of the new 3rd Generation wireless phones (and related community-wide antenna RF emissions in the Netherlands) caused almost immediate public complaints of illness.(5)

The reference is to a 2003 TNO study. [3] Both the statement and the reference to the TNO study are not correct. Long before UMTS networks were put into service some people already attributed

[2] Capitalization by the author.

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various health complaints to electromagnetic fields, especially those generated by GSM base stations. The TNO study indicated that exposure to an UMTS base station-like signal (but not to a GSM signal) might have a negative influence on wellbeing. Publication of this study led to public concern and an increase in the number of complaints, even without UMTS signals being transmitted. Four independent follow-up studies did not find any indications to confirm the TNO results.[4]

The Committee will not go into further detail here with regard to the many other shortcomings of the report, which runs to over 600 pages. If necessary, this can be done in another publication. All these deficiencies also do not add to the Committee's confidence in the quality of the BioInitiative report.

Conclusion

In view of the way the BioInitiative report was compiled, the selective use of scientific data and the other shortcomings mentioned above, the Committee concludes that the BioInitiative report is not an objective and balanced reflection of the current state of scientific knowledge. Therefore, the report does not provide any grounds for revising the current views as to the risks of exposure to electromagnetic fields.

The BioInitiative report argues that any effect of electromagnetic fields on biological systems should be avoided, thereby ignoring the distinction between effect and damage. The Committee does not agree with this approach, as documented in previous publications (for example, in the

[3] Zwamborn, APM, Vossen, SHJA, van Leersum, B, e.a. Effects of global communication system radio-frequency fields on well being and cognitive functions of human subjects with and without subjective complaints. The Hague: TNO Physics and Electronics Laboratory, 2003; FEL-03-C148.

[4] - Regel, SJ, Negovetic, S, Röösli, M, e.a. UMTS base station-like exposure, well-being, and cognitive performance. Environ Health Perspect, 2006; 114(8): 1270-1275.
- Riddervold, IS, Pedersen, GF, Andersen, NT, e.a. Cognitive function and symptoms in adults and adolescents in relation to rf radiation from UMTS base stations. Bioelectromagnetics, 2008; 29(4): 257-267.
- Eltiti, S, Wallace, D, Ridgewell, A, e.a. Does short-term exposure to mobile phone base station signals increase symptoms in individuals who report sensitivity to electromagnetic fields? A double-blind randomised provocation study. Environ Health Perspect, 2007;115(11): 1603-1608.
- Furubayashi, T, Ushiyama, A, Terao, Y, e.a. Effects of short-term W-CDMA mobile phone base stations exposure on women with and without mobile phone related symptoms. Bioelectromagnetics, 2008; in press.

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Health Council of the Netherlands

Subject : BioInitiative report
Our ref. : U-5601/EvR/iv/673-L1 Publication nr 2008/17E
Page : 5
Date : 2 September 2008

2002 advisory report entitled *Mobile telephones; an evaluation of health effects*). In the 2008 Annual Update on Electromagnetic Fields this topic will be further addressed.

Yours sincerely,

Prof M. de Visser
Vice-president

The following members served on the Electromagnetic fields committee while this advisory report was being produced: • Dr G.C. van Rhoon, physicist; Erasmus University Medical Centre Rotterdam, *chairman* • Dr L.M. van Aernsbergen, physicist; Ministry of Housing, Spatial Planning and the Environment, The Hague, *advisor* • Prof G. Brussaard, Emeritus Professor of Radio communication; Eindhoven University of Technology • Dr G. Kelfkens, physicist, National Institute for Public Health and the Environment, Bilthoven, *advisor* • Prof H. Kromhout, Professor of Occupational Hygiene and Exposure Determination, Institute for Risk Assessment Sciences, University of Utrecht • Prof F.E. van Leeuwen, Professor of Cancer Epidemiology; Free University Amsterdam, and Dutch Cancer Institute, Amsterdam • Dr H.K. Leonhard, physicist; Ministry of Economic Affairs, Groningen, *advisor* • Prof W.J. Wadman, Professor of Neurobiology, University of Amsterdam • D.H.J. van de Weerdt, MD, specialist in medical environmental affairs; Gelderland Midden emergency services / Arnhem mental health services • Prof A.P.M. Zwamborn, Professor of Electromagnetic Effects; Eindhoven University of Technology, and TNO, The Hague • Dr E. van Rongen, radiobiologist; Health Council, The Hague, *secretary*.

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4.1.1 Please confirm that the Health Council of Netherlands concluded on page 4 that:

"In view of the way the BioInitiative report was compiled, the selective use of scientific data and the other shortcomings mentioned above, the Committee concludes that the BioInitiative report is not an objective and balanced reflection of the current state of scientific knowledge. Therefore, the report does not provide any grounds for revising the current views as to the risks of exposure to electromagnetic fields."

Response:

Confirmed.

4.2 Please provide a copy of the report from European Commission Joint Research Centre EMF-Net, date October 30, 2007, that analyzed the scientific value of the BioInitiative report, and found at:

http://ihcp.jrc.ec.europa.eu/our_activities/public-health/exposure_health_impact_met/emf-net/docs/efrtdocuments/EMF-NET%20Comments%20on%20the%20BioInitiative%20Report%2030OCT2007.pdf

Response:

The requested document is provided as Attachment BCH 4.2.

EC FP6
Coordination Action

EFFECTS OF THE EXPOSURE TO ELECTROMAGNETIC FIELDS: FROM SCIENCE TO PUBLIC HEALTH AND SAFER WORKPLACE

Comments on the BioInitiative Working Group Report (BioInitiative Report)

October 30, 2007

The "BioInitiative Report: A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF)", co-edited by David Carpenter and Cindy Sage, was released on 31 August 2007. In the covering letter it is noted: "The information and conclusions in each chapter are the responsibilities of the authors of that chapter". This means that this report is not a consensus of a working group, but rather an assembly of chapters written by various scientists and consultants. There is no mention of who initiated this review or who funded the work, nor of potential conflicts of interest.
Ms Cindy Sage of Sage Associates (USA) is the author of the "Summary for the public" that is written in an alarmist and emotive language and whose arguments have no scientific support from well-conducted EMF research. She is also the author of five more chapters (with a total of 6 out of 17 chapters) and the co-author of the final key chapter on policy recommendations.

There is a lack of balance in the report; no mention is made in fact of reports that do not concur with authors' statements and conclusions. The results and conclusions are very different from those of recent national and international reviews on this topic (see Annex 1 and 2).

The stated purpose of the BioInitiative Report is to assess the scientific evidence of health effects of low-level EMF exposure below current international limits, and to establish which changes are needed to reduce public health risks from EMF exposure.
If this report were to be believed, EMF would be the cause of a variety of diseases and subjective effects, including: Sleeplessness, headache, fatigue, skin disorders and changes in skin sensitivity, loss of appetite, tinnitus, impairment of memory and concentration, Alzheimer's and Parkinson's disease, cardiac problems, changes in brain and nervous systems activity, stress reactions, inflammatory and allergic reactions, genotoxic effects, changes in immune system function, and many types of cancers.
None of these health effects has been classified as established in any national or international reviews that assessed biological and health effects from exposures below internationally accepted EMF limits when the whole database of scientific literature is reviewed according to well-accepted international risk assessment methods and criteria (see Annex 1 and 2).

Table 1.1 (pp. 34-49) gives the overall conclusions of the BioInitiative Report.
None of these conclusions is supported by the major national or international reviews as listed in Annexes 1 and 2, that have made use of the internationally accepted weight-of-evidence approach to study results. The BioInitiative Report advocates the use of precautionary measures. Consideration of precaution is also recommended by WHO, who notes however that it is the responsibility of national authorities to adopt precautionary measures if deemed appropriate, and that, if adopted, such measures should be based on local priorities and cost-effectiveness.

The chapter headings and their authors are given below:
1. Summary for the public and conclusions (Ms. Sage)
2. Statement of the problem (Ms. Sage)
3. The existing public exposure standards (Ms. Sage)
4. Evidence for inadequacy of the standards (Ms. Sage)
5. Evidence for effects on gene and protein expression (transcriptomic and proteomic research) (Drs. Xu, Chen)
6. Evidence for genotoxic effects – RFR and ELF DNA damage (Dr. Lai)
7. Evidence for stress response (stress proteins) (Dr. Blank)
8. Evidence for effects on immune function (Dr. Johansson)
9. Evidence for effects on neurology and behavior (Dr. Lai)
10. Evidence for brain tumors and acoustic neuromas (Drs Hardell, Mild, Kundi)
11. Evidence for childhood cancers (leukaemia) (Dr. Kundi)
12. Magnetic field exposure: melatonin production; Alzheimer's disease; breast cancer (Drs. Davanipour, Sobel)
13. Evidence for breast cancer promotion (melatonin links in laboratory and cell studies) (Ms. Sage)
14. Evidence for disruption by the modulating signal (Dr. Blackman)
15. Evidence based on EMF medical therapeutics (Ms. Sage)
16. The precautionary principle (Mr. Gee)
17. Key scientific evidence and public health policy recommendations (Dr. Carpenter, Ms. Sage)
18. List of participants and affiliations
19. Glossary of terms and abbreviations
20. Appendix - ambient ELF and RF levels, average residential and occupational exposures
21. Acknowledgements

Annex 1: Statements from Governments and Expert Panels Concerning Health Effects and Safe Exposure Levels of Radiofrequency Fields (RF)

EC FP6 EMF-NET Coordination Action

EMF-NET EFRT Comments on Health Risk Posed by Aerials of Mobile Telephone Base Stations (RE: Written Question QE4450/06), October 30, 2006, 4/2006

EMF-NET EFRT Opinion on the Possible Risk Concerned with Living at a Given Distance from Radio Base Stations, December 7, 2006

EMF-NET EFRT Opinion on Health Effects of Telephone Masts, December 16, 2004

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http://www.hpa.org.uk/radiation/publications/documents_of_nrpb/abstracts/absd15-3.htm

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U.S. Department of Health and Human Services, Centers for Disease Prevention and Control (2005) CDC Fact Sheet: Frequently Asked Questions about Cell Phones and Your Health

http://www.cdc.gov/nceh/radiation/factsheets/cellphone_facts.pdf

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http://emr.acma.gov.au/mobile_phone_health.pdf

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http://www.hc-sc.gc.ca/iyh-vsv/prod/cell_e.html

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http://www.ofta.gov.hk/en/freq-spec/radiation.pdf

Health Council of the Netherlands (*2007)*

http://www.healthcouncil.nl/pdf/Press%20release%20200706%20site.pdf

Ireland Expert Group on Health Effects of Electromagnetic Fields (2007)

http://www.dcmnr.gov.ie/NR/rdonlyres/9E29937F-1A27-4A16-A8C3-F403A623300C/0/ElectromagneticReport.pdf

European Commission Scientific Committee on Emerging and Newly Identified Health Risks. Possible Effects of Electromagnetic Fields (EMF) on Human Health (2007)

http://ec.europa.eu/health/ph_risk/committees/04_scenihr/docs/scenihr_o_007.pdf

World Health Organization (2007)

http://www.who.int/peh-emf/about/WhatisEMF/en/index1.html

http://www.who.int/peh-emf/meetings/ottawa_june05/en/index4.html

WHO Fact Sheet #304: Electromagnetic fields and public health: Base stations and wireless technologies

http://www.who.int/mediacentre/factsheets/fs304/en/index.html

Ministry of Internal Affairs and Communications (MIC), Japan (2007) On the Report Compiled by the Committee to Promote Research on the Possible Biological Effects of Electromagnetic Fields

http://www.soumu.go.jp/joho_tsusin/eng/Releases/NewsLetter/Vol18/Vol18_06/Vol18_06.pdf

Annex 2: Statement from the World Health Organization (WHO) Concerning Health Effects of Extremely Low Frequency Fields (ELF)

Reference: World Health Organization (2007) Fact sheet N°322 June 2007, Electromagnetic fields and public health: Exposure to extremely low frequency fields.

In 2002, IARC published a monograph classifying ELF magnetic fields as "possibly carcinogenic to humans". This classification is used to denote an agent for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence for carcinogenicity in experimental animals (other examples include coffee and welding fumes). This classification was based on pooled analyses of epidemiological studies demonstrating a consistent pattern of a two-fold increase in childhood leukaemia associated with average exposure to residential power-frequency magnetic field above 0.3 to 0.4 µT. The Task Group concluded that additional studies since then do not alter the status of this classification.

However, the epidemiological evidence is weakened by methodological problems, such as potential selection bias. In addition, there are no accepted biophysical mechanisms that would suggest that low-level exposures are involved in cancer development. Thus, if there were any effects from exposures to these low-level fields, it would have to be through a biological mechanism that is as yet unknown. Additionally, animal studies have been largely negative. Thus, on balance, the evidence related to childhood leukaemia is not strong enough to be considered causal.

Childhood leukaemia is a comparatively rare disease with a total annual number of new cases estimated to be 49,000 worldwide in 2000. Average magnetic field exposures above 0.3 µT in homes are rare: it is estimated that only between 1% and 4% of children live in such conditions. If the association between magnetic fields and childhood leukaemia is causal, the number of cases worldwide that might be attributable to magnetic field exposure is estimated to range from 100 to 2400 cases per year, based on values for the year 2000, representing 0.2 to 4.95% of the total incidence for that year. Thus, if ELF magnetic fields actually do increase the risk of the disease, when considered in a global context, the impact on public health of ELF EMF exposure would be limited.

A number of other adverse health effects have been studied for possible association with ELF magnetic field exposure. These include other childhood cancers, cancers in adults, depression, suicide, cardiovascular disorders, reproductive dysfunction, developmental disorders, immunological modifications, neurobehavioural effects and neurodegenerative disease. The WHO Task Group concluded that scientific evidence supporting an association between ELF magnetic field exposure and all of these health effects is much weaker than for childhood leukaemia. In some instances (i.e. for cardiovascular disease or breast cancer) the evidence suggests that these fields do not cause them.

For high-level short-term exposures to EMF, adverse health effects have been scientifically established (ICNIRP, 2003). International exposure guidelines designed to protect workers and the public from these effects should be adopted by policy makers. EMF protection programs should include exposure measurements from sources where exposures might be expected to exceed limit values.

Regarding long-term effects, given the weakness of the evidence for a link between exposure to ELF magnetic fields and childhood leukaemia, the benefits of exposure reduction on health are unclear. In view of this situation, the following recommendations are given:

Government and industry should monitor science and promote research programmes to further reduce the uncertainty of the scientific evidence on the health effects of ELF field exposure. Through the ELF risk assessment process, gaps in knowledge have been identified and these form the basis of a new research agenda.

Member States are encouraged to establish effective and open communication programmes with all stakeholders to enable informed decision-making. These may include improving coordination and consultation among industry, local government, and citizens in the planning process for ELF EMF-emitting facilities.

When constructing new facilities and designing new equipment, including appliances, low-cost ways of reducing exposures may be explored. Appropriate exposure reduction measures will vary from one country to another. However, policies based on the adoption of arbitrary low exposure limits are not warranted.

4.2.1 Please confirm that the European Commission Joint Research Centre concluded on page 1 that:

"None of these conclusions [of the Bio Initiative Working Group Report] is supported by the major national or international reviews as listed in Annexes 1 and 2, that have made use of the internationally accepted weight-of-evidence approach to study results."

Response:

Confirmed.

4.3 Please provide a copy of the report dated December 18, 2008 from the Australian Centre for Radiofrequency Bioeffects Research on the scientific validity of the BioInitiative report, found at:

http://acrbr.org.au/FAQ/ACRBR%20Bioinitiative%20Report%2018%20Dec%202008.pdf

Response:

The requested document is provided as Attachment BCH 4.3.

Australian Centre for Radiofrequency Bioeffects Research (ACRBR)

Rodney Croft, Michael Abramson, Irena Cosic
John Finnie, Ray McKenzie, Andrew Wood

"ACRBR Position Statement on *BioInitiative Report*"

ACRBR
Australian Centre for RF Bioeffects Research
An NHMRC Centre of Research Excellence

December 18, 2008

The ACRBR Perspective on The BioInitiative Report

In 2007 a group of interested individuals collated a series of views on the non-ionising radiation health debate. This was entitled the BioInitiative Report[1], a web document dated August 31, 2007. The BioInitiative Report presents a series of views that argue for a change in public exposure standards, but which are largely inconsistent with current scientific consensus. The ACRBR have received numerous queries about this report from the general public, and have provided this document to answer a few questions to clarify its perspective on the report.

Do the BioInitiative Report authors represent an authoritative international body?

Often in assessing public health issues, bodies are formed to evaluate evidence and offer recommendations about particular issues. The model that most scientific expert bodies in this area (e.g. World Health Organisation (WHO)) employ is to engage independent experts to provide a review and recommendations on an issue. Independent experts are engaged because it is meant to provide an objective evaluation of the issue. This contrasts strongly with the BioInitiative Report, which is the result of the opinions of a self-selected group of individuals who each have a strong belief that does not accord with that of current scientific consensus. An indication of this may be seen in the group's stated purpose, which is "*to document the reasons why current public exposure standards for non-ionizing electromagnetic radiation are no longer good enough to protect public health"* (Section 2, page 1), rather than to provide a scientific evaluation of the issue. Similarly, the standard model normally seeks a consensus view. In terms of the BioInitiative Report, the preface by Carpenter and Sage state that this is not a consensus document, but is rather a collection of individual views, where "*the information and conclusions in each chapter are the responsibilities of the authors of that chapter*" (Section i, page 1). Thus the 'Summary for the Public and Conclusions', released both independently and as part of the full Report, should be read as Sage's view on the matter, and there is no indication in the Report that the authors of other chapters share her views. This does not mean that what is written in the Report is invalid, but it means that we need to evaluate the content of the report itself, and cannot rely on there being a consensus from an independent authoritative body to help us judge the merits of these conclusions.

What is the scientific status of the BioInitiative Report?

In science we generally differentiate between peer-reviewed and non-peer-reviewed publications, where the peer-review comes from independent experts in the area. The reason for this is that peer-reviewed work is only published after independent scientific peers have reviewed the work and agreed with its scientific merit, making it easier for the reader to be confident with conclusions drawn in the publication. Conversely, without independent peer review, there is far less opportunity to correct errors and ensure that the conclusions are appropriate, and thus scientists treat peer-reviewed publications as their main scientific literature source. It should be noted that this does not mean that publications lacking independent peer review are flawed (or for that matter that peer-reviewed publications are perfect), it is more that scientists would typically withhold judgment about publications until peer review has occurred.

The BioInitiative Report has not undergone such independent peer review, and so the conclusions that it reaches would normally be viewed more as views of some of the authors, rather than strong contributions to science. In fact the Report does not identify the level of review that it has

undergone, merely mentioning that "*another dozen outside reviewers have looked at and refined the Report*" (Section 1, page 4). This is particularly important since many of the statements and conclusions in the Report are contrary to scientific consensus. Thus rigorous scientific evaluation would need to be performed to determine whether the inconsistencies are due to errors in the report, or errors in the scientific consensus. While such independent peer review would normally be undertaken prior to publication (to avoid misleading conclusions should problems be identified), some informal independent peer review has now occurred *in response to* publication of the BioInitiative Report. For example, the Health Council of the Netherlands (HCN) recently published a report that noted a number of inadequacies in the BioInitiative Report, inadequacies that would normally be addressed during the peer review process[2].

Of particular note is that the BioInitiative Report does not appear to apply principles consistently, which biases its conclusions. For example, in arguing for a link between 50/60 Hz power lines and breast cancer, the Report does not consider some of the evidence that argues against such an association. It also provides an argument for excluding other evidence (poor exposure assessment) that is not employed for studies arguing for an association between 50/60 Hz power lines and childhood leukemia (even though they are subject to the same exposure assessment limitations; see Section 12 of the Report). Another issue is that there are statements that do not accord with the standard view of science, and the Report does not provide a reasonable account of why we should reject the standard view in favour of the views espoused in the Report.

Should we be convinced by the BioInitiative Report?

Overall we think that the BioInitiative Report does not progress science, and would agree with the Health Council of the Netherlands[2] that the BioInitiative Report is "not an objective and balanced reflection of the current state of scientific knowledge" (page 4). As it stands it merely provides a set of views that are not consistent with the consensus of science, and it does not provide an analysis that is rigorous-enough to raise doubts about the scientific consensus.

It is worth noting that the state of science in this area is continually being debated and updated by a number of expert bodies comprised of the leading experts in this field. For example, the World Health Organisation (WHO) Electromagnetic Fields (EMF) project[3], the International Commission on Non-Ionizing Radiation Protection (ICNIRP)[4], the UK Mobile Telecommunications and Health Research (MTHR) programme[5], and here in Australia the Australian Radiation Protection and Nuclear Science Agency (ARPANSA)[6] have all provided authoritative analyses of the electromagnetic radiation bioeffects research. The WHO Environment Health Criteria 238 also provides a thorough analysis of the literature to date in relation to extremely low frequency (ELF, or powerline electromagnetic fields)[7]. We have provided some web links to these below, and would strongly urge the interested reader to consult these for a balanced perspective on this fascinating research domain.

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[1] BioInitiative Report: A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF), August 31, 2007 http://www.bioinitiative.org/report/index.htm.
[2] Health Council of the Netherlands. BioInitiative report. The Hague: Health Council of the Netherlands, 2008; publication no. 2008/17E. http://www.gr.nl/pdf.php?ID=1743&p=1
[3] http://www.who.int/peh-emf/en/
[4] http://www.icnirp.de/
[5] http://www.mthr.org.uk/documents/MTHR_report_2007.pdf
[6] http://www.arpansa.gov.au/mobilephones/index.cfm
[7] http://www.who.int/peh-emf/publications/Complet_DEC_2007.pdf

4.3.1 Please confirm that the Australian Centre for Radiofrequency Bioeffects Research states with respect to the BioInitiative report:

"Overall we think that the BioInitiative Report does not progress science, and would agree with the Health Council of the Netherlands2 that the BioInitiative Report is "not an objective and balanced reflection of the current state of scientific knowledge" (page 4). As it stands it merely provides a set of views that are not consistent with the consensus of science, and it does not provide an analysis that is rigorous-enough to raise doubts about the scientific consensus."

Response:

Confirmed.

U-17000 Report to the Commission

Prepared by the Staff of the Michigan Public Service Commission

June 29, 2012

EXECUTIVE SUMMARY

The smart grid encompasses technological improvements to the electric grid designed to increase reliability, reduce outage time, accommodate the integration of distributed generation sources, and improve electric vehicle charging capacity. Advanced Metering Infrastructure (AMI) systems "combine meters with two-way communication capabilities. These systems typically are capable of recording near-real-time data on power consumption and reporting that consumption to the utility at frequencies of an hour or less".[1] AMI meters are also known as smart meters, and they represent one component of an improved or smart grid.

On January 12, 2012, the Michigan Public Service Commission (Commission) issued an order in Case No. U-17000. This order directed the utilities to provide information by March 16, 2012, regarding their plans for smart meter deployment including proposed costs and benefits, scientific information addressing the safety of smart meter deployment, assurance of customer data privacy and other information. The order also allowed for public comments in response to the utilities' filings to be submitted by April 16, 2012.

Approximately 400 residential customer comments were received. The vast majority of these comments voice concerns about the installation of smart meters. The concerns can generally be categorized into the following topics: health and safety, privacy/data security, cyber security and bill impacts.

The Staff has engaged in a thorough review of resources in response to public concerns about smart meters. The resources fall into one or more of the following categories: technical in nature, relevant to smart meter technology, research focused, science based, peer reviewed, commentary and/or opinion.

The Staff's review supports the following conclusions:

- Smart meters are quickly becoming the primary replacement meter to the existing electromechanical meters because they are more accurate, enhance outage response and offer opportunities for customer energy management. The traditional electromechanical meter is obsolete and currently not in production.
- Smart meters are an important component to the success of a much larger picture, an emerging smart grid. As the United States Department of Energy (U.S. DOE) states "[a] smart grid uses digital technology to improve the reliability, security, and efficiency of the electricity system . . .."[2]
- After careful review of the available literature and studies, the Staff has determined that the health risk from the installation and operation of metering systems using radio transmitters is insignificant. In addition, the appropriate federal health and safety regulations provide assurance that smart meters represent a safe technology.

[1] Massachusetts Institute of Technology, *The Future of the Electric Grid*; An Interdisciplinary MIT Study, 2011, p.133. http://web.mit.edu/mitei/research/studies/documents/electric-grid-2011/Electric_Grid_Full_Report.pdf
[2] U.S. Department of Energy, *2010 Smart Grid System Report*, February 2012, Message from the Assistant Secretary. http://energy.gov/sites/prod/files/2010%20Smart%20Grid%20System%20Report.pdf

- Data privacy and cyber security continue to be priorities for customers, utilities and the Commission. Data protection procedures are continually being updated at the national and state levels. Michigan utilities currently have large amounts of critical customer information that they have safeguarded for years and will continue to adequately safeguard. Several national organizations are focused on monitoring and improving cyber security efforts that will continue to guide electric service providers' efforts.

The Staff's Recommendations

Smart Meter Implementation: Smart meters are part of the larger smart grid initiative that is being pursued by investor-owned and other utilities throughout the world. The smart grid initiative has been endorsed by federal laws and the technologies have been declared to be safe by accredited national agencies and industry councils. The Staff recommends that the Commission regulated utilities in Michigan continue to assess smart grid technologies as part of their efforts to improve the reliability and efficiency of the grid. AMI investments should continue to be reviewed by the Commission in contested rate cases.

Opt-out: A minority of customers have expressed concerns about smart meters. The Staff understands that some people remain opposed to the installation of smart meters for a number of reasons and should be allowed to opt-out. The Staff believes that ratemaking for the opt-out provision should be based on cost of service principles. If AMI meters result in a reduced cost of service, this could be accounted for by either an additional charge for those customers choosing to opt-out or a discount for those customers with an AMI meter.

Revised Rules and/or Tariffs: Several comments reflect concerns about customer privacy and data security. The Staff recommends there be additional consideration to ensure consistent protection of customer privacy and data.

Smart Grid Vision: The Staff has created a comprehensive smart grid vision which provides an all-inclusive perspective of the emerging smart grid. The vision will provide a framework for future grid modernization.

Details of these recommendations are contained in the body of this report.

SUMMARY OF DOCKET FILINGS

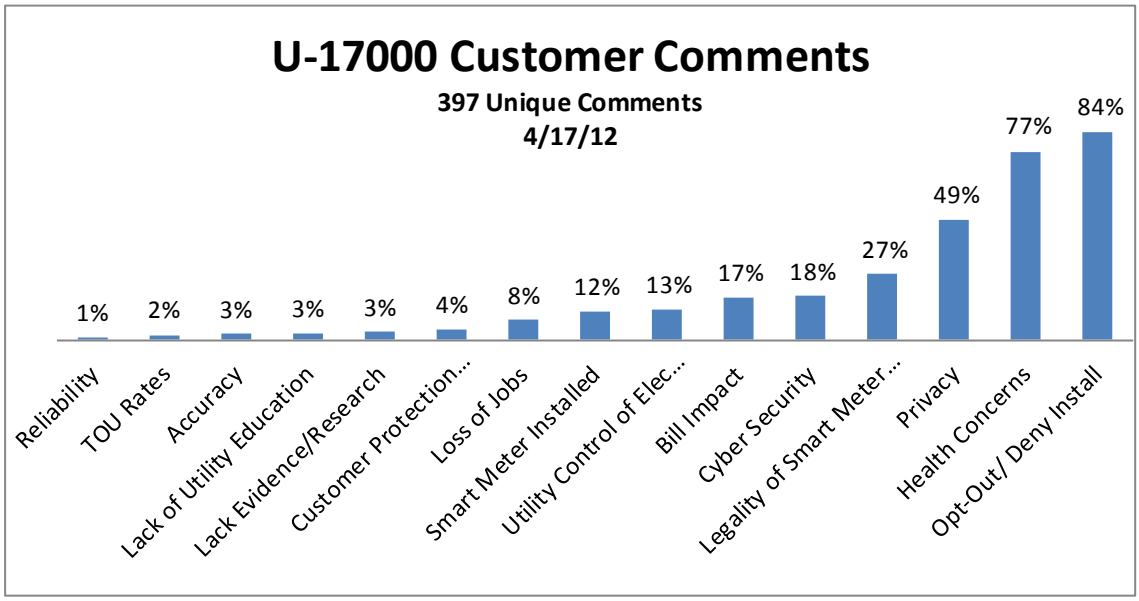
The Staff logged 397 entries received from *unique parties* during the comment period. (Several people submitted multiple entries; however, these were counted as one comment for purposes of this report.) Three comments were received from non-Michigan residents.

Report to the Commission
Case No. U-17000
June 29, 2012

Residential Customers

A number of topics were addressed in the comments. The dominant ones are shown in the chart below. Some customers addressed more than one topic in their submission. Of the customer commenters whose electric provider could be determined, the breakdown was: Detroit Edison (250), Consumers Energy (39), Cherryland Electric Cooperative (1), Clinton Board of Public Works (2), Indiana/Michigan Power Company (I&M) (4), Lansing Board of Water & Light (2), Upper Peninsula Power (4).

Chart 1: Residential Customer Comments



Reliability	TOU Rate	Accuracy	Lack of Education	Lack of Research	Customer Protection	Loss of Jobs	SM Installed	Utility Control of Power	Bill Impact	Cyber Security	Legality of SM Install	Privacy	Health	Opt-out/ Deny Install
4	9	10	11	13	17	32	46	50	69	71	106	193	304	334

Governmental Units

Seven resolutions were submitted by local governmental units:

- Townships of Harrison and Royal Oak,
- Villages of Almont and Grosse Pointe Shores,
- Cities of Farmington Hills and Madison Heights, and
- Macomb County Board of Commissioners.

Requested actions included: 1) further exploration into the health and safety of AMI meters, 2) delay/moratorium on further AMI installations until the Commission's review is completed, and 3) creation of an opt-out program for customers.

Although not formally submitted to the Case No. U-17000 docket, the Staff is aware of additional resolutions from other municipalities containing similar language to the resolutions filed in this docket.

Professional Organizations

Three professional organizations weighed in with submissions to the docket:

- American Academy of Environmental Medicine (AAEM) expresses concern with the levels of radio frequency (RF) radiation emitted by meters.
- Environmental Defense Fund (EDF) supports AMI deployment as a necessary element of grid modernization resulting in positive environmental impacts.
- TechNet also supports AMI deployment focusing on customer control of energy usage, data privacy and encouraging market innovation.

State of Michigan

A state agency and a state house representative filed comments:

- The Department of Attorney General asserts that smart meter benefits are not greater than the deployment costs for ratepayers.
- Representative Paul E. Opsommer states that filings for utilities with AMI meters were incomplete in the areas of meter function, cost and data privacy/protections.

Utilities

The order issued in Case No. U-17000 required utilities to provide specific information regarding smart meter deployment plans, investments, benefits, health and safety, data privacy, and opt-out options. The Commission received responses from investor-owned utilities (IOU) and Michigan electric cooperatives. Consumers Energy and Detroit Edison are the only Michigan utilities currently installing smart meters, so their responses are more thoroughly summarized.

Alpena Power plans to change to digital meters but does not intend to install smart meters. I&M has installed 10,000 AMI meters in South Bend, Indiana as a pilot. I&M has Automated Meter Reading (AMR)[3] at nearly all of its Michigan accounts and does not intend to replace those with smart meters. All of Northern States Power's Michigan customers have AMR, which send daily reads. Northern States

[3] Automated Meter Reading (AMR) "AMR technology allows utilities to read customer meters via short-range radio-frequency signals. These systems typically capture meter readings from the street using specially equipped vehicles." Massachusetts Institute of Technology, *The Future of the Electric Grid;* An Interdisciplinary MIT Study, 2011, p. 133. http://web.mit.edu/mitei/research/studies/documents/electric-grid-2011/Electric_Grid_Full_Report.pdf

Power does not intend to allow opt-out, but believes customers should pay for that option if an opt-out plan is required. Upper Peninsula Power uses electromechanical meters and is planning to continue this method**.** Wisconsin Electric Power Company (WEPCO) has installed AMR throughout its Michigan territory. WEPCO does not anticipate offering opt-out of AMR. Wisconsin Public Service Corporation has meters with both one and two-way communication. Its systems have been in place for over 10 years.

Alger Delta Cooperative, Cherryland Electric Cooperative, Cloverland Electric Cooperative, Great Lakes Energy Cooperative, HomeWorks Tri-county Cooperative, Midwest Energy Cooperative, Ontonagon County Rural Electrification Association, Presque Isle Electric & Gas Cooperative and Thumb Electric Cooperative filed a joint response and individual information. Most of the cooperatives have installed AMR that sends energy use data over power lines. Some of these meters have two-way communication. The cooperatives indicated they have experienced significant benefits from these meters. Presque Isle has a 10 meter AMI pilot. Cooperatives who have AMR do not intend to allow for opt-out.

Below are the responses from Consumers Energy and Detroit Edison regarding smart meter deployment plans as specified in the order in Case No. U-17000.

(1) The electric utility's existing plans for the deployment of smart meters in its service territory:

Consumers Energy Consumers Energy has completed Phase I of a four-phase pilot program, with the intention of full deployment by 2019 with 1.9 million total smart meters.
Detroit Edison Detroit Edison intends to install 2.6 million smart meters in a deployment plan that was initiated by a pilot in 2009. Detroit Edison currently has 650,000 meters installed and plans to have 1,000,000 installed by year end 2013.

(2) The estimated cost of deploying smart meters throughout its service territory and any sources of funding:

Consumers Energy The estimated cost is $750 million with no external funding (e.g., U.S. DOE ARRA grant); $398 million for smart meters and installation; $352 million for systems modifications, program management and other expenses.
Detroit Edison The estimated cost of smart meter deployment is $447 million for 2.6 million new electric meters, and the company received a U.S. DOE grant that reimbursed 50 percent of costs up to a pre-determined grant cap.

(3) An estimate of the savings to be achieved by the deployment of smart meters:

Consumers Energy Estimated savings over the anticipated 20-year life of the smart meters is $2 billion. Although benefits were described, no quantified breakdown of the savings total was provided.
Detroit Edison Detroit Edison estimates smart meter savings of $65 million per year, although this figure includes both electric and gas meters. Case No. U-16472, Exhibit A-18 was referenced for details.

(4) An explanation of any other non-monetary benefits that might be realized from the deployment of smart meters:

Consumers Energy Consumers Energy cited a U.S. DOE study (DOE/NETL-2010/1413) which summarizes the benefits tied to smart meter deployment. The study discusses societal benefits that include reduced outage times, as well as improvements in national security, environmental conditions, and economic growth.
Detroit Edison Proposed non-monetary benefits include an increase in customer satisfaction, the ability to identify voltage problems, new rate offerings, and the ability to expedite emergency disconnect response.

(5) Any scientific information known to the electric utility that bears on the safety of the smart meters to be deployed by that utility:

Consumers Energy Consumers Energy described its proposed system. No scientific information was provided.
Detroit Edison Detroit Edison provided a link to the report, *No Health Threat from Smart Meters*, Utilities Telecom Council, Q4 2010. The following studies were also included in an appendix:

Analysis of Radio Frequency Exposure Associated with Itron OpenWay® Communications Equipment, March 2011
Wireless Transmissions: An Examination of OpenWay® Smart Meter Transmissions in 24-Hour Duty Cycle, March 2011
Smart Meters and Smart Systems: A Metering Industry Perspective, Edison Electric Institute (EEI), Association of Edison Illuminating Companies (AEIC) and Utilities Telecom Council (UTC), March 2011
A Discussion of Smart Meters And RF Exposure Issues, Edison Electric Institute (EEI), Association of Edison Illuminating Companies (AEIC) and Utilities Telecom Council (UTC), March 2011

(6) An explanation of the type of information that will be gathered by the electric utility through the use of smart meters:

Consumers Energy The amount of kilowatt-hours (kWh) consumed each hour, kilovolts-ampere-reactive hours (kVARh) delivered, and actual voltage delivered will be collected every four-six hours. Some of this data is also added together and then sent once per day. Alarms and notification of field events will be sent out in real time.
Detroit Edison The data collected is accumulated Watt hour (Whr) consumption readings, load profile hourly interval watt-hour (Whr) and Volt Ampere hour (VAhr) energy data, load profile energy data, instantaneous voltage, meter messages, events, alarms, and network parameters. No customer-specific data such as addresses, phone numbers, account status or social security numbers will be gathered.

(7) An explanation of the steps that the electric utility intends to take to safeguard the privacy of the customer information so gathered:

Consumers Energy Safeguards for customer privacy include using data encryption and code division multiple access (CDMA). There is no personal customer information in the transmittal of data.

Detroit Edison Customer information is safeguarded through data encryption and internal confidentiality policies.

(8) Whether the electric utility intends to allow customers to opt out of having a smart meter:

Consumers Energy Consumers Energy proposes a future opt-out, but no details were provided.
Detroit Edison Detroit Edison is developing an opt-out for customers, but has yet to develop any details.

(9) How the electric utility intends to recover the cost of an opt-out program if one will exist:

Consumers Energy In accordance with utility cost of service principles, Consumers Energy suggests a future opt-out will be subject to a monthly maintenance fee. Fixed costs for opt-out would be recovered through a tariff-based, one-time charge and a monthly maintenance charge.
Detroit Edison Detroit Edison projects that customers choosing to opt-out will be responsible for all costs associated with an opt-out tariff provision.

Detroit Edison and Consumers Energy provided responses to the Commission's request in Case No. U-17000 regarding AMI deployment. The utilities could have provided additional details that would have been helpful for the Staff's analyses, including more specific information on savings calculations and privacy protections.

THE STAFF'S REVIEW OF AMI

The Staff reviewed the submitted comments, and the cited resources and literature provided by the electric utilities and the public. The Staff examined resources considered "technical" in nature. Many of these resources were published in reputable scientific or professional peer-reviewed journals or were based on reproducible, sound scientific methods and procedures. The Staff also examined many other resources and literature from a variety of sources. The Lawrence Berkeley National Laboratory (LBNL) document identifying resources was beneficial to the Staff in its review.[4] This report addresses some of the more frequently cited resources.

Safety and Health Concerns

The Federal Communications Commission (FCC) is charged with regulating international communications by radio, television, wire, satellite and cable within the United States and its territories. The FCC is responsible for providing licenses for RF emissions. The FCC regulations cover matters relating to public health and safety and have been designed to ensure that the levels of RF emissions that consumers are exposed to are not harmful.

[4] LBNL Website. http://smartresponse.lbl.gov/reports/sm-resourcelist041912.xlsx

In January 2011, the California Council on Science and Technology (CCST) completed a report titled *Health Impacts of Radio Frequency from Smart Meters*.[5] The CCST compiled a comprehensive overview of known information on human exposure to wireless signals, including the effectiveness of the FCC RF safety regulations. After evaluating numerous RF related publications and soliciting the opinions of technical experts in this and related fields, the CCST concluded that no additional standards are needed at this time and that FCC standards are adequate to ensure the health and safety of people from the known thermal effects of smart meters. The report also indicates that smart meters, when installed correctly and with FCC certification, emit only a fraction of the level that the FCC has determined to be safe.

In a recent report, *Radio-Frequency Exposure Levels from Smart Meters: A Case Study of One Model*,[6] the Electric Power Research Institute (EPRI) researched smart meter emission data that provides valuable insight into RF exposure scenarios for a widely used type of smart meter. There were three key findings: (1) exposure levels from individual meters declined rapidly as distance from the meter increased, (2) meters transmitted for only a small fraction of time, and (3) RF exposure levels remained well below the FCC exposure limits.

The Utilities Telecom Council (UTC), in an article titled *No Health Threat from Smart Meters*,[7] provided a review of the safety standards associated with RF emissions and stated that smart meters did not pose a health or safety threat. The UTC's research established that laptop computers using Wi-Fi transmit at levels similar to smart meters, although laptop transmitters are always "on" or transmitting and smart meters transmit for short intervals periodically throughout the day. After reviewing this and other common RF devices (cell phones, microwave ovens, etc.), the UTC concluded that the RF emissions from smart meters would not pose a threat to human health and safety.

The January 13, 2012, County of Santa Cruz Health Services Agency memorandum titled *Health Risks Associated with SmartMeters*[8] was drafted in response to the Santa Cruz County Board of Supervisors' request that the agency identify potential smart meter health effects and possible mitigation measures. The memorandum concluded that research addressing the health effects of electromagnetic fields (EMF) does not specifically address smart meters; there is no scientific data regarding non-thermal effects of smart meters; and government agencies should take precautionary avoidance measures. LBNL reviewed the agency's memorandum as part of the Smart Grid Technical Advisory Project.[9] LBNL's review focused on the objective of the memorandum, consistency of cited sources with agency established peer review criteria, and clarification of technical assumptions and claims. LBNL noted:

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[5] *Health Impacts of Radio Frequency from Smart Meters*, January 2011.
http://www.ccst.us/publications/2011/2011smartA.pdf

[6] *Radio-Frequency Exposure Levels from Smart Meters: A Case Study of One Model*, February 2011.
https://www.nvenergy.com/NVEnergize/documents/EPRI_1022270_caseStudy.pdf

[7] *No Health Threat From Smart Meters*, Fourth Quarter 2010 Issue of the UTC JOURNAL.
http://www.utc.org/utc/no-health-threat-smart-meters-says-latest-utc-study

[8] County of Santa Cruz, *Health Risks Associated with SmartMeters*, http://emfsafetynetwork.org/wp-content/uploads/2009/11/Health-Risks-Associated-With-SmartMeters.pdf

[9] The Smart Grid Technical Advisory Project provides technical assistance and training to state regulatory commissions on topics related to smart grid. The Smart Grid Technical Advisory Project does not participate in litigated or contested regulatory or other proceedings.

> [T]he Agency memorandum does not appear to provide a balanced representation of research, the risks, or mitigation options. Instead the Agency memorandum is largely focused on scientifically unsupported claims related to "electromagnetic hypersensitivity" (EHS).

Individuals with EHS report real symptoms; however, health research has been unable to consistently attribute those symptoms to EMF exposure.[10] LBNL's review of the Santa Cruz memorandum highlighted concerns with the methodology of the agency memorandum cited sources.[11]

On April 12, 2012, the AAEM submitted their position paper, *Electromagnetic and Radiofrequency Fields Effect on Human Health*, to Case No. U-17000.[12] The paper supports AAEM's position that emissions from smart meters are potentially harmful. LBNL also provided a response to the AAEM position paper. LBNL's primary concerns with the paper's findings are a) the research used to establish a cause and effect relationship does not address smart meters, b) the research citations and references are unrelated to smart meters, c) conclusions are about EHS, and d) the minimal amount of RF smart meters actually contribute to total environmental RF. LBNL explains that RF is distinguished by a number of characteristics including frequency, intensity and proximity.[13] There are multiple sources of RF exposure in our everyday environment such as cellular phones, wireless devices such as laptops and routers, microwave ovens, baby monitors, garage door openers, "walkie talkies," computer monitors, fluorescent lighting, and electrical wires within the home.[14] [15] Smart meters are a small contributor to the total environmental RF emissions to which the general public is exposed. Eliminating smart meters would result in a minimal reduction of total emissions.[16]

Several comments submitted in Case No. U-17000 cited the World Health Organization's (WHO) classification of RF EMF as a class 2B carcinogen in support of their smart meter health concerns. This classification means that RF EMF has been deemed as *possibly* carcinogenic to humans.[17] RF EMF was designated as a class 2B carcinogen due to limited evidence associating glioma and acoustic neuroma, two types of brain cancer, with wireless telephone users. The Staff was unable to identify research that associates AMI meters with any type of cancer.

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[10] LBNL, *Review of the January 13, 2012 County of Santa Cruz Health Services Agency memorandum: Health Risks Associated with Smart Meters* http://smartresponse.lbl.gov/reports/schd041312.pdf
[11] LBNL, *et al*. http://smartresponse.lbl.gov/reports/schd041312.pdf
[12] American Academy of Environmental Medicine, *Electromagnetic and Radiofrequency Fields Effect on Human Health*. http://efile.mpsc.state.mi.us/efile/docs/17000/0391.pdf
[13] LBNL, *Review of the April 12, 2012 American Academy of Environmental Medicine (AAEM) submittal to the Michigan Public Service Commission*, http://smartresponse.lbl.gov/reports/aaem041812.pdf
[14] Federal Communications Commission: *Radio Frequency Safety* http://transition.fcc.gov/oet/rfsafety/rf-faqs.html.
[15] Federal Communication Commission: *Interference – Defining the Source* http://www.fcc.gov/guides/interference-defining-source.
[16] City of Naperville, Naperville Smart Grid Initiative (NSGI), Pilot 2 RF Emissions Testing – Summary Report-V2.0, *Smart Meters, Household Equipment, and the General Environment*, November 10, 2011. http://www.naperville.il.us/emplibrary/Smart_Grid/Pilot2-RFEmissionsTesting-SummaryReport.pdf
[17] International Agency for Research on Cancer, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, January 2006. http://monographs.iarc.fr/ENG/Preamble/currentb6evalrationale0706.php

In May 2011, members of the WHO's International Agency for Research on Cancer's (IARC) Monographs Working Group reviewed roughly 900 studies that involved RF EMF and cancer.[18] The group categorized the studies by the following RF EMF sources: occupational exposure (i.e., radar installations), personal exposure associated with the use of wireless telephones, and environmental exposure (i.e., radio/television signals). For occupational exposure to RF EMF, the group determined that there are "some positive but inconsistent signals." With respect to environmental sources of RF EMF, the group determined that there was no "solid data" to conclude a link between cancer and RF EMF exposure. Lastly, regarding personal exposure, the group found there to be limited evidence linking glioma and acoustic neuroma to wireless phone use, with inadequate evidence for other cancer types.

Experts in the field of RF EMF have testified in front of public utility commissions outside of Michigan as to how the IARC classification correlates with smart meter technology. For example, Baltimore Gas & Electric provided the expert opinion of Dr. Peter Valberg to the Public Service Commission of Maryland, who testified on how the category 2B classification of RF EMF should be interpreted. Dr. Valberg stated that the IARC has not found any ". . . adverse health consequences established from exposure to RF fields at levels below the international guidelines on exposure limits published by the International Commission on Non-Ionizing Radiation Protection."[19] He goes on to state that the 2B classification of RF EMF was ". . . made with reference to the quantity of exposure, *e.g.*, no quantitative estimate as to how various uses of RF contribute to human exposure. . . ."[20] and that ". . . smart meters constitute one of the weakest sources of our RF exposure."

Dr. Yakov Shkolnikov and Dr. William H. Bailey, engineers from the consulting firm Exponent, provided expert testimony to the Public Utility Commission of Nevada concerning NV Energy's smart meter deployment, and addressed smart meter RF EMF emission concerns. These witnesses pointed out that although RF EMF was classified in group 2B ". . . the evidence is limited that cancer develops from exposures from RF fields."[21] They also make it clear that ". . . the indications of potential risk derive almost entirely from statistical associations in some studies between the use of mobile phones and certain types of cancer."[22]

The WHO's decision to classify RF EMF in the group 2B category was based on studies involving wireless phones, not smart meters. While both wireless phones and smart meters emit RF EMF, the

[18] International Agency for Research on Cancer, *Radiofrequency Electromagnetic Fields: evaluation of cancer hazards*. http://monographs.iarc.fr/ENG/Publications/REF_Poster2012.ppt

[19] *In the Matter of Baltimore Gas and Electric Company for Authorization to Deploy a Smart Meter Initiative and to Establish a Surcharge Mechanism for the Recovery of Cost*, Case No. 9208, Comments on an "Opt-Out" Option for Smart Meters, Testimony of Dr. Peter A. Valberg, April 6, 2012. http://webapp.psc.state.md.us/Intranet/Casenum/CaseAction_new1.cfm?CaseNumber=9208

[20] *In the Matter of Baltimore Gas and Electric Company for Authorization to Deploy a Smart Meter Initiative and to Establish a Surcharge Mechanism for the Recovery of Cost, et al.*

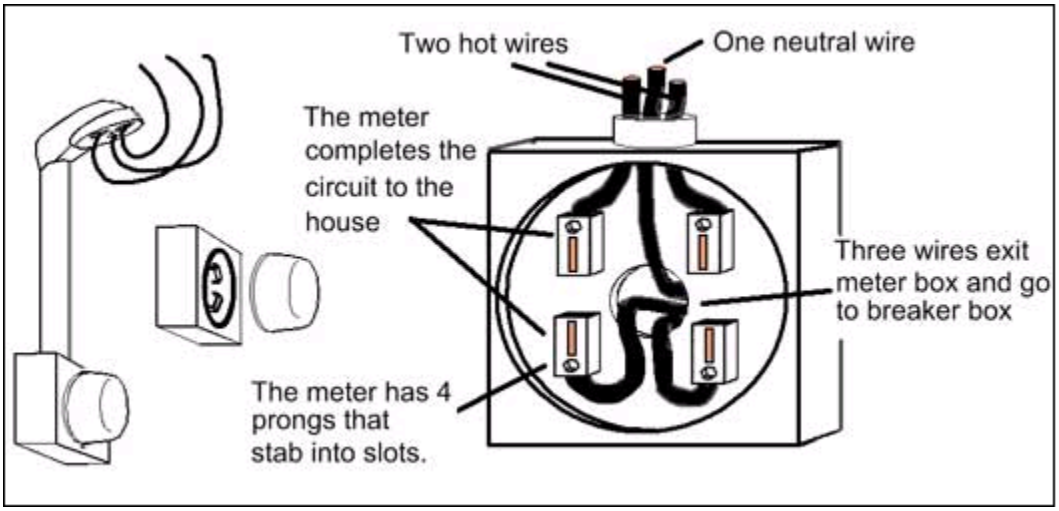
[21] *Investigation regarding NV Energy's Advanced Service Delivery Meter Program a/k/a Smart Meter and its implementation*, Docket No. 11-10007, Comment of S. Stirling, December 22, 2011.

[22] *Investigation regarding NV Energy's Advanced Service Delivery Meter Program a/k/a Smart Meter and its implementation, et al*

major difference between the two is the lower level of exposure to frequencies from smart meters. Low exposure levels from smart meters coupled with the fact that the IARC's classification is based on weak mechanistic evidence and limited evidence derived from different RF EMF emitting devices is important to consider when evaluating the substance of the group 2B classification. After careful review of the available literature and studies, the Staff believes that the health risk from the installation and operation of metering systems using radio transmitters is insignificant. In addition, the appropriate federal health and safety regulations provide assurance that smart meters represent a safe technology.

Some public comments stated a link between smart meters and house fires. Meter fires for any type of meter are a rare occurrence, according to the National Fire Protection Agency's 2012 annual report[23] on home electrical fires. This type of fire makes up only 1% of the average reported cause of home electrical fires. Factors associated with meter fires are not exclusive to smart meters but apply to all meters. Installation details for smart meters and electromechanical meters are the same. Both meter types have four prongs on the back. The four prongs attach to four slots known as stabs. These stabs, along with the wires from the power lines and meter itself, are housed inside a protective case known as a meter box. Once the meter is connected, the electrical circuit is complete. This is shown in the diagram below. Component failure (i.e. loose stab connection) can cause arcing, potentially resulting in a meter fire. It is the component failure, not the meter unit that is the cause of an arcing-induced fire.

Figure 1: Meter Connection



[23] *Home Electrical Fires*, National Fire Protection Association, January 2012. http://www.nfpa.org/assets/files//PDF/OS.electrical.pdf

Data Privacy

As smart meter deployments have become more prevalent throughout the United States, customer data privacy has become a priority issue. In order to address the concerns of the public regarding smart meter data privacy, multiple entities have engaged in efforts to identify and address the fundamental privacy issues. The Staff reviewed data privacy literature that specifically addressed or were clearly applicable to concerns arising from smart meters collection of customer electric usage information. Documents reviewed originated from the following entities: municipal utilities, state utility commissions, state legislation, standard development organizations, federal government and academia. The following table lists the literature reviewed in preparation of this section.[24]

Table 1: Data Privacy Policies

Entity:	***Document Name:***
Municipal Utilities	
City of Naperville	**Naperville Smart Grid Initiative Customer Bill of Rights**
State Utility Commissions	
State of California	**Privacy Protections For Energy Consumption Data**
State of Colorado	**Rules Regulating Electric Utilities**
State of New York	**Smart Grid Policy Statement**
State of Texas	**Customer Protection Rules For Retail Electric Service**
State Legislation	
State of Arizona	**Consumer Protections; Rules; Confidentiality; Unlawful Practice**
State of Oklahoma	**Electric Usage Data Protection Act**
State of Washington	**WAC 480-100-153 Disclosure of Private Information**
Standards Development Organizations	
NAESB	**Third Party Access To Smart Meter-Based Information**
NISTIR 7628	**Guidelines for Smart Grid Cyber Security**
Federal Government	
US Dept. of Energy	**Smart Grid Privacy Workshop Summary Report**
US Dept. of Homeland	**Fair Information Practice Principles**
Academia	
Vermont Law School	**A Model Privacy Policy for Smart Meter Data**

AMI necessitates a higher volume of data collected by utilities, therefore it is imperative that customer information be properly protected through appropriate regulations. Federal legislation protecting consumer data privacy is forthcoming;[25] however, it is important to identify ways to protect Michigan's ratepayers in the interim. States that feature more advanced AMI deployment such as California,

[24] Links to the table documents can be found in Appendix A.
[25] *U.S. Department of Energy Smart Grid Privacy Workshop Summary Report*. http://www.smartgrid.gov/sites/default/files/doc/files/Privacy%20report%202012_03_19%20Final.pdf

Colorado, Texas, Arizona, Oklahoma, and Washington have addressed customer data protection through state legislation or administrative rules adopted by the public utilities commissions. The Staff acknowledges that interim protections could be achieved through the development of utility tariffs that address customer data privacy. The Staff recommends including the following fundamental concepts when addressing privacy policy:

- Definitions of various types of data collected *(usage/billing, aggregate, customer identifiable),*
- Permitted usage of data types by utility *(sales, contractor work, emergency),*
- Customer consent and third-party disclosure rules *(notice, timeframe, records),*
- Availability of usage information to customer *(web portal, direct mail, email),*and
- Privacy breach requirements *(notification to customer/commission).*

The Staff recommends that there be further investigation into the most appropriate manner (administrative rules, legislation, tariffs, etc.) to ensure customer privacy. This process should include all relevant stakeholders. In the interim, the Staff recommends that utility tariffs include provisions to enhance customer privacy.

Cyber Security

As Michigan transitions to a more technologically advanced power grid, it is important that the proper actions are taken by utilities to address cyber security threats. Cyber security planning is defined as preventing damage to, unauthorized use of, or exploitation of electronic information and communications systems and the information contained therein to ensure confidentiality, integrity, and availability.[26] The attention cyber security has received at the national and state levels for many years indicates that utilities, regulators and consumers all share common concerns. Improving the electrical grid involves gathering more data and utilizing more technology. With every added piece of technology, the risk of vulnerabilities inherently increases. The U.S. DOE has stated that the smart grid of the future should be secure and resilient against all forms of attacks. A smarter grid includes more devices and connections that may become avenues for intrusions, error-caused disruptions, malicious attacks, destruction, and other threats.[27]

It is important to balance the need for a more digitally connected grid and the inherent risks of these new technologies and their interconnection. At the national level, several organizations are currently addressing this issue: North American Electric Reliability Corporation (NERC), National Institute of Standards and Technologies (NIST), Smart Grid Interoperability Panel Cyber Security Working Group (CSWG), National Electric Sector Cybersecurity Organization (NESCO), and the U.S. DOE. These

[26] National Association of State Energy Officials (NASEO), *Smart Grid and Cyber Security for Energy Assurance.* http://www.naseo.org/energyassurance/NASEO_Smart_Grid_and_Cyber_Security_for_Energy_Assurance_rev_November_2011.pdf

[27] Executive Office of the President, National Science and Technology Council, *A Policy Framework For The 21st Century Grid: Enabling Our Secure Energy Future*, June 2011. http://www.whitehouse.gov/sites/default/files/microsites/ostp/nstc-smart-grid-june2011.pdf

groups have published reports and compliance programs to provide utilities guidance on cyber security in the electric industry.

The overall goal is to develop a framework that ensures effective cyber security is appropriately implemented and that all stakeholders contribute to the security and reliability of the electrical grid.[28] The goal is not a compliance-based culture in which companies are expected to stand alone in this effort. Instead it should be a proactive, responsible and collaborative culture in the state of Michigan. The Staff reviewed multiple cyber security related documents published by the leading cyber security associations and found the following commonalities:

- Cyber security efforts should concentrate on rigorous open standards and guidelines through public-private partnerships for security,
- Effective cyber security will rely on data sharing and cooperation between regulatory, private and electric industry entities,
- A risk-based approach to cyber security planning should be implemented,
- A cyber security performance accountability system should be created to fulfill risk-based planning, and
- Regulatory bodies should be in constant contact with asset owners regarding cyber security.

Several states have taken positions on cyber security including California and Texas. The Public Utility Commission of Texas enacted a cyber security rule requiring electric utilities to have an independent security audit of the mechanism for customer and Retail Electric Provider (REP) access to meter data conducted within one year of initiating such access and promptly report the results to the commission.[29]

The Federal Trade Commission (FTC) has studied how entities collect and use personal information. They have compiled their findings in the Fair Information Practices (FIP), which has been used successfully across many industries. The California Public Utilities Commission (CPUC) cited the FIP as a proven model for data security that the electric industry should utilize. In regards to cyber security, the CPUC stated upon any breach[30] affecting 1000 or more customers, an electric provider has two weeks to notify a commission appointed cyber security representative.[31] They also required IOU's to file a year-end cyber security breach report with the cyber security representative at the commission.[32]

[28] Executive Office of the President, *et al*.
http://www.whitehouse.gov/sites/default/files/microsites/ostp/nstc-smart-grid-june2011.pdf
[29] Public Utility Commission of Texas, Electric Substantive Rules.
http://www.puc.state.tx.us/agency/rulesnlaws/subrules/electric/Electric.aspx
[30] A breach is any unauthorized use or exploitation of customer information.
[31] Order Instituting Rulemaking to Consider Smart Grid Technologies Pursuant to Federal Legislation and on the Commission's own Motion to Actively Guide Policy in California's Development of Smart Grid, *et al.*
[32] Order Instituting Rulemaking to Consider Smart Grid Technologies Pursuant to Federal Legislation and on the Commission's own Motion to Actively Guide Policy in California's Development of Smart Grid, *et al*.

The Staff proposes that the following cyber security measures be implemented in Michigan:

- Each utility should adopt an annual independent security audit of the mechanisms of customer access, third party access and internal cyber risk-management practices. The independent auditor should be approved by the Staff.
- As outlined in the National Association of Regulatory Utility Commissioners' (NARUC) resolution regarding cyber security, the Staff should maintain a dialogue with regulated utilities to ensure that they are in compliance with standards, and that preparedness measures are employed to deter, detect and respond to cyber attacks and to mitigate and recover from them.[33]
- Utilities should adopt the same breach notification policies as other states have adopted, namely the notification of any breach affecting 1000 or more customers within two weeks of the breach.
- Each utility should be required to file a yearly breach notification summary with the Staff, detailing all breaches of customer information, including any third party breach information.

Customer Education

Customer education and participation is an important component of the successful implementation of the smart grid. A portion of the smart meter benefits rely upon customer engagement. To facilitate customer engagement, utilities must provide customers with clear and accurate information about programs and services available both prior to and *throughout* the deployment of smart meters.[34] Within the 397 unique comments submitted to Case No. U-17000, 360 comments reference a lack of communication with customers about the functionality and benefits of smart meters.[35] As the Maryland Public Service Commission[36] stated:

> The negative experiences in other states . . . illustrate vividly that poor customer education will magnify small-scale problems and create disproportionate customer skepticism and unhappiness.

For this reason, the Staff reviewed customer education efforts in various states. Several states have supported the importance of customer education through both legislation and orders.

[33] NARUC, *Resolution Regarding Cybersecurity*, February 17, 2010. http://www.naruc.org/Resolutions/Resolution%20on%20Cybersecurity1.pdf
[34] Massachusetts Institute of Technology, *The Future of the Electric Grid*; An Interdisciplinary MIT Study, 2011, p. 164. http://web.mit.edu/mitei/research/studies/documents/electric-grid-2011/Electric_Grid_Full_Report.pdf
[35] Pg. 4, Chart 1 of this report (combined categories of lack of education, utility control of power, legality of smart meter install and privacy).
[36] *In the Matter of Baltimore Gas and Electric Company for Authorization to Deploy Smart Meter Initiative and to Establish a Surcharge Mechanism for the Recover of Cost*, Case No. 9208, Order No. 83531, pp. 42-43. http://webapp.psc.state.md.us/Intranet/Casenum/CaseAction_new1.cfm?CaseNumber=9208

- Colorado Public Utilities Commission concluded that utilities should submit a smart meter plan with a detailed customer education and outreach plan.[37]
- Nevada Public Utilities Commission concluded that NV Energy should enhance its consumer outreach efforts. The outreach efforts were to include a "media plan leading up to the deployment of smart meters that will frequently reach out into the community and use multiple channels to reach customers more effectively."[38]
- California Public Utility Commission (CPUC) was directed by California Public Utilities Code § 8360 (2009), to identify criteria to ensure that the utility smart grid deployment plans conform to best practices. Commission Rulemaking R 08-12-009 identifies the need for a smart grid strategy recognizing that customer participation is necessary for the demand-side benefits.[39] In addition, CPUC Decision 12-04-025 identifies metrics to use to track customer participation.[40]
- The Maryland Public Service Commission directly addressed customer education in Case No. 9208, Order No. 83531. The commission order states "[t]hat Baltimore Gas and Electric Company shall submit, for the Commission's approval, the Company's updated customer education plan and associated proposed messaging that it will provide customers prior to and during installation of the meters, before Peak Time Rebates begin, and before any other programmatic changes take effect. Baltimore Gas and Electric and other parties in the matter shall develop, and submit for Commission approval, a comprehensive set of metrics by which the Commission may measure the effectiveness of the customer education plan, . . ."[41]
- The Public Utility Commission of Texas met regularly with utilities to help develop radio ads, door hangers, billboards, etc. which were used to educate the public about smart meters. The education effort specifically targeted smart meter cost recovery, deployment, and implementation. The Texas Public Utility Commission also approved each utility's budget associated with smart meter customer education[42].
- Maine Public Utility Commission ordered Central Maine Power to ". . . develop and implement a customer communication plan that will explain the various opt-out options, describe the benefits of the AMI program, describe the functionality of the available meter options, describe the

[37] *In the Matter of the Investigation of the Issues Related to Smart Grid and Advanced Metering Technologies*, Docket No. 10I-099EG. Decision No. C11-0406, Order State Conclusions and Next Step, March 30, 2011, p. 5.
[38] *Investigation regarding NV Energy's Advanced Service Delivery Program a/k/a Smart Meters and its implementation*, Docket No. 11-10007, Interim Order, January 11, 2012, p. 8.
[39] California Public Utility Commission, R 08-12-009.
http://docs.cpuc.ca.gov/PUBLISHED/FINAL_DECISION/119902-02.htm#P201_29007
[40] California Public Utility Commission, Decision 12-04-025, April 24, 2012.
http://docs.cpuc.ca.gov/PUBLISHED/FINAL_DECISION/164808.htm
[41] *In the Matter of the Application of Baltimore Gas and Electric Company for Authorization to Deploy a Smart Grid Initiative and to Establish a Surcharge for the Recovery of Cost*, Case No. 9208, Order No. 83531, p. 50.
http://webapp.psc.state.md.us/Intranet/sitesearch/CN9208.pdf.
[42] Relevant Dockets include: Oncor Docket No. 35718, CenterPoint Docket No. 35639, AEP TX Docket No. 36928, TNMP Docket No. 38306.
http://interchange.puc.state.tx.us/WebApp/Interchange/application/dbapps/filings/pgSearch.asp.

charges associated with the opt-out, and describe the process by which a customer may opt-out."[43]

- In 2008, the Ohio legislature enacted changes to the Ohio Revised Code – Title XLIX Public Utilities which required utilities file a customer education plan; the purpose of which is to ". . . educated [*sic*] Ohio's consumers about their new choices for electric service."[44]

The transition to smart meters and related infrastructure will provide customers access to current data about their energy usage, creating an opportunity to better control energy consumption. Smart meters also provide the basic infrastructure for aggregate benefits related to reliability, outage identification, and reduced peak demand. These benefits have a positive effect on all customers including those who choose to opt-out.[45] A smooth transition to smart meters can be accomplished only through customer education. A well thought out education strategy allows customers to develop a sense of trust with the utility and an understanding of the available benefits.

The Staff recommends utilities develop and implement a new education strategy similar to those used in other jurisdictions. Education program results should reflect high levels of customer engagement, acceptance and enthusiasm with their smart meter program. The strategy should include metrics to measure the overall effectiveness of the education program.

National Policy

The United States Congress has passed several laws that support the upgrade of the electric grid, including deployment of smart meters for residential and other types of customers. These laws have provided a framework for smart grid, including smart meter deployment in the United States. Basically, these laws encourage states to proceed with modernizing the electric grid in order to be ready for the electric demands of the 21st Century.

The Energy Policy Act of 2005 (EPAct 2005) was the first piece of federal legislation that discussed smart grid. The statute strongly encourages demand response. It calls upon utilities to offer time-based rates with a time-of-use meter to all customer classes. It also requests that state public utility commissions investigate the installation in their state of time-of-use meters and communication devices to enable time-based pricing rate schedules and other demand response programs. The statute also mandates that, by October 2012, all federal buildings be individually metered for electricity consumption and, to the extent feasible, use advanced meters that measure energy use on an hourly basis.[46]

[43] Maine Public Utilities Commission, Docket No. 2010-345, Order (Part I), May 19, 2011, p. 2.
[44] *In the Matter of the Commission's Promulgation of Rules for Electric Transition Plans and of a Consumer Education Plan, Pursuant to Chapter 4928, Revised Code*, Case No. 99-1141-EL-ORD, Entry, June 8, 2000. http://www.puco.ohio.gov/emplibrary/files/docketing/ORDERS/2000/0604/99-1141.pdf
[45] Electric Power Research Institute *Advanced Metering Infrastructure*, February, 2007, p. 1. http://www.ferc.gov/eventcalendar/Files/20070423091846-EPRI%20-%20Advanced%20Metering.pdf
[46] Energy Policy Act of 2005, Pub. L. No. 109-58, 100 Stat. 567 (codified at 1 U.S.C. §§ 900-999).

The Energy Independence and Security Act of 2007 (EISA) is a major piece of federal legislation addressing smart grid and smart meters. Title XIII, Sections 1301 through 1309 supports modernizing the nation's electric grid and contains provisions giving the U.S. DOE a leadership role in all but two areas of smart grid advancement. Interoperability was assigned to the NIST and the Federal Energy Regulatory Commission (FERC), and recovery of smart grid investment was relegated to the state public service commissions. The statute contains a policy statement on United States' grid modernization that defines "smart grid;" establishes the Smart Grid Advisory Committee, the Smart Grid Task Force, and the Smart Grid Interoperability Framework; and institutes the Smart Grid Investment Matching Grant Program, which provides a 20% match for qualifying smart grid investments.[47]

The American Recovery and Reinvestment Act of 2009 (ARRA) amends EISA allowing U.S. DOE to provide financial support for smart grid demonstration projects and advanced grid technology investments, such as AMI. In total, the legislation provides $3.4 billion in funding for numerous smart grid projects across the nation, including smart meters, in-home energy management displays, smart thermostats, advanced transformers and load management equipment. The act establishes a smart grid information clearinghouse and requires that demonstration projects use open protocols and standards.[48]

In addition to federal laws, numerous prestigious agencies and institutions have considered the national outlook for the smart grid and indicate that installing smart grid technologies, including smart meters, will have a positive benefit on the United States' electric grid. These reports urge the United States to follow the directives of the federal law and update the electric grid.

In 2012, the U.S. DOE issued the 2010 Smart Grid System Report. The report, required by the EISA, outlines the current status of smart grid development, projects its future, and identifies obstacles to its progress. It describes the scope of smart grid, recognizes its stakeholders, and makes recommendations for future reports. The report states that recent progress has been significant due to funding from ARRA of 2009, including the provision of $812.6 million in federal grant awards for AMI deployments throughout the United States, the implementation or expansion of distributed resource interconnection policies in 14 states since 2008, and funding the deployment of 877 phasor measurement units. The report determines that correctly assessing the value proposition and obtaining capital for new technologies that communicate information between electricity sector participants are challenges that need to be overcome in order to continue development of the smart grid.[49]

Several NARUC initiatives support smart grid activities. NARUC and FERC have established the Smart Response Collaborative which provides a forum for federal and state regulators to share information about the smart grid to support the development of better and more effective policies. NARUC has also passed resolutions that address smart grid. A resolution passed on July 20, 2011, endorsed a foundational

[47] Energy Independence and Security Act of 2007 (EISA), Pub. L. No. 110-140, 121 Stat. 1492, 1783-84 (codified at 42 U.S.C. § 17381).
[48] American Recovery and Reinvestment Act of 2009 (ARRA), Pub. L. No. 111-5, 123 Stat. 115, 516.).
[49] *U.S. DOE 2010 Smart Grid System Report*, Report to Congress, Washington DC, February 2012. http://energy.gov/oe/downloads/2010-smart-grid-system-report-february-2012

set of principles related to advance metering and smart grid deployments. The principles encourage the continued installation of smart grid technologies including AMI, while also advising utility commissions to continue to assess the best strategies for their states.[50]

The Future of the Electric Grid was published by the Massachusetts Institute of Technology (MIT), the sixth in a series of reports that examine the "future of" energy and environmental issues. The report provides a snapshot of the current status of the United States' electric grid and a vision for the evolution of the grid over the next two decades. The study group, consisting of MIT professors and research assistants, with input from industry and government experts, reviewed and evaluated existing research and made recommendations that will help to ensure the future of the electric grid. One of the main findings is that regulatory policies and the technologies used to support the grid must change or it is likely to be difficult to maintain acceptable reliability and reasonable electric rates. An updated distribution system with the use of AMI is instrumental to a smarter grid. The study identifies the benefits of AMI including a reduced cost of meter reading, more accurate and timely billing, improved customer support, enhanced distribution monitoring and management, support for demand response and energy conservation, quicker response to outages and reduced outage times. With the decreasing availability of electromechanical meters, AMI will soon be the most viable metering option available to utilities. The study acknowledges that there have been health concerns raised by customers, but concludes that the scientific research does not suggest that radio waves from smart meters have adverse health effects. They acknowledge that utilities may have to consider these concerns when designing their programs by inclusion of opt-out or other provisions.

The study also reviewed the status of cyber security readiness on the United States' grid. The report recommends a heightened focus on detection, response, and recovery strategies, especially for the distribution system. Since there is currently more than one agency working on this issue, a single agency should be given responsibility to develop and enforce standards across the entire electric power system.[51]

A Policy Framework for the 21st Century was issued by the federal government to build on the policy directives set forth in the EISA and the ARRA by creating a pathway to a modernized grid. A smarter, modernized and expanded grid is pivotal to the United States, playing a lead role in a clean energy future. The electric grid in the United States is at an advanced age. This makes it imperative to upgrade the grid in three categories: advanced information and communication technologies that improve transmission and distribution; advanced metering; and equipment that accesses and leverages energy usage information. The study concludes that AMI can empower consumers to better manage their energy usage and reduce their energy bills.

[50] National Association of Regulatory Utility Commissioners, Smart Grid Resources. www.naruc.org/smartgrid/

[51] Massachusetts Institute of Technology, *The Future of the Electric Grid*, An Interdisciplinary MIT Study, 2011. http://web.mit.edu/mitei/research/studies/documents/electric-grid-2011/Electric_Grid_Full_Report.pdf

Ensuring the privacy of energy use data is also of primary concern to the study participants. Existing agencies, such as state public service commissions, may be able to set privacy rules for regulated utilities. The FTC's FIP principles can provide a framework for developing codes of conduct to protect this data.[52]

Policies and Practices

AMI has the potential to provide increased electric reliability while providing customers with the information and choices necessary to reduce or shift their electric consumption. Customers can only realize these benefits if utilities begin to collect more detailed usage data. While AMI does not transmit personal customer information, it does gather usage data more frequently than a traditional meter. Although utilities have been protecting customer data for many years, the collection, storage, use, access, and disclosure of customer consumption data have generated concerns about privacy, utility transparency, customer choice, and security. Attention to system reliability standards, electric technical standards and utility billing practices are warranted when addressing customer protection, data collection, customer privacy, cyber security, and system reliability benefits.

Several areas of current rules and tariffs will be affected by AMI deployment in Michigan. In some cases, the topic of concern is not a direct result of AMI. One example is privacy. Customers are more sensitive to privacy with the deployment of AMI, but the requirement for documented and clearly communicated utility privacy policies existed prior to AMI deployment. Consistently documenting privacy policies creates transparency and accountability as new technologies continue to evolve.

Electric utilities regulated by the Commission follow rules and standards for electric service set forth in administrative rules, tariffs, and Commission orders. All of these regulatory mechanisms should be considered and the most effective chosen to ensure customers have adequate protections.

The Staff conducted a preliminary investigation into national recommendations, rules from other states, and utility best practices. This investigation revealed Michigan's current policies are in need of review in order to address on-going customer issues.[53] Michigan should consider the following areas as the utility systems and utility/customer relationships change due to AMI.

- Customer Consent – Customers should have the option to authorize data collection and services not related to core billing and operational services.
- Individual Access and Participation – Customers should have easy, timely access to their detailed usage data in a standard downloadable format.
- Customer Choice –Utilities should clearly, fully, and accurately describe all choices available to customers.
- Notice and Purpose – Utilities should provide a detailed description of all purposes for which customer data will be used.

[52] *A Policy Framework for the 21st Century: Enabling Our Secure Energy Future, et al* http://www.whitehouse.gov/sites/default/files/microsites/ostp/nstc-smart-grid-june2011.pdf
[53] A complete list of research sources is available in Appendix A

- Collection and Scope – Only information that is required to fulfill the stated purpose specified under Notice and Purpose should be collected.
- Security – Personal information in all forms should be protected from loss, theft, unauthorized access, inappropriate disclosure, copying, use, or modification. Utilities should implement breach notification policies and independent third party privacy and security audits.
- Management and Accountability – Utilities should develop and appoint personnel to ensure that information security, privacy policies, and privacy practices exist and are followed, including ongoing training and audits.
- Utility Processes – Utilities should provide a process for individuals to see and easily correct inaccuracies in their information. Utilities should estimate customer bills only if they are able to demonstrate that there was an unavoidable circumstance. Prepayment is an option that may be preferred by some customers.
- Meter Accuracy – Standards that ensure the accuracy of AMI meters should be developed.
- Service Reliability – Performance measures should reflect system reliability and outage support provided through AMI implementation.

The Staff examined current Commission rules and technical standards and found that some AMI related areas are not covered. For example, there is no definition for AMI. There are, however, current rules that address AMI capabilities such as remote shutoff (2007 AACS R 460.142). In a larger review of methodologies, rules and standards should be evaluated further.

It is recommended that all stakeholders work to analyze and identify the most appropriate implementation methods for addressing the policy considerations listed above. Stakeholders should routinely review all policies related to smart grid as smart grid technologies continue to develop.

Smart Grid Vision

When considering the deployment of AMI in Michigan, it is important to recognize that smart meters and their supporting communications infrastructure represent a single component of a fully modernized grid. AMI introduces a communications platform that can support a multitude of smart grid applications resulting in improved efficiency and reliability, as well as increased longevity of Michigan's aging electric infrastructure. When properly designed and implemented, AMI presents a unique opportunity for Michigan ratepayers to take control of their energy consumption and their energy bills.

The smart grid will enhance electric service in Michigan. Real time outage identification, through AMI, will result in a quicker response to outage situations. Areas without service can be identified almost immediately and individual customers who are still out after their neighborhood has been restored will be easily located. The smart grid technologies will reduce operations and maintenance costs, primarily through reduced meter reading costs, more accurate billing, reduced outage time and monitoring tools that help the utility anticipate equipment failure. AMI meters, with the use of dynamic and time-of-use rates, can reduce peak demand and increase energy conservation. The result could curtail the need for future

capital investment in electrical system capacity and lead to other grid efficiencies. This would result in lower capital costs for all ratepayers.

A Michigan smart grid vision should provide direction to implement technology that will enhance the functionality of the electric grid. It is difficult to have all utilities, vendors, regulators and customers share a succinct vision of what the future electric grid will look like. Therefore, it is important to identify electric grid "objectives" that outline a more reliable grid, improve power quality and incorporate cleaner power sources for electricity generation. All components of electric grid improvements, including AMI installation, distribution infrastructure replacement, and electric generation should reflect the larger objectives of a smart grid vision.

The Staff proposes that future smart grid investments from utilities must correlate with the following objectives aimed at delivering transparent and identifiable benefits to ratepayers:

- Accommodate advanced generation and storage options
- Enable informed participation by all customers
- Support new products, services, and markets
- Optimize existing assets, increase efficiency and improve reliability
- Operate resiliently against physical and cyber attacks

Michigan's current electric grid is characterized by centralized fossil fuel generation plants delivering electricity over long distances to meet customer needs. This model has been dominant for over a century and has provided an economical and reliable means of providing energy to Michigan citizens. However, increased investment and technological advances in decentralized generation and storage options such as gas turbines, diesel engines, solar photovoltaic, wind turbines, biomass generators and plug-in electric vehicles present potential generation options in the future. The Staff supports future grid investments that promote a more flexible grid that is capable of integrating any and all generation, two-way power flows and storage options. These investments will help ensure that Michigan ratepayers have access to the most cost effective generation in the future.

The traditional relationship that has existed between the utility and its ratepayers was limited to customers consuming energy and then receiving a monthly bill for the service. As the smart grid takes form in Michigan, the Staff envisions a much more interactive relationship developing between utility and customer. Utilities need to develop communications avenues and program incentives capable of informing, engaging, empowering, and motivating customers to change their behavior. The Staff believes that an extensive customer education campaign that coincides with technology deployment is pivotal to a successful implementation strategy. The Staff also believes that in the future, piloting a variety of customer programs (dynamic rates, prepay, demand response) to measure their effectiveness will be key to realizing the full spectrum of utility and customer benefits.

Consistent standards are necessary for new products, services and markets to be successful. Effective implementation of a smart grid in Michigan will bring an abundance of new products, services, and

markets that accommodate a variety of customer needs. Michigan customers should have access to the full potential of these innovations. For this reason, smart grid deployment in Michigan should be standards based. Nationally and globally recognized standards play a critical role in the ongoing development of these products, services and markets. The development and adoption of smart grid standards can help investments made today remain valuable into the future, remove barriers to innovation, maximize customer choice, create economies of scale, emphasize best practices, and open global markets. A standard based framework will promote interoperability and accommodate advances in technology.

The two-way flow of system information made possible by the implementation of AMI has multiple applications outside of metering. In the future, the Staff expects to see numerous efficiency applications made possible by the availability of real-time information. Using this system information to recognize and avoid issues such as power line congestion, transformer overheating, and other detrimental grid conditions, will lower the cost of transporting energy from the power plant to the customer meter and improve reliability. Optimizing the efficiency of existing assets already in rate base will help meet increasing electric demand while minimizing investment in new generation facilities and distribution assets.

The transition to a modern grid utilizing digital technology will require a large emphasis on security. The modernized grid must be capable of providing a greater level of reliability to prevent cyber-attacks and sabotage of utility equipment. Grid modernization plans should be developed concurrently with cyber security and outage mitigation strategies. Providing adequate focus on these threats prior to their occurrence will help mitigate the overall effect on Michigan customers. The longevity of a digitalized grid will rely on a utility's ability to plan for and react to both physical and cyber-attacks. Developing robust risk based management strategies can mitigate, if not eliminate, the potential of these threats coming to fruition.

The above objectives provide a glimpse of the potential benefits of moving to a modernized electric grid. Many of the benefits outlined above are being achieved in other jurisdictions throughout the country and the world. These benefits could be realized in Michigan with proper utility implementation strategies. The Staff sees prudent utility investments in AMI as a first step toward realizing a modern grid. The Staff will continuously evaluate requests from utilities for recovery of advanced digital technology for consistency with prudency principles.

Opt-Out Policies in Other Jurisdictions

A few state commissions have adopted opt-out policies for their regulated utilities. California and Maine have the two most prominent examples of commission approved opt-out policies. Costs vary across jurisdictions and service providers. Generally, an initial fee is charged to cover the fixed costs of retaining or replacing an electromechanical meter along with a monthly fee associated with the ongoing meter reading costs. For example: there is a $75 up-front charge and a $10 monthly meter reading charge associated with the opt-out tariff of Pacific Gas and Electric in California. NV Energy of Nevada charges a monthly opt-out fee, which is higher for customers in the northern part of the state and lower to south Nevada customers.

States and municipalities feature a variety of opt-out meter choices. Some states allow customers to retain their electromechanical meter, while others provide a smart meter with the radio transmitter turned off. When more than one opt-out method is offered (such as in Maine), the charge for retaining an electromechanical meter is greater than the radio disabled smart meter to reflect the actual increased cost of maintenance incurred by the utility. Also, NV Energy offers AMR meters to those who choose to opt-out. Using AMR infrastructure, while not optimal, does reduce the cost of an opt-out policy for both the customer and utility.

Not all utilities or states with AMI have an opt-out policy. The Public Service Commission of Washington D.C. denied a request for an investigation into opt-out, and earlier in 2012, an order from the Idaho Public Utilities Commission dismissed a pair of complaints from customers who demanded that an opt-out policy be created. Opt-out plans are not offered in the Canadian provinces of British Columbia and Ontario, while Hydro-Québec proposed a radio-off option with an up-front and monthly charge.

Some state regulators are in the process of discussing whether or not to offer AMI opt-out, while others are working through the process of reviewing proposals for utility opt-out policies and evaluating costs. Commissions in Texas and Arizona are currently investigating smart meter opt-out options. Lawmakers in Georgia and Pennsylvania have introduced legislation that requires opt-out. A senate bill in New Hampshire aims to make smart meter deployment strictly opt-in. Vermont's opt-out legislation was signed into law in May, and requires opt-out and smart meter removal free of charge. Table 2 shows the status of opt-out policies across the United States and Canada as of June 2012. It is important to note that the opt-out debate is constantly changing in light of commission findings, legislative actions, and utility planning across the country. There is no universal opt-out program.

Table 2: Smart Meter Opt-Out Policies

Jurisdiction	Opt-Out Activity	Opt-Out Cost to consumers
Arizona E-00000C-11-0328	Opened a generic docket for the investigation of smart meters. (8/29/11)	
Colorado Docket 10R-799E	The commission intends to address opt-out in future proceeding. (10/17/11)	
California Decision #D1202014	California PUC approved opt-out. (2/9/12)	Analog meter: $75 initial fee, $10 monthly fee, low income customers pay reduced fees.
District of Columbia Order-16708	DC PSC denied Office of the People's Counsel's request for opt-out investigation. (4/13/12)	
Georgia Senate Bill 459	Opt-out bill passed Georgia senate. (3/13/12)	Proposes no fee.
Idaho Order-32500	Consumer request for opt-out is dismissed. (3/27/12)	
Illinois, City of Naperville	Municipal utility approved opt-out.	Radio-off smart meter: $68.35 + $24.75/mo.
Maryland Cases 9207, 9208	Interim order allows customers to defer smart meter installation pending the commission's final decision. (5/24/12)	
Maine Docket 7307	Maine PUC approved opt-out. (5/19/11)	Radio-off smart meter: $20+$10.50/mo. Electromechanical meter: $40+ $12/mo.
Nevada Docket 11-10007	NV Energy proposed opt-out tariff: AMR w/ monthly reporting. (5/1/12)	South Nevada: $98.75 + $7.61/mo. North Nevada: $107.66+$11.01/mo.
New Hampshire Senate Bill 266	Bill prohibiting electric utilities from installing smart meters without the property owner's consent. Passed by house and senate. (5/16/12)	
Oregon Advice # 11-15 Tariff Sheet # 300	Allows PGE customers to opt-out of a digital meter. Idaho Power has digital meters in Oregon with no opt-out option. (8/10/11)	Portland GE: $254 + $51/mo.
Pennsylvania House Bill 2188	A bill allowing opt-out is in committee. (2/8/12)	
Quebec	Régie de l'énergie considering Hydro-Québec's proposed opt-out rates. (3/14/12)	Hydro-Quebec: $98 + $17/mo.
Texas Filing 40190	Petition requesting an opt-out being considered by the PUC. (2/16/12)	
Vermont Act 170	Law does not allow opt-out fees or smart meter removal fees. (5/18/12)	No cost for opt-out.

Opt-out Options

The Staff concludes that providing an opt-out option is the best solution for customers who have concerns about smart meters. The Staff recommends that utilities investigate a variety of opt-out options. Electromechanical meters may be a viable opt-out option for some customers; however, maintaining electromechanical test facilities, inventory, and manual meter reading could result in higher incremental costs.[54] The traditional electromechanical meter is obsolete and currently not in production. Offering customers an electromechanical meter as an alternative to a smart meter is not a long-term solution.

Other options are the installation of a smart meter that does not have a communicating radio, relocating a smart meter on the customer's premise, or hard-wiring a smart meter into the network. A smart meter without a communicating radio allows the utility to maintain one type of meter. However, manual meter reading would still be required. Customers with a non-communicating meter will not receive some benefits of AMI, and would not, for example, be able to fully participate in new rate structures.

Smart meter relocation would allow customers to still receive all the benefits of AMI. Meter relocation may result in a higher initial cost and may not be feasible at some locations. Currently, administrative rules governing meter relocation allow the customer to request meter relocation at the customer's expense.[55]

A wired smart meter also permits opt-out customers to receive all AMI benefits by allowing two-way communication with the utility without using radio frequency (i.e. power line carrier, fiber optic cable, etc.). This option may be costly and may not be feasible within the confines of the utility infrastructure or of the customer's premises.

As discussed above, there are costs associated with allowing a customer to opt-out. Most states have acknowledged these costs by assessing charges that reflect the actual cost of maintaining a non-AMI meter.

No opt-out tariffs have been submitted to the Commission by any Michigan utilities as of June 2012. The Staff believes that ratemaking for the opt-out provision should be based on cost-of-service principles. If AMI meters result in a reduced cost of service, this could be accounted for by either an additional charge for those customers choosing to opt-out or a discount for those customers with an AMI meter.

[54] Commission billing rules allow for customers to read their own meters. However, the utility must verify the meter reading once a year. (Consumer Standards and Billing Practices for Electric and Gas Residential Services, R 460.115)

[55] Consumer Standards and Billing Practices For Electric and Gas Residential Services, 1999 AC, R 460.116

RECOMMENDATIONS AND CONCLUSIONS

Health and Safety

- After careful review of the available literature and studies, the Staff has determined that the health risk from the installation and operation of metering systems using radio transmitters is insignificant.

- The appropriate federal health and safety regulations provide assurance that smart meters represent a safe technology.

Data Privacy

- The Staff recommends that all stakeholders identify and implement privacy policy considerations through administrative rules, tariffs, orders and/or other means.

- Customer data privacy policies should include provisions addressing customer consent, individual access, customer choice, notice and purpose, collection and scope, data retention and management and accountability.

Cyber Security

- Each utility should adopt an annual independent security audit of the mechanisms of customer access, third party access and internal cyber risk-management practices.

- As outlined in the NARUC resolution regarding cyber security, the Staff intends to maintain a dialogue with regulated utilities to ensure that they are in compliance with standards, and that preparedness measures are employed to deter, detect and respond to cyber-attacks and to mitigate and recover from them.[56]

- Utilities should adopt the same breach notification policies as other states have adopted, namely the notification of any breach affecting 1000 or more customers within two weeks of the breach.

- Each utility should be required to file a yearly breach notification summary with the Staff, detailing all breaches of customer information, including any third party breach information.

Customer Education

- The Staff recommends utilities develop and implement a new education strategy similar to those used in other jurisdictions. Education program results should reflect high levels of customer engagement, acceptance and enthusiasm with their smart meter program.

[56] NARUC, *Resolution Regarding Cybersecurity, et al.*

- The strategy should include metrics to measure the overall effectiveness of the education program.

National Policy

- The United States Congress has passed several laws that support the upgrade of the electric grid, including deployment of smart meters for residential and other types of customers. These laws have provided a framework for smart grid, including smart meter deployment in the United States.

- Numerous prestigious agencies and institutions have considered the national outlook for the smart grid and indicate that installing smart grid technologies, including smart meters, will have a positive benefit on the United States' electric grid. These reports urge the United States to follow the directives of the federal law and update the electric grid.

Policies and Practices

- Several areas of current rules and tariffs will be affected by AMI deployment in Michigan. Administrative rules, tariffs, and Commission orders should be considered, and the most effective methodology should be employed to ensure customers have adequate protections.

- It is recommended that all stakeholders work to analyze and identify the most appropriate implementation methods for addressing the policy considerations. Stakeholders should routinely review all policies related to smart grid as smart grid technologies continue to develop.

Smart Grid Vision

- A Michigan smart grid vision should provide direction to implement technology that will enhance the functionality of the electric grid. All components of electric grid improvements, including AMI installation, distribution infrastructure replacement, and electric generation should reflect the larger objectives of a smart grid vision.

- The Staff proposes that future smart grid investments from utilities must correlate with the following objectives aimed at delivering transparent and identifiable benefits to ratepayers: accommodate advanced generation and storage options; enable informed participation by all customers; support new products, services, and markets; optimize existing assets, increase efficiency and improve reliability; and operate resiliently against physical and cyber-attacks.

Opt-Out

- The Staff concludes that an opt-out option or options is the best solution for customers who have concerns about smart meters.

- The Staff believes that ratemaking for the opt-out provision should be based on cost of service principles. If AMI meters result in a reduced cost of service, this could be accounted for by either an additional charge for those customers choosing to opt-out or a discount for those customers with an AMI meter.

Appendix A

Additional Resources:

- *National Institute of Standards and Technology Interagency Report 7628, Guidelines for Smart Grid Cyber Security: Vol. 1, Privacy and the Smart Grid*, August 2010. http://csrc.nist.gov/publications/nistir/ir7628/nistir-7628_vol1.pdf
- *National Institute of Standards and Technology Interagency Report 7628, Guidelines for Smart Grid Cyber Security: Vol. 2, Privacy and the Smart Grid*, August 2010.
- http://csrc.nist.gov/publications/nistir/ir7628/nistir-7628_vol2.pdf
- North American Energy Standards Board, *Third Party Access to Smart Meter-based Information*, April 20, 2012.
- Oklahoma Electric Usage Data Protection Act, H.B. 1079, May 20, 2011.
- C. Hagan & K. Thomas, *A Model Privacy Policy for Smart Grid Data Institute for Energy and the Environment*, Vermont Law School, November 4, 2011.
- Public Utility Commission of Texas, Electric Substantive Rules, Chapter 25 Rules. http://www.puc.state.tx.us/agency/rulesnlaws/subrules/electric/Electric.aspx
- Federal Trade Commission, Fair Information Practice Principles. http://www.ftc.gov/reports/privacy3/fairinfo.shtm
- Colorado Department of Regulatory Agencies Public Utilities Commission, 4 Code of Colorado Regulations 723-3 Part 3, Rules Regulating Electric Utilities, February 14, 2012.
- United States Code 47 §222, Privacy of Customer Information, January 7, 2011.
- Naperville Smart Grid Initiative, Naperville Smart Grid Customer Bill of Rights, Ordinance No. 11-029, February 16, 2011.
- Washington Administrative Code, Chapter 480-100, Electric Companies, February 15, 2012. http://apps.leg.wa.gov/wac/default.aspx?cite=480-100
- California Public Utility Commission, Public Utility Code Chapter 4-5. http://www.leginfo.ca.gov/cgi-bin/calawquery?codesection=puc
- NAESB Data Privacy Task Force, Team Five-State and Province Law. www.naesb.org/pdf4/data_privacy_042111w3.doc
- Arizona State Legislature, Consumer Protections; rules; confidentiality; unlawful practice http://www.azleg.gov/FormatDocument.asp?inDoc=/ars/30/00806.htm&Title=30&DocType=ARS
- California Public Utilities Commission, Decision Adopting Rules To Protect The Privacy And Security Of The Electricity Usage Data Of The Customers Of Pacific Gas And Electric Company, Southern California Edison Company, And San Diego Gas & Electric Company
- http://www.azleg.gov/FormatDocument.asp?inDoc=/ars/30/00806.htm&Title=30&DocType=ARS

- Colorado Department Of Regulatory Agencies, Public Utilities Commission, 4 Code of Colorado Regulations (CCR) 723-3, Part 3, Rules Regulating Electric Utilities. http://www.dora.state.co.us/puc/rules/723-3.pdf

- New York Department of Public Services, Smart Grid Privacy Statement. http://documents.dps.ny.gov/public/MatterManagement/CaseMaster.aspx?MatterCaseNo=10-E-0285

- Oklahoma State Legislature, Electric Usage Data Protection Act. http://www.oklegislature.gov/BillInfo.aspx?Bill=HB1079&Tab=0

- United States Department of Energy, Smart Grid Privacy Workshop Summary Report. http://www.smartgrid.gov/sites/default/files/doc/files/Privacy%20report%202012_03_19%20Final.pdf

- United States Department of Homeland Security, Privacy Policy Guidance Memorandum, December 29, 2008. http://www.dhs.gov/xlibrary/assets/privacy/privacy_policyguide_2008-01.pdf

- United States Department of Energy, Electricity Subsector cyber security risk management process, March 2012: Public Comment Draft. http://energy.gov/sites/prod/files/RMP%20Guideline%20Second%20Draft%20for%20Public%20Comment%20-%20March%202012.pdf

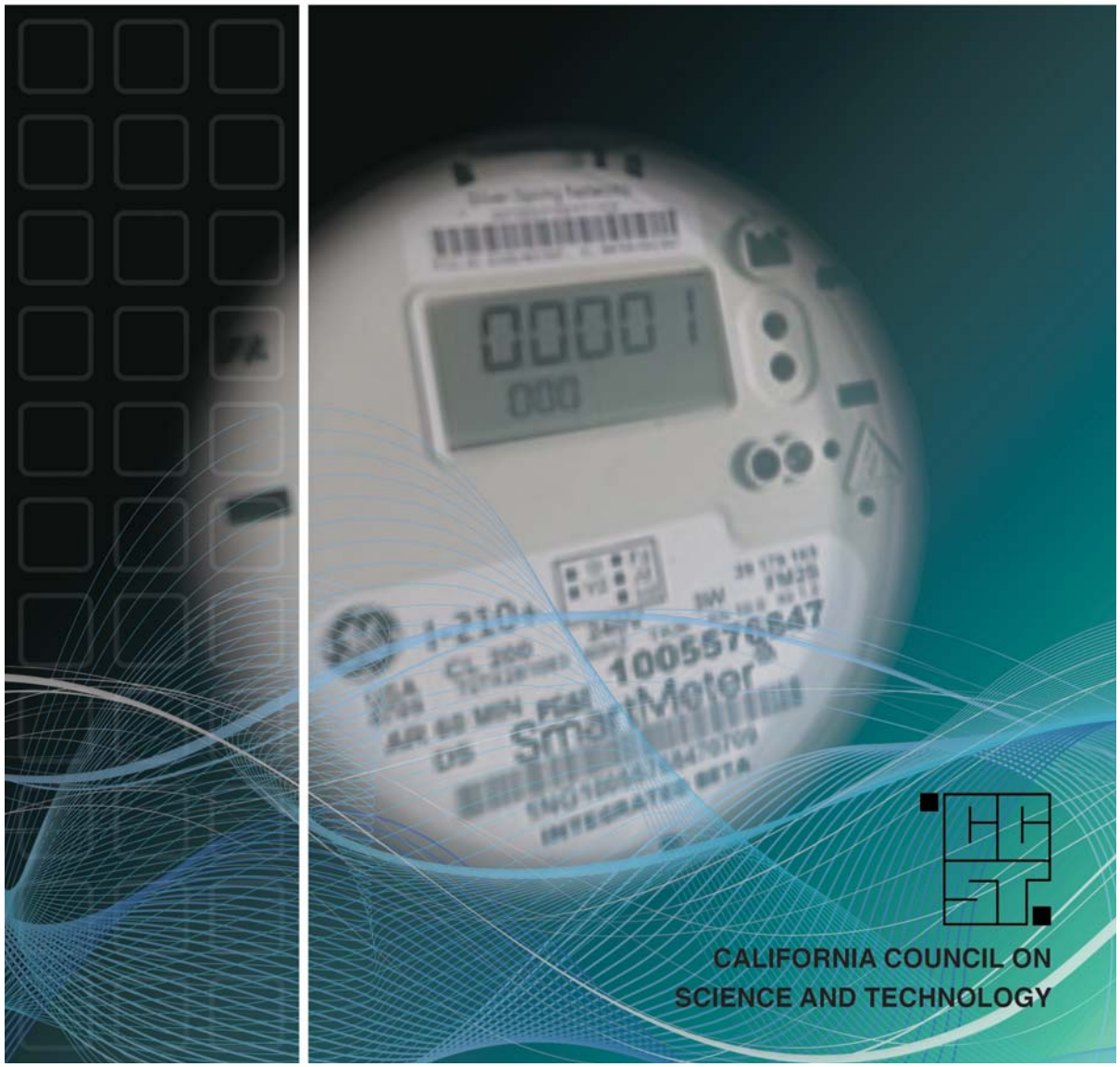
- Executive Office of the President, A Policy Framework For the 21st Century Grid, June 2011. http://www.whitehouse.gov/sites/default/files/microsites/ostp/nstc-smart-grid-june2011.pdf

- National Institute of Science and Technology, NIST Framework and Roadmap for Smart Grid Interoperability Standards Release 2.0. http://www.nist.gov/smartgrid/upload/NIST_Framework_Release_2-0_corr.pdf

- ASIS International, Utility and Smart Grid Security: The impact of NERC CIP Standards and NISTIR 7628 to the Utility Industry. http://www.asisonline.org/councils/documents/UtilitySmartGridSecurity.pdf

HEALTH IMPACTS OF RADIO FREQUENCY EXPOSURE FROM SMART METERS

APRIL 2011 - FINAL REPORT



CALIFORNIA COUNCIL ON
SCIENCE AND TECHNOLOGY

ACKNOWLEDGMENTS

We would like to thank the many people who provided input and feedback towards the completion of this report. Without the insightful feedback that these individuals generously provided, this report could not have been completed. We would like to give special thanks to the California Smart Grid Center, College of Engineering and Computer Science at the California State University, Sacramento and to the University of California's Center for Information Technology Research in the Interest of Society (CITRIS).

This report was conducted with the oversight of a CCST Smart Meter Project Team, whose members include: Rollin Richmond (Chair), Emir Macari, Patrick Mantey, Paul Wright, Ryan McCarthy, Jane Long, David Winickoff, and Larry Papay. We also thank J.D. Stack for his technical contributions and Lora Lee Martin for the overall coordination of this report response. We express gratitude to CCST's members and colleagues for their many contributions to the report. Comments on the January 2011 draft of this report were solicited from the public. Many very thoughtful and informed comments were received. All public comments were reviewed and taken into consideration as this final report was completed.

ISBN-13: 978-1-930117-42-6

CCST is a non-profit organization established in 1988 at the request of the California State Government and sponsored by the major public and private postsecondary institutions of California and affiliate federal laboratories in conjunction with leading private-sector firms. CCST's mission is to improve science and technology policy and application in California by proposing programs, conducting analyses, and recommending public policies and initiatives that will maintain California's technological leadership and a vigorous economy.

Note: The California Council on Science and Technology (CCST) has made every reasonable effort to assure the accuracy of the information in this publication. However, the contents of this publication are subject to changes, omissions, and errors, and CCST does not accept responsibility for any inaccuracies that may occur.

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Letter from CCST

With rapidly emerging and evolving technologies, lawmakers at times find themselves pressed to make policy decisions on complex technologies. Smart meters are one such technology.

Smart meters are being deployed in many places in the world in an effort to create a new generation of utility service based on the concepts of a smart grid, one that is agile, efficient and cost effective.

The electricity crisis of 2000 and 2001 helped force the issue here in California, lending significant urgency to the need for better management of power generation and distribution. In 2006, the California Public Utilities Commission authorized the Pacific Gas and Electric Company to implement a relatively new technology, smart meters, to gather much more precise information about power usage throughout the state. The process of installing the meters throughout the state is still underway.

As with any new technology, there are unknowns involved. Smart meters generally work by transmitting information wirelessly. Some people have expressed concerns about the health effects of wireless signals, particularly as they become virtually ubiquitous. These concerns have recently been brought to the attention of state legislators, with some local municipalities opting to ban further installation of the meters in their communities.

We are pleased that Assembly Members Huffman and Monning have turned to CCST for input on this issue. It is CCST's charge to offer independent expert advice to the state government and to recommend solutions to science and technology-related policy issues. In this case, we have assembled a succinct but comprehensive overview of what is known about human exposure to wireless signals and the efficacy of the FCC safety standards for these signals. To do so, we assembled a project team that consulted with over two dozen experts and sifted through over a hundred articles and reports, providing a thorough, unbiased overview in a relatively rapid manner.

In situations where public sentiment urges policy makers to make policy decisions with potentially long-term consequences, access to the best information possible is critical. This is the role that CCST was created to fulfill.

Susan Hackwood
Executive Director, CCST

Rollin Richmond
Project Team Chair, CCST

Health Impacts of Radio Frequency from Smart Meters
Response to Assembly Members Huffman and Monning

California Council on Science and Technology
April 2011

KEY REPORT FINDINGS
1. Wireless smart meters, when installed and properly maintained, result in much smaller levels of radio frequency (RF) exposure than many existing common household electronic devices, particularly cell phones and microwave ovens. 2. The current FCC standard provides an adequate factor of safety against *known thermally* induced health impacts of existing common household electronic devices and smart meters. 3. To date, scientific studies have not identified or confirmed negative health effects from *potential non-thermal* impacts of RF emissions such as those produced by existing common household electronic devices and smart meters. 4. Not enough is currently known about potential non-thermal impacts of radio frequency emissions to identify or recommend additional standards for such impacts

OTHER CONSIDERATIONS
Smart electricity meters are a key enabling technology for a "smart grid" that is expected to become increasingly clean, efficient, reliable, and safe at a potentially lower cost to the consumer. The CCST Smart Meter Project Team offers the following for further consideration by policy makers, regulators and the utilities. We appreciate that each of these considerations would likely require a cost/benefit analysis. However, we feel they should be considered as the overall cumulative exposure to RF emissions in our environment continues to expand. 1. As wireless technologies of all types increase in usage, it will be important to: (a) continue to quantitatively assess the levels of RF emissions from common household devices and smart meters to which the public may be exposed; and (b) continue to investigate potential thermal and non-thermal impacts of such RF emissions on human health. 2. Consumers should be provided with clearly understood information about the radiofrequency emissions of all devices that emit RF including smart meters. Such information should include intensity of output, duration and frequency of output, and, in the cases of the smart meter, pattern of sending and receiving transmissions to and from all sources. 3. The California Public Utilities Commission should consider doing an independent review of the deployment of smart meters to determine if they are installed and operating consistent with the information provided to the consumer. 4. Consideration could be given to alternative smart meter configurations (such as wired) in those cases where wireless meters continue to be concern to consumers.

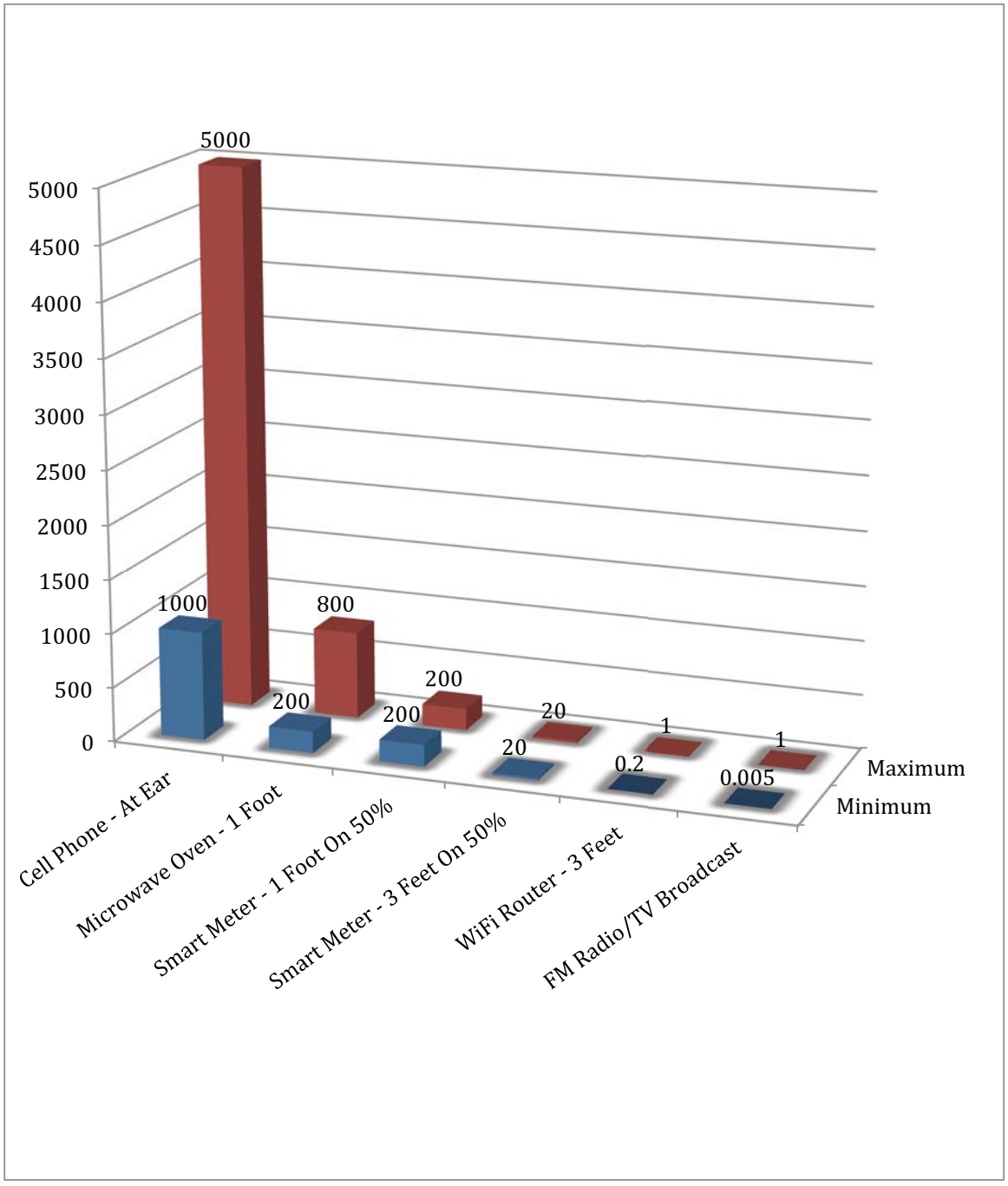

Figure 1. Instantaneous Radio Frequency Power Density Levels of Common Devices (in microWatts/cm^2)
About this figure**:** This figure was developed by the CCST project team**.** Quantities for different distances calculated using Inverse Square Law. Assumes distances in far-field, where power density reduces as the square of the distance from the source. Smart meter power scaled to obtain output for 50% duty cycle. The source for the various starting measurements came from Electric Power Research Institute (EPRI), Radio-Frequency Exposure Levels from Smart Meters: A Case Study of One Model (February 2011)

Legislative Request

On July 30, 2010, California Assembly Member Jared Huffman wrote to the California Council on Science and Technology (CCST) to request that the Council perform an "independent, science-based study…[that] would help policy makers and the general public resolve the debate over whether smart meters present a significant risk of adverse health effects." California Assembly Member Bill Monning signed onto the request with his own letter to CCST on September 15, 2010. The City of Mill Valley also sent a letter on September 20th supporting Assembly Member Huffman's request for the study.

Approach

Reflecting the requests of the Assembly Members, CCST agreed to compile and assess the evidence available to address:

> **1. Whether Federal Communications Commission (FCC) standards for smart meters are sufficiently protective of public health, taking into account current exposure levels to radiofrequency and electromagnetic fields.**
>
> **2. Whether additional technology-specific standards are needed for smart meters and other devices that are commonly found in and around homes, to ensure adequate protection from adverse health effects.**

CCST convened a Smart Meter Project Team composed of CCST Council and Board members supplemented with additional experts in relevant fields (see Appendix A for Project Team members). The Project Team identified and reviewed over 100 publications and postings about smart meters and other devices in the same range of emissions, including research related to cell phone RF emissions, and contacted over two dozen experts in radio and electromagnetic emissions and related fields to seek their opinion on the two identified issues.

It is important to note that CCST has not undertaken primary research of its own to address these issues. This response is limited to soliciting input from technical experts and to reviewing and evaluating available information from past and current research about health impacts of RF emitted from electric appliances generally, and smart meters specifically. This report has been extensively reviewed by the Project Team, experts in related fields, and has been subject to the CCST peer review process (see Appendix B). It has also been made available to the public for comment.

Two Types of Radio Frequency Effects: Thermal and Non-thermal

Household electronic devices, such as cellular and cordless telephones, microwave ovens, wireless routers, and wireless smart meters produce RF emissions. Exposure to RF emissions may lead to thermal and non-thermal effects. Thermal effects on humans have been extensively studied and appear to be well understood. The Federal Communications Commission (FCC) has established guidelines to protect public health from known hazards associated with the thermal impacts of RF: tissue heating from absorbing energy associated with radiofrequency emissions. Non-thermal effects, however, including cumulative or prolonged exposure to lower levels of RF emissions, are not well understood. Some studies have suggested non-thermal effects may include fatigue, headache, irritability, or even cancer. *But these findings have not been scientifically established, and the mechanisms that might lead to non-thermal effects remain uncertain.* Additional research and monitoring is needed to better identify and understand potential non-thermal effects.

Findings

Given the body of existing, *generally accepted scientific knowledge* regarding smart meters and similar electronic devices, CCST finds that:

1. **The FCC standard provides an adequate factor of safety against known RF induced health impacts of smart meters and other electronic devices in the same range of RF emissions.**
   The potential for behavioral disruption from increased body tissue temperatures is the only biological health impact that has been consistently demonstrated and scientifically proven to result from absorbing RF within the band of the electromagnetic spectrum (EMF) that smart meters use. The Federal Communications Commission (FCC) has set a limit on the Standard Absorption Rate (SAR) from electronic devices, which is well below the level that has been demonstrated to affect behavior in laboratory animals. Smart meters, including those being installed by Pacific Gas and Electric Company (PG&E) in the Assembly Members' districts, if installed according to the manufacturers instructions and consistent with the FCC certification, emit RF that is a very small fraction of the exposure level established as safe by the FCC guidelines.

   FCC staff has recently confirmed that it "relied on the expert opinions of EPA, NCRP, and others to conclude that the RF exposure limits it adopted were adequately protective of human health from all known adverse effects, regardless of whether these effects were thermal or athermal in origin".[1]

   The FCC guidelines provide a significant factor of safety against known RF impacts that occur at the power levels and within the RF band used by smart meters. Given current

[1] Statement provide by Robert Weller regarding FCC regulations on February 3, 2011. Robert Weller, Chief, Technical Analysis Branch, Office of Engineering and Technology, Federal Communications Commission.

scientific knowledge, the FCC guideline provides a more than adequate margin of safety against known RF effects.

2. **At this time there is no clear evidence that additional standards are needed to protect the public from smart meters or other common household electronic devices.** Neither the relevant scientific literature nor our expert consultations support that there is a causal relationship between RF emissions and non-thermal human health impacts. Nor does the relevant evidence convincingly describe mechanisms for such impacts, although more research is needed to better understand and verify these potential mechanisms. Given the absence of evidence supporting a real hazard, the benefits of elevating existing standards are highly speculative. Further, there is not an existing basis from which to understand what types of standards could be helpful or appropriate**.** Without a clearer understanding of the biological mechanisms involved identifying additional standards or evaluating the relative costs and benefits of those standards cannot be determined at this time.

   Given the existing significant scientific uncertainty around non-thermal effects, there is currently no generally accepted definitive, evidence-based indication that additional standards are needed. Because of the lack of generally accepted evidence, there is also not an existing basis from which to understand what types of standards could be helpful or appropriate**.** Without a clearer understanding of the biological mechanisms involved identifying additional standards or evaluating the relative costs and benefits of those standards cannot be determined at this time.

   CCST notes that in some of the studies reviewed, contributors have raised emerging questions from some in the medical and biological fields about the potential for biological impacts other than the thermal impact that the FCC guidelines address. A report of the National Academies identifies research needs and gaps and recommended areas of research to be undertaken to further understanding of long-term exposure to RF emissions from communication devices, particularly from non-thermal mechanisms.[2] In our increasingly wireless society, smart meters account for a very small portion of RF emissions to which we are exposed. Concerns about human health impacts of RF emissions from smart meters should be considered in this broader context.

[2] National Research Council (2008) *Identification of Research Needs Relating to Potential Biological or Adverse Health Effects of Wireless Communication,* The National Academies Press, Washington, D.C.

THE SCIENTIFIC METHOD
"Scientifically established", "generally accepted scientific knowledge" and other such references throughout this document are referencing information obtained through the scientific method. A scientific method consists of the collection of data through observation and experimentation, and the formulation and testing of hypotheses. These steps must be repeatable in order to predict future results. Scientific inquiry is generally intended to be as objective as possible, to reduce biased interpretations of results. Another basic expectation is to document, archive and share all data and methodology so they are available for careful scrutiny by other scientists, giving them the opportunity to verify results by attempting to reproduce them. This practice, called full disclosure, also allows statistical measures of the reliability of these data to be established.

INTERPRETING THE SCIENTIFIC LITERATURE
In our review of the relevant scientific evidence, we privileged those studies that had as many of the following indicia of scientific reliability as possible: (1) Empirical testing; (2) Peer review and publication; (3) The use of accepted standards and controls; (4) Degree to which the finding is generally accepted by a relevant scientific community. These criteria of scientific reliability are broadly based on the standards of expert testimony and evidence in the US Federal Courts.

Health concerns surrounding RF from smart meters are similar to those from many other devices that we use in our daily lives, including cordless and cellular telephones, microwave ovens, wireless routers, hair dryers, and wireless-enabled laptop computers. As detailed in the report, a comparison of electromagnetic frequencies from smart meters and other devices shows that the exposure level is very low.

Standards of Proof or Certainty in Public Health

In this report, scientific evidence is the primary consideration. Upon consulting with the California Department of Public Health, it is noted that using scientific evidence to shape public policy is always challenging. The standards for declaring certainty within a scientific discipline, which are based on the results of statistical testing, may be unrealistic or inappropriate for making public policy decisions, particularly those with potential impacts on population health. Statistical tests usually rely on the convention of whether the results of a given study are sufficient to reject the null hypothesis of no effect (i.e., of a given exposure). This is effectively a standard of 95% certainty, analogous to the legal standard of proof "beyond a reasonable doubt."

In public health, five factors are generally considered when reviewing scientific evidence for policy decisions related to specified exposures:

1. Severity of potential effect(s): e.g., cancer or serious birth defects would be considered more severe than skin irritation;
2. Number of people with potential exposure;
3. Levels of likely and possible exposures;
4. Degree of certainty of the specific effect(s) at different exposure levels; certainty just above 50% might be characterized as "more likely than not."
5. Cost to mitigate potential effect(s), typically considered in light of the other factors.

Policy makers constantly weigh these factors consciously or unconsciously as they interact with stakeholders to craft good public policy. In one situation, they might consider high-cost mitigations for high-severity effects with high-certainty evidence. In another situation with high-severity effects and "more likely than not" certainty of those effects, they might choose low-cost mitigations. This report did not extend beyond the scientific evidence realm with which we were charged leaving those issues to the policy makers to whom this report has been delivered.

What are Smart Meters?

Smart meters measure attributes of electricity, natural gas, or water as delivered to consumers and transmit that information (e.g., usage) digitally to utility companies. Some smart meters are also designed to transmit real-time information to the consumer. These smart meters replace traditional, analog meters and meter readers with an automated process that is expected to reduce operating costs for utilities, and potentially, costs for customers (see Figure 2). Each of California's major electricity utilities has begun deploying smart meter infrastructure.

a. Analog Meter b. Digital Meter

Figure 2. a) An analog, conventional meter and a (b) digital smart meter (Source: PG&E)

There are many kinds of smart meters manufactured by a variety of companies. The meter, including sensors and the housing or casing, may be manufactured by one company while the communications device (installed within the meter) is manufactured by another. Depending upon the internal communications device employed, meters are configured to operate in a wired or in wireless environment. The smart meters used by PG&E are made by General Electric and Landis + Gyr and use a wireless communications technology from Silver Spring Networks. Each of these PG&E meters has two transmitters to provide two different communications of data from these meters.[3] The first provides for the "automatic meter reading" (AMR) function of the meter (and for more detailed and real time monitoring of the characteristics of the

[3] Tell, R. (2008) "Supplemental Report on An Analysis of Radiofrequency Fields Associated with Operation of the PG&E Smart Meter Program Upgrade System," Prepared for Pacific Gas & Electric Company, Richard Tell Associates, Inc., October 27.

electrical energy delivered to the consumer) and sends this data to an access point, where it is collected along with data from many other customers and transmitted to PG&E using a wireless area network (WAN) (similar to the way cell phone communication works).

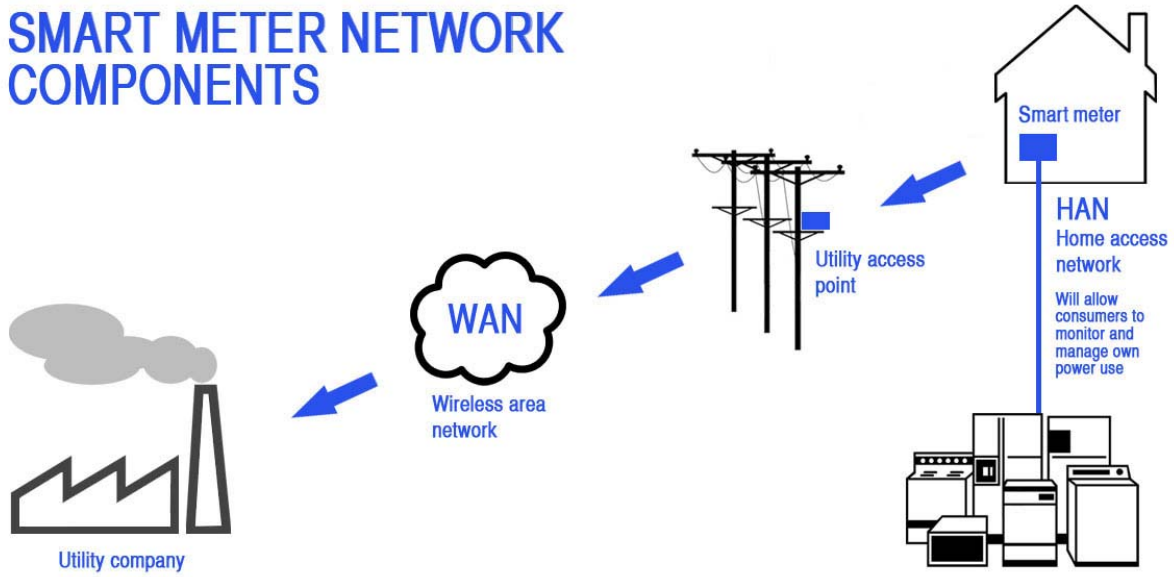

Figure 3. Simplified depiction of Smart Meter system network. Arrows show the use of radiofrequency (RF) signals for automated meter reading, communications among electric power meters, relays, access points, the company's enterprise management systems. The future home access network will operate within the house.

Smart meters have evolved from automatic meter reading (AMR; i.e., replacing meter readers) to a real time monitoring of power as delivered to the consumer by the utility company. CCST obtained from PG&E the Richard Tell Associates report, which describes the operation of the smart meter from the 2008 perspective of AMR, not a fully deployed real time smart grid. The Richard Tell Associates reports describe the use of the smart meter radios being deployed by PG&E as licensed by the FCC for a maximum power output of 1 W (watt) and within the 902-928 MHz (mega-hertz) frequency band. In its initial deployment, PG&E reports that it will configure the radios to transmit data from the meter to the access point once every four hours, for about 50 milliseconds at a time.[4] Accounting for this, the current duty cycles of the smart meter transmitter (that is, the percent of time that the meter operates) would then typically be 1 percent, or in some cases where the meter is frequently used as a relay, as much as 2-4 percent. *This means that the typical smart meter in this initial (AMR) use would not transmit any RF signal at least 96-98 percent of the time.*

It is important to note that any one smart meter is part of a broader "mesh" network and may act as a relay among other smart meters and utility access points. In addition, when the smart

[4] Tell, R. (2008) "Supplemental Report on An Analysis of Radiofrequency Fields Associated with Operation of the PG&E Smart Meter Program Upgrade System," Prepared for Pacific Gas & Electric Company, Richard Tell Associates, Inc., October 27. http://www.pge.com/includes/docs/pdfs/shared/edusafety/systemworks/rfsafety/rf_fields_supplemental_report_2008.pdf)

grid is fully functional the smart meters would be expected to be transmitting much more than once every four hours, providing data in near real-time, which will result in a much higher duty cycle. For purposes of this report we include a hypothetical scenario where the smart meter is transmitting 50 percent of the time (i.e., transmitting half the time and receiving half the time). Even in this 50% duty cycle situation the power output would be well below the FCC limits.

Smart meters are designed to transmit data to a utility access point that is usually 25 feet above ground, on utility or light poles. These access points are designed to transmit data from up to 5,000 smart meters to the utility company. Access points have a similar AMR transmitter as smart meters, as well as an additional *AirCard,* which communicates with utilities and is similar to wireless cards used in laptop computers. *AirCard*s typically operate at 0.25-1 W, in the 800-900 MHz or 1.9 GHz range.

In some cases, data is moved through the mesh network, relaying the data through other meters to the utility access point. This may occur when the topography or built environment interferes with the transmission of data from a smart meter to the access point. In these cases, the relaying of data may occur between one smart meter and another before the signal is sent to the utility access point (e.g., hops along a set of meters). Additionally, some non-meter data relays will also exist in the system to connect some smart meters to utility access points.

Many smart meters, including those from PG&E, also have a second transmitter that, at some future point in time, will allow customers to enable a home access network (HAN). The HAN will allow increased consumer monitoring of electricity use and communication among appliances and the future smart grid. This functionality is important to achieve the full potential of the smart grid. This second internal transmitter, for delivery of smart meter data to the consumer, reportedly will operate at a rated power of 0.223W, at frequency of about 2.4 GHz (again, similar to that of cell phones and wireless phones). The actual duty cycle of this transmitter will depend on the design and operation of the home area network.

Why are Smart Meters Being Installed Throughout California?

It is anticipated, when fully operational, that smart electricity meters are a key enabling technology for a "smart grid" that is expected to become increasingly clean, efficient, reliable, and safe (see Figure 3) at a potential lower cost to the consumer. (Digital meters are also being used for reading of natural gas and water consumption). Smart electrical meters allow direct two-way communication between utilities and customers, which is expected to help end users adjust their demand to price changes that reflect the condition of the electricity grid. These end user adjustments can help to protect the overall reliability of the electricity grid, cut costs for utility customers, and improve the operation and efficiency of the electricity grid. The smart grid will enable grid operators to better balance electricity supply and demand in real-time, which becomes increasingly important as more intermittent wind and solar generation resources are added to the grid.

Figure 4 depicts the potential operation of a smart grid.

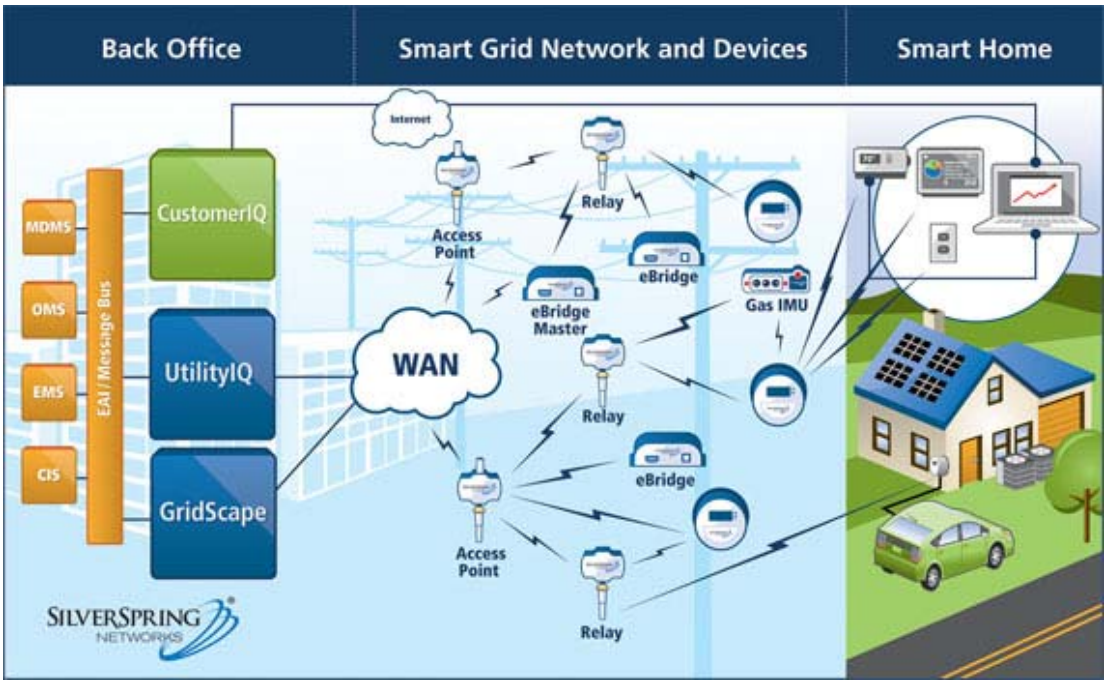

Figure 4. Illustration of components of the PG&E Smart Meter Program Upgrade showing the use of radiofrequency (RF) signals for communications among electric power meters, relays, access points and, ultimately, the company's enterprise management systems. (Source Silver Spring Network[5])

Smart meters will also allow utilities to communicate grid conditions to customers through price signals, so that consumers, via their HAN, can delay non-time sensitive demands (such as clothes drying) to a time when electricity is cheapest or has the most benefit to the reliability of the system. In some cases wireless signals interior to the structure will also be able to automatically adjust the heating and ventilation systems and to adjust heat or air conditioning units. This adaptation to price or reliability signals could reduce overall electricity costs for customers, improve the utilization of renewable and non-renewable power plants, and cut costs associated with adding intermittent wind and solar resources to the grid.

While such long-term value of smart meters will take years to fully realize, they are sufficiently promising that the federal government has required utilities to take steps to implement smart

[5] See http://www.silverspringnet.com/products/index.html for component descriptions. Network infrastructure includes the Silver Spring Access Points (APs) and Relays that forward data from endpoints across the utility's backhaul or WAN infrastructure into the back office.
The UtilityIQ application suite incorporates both utility applications such as Advanced Metering and Outage Detection as well as administrative programs for managing and upgrading the network. GridScape provides management for DA communications networks.
The CustomerIQ web portal enables utilities to directly communicate usage, pricing, and recommendations to consumers. Silver Spring works with each utility to customize the information portrayed and to import utility-specific information such as rate schedules.

grid networks, including the use of smart meters.[6] After review and authorization from the California Public Utilities Commission,[7] utilities in California have begun to install smart meters throughout the state. Some California utilities (such as Sacramento Municipal Utility District) have received significant federal funding for smart meter deployment from the American Recovery and Reinvestment Act (federal stimulus package). Many countries around the world are actively deploying smart meters as well. Digital smart meters are generally considered to be the fundamental technology required to enable widespread integration of information technology (IT) into the power grid (i.e., the smart grid). The following table (table 1) summarizes some potential societal benefits expected to result from the smart grid.

Table 1: Smart Grid Benefits

Consumers	**Environment**
1. Cost Savings Resulting from Energy Efficiency 2. Increased Consumer Choice and Convenience 3. More Transparent, Real-Time Information and Control for Consumers	1. Widespread Deployment of Renewable Energy (Solar, Wind, Biofuels) and Electric Vehicles (EVs) 2. Reduced Need to Build More Fossil Fueled Power plants 3. Reduced Carbon Footprint and Other Pollutants (via Renewables, Energy Efficiency, Electric Vehicles)
Utilities	**Economy**
1. Reduced Cost Due to Increased Efficiencies in Delivering Electricity and Reduction in Manpower to Read Meters. 2. Improved Reliability and More Timely Outage Response 3. Increased Customer Satisfaction Due to Cost Savings and Self-Control *Source: California Smart Grid Center*	1. Creates New Market for Goods and Services (i.e., New Companies, New Jobs) 2. Up-skilling Workforce to be Prepared for New Jobs 3. Reduced Dependence on Foreign Oil, Keeps Dollars at Home

[6] The federal Energy Independence and Security Act of 2007 directs states to encourage utilities to initiate smart grid programs, allows recovery of smart grid investments through utility rates, and reimburses 20% of qualifying smart grid investments. The American Recovery and Reinvestment Act of 2009 provided $4.5 billion to develop smart grid infrastructure in the U.S. For more information, see: Congressional Research Service (2007) "Energy Independence and Security Act of 2007: A Summary of Major Provisions," CRS Report for Congress, Order Code RL34l294, December 21. (http://energy.senate.gov/public/_files/RL342941.pdf)

[7] California Public Utilities Commission decision on Application 07-12-009 (March 12, 2009). Decision on Pacific Gas and Electric Company's Proposed Upgrade to the Smartmeter Program.

What Health Concerns are Associated with Smart Meters?

Human health impacts from exposure to electromagnetic frequency (EMF) emissions vary depending on the frequency and power of the fields. Smart meters operate at low power and in the RF portion of the electromagnetic spectrum. At these levels, RF emissions from smart meters are unlikely to produce *thermal effects; however it is not scientifically confirmed whether or what the non-thermal effects* on living organisms, and potentially, human health might be. These same concerns over potential impacts should apply to all other electronic devices that operate with similar frequency and power levels, including cell phones, computers, cordless phones, televisions, and wireless routers. Any difference in health impacts from these devices is likely to be a result of differences in usage patterns among them.

Thermal Effects

Electromagnetic waves carry energy, and EMF absorbed by the body can increase the temperature of human tissue. The scientific consensus is that body temperatures must increase at least 1°C to lead to potential biological impacts from the heat. The only scientifically verified effect that has been shown to occur in the power and frequency range that smart meters are designed to occupy is a disruption in animal feeding behavior at energy exposure levels of 4 W/kg and with an accompanying increase in body temperature of 1°C or more.[8] The exposure levels from smart meters even at close range are far below this threshold. The FCC has set limits on power densities from electronic devices that are well below the level where demonstrated biological impacts occur, and the limits are tens or hundreds of times higher than likely exposure from smart meters.[9]

Non-thermal Effects

There are emerging questions in the medical and biological fields about potential harmful effects caused by non-thermal mechanisms of absorbed RF emissions. Complaints of health impacts from "electromagnetic stress" have been reported, with symptoms including fatigue, headache, and irritability. Some studies have suggested that RF absorption from mobile phones may disrupt communication between human cells, which may lead to other negatives impacts on human biology.[10,11] While concerns of brain cancer associated with mobile phone usage persist, there is currently no definitive evidence linking cell phone usage with increased

[8] D'Andrea, J.A., Adair, E.R., and J.O. de Lorge (2003) Behavioral and cognitive effects of microwave exposure, *Bioelectromagnetics* Suppl 6, S39-62 (2003).

[9] Tell, R. (2008) "Supplemental Report on An Analysis of Radiofrequency Fields Associated with Operation of the PG&E Smart Meter Program Upgrade System," Prepared for Pacific Gas & Electric Company, Richard Tell Associates, Inc., October 27. (http://www.pge.com/includes/docs/pdfs/shared/edusafety/systemworks/rfsafety/rf_fields_supplemental_report_2008.pdf)

[10] Markova, E., Malmgren, L., and I.Y. Belyaev (2009) Microwaves from mobile phones inhibit 53PB1 focus formation in human stem cells stronger than in differentiated cells: Possible mechanistic link to cancer risk. Environmental Health Perspectives, doi:10.1289/ehp.0900781.

[11] Nittby, H., Grafstrom, G., Eberhardt, J.L., Malmgren, L., Brun, A., Persson B.R.R., and L.G. Salford (2008) Radiofrequency and Extremely Low-Frequency Electromagnetic Field Effects on the Blood-Brain Barrier Electromagnetic Biology and Medicine, 27: 103–126, 2008.

incidence of cancer.[12] But due to the recent nature of the technology, impacts of long-term exposure are not known. Ongoing scientific study is being conducted to understand non-thermal effects from long-term exposure to mobile phones and smart meters, etc., especially the cumulative impact from all RF emitting devices including that of a network of smart meters operating throughout a community.[13]

There currently is no conclusive scientific evidence pointing to a non-thermal cause-and-effect between human exposure to RF emissions and negative health impacts. For this reason, regulators and policy makers may be prudent to call for more research while continuing to base acceptable human RF exposure limits on currently proven scientific and engineering findings on known thermal effects, rather than on general concerns or speculation about possible unknown and as yet unproven non-thermal effects. Such questions will likely take considerable time to resolve. The data that are available strongly suggest that if there are non-thermal effects of RF absorption on human health, such effects are not so profound as to be easily discernable.

FCC Guidelines

In 1985, the FCC first established guidelines to limit human exposure and protect against thermal effects of absorbed RF emissions. The guidelines were based on those from the American National Standards Institute (ANSI) that were issued in 1982.[14] In 1996, the FCC modified its guidelines,[15] based on a rulemaking process that began in 1993 in response to a 1992 revision of the ANSI guidelines[16, 17] and findings by the National Council on Radiation Protection and Measurements (NCRP).[18] The 1996 guidelines are still in place today.

> In its rulemaking process to set SAR and MPE limits, the FCC relied on many federal health and safety agencies, including the U.S. Environmental Protection Agency and the Food and Drug Administration.

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[12] Ahlbom, A., Feychting, M., Green, A., Kheifets, L., Savitz, D. A., and A. J. Swerdlow (2009) Epidemiologic evidence on mobile phones and tumor risk: a review. *Epidemiology* 20, 639-52 (2009).

[13] National Research Council (2008) *Identification of Research Needs Relating to Potential Biological or Adverse Health Effects of Wireless Communication,* The National Academies Press, Washington, D.C. (http://www.nap.edu/catalog/12036.html)

[14] American National Standards Institute (1982) "American National Standard Radio Frequency Radiation Hazard Warning Symbol," ANSI C95.2-1982, Institute of Electrical and Electronics Engineers, Inc.

[15] FCC (1997) "Evaluating Compliance with FCC Guidelines for Human Exposure to Radiofrequency Electromagnetic Fields," OET Bulletin 65 (Edition 97-01), Federal Communications Commission, August. (http://www.fcc.gov/Bureaus/Engineering_Technology/Documents/bulletins/oet65/oet65.pdf)

[16] American National Standards Institute (1992) "Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz," ANSI/IEEE C95.1-1992 (previously issued as IEEE C95.1-1991), Institute of Electrical and Electronics Engineers, Inc.

[17] American National Standards Institute (1992) "Recommended Practice for the Measurement of Potentially Hazardous Electromagnetic Fields – RF and Microwave," ANSI/IEEE C95.3-1992, Institute of Electrical and Electronics Engineers, Inc.

[18] NCRP (1986) "Biological Effects and Exposure Criteria for Radiofrequency Electromagnetic Fields," NCRP Report No. 86 (1986), National Council on Radiation Protection Measurements.

While the FCC guidelines appear to provide a large factor of safety against known thermal effects of exposure to radiofrequency, they do not necessarily protect against potential non-thermal effects, nor do they claim to.[19] Without additional understanding of these effects, there is inadequate basis to develop additional guidelines at this time.

The FCC guidelines measure exposure to RF emissions in two ways. Specific absorption rate (SAR) measures the rate of energy absorption and is measured in units of watts-per-kilogram of body weight (W/kg). It accounts for the thermal effects on human health associated with heating body tissue and is used as a limiting measurement for wireless devices, such as mobile phones, that are used in close proximity to human tissue.[20] The FCC limits, as well as the underlying ANSI and NCRP limits, are based on a SAR threshold of 4 W/kg. At the time of the FCC rulemaking, and still today, behavioral disruption in laboratory animals (including non-human primates) at this absorption rate is the only adverse health impact that has been clearly linked to RF at levels similar to those emitted by smart meters. This finding is supported in scientific literature[21, 22] and by the World Health Organization and many health agencies in Europe.[23, 24] The FCC limit of 1.6 W/kg provides a significant factor of safety against this threshold.

Limits on SAR provide the basis for another measurement of exposure, maximum permissible exposure (MPE). MPE limits average exposure over a given time period (usually 30 minutes for general exposure) from a device and is often used for exposure to stationary devices and where human exposure is likely to occur at a distance of more than 20 cm. It is measured in micro (10^{-6}) watts-per-square-centimeter ($\mu W/cm^2$), and accounts for the fact that the human body absorbs energy more efficiently at some radiofrequencies than others. The human body absorbs energy most efficiently in the range of 30-300 MHz, and the corresponding MPE limits for RF emissions in this range are consequently the most stringent. In the frequency bands where smart meters operate, including PG&E's, namely the 902-928 MHz band and 2.4 GHz range, the human body absorbs energy less efficiently, and the MPE limits are less restrictive.

[19] The U.S. EPA confirmed this in a letter to The Electromagnetic Radiation Policy Institute, dated March 8, 2002. (http://www.emrpolicy.org/litigation/case_law/docs/noi_epa_response.pdf)

[20] FCC (2001) "Additional Information for Evaluating Compliance of Mobile and Portable Devices with FCC Limits for Human Exposure to Radiofrequency Emissions," Supplement C (Edition 01-01) to OET Bulletin 65 (Edition 97-01), Federal Communications Commission, June. (http://www.fcc.gov/Bureaus/Engineering_Technology/Documents/bulletins/oet65/oet65c.pdf)

[21] D'Andrea, J.A., Adair, E.R., and J.O. de Lorge (2003) Behavioral and cognitive effects of microwave exposure, *Bioelectromagnetics* Suppl 6, S39-62 (2003).

[22] Sheppard, A.R, Swicord, M. L., and Q. Balzano (2008) Quantitative evaluations of mechanisms of radiofrequency interactions with biological molecules and processes, *Health Phys* 95, 365-96 (2008).

[23] The World Health Organization has reviewed international guidelines for limiting radiofrequency exposure and scientific studies related to human health impacts and concludes that exposure below guideline limits don't appear to have health consequences. (http://www.who.int/peh-emf/standards/en/)

[24] Committee on Man and Radiation (COMAR) (2009) "Technical Information Statement: Expert reviews on potential health effects of radiofrequency electromagnetic fields and comments on The Bioinitiative Report," Health Physics 97(4):348-356 (2009).

The FCC limits on MPE are summarized in Figure 5.[25, 26] At 902 MHz, appropriate for operation of the AMR transmitter of the smart meter; the FCC limit is 601 μW/cm^2. At higher frequencies, the human body absorbs even less energy, and the threshold for the 2.4 GHz transmitter for home area network communications is consequently higher, 1000 μW/cm^2.

PG&E commissioned a 2008 study by Richard Tell Associates, "Supplemental Report on An Analysis of Radiofrequency Fields Associated with Operation of the PG&E Smart Meter Program Upgrade System." In this study of PG&E's proposed smart meter network it is noted that the FCC limits on MPE include a factor of safety, and the perceived hazardous exposure level is 50 times higher than the FCC limits.[27] The study estimates that the highest exposure from smart meters, if an individual were standing directly in front of and next to the meter, would be 8.8 μW/cm^2 transmitting at 2 to 4% of the time. The study notes that this is almost 70 times less than the FCC limit and 3,500 times less than the demonstrated hazard level. In all likelihood, individuals will be much farther away from smart meters and likely behind them, (within a structure) where power density will be much lower. The highest exposure from the entire smart meter system would occur immediately adjacent to an access point. It is very unlikely that an individual would be immediately adjacent to an access point, as they are normally located 25 feet above the ground on a telephone or electrical pole or other structure. The peak power density from an access point is estimated to be 24.4 μW/cm^2, or about 25 times less than the FCC limit. From the ground, exposure to power density from access points is estimated to be 15,000 times less than the FCC limit in great part due to the distance from the device.

The PG&E commissioned report by Richard Tell Associates is based only on an AMR duty cycle of transmitting data once every four hours which results in this very low estimated peak power. However, we are not aware of the justification for using averaging over a four-hour period. We do know the FCC[28] allows averaging of exposure over a designated period (30 minutes). To truly be a smart grid the data will be transmitted at a much more frequent rate than this. In this report we look at the worst-case scenario, a meter that is stuck in the "on" position, constantly relaying, at a 100% duty cycle. Even in this 100% scenario the RF emissions would be measurably below the FCC limits for thermal effects.

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[25] FCC (1997) "Evaluating Compliance with FCC Guidelines for Human Exposure to Radiofrequency Electromagnetic Fields," OET Bulletin 65 (Edition 97-01), Federal Communications Commission, August. (http://www.fcc.gov/Bureaus/Engineering_Technology/Documents/bulletins/oet65/oet65.pdf)

[26] FCC (1999) "Questions and Answers about Biological Effects and Potential Hazards of Radiofrequency Electromagnetic Fields," OET Bulletin 56 (Fourth Edition), Federal Communications Commission, August. (http://www.fcc.gov/Bureaus/Engineering_Technology/Documents/bulletins/oet56/oet56e4.pdf)

[27] Tell, R. (2008) "Supplemental Report on An Analysis of Radiofrequency Fields Associated with Operation of the PG&E Smart Meter Program Upgrade System," Prepared for Pacific Gas & Electric Company, Richard Tell Associates, Inc., October 27. (http://www.pge.com/includes/docs/pdfs/shared/edusafety/systemworks/rfsafety/rf_fields_supplemental_report_2008.pdf)

[28] http://www.fcc.gov/Bureaus/Engineering_Technology/Documents/bulletins/oet56/oet56e4.pdf

Power Density (and Exposure Level) Declines Rapidly with Distance

The power density from smart meters, or other devices that emit RF, falls off dramatically with distance. Figure 6 illustrates this affect for an example smart meter. While the estimated maximum exposure level at 1 foot from the meter with a duty cycle of 50% is 180 µW/cm^2 (far below the FCC guidelines), at a distance of about 10 feet, the power-density exposure approaches zero.

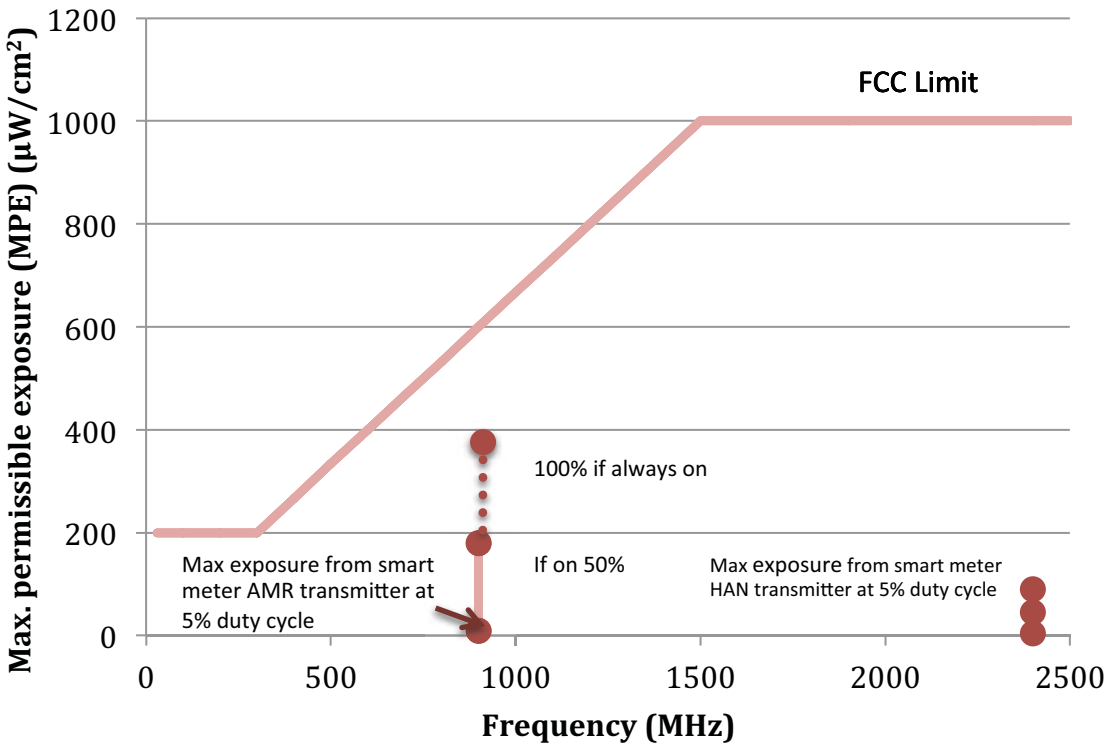

Figure 5. FCC maximum permissible exposure limits on power density rise with frequency because the human body can safely absorb more energy at higher frequencies. The estimated maximum exposure from a 1-Watt AMR transmitter at 5% duty cycle (i.e., 72 minutes/day) and one-foot distance is 18 µW/cm^2, or 3% of the FCC limit. Even if a meter malfunctioned and was stuck in the always-on transmit mode (i.e., 100% duty cycle), exposure levels would be 60% of the FCC limit for an AMR transmitter. For a 250mW HAN transmitter at a 5% duty cycle, the level would be .45% of the FCC limit and 9% of the FCC limit if the transmitter were on 100%. Exposure figures derived from February 2011 Electric Power Research Institute (EPRI) field measurement study entitled "Radio Frequency Exposure Levels from Smart Meters: A Case Study of One Model".[29]

[29] EPRI (2011) "Radio-Frequency Exposure Levels from Smart Meters: A Case Study of One Model," Electric Power Research Institute, February 2011.

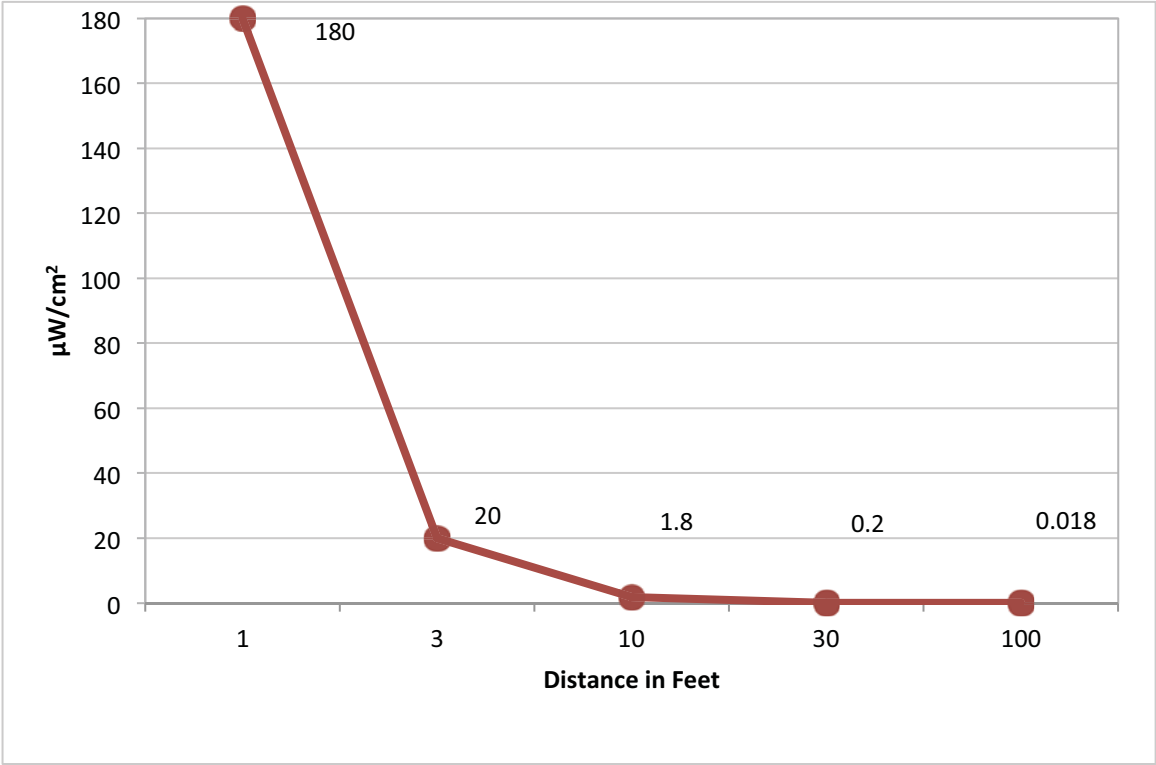

Figure 6. Power density from a sample smart meter versus distance;[30] 1-Watt emitter at 50% duty cycle. Typical smart meter AMR transmitter power density declines rapidly with distance. The rapid drop of power density with distance (inverse-square law) is similar for various duty cycles and different sets of source data.

Comparison of Electromagnetic Frequencies from Smart Meters and Other Devices

Health concerns surrounding RF from smart meters are similar to those from many other devices that we use in our daily lives, including cordless and mobile telephones, microwave ovens, wireless routers, hair dryers, and wireless-enabled laptop computers.

In addition to slight differences in frequency and power levels, which affect human absorption of RF from these devices, the primary difference among them is how they are used. Cell phones, for example, are often used for many minutes at a time, several times over the course of a day, and held directly next to one's head.

For perspective, microwave ovens operate at a similar frequency as the HAN transmitter of smart meters (2.45 GHz), and the U.S. Food and Drug Administration has set limits on leakage levels that are five times higher (5,000 µW /cm^2) than the FCC limit for smart meters and other

[30] EPRI (20110) "Radio- Frequency Exposure Levels from Smart Meters; A Case Study of One Model, "" Electric Power Research Institute, February 2011.

devices operating at 2.4 GHz.[31] Wireless routers and Wi-Fi equipment produce radiofrequency fields of about 0.2 – 1.0 µW /cm^2.[32, 33, 34] People in metropolitan areas are exposed to radiofrequency from radio and television antennas, as well, although for most of the population, exposure is quite low, around 0.005 µW /cm^2.[35]

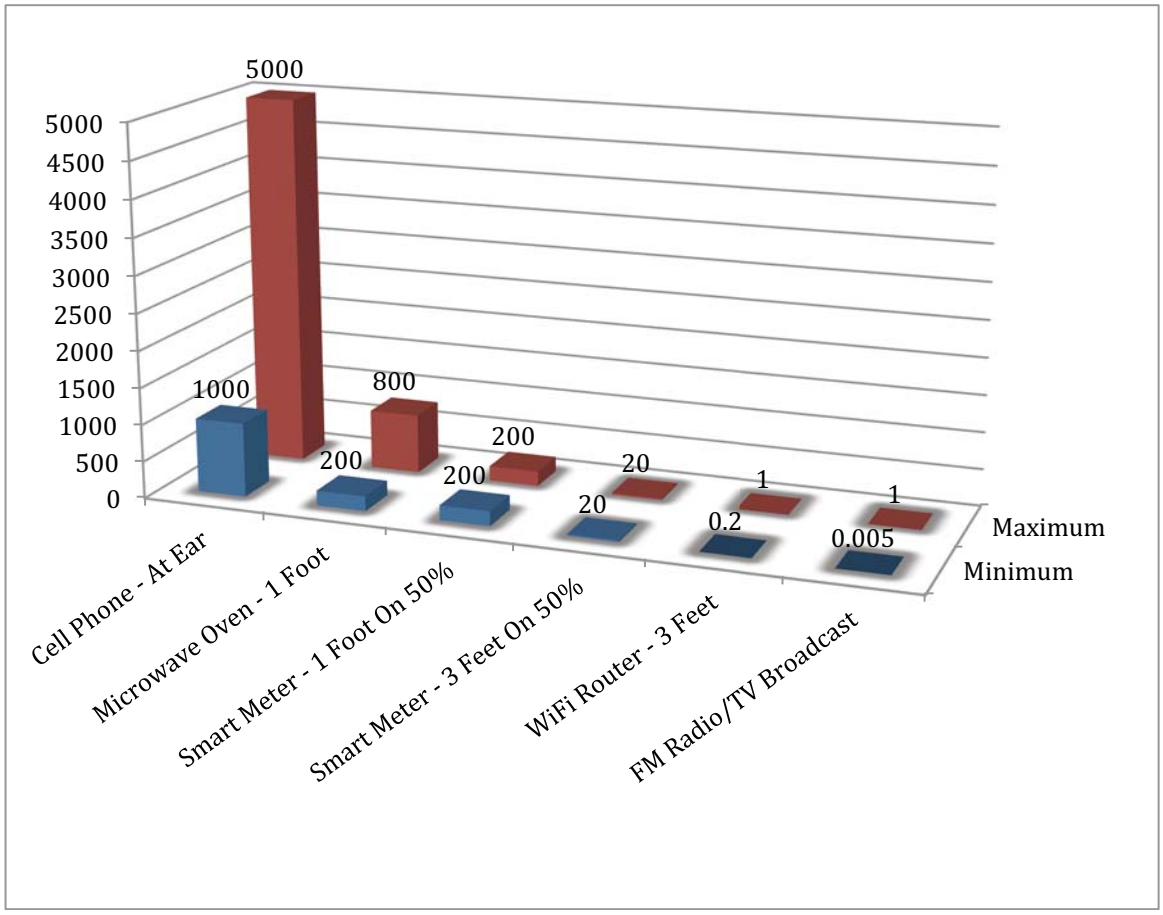

Figure 7. Instantaneous Radio Frequency Power Density Levels of Common Devices (in microWatts/cm^2)
About this figure**:** This figure was developed by the CCST project team**.** Quantities for different distances calculated using Inverse Square Law. Assumes distances in far-field, where power density reduces as the square of the distance from the source. Smart meter power scaled to obtain output for 50% duty cycle. The source for the various starting measurements came from Electric Power Research Institute (EPRI), Radio-Frequency Exposure Levels from Smart Meters: A Case Study of One Model (February 2011)

[31] FDA, "Summary of the Electronic Product Radiation Control Provisions of the Federal Food, Drug, and Cosmetic Act," U.S. Food and Drug Administration. (http://www.fda.gov/Radiation-EmittingProducts/ElectronicProductRadiationControlProgram/LawsandRegulations/ucm118156.htm)
[32] EPRI (2011) "Radio-Frequency Exposure Levels from Smart Meters; A Case Study of One Model, "Electric Power Research Institute, February 2011.
[33] Foster, K.R. (2007) Radiofrequency exposure from wireless LANS utilizing WI-FFI technology. *Health Physics*, Vol. 92, No. 3, March, pp. 280-282.
[34] Schmidt, G. et al. (2007) Exposure of the general public due to wireless LAN applications in public Places, *Radiation Protection Dosimetry*, Vol. 123, No. 1, Epub June 11, pp. 48-52.
[35] EPA (1986) The Radiofrequency Radiation Environment: Environmental Exposure Levels and RF Radiation Emitting Sources, EPA 520/1-85-014, U.S. Environmental Protection Agency, July.

Table 2: Radio-Frequency Levels from Various Sources

Source	Frequency	Exposure Level (mW/cm^2)	Distance	Time	Spatial Characteristic
Mobile phone	900 MHz, 1800 MHz	1—5	At ear	During call	Highly localized
Mobile phone base station	900 MHz, 1800 MHz	0.000005—0.002	10s to a few thousand feet	Constant	Relatively uniform
Microwave oven	2450 MHz	~50.05-0.2	2 inches2 feet	During use	Localized, non-uniform
Local area networks	2.4—5 GHz	0.0002—0.001 0.000005—0.0002	3 feet	Constant when nearby	Localized, non-uniform
Radio/TV broadcast	Wide spectrum	0.001 (highest 1% of population) 0.000005 (50% of population)	Far from source (in most cases)	Constant	Relatively uniform
Smart meter	900 MHz, 2400 MHz	0.0001 (250 mW, 1% duty cycle) 0.002 (1 W, 5% duty cycle) 0.000009 (250 mW, 1% duty cycle) 0.0002 (1 W, 5% duty cycle)	3 feet 10 feet	When in proximity during transmission	Localized, non-uniform

Source: Electric Power Research Institute (EPRI), Radio-Frequency Exposure Levels from Smart Meters: A Case Study of One Model (February 2011)

What is Duty Cycle and How Does it Relate to RF Exposure?

Duty cycle refers to the fraction of time a device is transmitting. For instance, a duty cycle of 1% means the device transmits RF energy 1% of a given time period. One percent of the time in a day is equivalent to 14.4 minutes per day. *The duty cycle, or signal duration is an often-overlooked factor when comparing exposures from different kinds of devices (e.g., mobile phones, Wi-Fi routers, smart meters, microwave ovens, FM radio/TV broadcast signals).*

Duty cycles of various devices vary considerably. The duty cycle of AM/FM radio/TV broadcasts, are 100%; in other words, they are transmitting continuously. Mobile phones usage varies widely from user to user, of course. However, the national average use is about 450 minutes per month. This usage equates to a 1% duty cycle for the "average" user.

From information that CCST was able to obtain we understand that the smart meter transmitter being used by PG&E operates with a maximum power output of 1 W (watt) and within the 902-928 MHz (mega-hertz) frequency band. Each smart meter is part of a broader "mesh" network and may act as a relay between other smart meters and utility access points. The transmitter at each smart meter will be idle some of the time, with the percent of time idle (not transmitting) depending on the amount and schedule of data transmissions made from each meter, the relaying of data from other meters that an individual meter does, and the networking protocol (algorithm) that manages control and use of the communications paths in the mesh network.

Theoretically the transmit time could increase substantially beyond today's actual operation level if new applications and functionality are added to the meter's communication module in the future. For a hypothetical illustration (i.e., the meter transmits half the time and receives half the time), an upper end duty cycle would be 50%,. The table below compares the effect of different duty cycles against the FCC guidelines for human exposure limits.

Typical Smart Meter Operation With Repeater Activity	**Scaled Hypothetical Maximum Use Case (i.e., always on)**
5% Duty Cycle	50% Duty Cycle
72 minutes/day	12 hours/day
3% of FCC limit	30% of FCC limit

Source data on operating duty cycles (i.e., first column) from Electric Power Research Institute (EPRI) actual field testing of smart meters, as reported in *Radio-Frequency Exposure Levels from Smart Meters: A Case Study of One Model*, February 2011. Second column hypothetical maximum case derived through extrapolation of first column data. Both exposure levels at 1-foot distance.

In summary, the duty cycles of smart meters in typical meter-read operation and added maximum-case repeater operation result in exposures that are 3% of the FCC exposure guidelines. Even in a hypothetical extreme and unusual case of half-transmit and half-receive scenario the maximum exposure would be about 30% of the FCC limit, which provides a wide safety margin from known thermal effects of RF emissions.

What About Exposure Levels from a Bank of Meters and from Just Behind the Wall of a Single Meter?

In a February 2011 study Electric Power Research Institute (EPRI)[36] field tested exposure levels from a bank of 10 meters of 250 mW power level at one foot distance in order to simulate a bank of smart meters located at a multifamily building, such as an apartment house. The exposure level was equivalent to 8% of the FCC standard.

In the same study EPRI measured exposure of one meter from eight inches *behind* the meter panel box in order to simulate proximity on the opposite site of the meter wall. At 5% duty cycle it yielded an exposure of only 0.03% of the FCC standard. Even at 100% duty cycle (i.e., always transmitting), exposure at eight inches behind the meter was 0.6% of the FCC limit.

Is the FCC Standard Sufficient to Protect Public Health?

The FCC guidelines do provide a significant factor of safety against thermal impacts the only currently understood human health impact that occurs at the power level and within the frequency band that smart meters use. In addition to the factor of safety built into the guidelines, at worst, human exposure to RF from smart meter infrastructure operating at even 50% duty cycle will be significantly lower than the guidelines. While additional study is needed to understand potential non-thermal effects of exposure to RF and effects of cumulative and prolonged exposure to several devices emitting RF, given current scientific knowledge the FCC guideline provides an adequate margin of safety against known RF effects.

Are Additional Technology-specific Standards Needed?

FCC guidelines protect against thermal effects of RF exposure. Many non-thermal effects have been suggested, and additional research is needed to better understand and scientifically validate them.

Given the scientific uncertainty around non-thermal effects of all RF emitting equipment, at this time there is no clear indication of what, if any, additional standards might be needed. Neither is there a basis from which to understand what types of standards could be helpful or appropriate**.** Without a clear understanding of the biological mechanisms at play, the costs and benefits of additional standards for RF emitting devices including smart meters, cannot be determined at this time.

[36] EPRI (2010) "A perspective on radio-frequency exposure associated with residential automatic meter reading technology," Electric Power Research Institute, February, 2011.

Public Information and Education

It is important that consumers have clear and easily understood information about smart meter emissions as well as readily available access to clear, factual information and education on known effects of RF emissions at various field strengths and distances from an array of devices commonly found in our world.

Equipped with this information, people can make knowledgeable judgments about how to prudently minimize possible risks to themselves and their families by utilizing standards-compliant devices at known safe distances. Also, people will be better able to gauge relative field strengths of various RF sources in our everyday environment (e.g., mobile phones, electric blankets, clock radios, TV and radio, computers, smart meters, power lines, microwave ovens, etc.). An ongoing regularly updated source of unbiased information on the state of scientific research, both proven and as-yet-unproven causal effects being studied, if presented by an independent entity, would provide consumers a credible and transparent source from which to obtain facts about RF in our environment.

CCST is not currently aware of a single website with up-to-date consumer information which we are able to endorse as impartial.

Alternatives to Wireless?

Assembly Member Huffman has inquired about potential alternatives to wireless communication with smart meters. There are currently several other methods of transmitting data from some smart meters to the utility company. These methods include transmitting over a power line or wired through phone lines, fiber-optic or coaxial cable. Each method has tradeoffs among cost and performance (e.g., how much data can be carried, how far, how fast). The ability to have a transmission protocol alternative to wireless depends upon the type and configuration of the meter used. Some existing smart meters can be hard-wired, while others would have to be modified or replaced. The communications board plugs into a digital meter. The current PG&E meters use a SilverSpring communications board that only supports wireless protocol. SilverSpring or another vendor could provide an alternative communications means if such were warranted and cost effective. The related costs of an alternative approach would need to be factored into the decision making process related to different options.

If future research were to establish a causal relationship between RF emissions and negative human health impacts, industries and governments worldwide may be faced with difficult choices about practical alternatives to avoid and mitigate such effects. This would greatly affect the widespread use of mobile phones, cordless phones, Wi-Fi devices, smart meters, walkie-talkies, microwave ovens, and many other everyday appliances and devices emitting RF. If such a hypothetical scenario were to occur, smart meters could conceivably be adapted to non-wireless transmission of data. However, retrofitting millions of smart meters with hard-wired technology could be difficult and costly. Perhaps more importantly, retrofitting smart

meters would not address the significantly greater challenge presented by the billions of mobile phones in use globally.

Key Factors to Consider When Evaluating Exposure to Radiofrequency from Smart Meters

1. Signal Frequency	Compare to devices in the 900 MHz band and 2.4 GHz band	Frequency similar to mobile phones, Wi-Fi, laptop computers, walkie-talkies, baby monitors, microwave ovens
2. Signal Strength (or Power Density)	Microwatts/square centimeter ($\mu W/cm^2$)	Meter signal strength very small compared to other devices listed above
3. Distance from Signal	Signal strength drops rapidly (doubling distance cuts power density by four)	Example: 1 ft. – 8.8 $\mu W/cm^2$ 3 ft. – 1.0 $\mu W/cm^2$ 10 ft. – 0.1 $\mu W/cm^2$
4. Signal Duration	- Extremely short amount of time (2.0-5.0%, max.) - No RF signal 95-98% of the time (over 23 hours/day)	- Often overlooked factor when comparing devices. - Short duration combined with weak signal strength yields tiny exposures
5. Thermal Effects	- Scientific consensus on proven effects from heat at high RF levels	- FCC "margin-of-safety" limits 50 times lower than hazardous exposure level - Typical meter operates at 70 times less than FCC limit and 3,500 times less than the demonstrated hazard level
6. Non-thermal Effects	- Inconclusive research to date - No established cause-and-effect pointing to negative health impacts	Continuing research needed

Conclusion

The CCST Project Team, after carefully reviewing the available literature on the current state of science on health impacts of radiofrequency from smart meters and input from a wide array of subject matter experts, concludes that:

1. **The FCC standard provides a currently accepted factor of safety against known thermally induced health impacts of smart meters and other electronic devices in the same range of RF emissions. Exposure levels from smart meters are well below the thresholds for such effects.**

2. **There is no evidence that additional standards are needed to protect the public from smart meters.**

The topic of potential health impacts from RF exposure in general, including the small RF exposure levels of smart meters, continues to be of concern. This report has been developed to provide readers and consumers with factual, relevant information about the:

- Scientific basis underpinning current RF limits
- Need for further research into RF effects
- Relative nature of RF emissions from a wide array of devices commonly used throughout world (e.g., cellular and cordless phones, Wi-Fi devices, laptop computers, baby monitors, microwave ovens).

CCST encourages the ongoing development of unbiased sources of readily available and clear facts for public information and education. A web-based repository of written reports, frequently asked questions and answers, graphics, and video demonstrations would provide consumers with factual, relevant information with which to better understand RF effects in our environment.

Appendix A – Letters Requesting CCST

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Assembly
California Legislature

JARED HUFFMAN
ASSEMBLYMEMBER, SIXTH DISTRICT

COMMITTEES
CHAIR, WATER, PARKS AND WILDLIFE
NATURAL RESOURCES
UTILITIES AND COMMERCE

SUBCOMMITTEE NO.3
ON RESOURCES

July 30, 2010

Karl Pister, Chair
Susan Hackwood, Executive Director
California Council on Science and Technology
1130 K Street, Suite 280
Sacramento, CA 95814-3965

Dear Chair Pister and Ms. Hackwood:

I am writing to request a study by the California Council on Science and Technology in response to the many concerns and questions that have been raised by constituents in my Assembly District including the Marin County Board of Supervisors, City of Sebastopol, City of Fairfax, and Marin Association of Realtors relating to potential negative health effects from SmartMeters, the electronic monitoring devices that Pacific Gas and Electric Company (PG&E) is installing statewide to continuously measure the electricity output from each household and business.

SmartMeters are currently being installed throughout the state under the authority of the California Public Utilities Commission (CPUC) pursuant to a series of decisions that span from 2006 through 2009. The authority for PG&E to deploy SmartMeters in its territory is embodied in two decisions: D.06-07-027 (the initial deployment) and D.09-03-026 (the upgrade). On the question of health effects of radiation from the devises, PG&E and CPUC maintain that electromagnetic fields emitted from these SmartMeters and the radio frequency power associated with the wireless radios fall within the Federal Communications Commission's (FCC) regulations, pointing out that SmartMeters emit fewer radio frequencies than the amount allowable for cellular telephones, microwave ovens, and wireless Internet Services.

Critics claim, among other things, that FCC standards are not sufficiently protective of public health and do not take into account the cumulative effect of radiation exposure from a growing number of sources and devices, including continuous exposure from some sources. For example, they cite a letter from the Radiation Protection Division of the Environmental Protection Agency (attached), they argue, ..."these standards were thermally based and do not apply to chronic, nonthermal exposure situations, ... and that … the current exposure guidelines are based on the effects resulting from whole-body heating, not exposure of and effect on critical organs including the brain and the eyes." Therefore, they argue the "safety" standards were not designed to protect the public from health problems under the circumstances which the meters are being used.

An independent, science-based study by the California Council on Science and Technology would help policy makers and the general public resolve the debate over whether SmartMeters present a significant risk of adverse health effects. Toward that end, I request that the Council specifically determine whether FCC standards for SmartMeters are sufficiently protective of public health taking into account current exposure levels to radiofrequency and electromagnetic fields, and further to assess whether additional technology specific standards are needed for SmartMeters and other devises that are commonly found in and around homes, to ensure adequate protection from adverse health effects.

Thank you for your serious consideration of this important and time-sensitive request. Please do not hesitate to contact me if I can be of assistance going forward

Sincerely,

JARED HUFFMAN
Assemblymember, 6th District

COMMITTEES
CHAIR, HEALTH
ARTS, ENTERTAINMENT, SPORTS, TOURISM & INTERNET MEDIA
ENVIRONMENTAL SAFETY & TOXIC MATERIALS
JOINT LEGISLATIVE AUDIT COMMITTEE
JUDICIARY
LABOR AND EMPLOYMENT
WEBSITE: www.assembly.ca.gov/monning

Assembly
California Legislature

WILLIAM W. MONNING
ASSEMBLYMEMBER, TWENTY-SEVENTH DISTRICT

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MONTEREY, CA 93940
(831) 649-2832
(831) 649-2935

SANTA CLARA COUNTY DIRECT LINE
(408) 782-0647

September 15, 2010

Karl Pister, Chair
California Council on Science and Technology
1130 K Street, Suite 280
Sacramento, CA 95814-3965

Dear Chair Pister:

This letter is to formally request that I be included in the response from the California Council on Science and Technology (CCST) regarding the health safety evaluation of the new electronic metering devices, otherwise known as Smart Meters, currently being installed by Pacific Gas and Electric Company (PG&E) which will be available by October 15, 2010.

Numerous concerns and questions have been raised by PG&E customers throughout the state, as well as local government entities such as the County of Santa Cruz, the City of Capitola, City of Santa Cruz, City of Scotts Valley, and the City of Watsonville, relating to potential health effects of the radio frequency (RF) emitted from Smart Meters.

As you know, the federal Energy Independence and Security Act of 2007 required each state to initiate a smart grid system. In response to this federal mandate, the State of California enacted Senate Bill 17, Chapter 327, Statutes of 2009, granting the California Public Utilities Commission (CPUC) smart grid oversight authority. While the CPUC has authorized PG&E to install their current Smart Meter system, CPUC has not addressed the question of whether the RF emissions from Smart Meter devices have potential health impacts.

While PG&E maintains that Smart Meters comply with the Federal Communications Commission (FCC) safety standards, there is still public concern that the FCC standards do not sufficiently protect the public's health and do not take into account the cumulative effect of radiation exposure from the growing number of sources and devices emitting RF.

The scientific evaluation by the California Council on Science and Technology will help to inform both elected officials and the public about the safety of PG&E's Smart Meters and I appreciate the Council taking the time to assess this very important issue.

Thank you for your time and assistance on this issue.

Sincerely,

WILLIAM W. MONNING
Assemblymember, 27th District

WWM:rog

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MILL VALLEY

September 20, 2010

Karl Pister, Chair
Susan Hackwood, Executive Director
California Council on Science and Technology
1130 K Street, Suite 280
Sacramento, CA 95814-3965

Dear Chair Pistel and Ms. Hackwood:

On behalf of the Mill Valley City Council, I am writing to support Assemblymember Jared Huffman's request for a study by the California Council on Science and Technology (CCST) to specifically determine whether Federal Communications Commission (FCC) standards for Pacific Gas and Electric (PG&E) SmartMeters are sufficiently protective of public health.

This request is in response to the many concerns and questions that have been raised by Mill Valley residents relating to potential negative health effects from SmartMeters. Mill Valley residents have expressed their concerns that these devices, which are regulated by the California Public Utilities Commission (CPUC), emit levels of radiation that may be harmful to public health, especially with consideration to the long-term and cumulative impacts of the devices. The CPUC maintains that SmartMeters emit radiation well below the FCC-established safety standards, and have therefore not ordered PG&E to halt the installation of the advanced metering devices.

Critics argue that the safety standards determined by the FCC are not sufficient and specifically not designed to protect the public from health problems under the circumstances which the meters will be used. The FCC standards, they claim, do not take into consideration long-term and cumulative exposures to these devices.

The City of Mill Valley City Council therefore join Assemblymember Huffman in requesting the CCST undertake a study to specifically determine whether FCC standards for SmartMeters are sufficiently protective of public health, taking into account current exposure levels to radiofrequency and electromagnetic fields, and further to assess whether additional technology

City of Mill Valley, 26 Corte Madera Avenue, Mill Valley, California 94941 • 415-388-4033

specific standards are needed for SmartMeters and other devices that are commonly found in and around homes, to ensure adequate protection from adverse health effects.

Thank you for your consideration.

Sincerely,

Stephanie Moulton-Peters, Mayor
City of Mill Valley

Cc: Mill Valley City Council
Assemblymember Jared Huffman
Joshua Townsend, PG&E Public Affairs Manager
Marzia Zafar, CPUC Business and Community Outreach Division Manager

Appendix B – Project Process

CCST Smart Meter Project Approach

Assembly Member Huffman (Marin) (July 30, 2010 letter) and Assembly Member Monning (Santa Cruz) (September 17, 2010 letter) requested CCST's assistance in determining if there are health safety issues regarding the new SMART meters being installed by the utilities. In addition, the City of Mill Valley sent a letter to CCST (September, 2010) in support of Mr. Huffman's request. (Appendix A - letters)

The CCST Executive Committee appointed a Smart Meter Project Team that oversaw the development of a response on the issue (Appendix C):

- Rollin Richmond (Chair), President Humboldt State University, CSU
- Jane Long, Associate Director at Large, Global Security Directorate Fellow, Center for Global Security Research Lawrence Livermore National Laboratory
- Emir Macari, Dean of Engineering and Computer Science, California State University, Sacramento and Director of the California Smart Grid Center
- Patrick Mantey, Director, CITRIS @ Santa Cruz
- Ryan McCarthy, 2009 CCST Science and Technology Policy Fellow
- Larry Papay, CEO, PQR, LLC, mgmt consulting firm
- David Winickoff, Assistant Professor of Bioethics and Society, Department of Environmental Science, Policy and Management, UC Berkeley
- Paul Wright, Director, UC Center for Information Technology Research in the Interest of Society (CITRIS)

In addition to those on the project team, CCST approached over two dozen technical experts to contribute their opinion to inform CCST's response. The experts were referred from a variety of sources and were vetted by the Smart Meter Project Team. Efforts were made to include both biological and physical scientists and engineers to help provide broad context and perspective to the response. Many of the experts approached indicated they did not time to provide a written response however they provided references to additional experts and/or literature for review. A few experts identified were not asked to contribute due to affiliations that were felt to be a conflict of interest. Experts were asked to provide written comment on two issues, to provide referral to other experts, and to suggest literature that should be reviewed. Appendix D provides a list of those experts who provided written comment.

Smart Meter Project Team members and the experts providing written technical input completed a conflict of interest disclosure form to reveal any activities that could create the potential perception of a conflict.

In addition to written and oral input from technical experts, CCST identified relevant reports and other sources of information to inform the final report. This material can be found listed in Appendix E and on a CCST website: http://ccst.us/projects/smart/.

Peer Review: After the draft report was vetted in great detail by the Smart Meter Project Team, it was forwarded to the CCST Board and Council for peer review.

Public Comment: Comments on the January 2011 draft of this report were solicited from the public. The report was posted to the CCST website to allow the general public to easily comment. Many very thoughtful and informed comments were received. All public comments were reviewed and taken into consideration as this final report was completed.

Appendix C – Project Team

The California Council on Science and Technology adheres to the highest standards to provide independent, objective, and respected work. Board and Council Members review all work that bears CCST's name. In addition, CCST seeks peer review from external technical experts. The request for rigorous peer review results in a protocol that ensures the specific issue being addressed is done so in a targeted way with results that are clear and sound.

In all, this report reflects the input and expertise of nearly 30 people in addition to the project team. Reviewers include experts from academia, industry, national laboratories, and non-profit organizations.

We wish to extend our sincere appreciation to the project team members who have helped produce this report. Their expertise and diligence has been invaluable, both in rigorously honing the accuracy and focus of the work and in ensuring that the perspectives of their respective areas of expertise and institutions were taken into account. Without the insightful feedback that these experts generously provided, this report could not have been completed.

Rollin Richmond, Smart Meter Project Chair, CCST Board Member
President Humboldt State University, CSU

Prior to Richmond's appointment at Humboldt State University in 2002, he had a distinguished career as a faculty member, researcher in evolutionary biology and academic administrator. Richmond received a Ph.D. in genetics from the Rockefeller University and a bachelor's degree in zoology from San Diego State University. Dr. Richmond's career has included: Chairperson of biology at Indiana University, founding Dean of the College of Arts and Sciences at the University of South Florida, Provost at the State University of New York at Stony Brook, and Provost and Professor of Zoology and Genetics at Iowa State University. He was named the sixth President of Humboldt State University in July of 2002. Dr. Richmond is a fellow of the American Association for the Advancement of Science and a member of Phi Beta Kappa. His research interests are in evolutionary genetics.

Jane Long, CCST's California's Energy Future Project Co-Chair and CCST Sr. Fellow
Associate Director at Large, Global Security Directorate Fellow, Center for Global Security Research Lawrence Livermore National Laboratory

Dr. Long is the Principal Associate Director at Large for Lawrence Livermore National Laboratory working on energy and climate. She is also a Fellow in the LLNL Center for Global Strategic Research. Her current interests are in reinvention of the energy system in light of climate change, national security issues, economic stress, and ecological breakdown. She holds a bachelor's degree in engineering from Brown University and Masters and Ph.D. from UC Berkeley.

Patrick Mantey

Director, UC Center for Information Technology Research in the Interest of Society (CITRIS) @ Santa Cruz, University of California, Santa Cruz

Mantey holds the Jack Baskin Chair in Computer Engineering and was the founding Dean of the Jack Baskin School of Engineering. He is now the director of CITRIS at UC Santa Cruz and of ITI, the Information Technologies Institute in the Baskin School of Engineering. In 1984, he joined the UCSC faculty to start the engineering programs, coming from IBM where he was a senior manager at IBM Almaden Research. His research interests include system architecture, design, and performance, simulation and modeling of complex systems, computer networks and multimedia, real-time data acquisition, and control systems. Mantey is a Fellow of the Institute of Electrical and Electronics Engineers. His current projects at CITRIS include the Residential Load Monitoring Project and work on power distribution system monitoring and reliability. Mantey received his B.S. (magna cum laude) from the University of Notre Dame, his M.S. from the University of Wisconsin-Madison, and his Ph.D. from Stanford University, all in electrical engineering. He is a Fellow of the Institute of Electrical and Electronics Engineers (IEEE).

Emir José Macari

Dean of Engineering and Computer Science, California State University, Sacramento and Director of the California Smart Grid Center

Prior to his appointment as dean at CSU Sacramento, Macari was dean of the College of Science, Mathematics and Technology at the University of Texas at Brownsville. Prior to that, he served as the program director for the Centers of Research Excellence in Science and Technology at the National Science Foundation. From 1999-2001 he served as the Chair and Bingham C. Stewart Distinguished Professor in the Department of Civil and Environmental Engineering at Louisiana State University. At the Georgia Institute of Technology he taught both engineering and public policy and at the University of Puerto Rico he was a professor and director of Civil Infrastructure Research Center. He has also worked as a civil engineer in private industry and has been a fellow at NASA. Macari holds both a doctorate and a master's degree in civil engineering geomechanics from the University of Colorado. He has a bachelor's degree in civil engineering geomechanics from Virginia Tech University.

Larry Papay CCST Board Member

CEO, PQR, LLC, mgmt consulting firm

Papay is currently CEO and Principal of PQR, LLC, a management consulting firm specializing in managerial, financial, and technical strategies for a variety of clients in electric power and other energy areas. His previous positions include Sector Vice President for the Integrated Solutions Sector, SAIC; Senior Vice President and General Manager of Bechtel Technology & Consulting; and Senior

Vice President at Southern California Edison. Papay received a B.S. in Physics from Fordham University, a M.S. in Nuclear Engineering from MIT, and a Sc.D. in Nuclear Engineering from MIT. He is a member of the National Academy of Engineering and served on its Board of Councilors from 2004-2010. He served as CCST Council Chair from 2005 through 2008, after which he was appointed to the Board.

David E Winickoff

Associate Professor of Bioethics and Society, Department of Environmental Science, Policy and Management, UC Berkeley

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McCarthy recently completed the CCST Science and Technology Policy Fellowship in the office of California Assembly Member Wilmer Amina Carter, where he advised on issues associated with energy, utilities, and the environment, among others. McCarthy holds a master and doctorate degree in civil and environmental engineering from UC Davis, and a bachelor's degree in structural engineering from UC San Diego. His expertise lies in transportation and energy systems analysis, specifically regarding the electricity grid in California and impacts of electric vehicles on energy use and emissions in the state.

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Asher Sheppard, Ph.D., Asher Sheppard Consulting, trained in physics, environmental medicine, and neuroscience
Magda Havas, B.Sc., Ph.D., Environmental & Resource Studies, Trent University, Peterborough, Canada
Cindy Sage, MA, Department of Oncology, University Hospital, Orebro, Sweden and Co-Editor, BioInitiative Report

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- stopsmartmeters.org

Appendix F – Glossary

Access point - A term typically used to describe an electronic device that provides for wireless connectivity via a WAN to the Internet or a particular computer facility.

Duty cycle – A measure of the percentage or fraction of time that an RF device is in operation. A duty cycle of 100% corresponds to continuous operation (e.g., 24 hours/day). A duty cycle of 1% corresponds to a transmitter operating on average 1% of the time (e.g., 14.4 minutes/day).

Electromagnetic field (EMF) - A composition of both an electric field and a magnetic field that are related in a fixed way that can convey electromagnetic energy. Antennas produce electromagnetic fields when they are used to transmit signals.

Far-field - A distance which extends from about two wavelengths distance from the antenna to infinity, is the region in which the field acts as "normal" electromagnetic radiation. The power of this radiation decreases as the square of distance from the antenna. By contrast, the **near-field**, which is inside about one wavelength distance from the antenna, is a region in which there are effects from the currents and charges in the antenna, which do not behave like far-field radiation. These effects decrease in power far more quickly with distance, than does the far-field radiation power.

Federal Communications Commission (FCC) - The Federal Communications Commission (FCC) is an independent agency of the US Federal Government and is directly responsible to Congress. The FCC was established by the Communications Act of 1934 and is charged with regulating interstate and international communications by radio, television, wire, satellite, and cable. The FCC also allocates bands of frequencies for non-government communications services (the NTIA allocates government frequencies). The guidelines for human exposure to radio frequency electromagnetic fields as set by the FCC are contained in the Office of Engineering and Technology (OET) Bulletin 65, Edition 97-01 (August 1997). Additional information is contained in OET Bulletin 65 Supplement A (radio and television broadcast stations), Supplement B (amateur radio stations), and Supplement C (mobile and portable devices).

Gigahertz (GHz) - One billion Hertz, or one billion cycles per second, a measure of frequency.

Hertz - The unit for expressing frequency, one Hertz (Hz) equals one cycle per second.

Maximum permissible exposure (MPE) limit. An exposure limit or guideline for RF energy exposure published by a recognized consensus standards organization.

Megahertz (MHz) - One million Hertz, or one million cycles per second, a unit for expressing frequency.

Mesh network - A network providing a means for routing data, voice and instructions between nodes. A mesh network allows for continuous connections and reconfiguration around broken or blocked data paths by “hopping” from node to node until the destination is reached.

Milliwatt per square centimeter (mW/cm^2) - A measure of the power density flowing through an area of space, one thousandth (10^{-3}) of a watt passing through a square centimeter.

Microwatt per square centimeter (µW/cm^2) - A measure of the power density flowing through an area of space, one millionth (10^{-6}) of a watt passing through a square centimeter.

Radiofrequency (RF) - The RF spectrum is formally defined in terms of frequency as extending from 0 to 3000 GHz, the frequency range of interest is 3 kHz to 300 GHz.

Repeater unit - A device that can simultaneously receive a radio signal and retransmit the signal. Repeater units are used to extend the range of low power transmitters in a geographical area.

Router - An electronic computer device that is used to route and forward information, typically between various computers within a local area network or between different local area networks.

Smart meter - A digital device for measuring consumption, such as for electricity and natural gas, and sending the measurement to a utility company. Automated meter reading (AMR) meters send information one-way only. Automated meter infrastructure (AMI) meters are capable of two-way communications.

Specific absorption rate (SAR) - The incremental energy absorbed by a mass of a given density. SAR is expressed in units of watts per kilogram (or milliwatts per gram, mW/g).

Transmitter - An electronic device that produces RF energy that can be transmitted by an antenna. The transmitted energy is typically referred to a radio signal or RF field.

Wide area network (WAN) - A computer network that covers a broad area such as a whole community, town, or city. Commonly, WANs are implemented via a wireless connection using radio signals. High-speed Internet connections can be provided to customers by wireless WANs.

Wi-Fi - An name given to the wireless technology used in home networks, mobile phones, and other wireless electronic devices that employ the IEEE 802.11 technologies (a standard that defines specific characteristics of wireless local area networks).

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A Discussion of Smart Meters And RF Exposure Issues

An EEI-AEIC-UTC White Paper

A Joint Project of the EEI and AEIC Meter Committees

March 2011

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Published by:
Edison Electric Institute
701 Pennsylvania Avenue, N.W.
Washington, D.C. 20004-2696
Phone: 202-508-5000
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Table of Contents

Acknowledgements

This report was conducted with the oversight of the Edison Electric Institute (EEI) and Association of Edison Illuminating Companies (AEIC) Metering Committees and the Smart Meter Report Project Team. We would like to acknowledge the contributions of the core team consisting of:

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Technical Contributor	Tim Morgan	Duke Energy
Technical Contributor	Larry Barto	Georgia Power Company

We would like to give special thanks to the leadership of the EEI and AEIC Meter and Service Committees for the support necessary to make this project a reality. We also thank the many EEI & AEIC members, vendors, and other meter professionals who provided input and feedback towards the completion of this report. The report could not have been completed without the input this group so generously provided.

1: Introduction

The following industry discussion of Smart Meters and Radio Frequency (RF) Issues was prepared by the member company representatives from the following organizations.

Edison Electric Institute (EEI)

The Edison Electric Institute (EEI) is the association of U.S. Shareholder-Owned Electric Companies. Our members serve 95 percent of the ultimate customers in the shareholder-owned segment of the industry, and represent approximately 70 percent of the U.S. electric power industry.
Organized in 1933, EEI works closely with all of its members, representing their interests and advocating equitable policies in legislative and regulatory arenas.

The Association of Edison Illuminating Companies (AEIC)

AEIC was founded by Thomas Edison and his associates in 1885. AEIC encourages research and the exchange of technical information through a committee structure, staffed with experts from management of member companies.

AEIC's members are electric utilities, generating companies, transmitting companies, and distributing companies – including investor-owned, federal, state, cooperative and municipal systems – from within and outside the United States. Associate members include organizations responsible for technical research and for promoting, coordinating and ensuring the reliability and efficient operation of the bulk power supply system.

AEIC's Six Technical Committees are: Load Research, Meter and Service, Power Apparatus, Power Delivery, Power Generation and Cable Engineering. AEIC also provides highly valued literature on load research and underground cable specifications and guidelines.

Utilities Telecom Council

The Utilities Telecom Council (UTC) is a global, full-service trade association dedicated to creating a favorable business, regulatory, and technological environment for members. Founded in 1948, UTC has evolved into a dynamic organization that represents the broad communications interests of electric, gas, and water utilities; natural gas pipelines; other critical infrastructure entities and other industry stakeholders. Visit www.utc.org for more information on UTC and its services.

2. Executive Summary

Smart Meters and Smart Meter Systems are being deployed throughout North America, and utilities are continuing their efforts to improve grid reliability and promote energy efficiency while providing improved services to their customers. However, concerns have been raised regarding the potential impacts of radio frequency exposure from these meters on the public. The purpose of this paper is to give an overview of the issues raised recently concerning RF exposure due to the deployment of Smart Meter and Smart Meter Systems. The paper provides a basic overview for understanding how the electric utility industry seeks to ensure the appropriate level of accuracy and safety. It also makes evident that before being accepted and deployed Smart Meters must meet a number of national standards and comply with state and local codes designed to ensure proper operation, functionality and safety.

Radio Frequency (RF) Exposure

Several Smart Meter Systems operate by transmitting information wirelessly. This has raised some concern about the health effects of wireless signals on electric consumers and the general public. Accordingly, this document explains that the RF exposures of Smart Meters are lower compared to other common sources in the home and operate significantly below Federal Communications Commission (FCC) exposure limits.[1] The paper discusses how the location, distance from the transmitter, shielding by meter enclosures, attenuation of building materials, direction of RF emissions, and transmit duty cycle significantly reduce exposure to consumers. It also includes a review of the conclusions of several Smart Meter RF studies and actual measurements of Smart Meter RF emissions to support this conclusion. Other observations include:

- All smart meter radio devices must be certified to the FCC's rules.
- Tests simulating multi-family metering locations containing several meters in close proximity have shown RF exposure levels dramatically less than FCC limits.
- The FCC limits on maximum permissible exposure (MPE) for application to the general public were set using safety factors fifty times lower than the levels of known effects.
- Exposure levels drop significantly (1) with the distance from the transmitter, (2) with spatial averaging, and (3) in living spaces due to the attenuation effects of building materials.
- Due to shielding of the meter enclosure and signal patterns, RF exposure from the rear of a metering location is nominally 10 times less than in front of the meter and dramatically below FCC limits, not including the spatial averaging and building material attenuation reductions.[2]
- For measurement and calculation purposes some studies use a 100% duty cycle parameters. However, the maximum operational Duty Cycle for Smart meter systems is less than 50% to prevent message traffic congestion and collisions. The typical Duty Cycles for Smart Meter Systems is between 1% and 5%.

[1] See Section 5, RF Exposure in Smart Meter Systems

[2] "An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter", EPRI (2010),December 2010

- An RF exposure comparison of a person talking on a cell phone and a person 3 and 10 feet from a continuously operating Smart Meter would result in Smart Meter RF exposure 125 to 1250 times less than the cell phone.[3]
- In test environments simulating operational conditions, for power (250 mWatt - 2 Watt), duty cycle (2%-5%) at close distance (1 foot) from in front of the transmitter, Smart Meters produce very low RF exposure to the consumer, typically well under 10 % of the FCC exposure regulations.

Additionally, before utilities accept and deploy Smart Meters, these devices must meet a number of national standards and comply with state and local codes designed to ensure proper operation, functionality and safety. In particular, Smart Meter and Smart Meter installations are typically designed to conform with and certified to comply with: [4]

- *ANSI C12 .1, 12.10, and 12.20* standards for accuracy and performance
- *NEMA SG-AMI 1 "Requirements for Smart Meter Upgradeability"*
- FCC standards for intentional and unintentional radio emissions and safety related to RF exposure, *Parts 1 and 2 of the FCC's Rules and Regulations [47 C.F.R. 1.1307(b), 1.1310, 2.1091, 2.1093]*.
- Local technical codes and requirements
- Utility specific and customer beneficial business and technical requirements

The electric utility industry is continuously developing standards and guidelines to improve the safety, accuracy and operability of meters and associated metering devices. An example of these continuing improvement is *NEMA SG-AMI 1 "Requirements for Smart Meter Upgradeability"* released in September 2009 to support the needs of developing the Smart Grid.

Finally, the paper discusses how manufacturers conduct complete performance and life cycle testing for all meter types and for major design changes to existing meter types, including hardware and firmware. Once the testing is successfully completed, the Smart Meter System components are utility or third party certified for production and purchase. Furthermore, after certification and purchasing, the paper discusses the utility materials acceptance process to evaluate each shipment of equipment for quality and compliance to specification. Completion of this process by utilities allows for receipt of equipment for field installation.

The deployment of a Smart Meter System begins with selection of the technology and the planning for installation, operation and maintenance. Utilities have integrated within the deployment process many elements of management, control and compliance to support successful project implementation.

[3] "*Health Impacts of Radio Frequency (RF) from Smart Meters*"; California Council on Science and Technology (CCST); January 2011; page 20

[4] This list is not exhaustive, and there may be other sets of rules/standards/requirements not reflected but applicable.

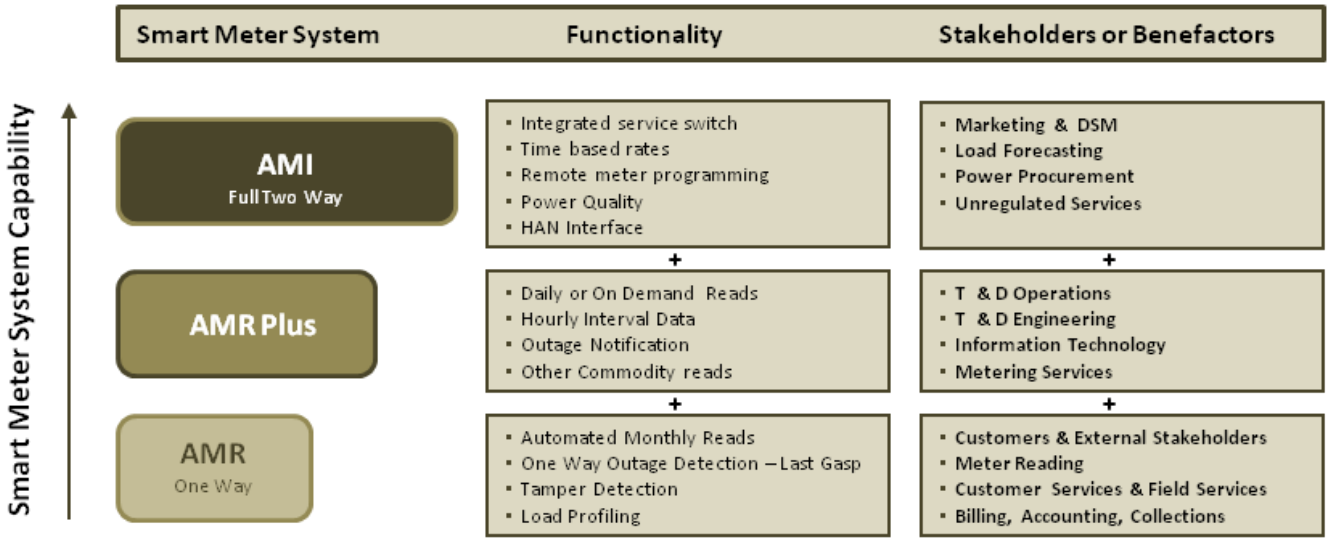
3. An Overview of Smart Meters and Smart Meter Systems

3.1 Definition of Smart Meter and Smart Meter Systems

Smart Meters are electronic measurement devices used by utilities to remotely communicate information for billing customers and operating their electric systems. For over fifteen years electronic meters have been used effectively by utilities in delivering accurate billing data for at least a portion of their customer base. Initially, the use of this technology was applied to commercial and industrial customers due to the need for more sophisticated rates and more granular billing data requirements. The use of electronic meters came into service to the largest customers of the utility and over time gradually expanded to all customer classes. This migration was made possible by decreasing cost of the technology and advanced billing requirements for all customer classes.

The combination of the electronic meters with two-way communications technology for information, monitor and control is commonly referred to as Advanced Metering Infrastructure (AMI). Previous systems which utilized one way communication and were referred to as AMR (Automated Meter Reading) Systems. AMI has developed over time, from its roots as a meter reading substitute (AMR) to today's two-way communication and data system. The evolution from AMR to AMI is shown in Figure 1 with lists of stakeholders and benefactors for each step in the Smart Meter evolution.[5]

Figure 1 – Smart Meter Technology Evolution



[5] Note: All functionality and stakeholder interests are additive, progressing up the chart

Not until the Smart Grid initiatives were established did AMI meters and systems become referred to as "Smart Meters and Smart Meter Systems". Thus, the present state of these technologies should be more appropriately referred to as "an evolution, not a revolution" because of the development and use of Smart Meter technology and communications over the last fifteen years. The combined technologies are also required to meet national standards for accuracy and operability essential in the industry.

Although the Smart Meters are relatively new to the utility industry, they have still been treated with the same due diligence and scrutiny associated with the older electro- mechanical counterparts. These meters have always met or exceeded national standards such as American National Standards Institute (ANSI) C12.1 for meter accuracy and performance. Another quality control is that equipment used to certify meter performance must be traceable to the National Institute of Standards and Technology (NIST), a federal technology agency that works with industry to properly apply technology and measurements.

Other standards in use for the Smart Meter installations include National Electric Code (NEC) for home electrical wiring, National Electrical Manufacturers Association (NEMA) and Underwriters Laboratories (UL) for meters, enclosures and devices, and National Electric Safety Code (NESC) for utility wiring. Through the leadership of utility metering professionals and metering manufacturers, the meticulous and deliberate development of these solid state electronic measurement devices has resulted in meter products that have advanced functionality, are stable and have tighter accuracy tolerances, and are more cost effective for advanced features than the legacy mechanical technologies.

3.2 Smart Meter System Benefits

The benefits of Smart Metering installations are numerous for many different Stakeholders of the systems. The table below lists some of the major benefits for utility stakeholders.

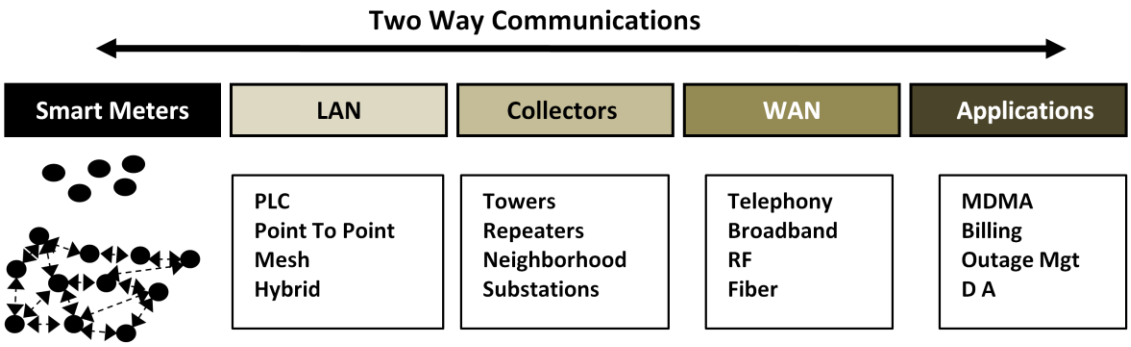
Stakeholder	Benefits
Utility Customers	• Better access and data to manage energy use • More accurate and timely billing • Improved rate options • Improved outage restoration • Power quality data
Customer Service & Field Operations	• Reduced cost of Meter reading • Reduced trips for off-cycle reads • Eliminates handheld meter reading equipment • Reduced call center transactions • Reduced collections and connects/disconnects
Revenue Cycle Services - **Billing, Accounting, Revenue Protection**	• Reduced back office rebilling • Early detection of meter tampering and theft • Reduced estimated billing and billing errors
Transmission and Distribution	• Improved transformer load management • Improved capacitor bank switching • Data for improved efficiency, reliability of service, losses, and loading • Improved data for efficient grid system design • Power quality data for the service areas
Marketing & Load Forecasting	• Reduced costs for collecting load research data

Stakeholder	Benefits
Utility General	• Reduced regulatory complaints • Improved customer premise safety & risk profile • Reduced employee safety incidents
External Stakeholders	• Improved environmental benefits • Support for the Smart Grid initiatives

3.3 Smart Meter Technologies

Smart Meter Systems are varied in technology and design but operate through a simple overall process. The Smart Meters collect data locally and transmit via a Local Area Network (LAN) to a data collector. This transmission can occur as often as 15 minutes or as infrequently as daily according to the use of the data. The collector retrieves the data and may process it or simply pass it on for processing upstream. Data is transmitted via a Wide Area Network (WAN) to the utility central collection point for processing and use by business applications. Since the communications path is two way, signals or commands can be sent directly to the meters, customer premise or distribution device. The utility selects the best technology to meet its demographic and business needs. Figure 3 shows the basic architecture of Smart Meter System operations.

Figure 3: Smart Meter System Basic Architecture



4. Deployment of Smart Meter Systems

4.1 Meter and System Certification & Acceptance

A plan to certify the meters and other system components for purchase and installation is essential to the deployment of the Smart Meter System. The technical requirements developed by the utility will include the Smart Meter equipment specifications for meeting national standards for safety, accuracy, compliance, and functionality criterion.

Smart Meter hardware to be certified must be production units and must conform to or exceed:[6]

- Federal Communications Commission (FCC) standards for intentional and unintentional radio emissions, and safety related to RF exposure, *parts 1 and 2 of the FCC's Rules and Regulations [47 C.F.R. 1.1307(b), 1.1310, 2.1091, 2.1093*.
- *ANSI C12 .1, 12.10, and 12.20* standards for meter accuracy and performance
- Local technical codes and requirements
- A functional test designed to verify the compliance to utilities technical and business requirements
- Utility specified requirements that are expected to exceed the standards. Examples:
  - Higher surge requirements for areas with lightning issues
  - Stainless steel enclosures for close seaside locations

The electric utility industry is continuously developing standards and guidelines to improve the safety, accuracy and functionality of meters and associated metering devices. An example of these continuing improvements is the release of *NEMA SG-AMI 1 "Requirements for Smart Meter Upgradeability" published in September 2009* in conjunction with NIST and Smart Grid Interoperability Panel (SGIP).

Complete performance testing is done by manufacturers and utilities for all meter types and for major design changes to existing meter types, including hardware and firmware. Once the testing is completed successfully, the Smart Meter System components are certified for production and purchase.

After certification and purchasing, the utility establishes a materials acceptance process to evaluate each shipment of equipment for quality and compliance with specification. The acceptance plan is usually a combination of vendor manufacturing test data and a sample test plan designed by the utility to meet its risk criteria. In addition to testing items included in the certification phase, other items may be evaluated. These may include items such as binding of the communication module to the meter, accuracy of the face plate data and data format, and quality of the meter data received, etc. Completion of this process allows receipt of equipment for field installation.

[6] This list is not exhaustive, and there may be other sets of rules/standards/requirements not reflected but applicable.

4.2 Smart Meter Installation

The planning for the installation of Smart Meters is just as important as the actual installation itself. This part of the process, if done correctly, can lead to a smooth installation process with a minimum of errors, customer issues or installation delays. The safety aspects of the installation conform to:

- The National Electric Safety Code (NESC) for utility wiring
- The National Electric Code (NEC) for home wiring
- ASNI C12.1 – Code for Electricity Metering
- Local building codes

The customer is notified of the installation if they are present and the installation process begins. The first step in the installation process involves the assessment of access to the meter location and safety of the existing equipment. After proper access has been established, actions include:

- Check meter location for safety issues, damage, and diversion
- Verify meter data for service voltage and meter form type
- Verify premise information for correct address, meter number, GPS Location, etc
- Safely replace old meter with Smart Meter and re-seal
- Update customer premise information for new installation

National demographics show a housing unit split of approximately 74% single family and 26% multi-family homes, with percentages varying from state to state. [7] Therefore, the vast majority of the Smart Meter installations will be to single family homes with single meter base designs. Typically the meter base is mounted to the surface of an exterior wall [8] where the service entrance attaches to the house. Gang meter socket designs are used to consolidate multiple meters to a few locations for the multi-family dwelling units. Generally, these gang sockets are located in designated meter rooms, on the outside wall of apartment buildings, or in the basement of high rise apartment buildings. Both single family and multi-family installation processes are designed to address physical access and safety concerns, to make sure the proper type of Smart Meter is installed safely and correctly, and to ensure the correct information is obtained and delivered for accurate setup of customer billing.

After the Smart Meter is installed, it is usually ready for operation and is automatically registered with the network system. If the customer is not present and the installation cannot be completed, a notification is left detailing the process to schedule the installation for a later date.

[7] "*Historical Census of Housing Tables, Units in Structure";* U.S. Census Bureau, Housing and Household Economic Statistics Division; December 16, 2005

[8] See section 4 for further discussion

5. RF Exposure in Smart Meter Systems

The implementation of Smart Meter Systems has generated some concerns about RF exposure that the local jurisdictions and serving utilities have addressed or are addressing. In this regard, utilities have used verification, technical data, and numerous third party investigations to address the customer concerns appropriately.

5.1 Radio Frequency (RF) Exposure

Various Smart Meter Systems work by transmitting information wirelessly. The Federal Communications Commission (FCC) has jurisdiction over the approval and use of radio frequency devices, whether a license is required for the devices or if unlicensed operation is allowed. The FCC has a twofold role in ensuring safety:

- The FCC has allocated the radio spectrum into a variety of pieces, most of which needs coordination and a license before operation is permitted. At the same time, the FCC has allocated some frequencies for unlicensed operation (e.g., allowing consumers to purchase products at retail outlets and install them in their homes). These devices operate at low power levels, enabling communications but posing no known health effects to humans. Examples include the WiFi routers already discussed, wireless baby monitors and garage door openers. For the most part, Smart Meters fall under this low power, unlicensed criteria.
- The FCC's second role is to approve radio devices for manufacture, import and sale. Regardless of whether the equipment operates on low power unlicensed channels or at higher power levels that require authorization, each device must be tested to meet FCC standards. The sale of untested and unapproved equipment is a serious offense and the FCC aggressively prosecutes violators. FCC Rules governing the approval and sale of radio devices can be found in the Code of Federal Regulations (CFR) title 47, Part 15. These rules govern all aspects of radio emission, including both intentional and unintentional radiators.

Specific to RF safety issues, the FCC is required by the National Environmental Policy Act of 1969, among other things, to evaluate the effect of emissions from FCC-regulated transmitters on the quality of the human environment. Several organizations, such as the American National Standards Institute (ANSI), the Institute of Electrical and Electronics Engineers, Inc. (IEEE), and the National Council on Radiation Protection and Measurements (NCRP) have issued recommendations for human exposure to RF electromagnetic fields.

On August 1, 1996, the Commission adopted the NCRP's recommended Maximum Permissible Exposure (MPE) limits for field strength and power density for the transmitters operating at frequencies of 300 kHz to 100 GHz. The Commission's requirements are detailed in Parts 1 and 2 of the FCC's Rules and Regulations [47 C.F.R. 1.1307(b), 1.1310, 2.1091, 2.1093]. The FCC also presents OET Bulletin 65 on this topic. The revised OET Bulletin 65 has been prepared to provide assistance in determining whether proposed or existing transmitting facilities, operations or devices comply with limits for human exposure to RF fields adopted by the FCC. This bulletin offers guidelines and suggestions for evaluating compliance.

All Smart Meter radio devices must be certified to the FCC's Rules. Vendors develop products based on technical and regulatory specifications. Often, radio transmitters are integral parts of the meter itself;

integrated into the circuit board of the device. The manufacturer tests the devices to FCC specifications and then presents the test results to an independent certification laboratory, or the FCC directly. Only when the FCC reviews the detailed report and certifies the device can the manufacturer market and sell the devices. The same procedures are used for Wi-Fi network equipment in PCs and wireless routers located nearly everywhere in our homes and offices.

There are two types of potential effects due to RF emissions, non-thermal and thermal. To date, there is no conclusive research that confirms negative non-thermal health impacts caused by non-ionizing RF emissions. There is, however, scientific consensus that for certain RF signal strengths there could be negative health effects. Therefore, most health studies have focused solely on the thermal effects of RF.[9,10] Several studies have been prepared to investigate the RF exposures of Smart Meters with relatively consistent conclusions:

- Smart Meter exposures even at close range and with exaggerated duty cycle are many times less than other household devices and are compliant with FCC limitations.
- As an example, an RF exposure comparison of a person talking on a cell phone and a person 3 and 10 feet from a continuously operating smart meter would result in Smart Meter RF exposure of 125 to 1250 times less exposure than the cell phone.[11]

Utility installation and operational practices and the impacts of all equipment used in the premise service location affect the exposure levels of RF greatly. Smart Meters are universally mounted in metal enclosures referred to as sockets or bases. These enclosures are generally mounted outside and facing away from the living space of a home. Single family dwellings typically have one socket located at the point of service. For multi-family housing such as apartments, condominiums, and townhouses, the sockets are a single unit with multiple meters. They are usually located in designated meter rooms, on the outside structure wall, or in the basement of high rise apartment buildings. Most of these typical mounting locations are either facing away from or are not adjacent to living areas. In addition, local fire codes and practical construction techniques limit the number of meters that are typically wall mounted, as described above, for multi-family dwellings and are not usually readily accessible. In larger multi-family buildings, i.e. mid-rise and high-rise units, the meters are typically located in meter rooms or in the basements and are ordinarily secured for limited access.

Even in a meter room or basement with large numbers of meters, it is impossible to obtain peak exposure from every meter. For example, if the meter room is 12 feet wide and the body is 2 foot wide, a person could only be within one foot of 17 % of the meters. Typical exposure to Smart Meter fields is usually at some considerable distance. But for those relatively rare instances that result in close proximity to the meters, measurements have shown exposure well below FCC standard limits. Exposure in living spaces will be even less due to the attenuation of RF signal caused by building materials in the walls and other structures. A typical building wall construction combined with a surface mounted meter base will represent a nominal minimum 10 inch (25 cm) distance between the transmitter and the interior wall surface and

[9] "*Health Impacts of Radio Frequency (RF) from Smart Meters*"; California Council on Science and Technology (CCST); January 2011

[10] "*Evaluating Compliance with FCC Guidelines for Human Exposure to Radiofrequency Electromagnetic Fields";* OET Bulletin 65; Edition 97-01; August; Federal Communications Commission, Office of Engineering & Technology

[11] "*Health Impacts of Radio Frequency (RF) from Smart Meters*"; California Council on Science and Technology (CCST); January 2011; page 20

potential internal dwelling RF exposure to humans. Actual measurements directly behind the meter on the inside of the wall have produced MPE's of 0.01 % of the FCC limits. [12]

At all meter premise locations, the meter socket acts as a barrier for RF emissions entering the home. Manufacturers point out that the area behind the meter socket is virtually a dead spot for RF emissions. In addition, measurements have shown that at 8 inches behind gang meter sockets, the RF exposure is over 10 times less than the same distance in front of the sockets and less than 1% of the FCC exposure limits.[13] The metal meter socket reflects almost all of the RF out of the front of the meter. The only path for RF to get into a building is by first bouncing off the ground or an adjacent house and then back into the building. The distances required for this to happen dramatically reduce the power signal by the time it has traveled a minimum of 4-5 feet to the ground and into the living space.

The following are examples of measured RF exposure level with transmitter at continuous operation (an unrealistic condition) from a gang meter arrangement simulating an apartment metering location.

Example 1

Duty Cycle	% FCC Limit @ 1 ft $\mu W/cm^2$	% FCC Limit @ 2ft	% FCC Limit @ 3 ft	% FCC Limit @ 5 ft
100%	8.1 %	3.9%	2.5%	1.4%

A 10 meter rack with a 250 mWatt 915 MHz[13] Smart Meter transmitter simulating an apartment wall meter installation demonstrating of exposure variance with distance[14]

Example 2

Duty Cycle	Front Exposure @ 1 ft % FCC MPE	Rear Exposure @ 8 in % FCC MPE	Rear Exposure @ 5 ft % FCC MPE
100%	8.1%	0.6%	0.25%

A 10 meter rack with a 250 mWatt 915 MHz Smart Meter transmitter front and rear measurement RF exposure comparison[15]

The FCC limits on maximum permissible exposure (MPE) for application to the general public were set using safety factors fifty times lower than the levels of known effects. The MPE's are those values of RF field strength, or power density that have been averaged over any 30-minute period (time averaging) and

[12] "An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter", EPRI (2010),December 2010

[13] Generally refers to the FCC's "license free" band of 902-928 MHz

[14] "An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter", EPRI (2010),December 2010

[15] "An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter", EPRI (2010),December 2010

averaged over the dimensions of the body (spatial averaging). Discussed below are several basic factors that affect RF exposure:

RF frequency

Most Smart Meters use the same frequencies as other RF devices in the home, the 915 MHz band and 2.4 GHz band. The RF exposure limits, MPE, set by the FCC for Smart Meters are rated at the frequencies they use to communicate:

- 915 MHz 601 $\mu W/cm^2$ avg.
- 2.4 - 100 GHz[16] 1000 $\mu W/cm^2$

Transmitter Power

Smart Meters use low power transmitters, generally one watt or less for unlicensed frequency, 2 watts licensed, and produce relatively weak RF signals.

Distance

The power density decreases proportional to the square of the distance from the RF source at single meter locations. At multi-meter sites, the power density decreases significantly but at a lesser rate, proportional to the distance.

Duty Cycle (RF Exposure time)[17]

The percentage of time an RF device is in operation is called the duty cycle. The actual percent of time the Smart Meter is transmitting, especially in the initial years of operation, is very small, usually less than 1% (less than 15 minutes accumulated total per day). There are several other factors that affect the duty cycle for Smart meter systems.

The first factor of the duty cycle is how many meters communicate at the same time. As a practical design matter, when several Smart Meters are placed in a cluster, they generally have to communicate with a single controller. In order to ensure that the controller receives the information properly, transmitters are typically programmed to communicate with a controller in a random fashion, significantly decreasing the potential for exposure to multiple signals at the same time.

The second factor is the length of the communication. Smart Meter communications are typically less than a second and under normal operations, the programmed interval for randomized transmissions is 4 to 6 hours or longer. Over time, while it is possible that the duty cycle could rise due to additional use of the system for Smart Grid initiatives, the use of higher data transfer rates could, in fact, diminish the duty cycle.[18] All meters transmitting continuously will disrupt the system from functioning properly due to message traffic congestion and collisions. Therefore, the practical operational limit is less than 50%; well below 100% duty cycle sometimes used for comparisons. In spite of this, several RF exposure studies consider 2% -5% duty

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[16] To date there are no known Smart Meter Systems that operate above 6 GHz.

[17] "An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter", EPRI (2010),December 2010

[18] *"Wireless Transmissions: An Examination of OpenWay Smart Meter Transmissions in a 24 hour Duty Cycle*"; Itron Inc.; 2011; page 6, note #2.

cycle operational scenarios, and a 100% duty cycle, continuous operation, scenario to establish an absolute maximum exposure value.

Spatial Averaging

MPE values are measured by averaging the exposure value over the dimensions of the body. Since different parts of a person's body are at varying distances from the transmitter, the RF exposure will vary at different parts of the body. At the typical 5 foot mounting height, a person's head may have maximum exposure but the person's knee will receive less exposure. The spatial average MPE is 18% to 24% of the peak value MPE on the body.[19]

In summary, the RF exposure effects of Smart Meters are very small compared to exposure from other sources in the home. Smart Meters operate significantly below FCC exposure limits. In addition, the location, distance from the transmitter, shielding by meter enclosures, attenuation of building materials, direction of RF emissions, and limited duty cycles even further reduce exposure to consumers. A review of the results of several Smart Meter RF studies and actual measurements of Smart Meter RF emissions support these observations. Other summary observations include:

- All smart meter radio devices must be certified to the FCC's rules.
- Exposure levels drop significantly with the distance from the transmitter, with spatial averaging, and in living spaces due to the attenuation effects of building materials.
- The FCC limits on maximum permissible exposure (MPE) for application to the general public were set using safety factors fifty times lower than the levels of known effects.
- Tests simulating multi-family metering locations containing several meters in close proximity have shown RF exposure levels dramatically less than FCC standards.
- Due to shielding of the meter enclosure and signal patterns, RF exposure from the rear of a metering location is nominally 10 times less than in front of the meter and dramatically below FCC limits, not including the spatial averaging and building material attenuation reductions.[20]
- For measurement and calculation purposes some studies use a 100% duty cycle parameter. However, the maximum operational Duty Cycle for Smart meter systems is less than 50% due to message traffic congestion and collisions. The typical Duty Cycles for Smart Meter Systems is between 1% and 5%.
- An RF exposure comparison of a person talking on a cell phone and a person 3 and 10 feet from a continuously operating Smart Meter would result in Smart Meter RF exposure 125 to 1250 times less than the cell phone.[21]
- In test environments simulating operational conditions, for power (250 mWatt - 2 Watt), duty cycle (2%-5%) at close distance (1 foot) from the transmitter, Smart Meters cause very low RF exposure to the consumer, typically well under 10 % of the FCC exposure regulations.

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[19] "An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter", EPRI (2010),December 2010

[20] "An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter", EPRI (2010),December 2010

[21] "*Health Impacts of Radio Frequency (RF) from Smart Meters*"; California Council on Science and Technology (CCST); January 2011; page 20

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2010 TECHNICAL REPORT

An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter

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1021126
Final Report, December 2010

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The following organization, under contract to EPRI, prepared this report:

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NOTE

Acknowledgments

The following organization, under contract to the Electric Power Research Institute (EPRI), prepared this report:

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This report describes research sponsored by EPRI and overseen by Gabor Mezei, MD, Ph.D, EPRI's Program Manager for the EMF and RF Health Assessment and Safety Program. Expertise, review and comment were provided by a cross-functional team at EPRI including Rob Kavet, D.Sc., Senior Technical Executive; Chris Melhorn, Program Manager; Kermit Phipps, Project Engineer/Scientist and Brian Seal, Senior Project Manager, EPRI.

Numerous individuals from Southern California Edison (SCE) Electric Company conducted and supported the measurement work reported here in both South Carolina and California: Glenn Sias, Manager, SCE EMF and Energy Group; Al Vazquez, Enterprise Network Architect, SmartGrid Engineering; Kevin Nesby, Project Manager in the Edison SmartConnect™ Deployment Program; Brian Thorson, EMF Specialist in the Operations Support Business Unit; Jack Sahl, Director of Operations Support in the SCE Operations Support Business Unit; and John Bubb, Manager of Product Management for SCE SmartConnect.

Background information on operational characteristics of smart meters was provided by Vijay Lakkasetty, System Engineering & Operations - Smart Meter Project, Jay Xu, Principal Engineer, and Jim Turman, Program Manager, Department & Employee Readiness, Safety and Emergency Services, all of San Diego Gas and Electric Company.

As further described within the report, testing support, facility access and cooperation was provided by Itron, which facilitated this work, particularly by Dan Lakich, Design & Development Manager, Electricity Metering; Mike Belanger, Product Line Manager, Itron OpenWay Network Communications; Sudhir Thumaty, R&D Manager; Suresh Kumar Yada, Design Engineer; Ben Smith, Engineer; Ken Prevatte, Project Director; and Matthew DeCubellis, Application Engineer.

This publication is a corporate document that should be cited in the literature in the following manner:

An Investigation of Radiofrequency Fields Associated with the Itron Smart Meter
EPRI, Palo Alto, CA: 2010. 1021126

Product Description

Smart meters represent one component of the advanced metering infrastructure (AMI). Although data to and from smart meters may be transmitted through wired connections, many smart meters make use of miniature, low power radio transceivers to wirelessly communicate with the electric utility and with the Home Area Network (HAN) that provides home owners with the ability to interact with electrical appliances and systems within the home. Deployment of smart meters has raised concerns by members of the public about possible adverse health effects that could be related to exposure to the radiofrequency (RF) emissions of the meters. As part of on-going efforts to address public concerns on this issue, this report documents the collection of information on RF exposure related to the operation of two particular models of Smart Meter produced by Itron Inc.

Results & Findings

The smart meters studied in this report are currently being deployed by two electric utilities in California. The meters are part of wireless mesh networks in which one meter is configured as a collector point, referred to as a "cell relay" by Itron, for each of approximately 500 to 750 "end point meters." The cell relay collects data from the various end point meters and conveys these data onto the cellular wireless wide area network (WWAN) for communication back to the electric utility company's data management system. Mesh network communication among the many meters is provided by the 900 MHz band transceiver RF LAN (local area network). A HAN feature is supported by a 2.4 GHz transceiver.

Data collection was carried out in a laboratory setting and at residences and in neighborhoods in southern California and Colville, Washington, supplemented with theoretical modeling studies. The results indicate that RF field from the investigated smart meter are well below the maximum permitted exposure (MPE) established by the Federal Communications Commission (FCC). For instance, at one foot, the RF field from an end point meter would be expected to not exceed 0.8% of the MPE) established by the Federal Communications Commission (FCC). For the cell relay, the RF field would not exceed 0.2% of the MPE. Even at very close distances, such as one foot directly in front of the meter, with an unrealistic assumption that the transmitters operate at 100% duty cycle, the resulting exposure is less than the FCC MPE. When viewed in the context of a typical, realistic exposure distance of 10 feet, the RF fields are much smaller, about 0.008% for the end point meter and about 0.002% of MPE for the cell relay. For occupants of a home equipped with a Smart Meter, interior RF fields would be expected to be at least ten times less intense simply due to the directional properties of the meter. When the attenuation afforded by a stucco home's construction is included, a realistic

value of the interior RF field would be about 0.023% of the MPE for an end point meter and about 0.065% for a cell relay. Regardless of duty cycle values for end point and cell relay meters, typical exposures that result from the operation of smart meters are very low and comply with scientifically based human exposure limits by a wide margin.

Challenges & Objective(s)

This report is focused on the RF aspects of smart meters and in particular, the strength of the transmitted RF fields that may be produced by the meters from a human exposure perspective. The greatest difficulty in arriving in determining realistic time-averaged exposure from smart meters is associated with determining transmitter duty cycles since the meters only emit RF radiation at intervals

Applications, Values & Use

This report documents an investigation of the characteristics of RF fields associated with Itron Smart Meter. The project was undertaken to improve understanding of public exposure to the RF emissions produced by smart meters and to respond to public concerns about potential health effects.

EPRI Perspective

Measuring electric energy consumption with so-called smart meters in residential and commercial environments is becoming more commonplace as part of the development of Advanced Metering Infrastructure (AMI) in the electric utility industry. With the deployment of smart meters public concern was raised about potential health effects associated with RF emissions from smart meters EPRI is responding to these concerns with research efforts to provide objective information on RF emissions related to smart meters.

Approach

The project team conducted laboratory and field measurements of the RF emissions of Itron smart meters. A key objective was to determine realistic estimates of the operational duty cycle of meter transmitters. The team also investigated the effectiveness of metal meshes and stucco walls in shielding smart meters.

Keywords

Smart meters
Radiofrequency emissions
EMF health assessment
Environmental issues

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Section 1: Summary

Measuring electric energy consumption with so-called Smart Meters in residential and commercial environments is becoming more commonplace. Smart Meters represent one component of what is referred to as Advanced Metering Infrastructure (AMI) in the electric utility industry. AMI systems comprise both wired and wireless technologies with each exhibiting their own advantages. Electric utility companies, thus, have options to implementing AMI systems. Even within the wireless category of AMI system, equipment can operate over a wide range of frequencies and powers and levels of activity. The Smart Meters, based on wireless technology, make use of miniature, low power radio transceivers, typically inside the meter, to wirelessly communicate with the electric utility. Two-way radio communication provided by Smart Meters allows for transmission of energy consumption data from a residence or business to the utility company and reception of data pertaining to time-of-day pricing of electric energy.

As wireless AMI technology is projected to become widely distributed, it becomes prudent to quantitatively assess the levels of RF emissions fro meters to which the public may be exposed. Nearly two dozen communities have placed moratoria on further deployment of Smart Meters in northern California and more than 2000 health-related complaints have been received by the California Public Utilities Commission[1]. This report documents the collection of information related to the operation of two particular models of Smart Meters[2] produced by Itron Inc. for purposes of supporting exposure assessment exercises that can address public concerns about exposure. The Itron products are currently being deployed by Southern California Edison Electric Company (SCE) and San Diego Gas and Electric Company (SDG&E) and both companies provided support to EPRI (the Electric Power Research Institute) for this activity. A number of companies currently manufacture different forms of Smart Meters and, most commonly, these meters employ radio transmitters that operate in Federal Communications Commission (FCC) designated license free bands[3]. The Itron meters in this study use transmitters that operate in the license free bands of 902 MHz to 928 MHz (the "900 MHz band") and 2400 MHz to 2500 MHz (the "2.4 GHz band").

The Smart Meters studied here act as nodes in wireless mesh networks consisting of approximately 500 residences (for SCE) or 750 residences (for SDG&E); these are referred to as "end point meters." Within each mesh network, one residence, designated as a "collection point," is equipped with a Smart Meter having an additional internal transmitter (referred to as a "cell relay" for communicating data to the utility over a wireless wide area network (WWAN). The cell relay collects data from the various end point meters and conveys these data onto the cellular wireless wide area network (WWAN) for communication back to the electric utility company's data management system. Mesh network communications among the many meters is provided by the 900 MHz band transceiver RF LAN (local area network). A HAN feature is supported by the 2.4 GHz transceiver. A data protocol used by the HAN called Zigbee is used to refer to the 2.4 GHz transceiver as in "the 2.4 GHz Zigbee radio".

The data collection effort included gathering of information and working with the manufacturer at their facility in West Union, South Carolina, measurements at residences and in neighborhoods in southern California and some more limited measurements in Colville, Washington. Itron graciously provided technical support and access to its facilities and personnel to assist in this effort. Data included transmitter power levels, radiation patterns, RF field strengths or power densities of individual meters and groups of meters, spatial variations of RF fields in a vertical plane near Smart Meters, attenuation of Smart

[1] See, for example, "Smart Meters - They're Smart, But Are They Safe?". http://www.publicnewsservice.org/index.php?/content/article/16846-1 (November 8, 2010).

[2] Itron model CL200 (end point meter) and model C2SORD (cell relay).

[3] Some Smart Meters are designed to operate in FCC licensed bands and may operate with higher powers.

Meter RF fields by building materials, and information potentially useful for assessing transmitter duty cycles. To characterize the systems currently operating, parallel efforts included modeling of RF fields based on measured values of maximum equivalent isotropic radiated power (EIRP) of both end point and cell relay meters and analysis of end point meter transmission statistics for estimating duty cycles. Antenna patterns were determined for the 900 MHz RF LAN and 2.4 GHz Zigbee transmitter in both end point and cell relay meter configurations. Patterns were also measured for both the 850 MHz and 1900 MHz cellular bands from a cell relay.

Antenna pattern measurements revealed that RF fields are emitted preferentially toward the frontal region of the meters; the direction of maximum EIPR, however, might not be directly normal to the front of the meter. Apparent antenna gain values were modest, ranging between 0.88 dBi and 5.08 dBi, depending on the frequency band and the configuration (end point vs. cell relay). Patterns typically exhibited a reduced RF field behind the meter of approximately 10 dB down from the maximum frontal value of field with relatively narrow notches in the pattern directly behind the meter of as much as 20-30 dB less than in front.

Transmitter power data were obtained on 200,000 RF LAN 900 MHz transmitters with a most likely value of approximately 24.5 dBm (282 mW) with a 99th percentile power of 26.0 dBm (298 mW). Based on a sample size of 200,000 2.4 GHz radios, the most likely power was found to be 18.5 dBm (70.8 mW) with a 99th percentile power of 20.8 dBm (114.8 mW). Cellular transmitters were specified as 31.8 dBm in the 850 MHz band and 28.7 dBm in the 1900 MHz band.

Because of the very intermittent nature of transmissions from Smart Meters and their frequency hopping spread spectrum transmitters, accurate measurement of RF fields can be challenging. To facilitate the measurements, Smart Meters were programmed to transmit continuously on a single frequency. RF field measurements were performed on a single meter inside the Itron anechoic chamber and on ten individual meters installed in the Itron meter farm. These measurements were obtained with two different instruments including an isotropic, broadband, frequency conformal electric field probe (Narda Model B8742D) and a spectrum analyzer based selective radiation meter (Narda Model SRM-3006). Measurement data for the 900 MHz RF LAN transmitters showed RF fields in the range of a few percent of the FCC MPE for the general public at 30 cm (approximately 1 foot) in front of the meters (0.7 to 5.5%) with the broadband probe depending on frequency. Similar measurements for the 2.4 GHz Zigbee radios at a distance of 20 cm showed 0.75% to 1.7% of the MPE, again depending on the frequency of the transmitter.

Using the SRM-3006 instrument, RF fields were measured as a function of distance from the rack of ten meters in both the 900 MHz and 2.4 GHz bands. These measurements produced readings ranging between approximately 8% at 1 foot to less than 0.1% at 75 feet from the meters in the 900 MHz band and approximately 4.5% at 1 foot to less than 0.01% at 75 feet in the 2.4 GHz band. 900 MHz field measurements showed that the emissions associated with the ten meters dropped into the background produced by other meters in the meter farm at a distance of approximately 50 feet.

By using the maximum hold and average measurement feature of the SRM-3006, a measurement in the meter farm obtained by walking along two rows of meter racks resulted in an integrated peak RF field equivalent to 0.114% of MPE and an average value of 0.00023% of MPE. The ratio of average to peak readings corresponds to an apparent duty cycle of about 0.2%. In measurements taken at two apartment houses in Downey, California, ratios of average to peak values of RF field obtained over five-minute monitoring periods resulted in estimated duty cycles of approximately 0.001%. Using a tiny USB spectrum analyzer designed specifically for just the 900 MHz band in the Itron meter farm, spectral measurements were captured for approximately one hour. This measurement resulted in an apparent duty cycle of approximately 0.02%.

Interior residential measurements were performed in two homes in Downey, California after temporarily replacing the existing Smart Meter with specially programmed units that would transmit continuously in the 900 MHz and 2.4 GHz bands. Inside measurements ranged from approximately 0.006% to 22% of MPE, the highest value associated with operation of a microwave oven in the kitchen at 2 feet from the oven. The greatest value immediately behind the Smart Meter, inside the home, was 0.009% of MPE. Wireless routers found in both homes resulted in RF fields in the range of 0.02 to 0.03% of MPE.

Residential neighborhood surveys were performed in areas with and without deployed Smart Meters while driving the streets of two communities, one in Downey, CA and one in Santa Monica, CA respectively. The exercise demonstrated that the emissions of randomly emitting Smart Meters could be detected in the Downey neighborhood but virtually no signals were detected in Santa Monica with the exception that when driving through a commercial district, the 900 MHz band came alive with noticeable activity, presumably caused by various 900 MHz sources, such as cordless telephones, etc. Spectrum measurements in several other band were also performed including the FM radio broadcast band, two cellular telephone bands and the 2.4 GHz Wi-Fi band.

The insertion loss of three different metal meshes was evaluated in California at one of the residences in which RF measurements were obtained. Three different sizes of mesh were used in the tests by inserting the mesh between a specially prepared, portable Smart Meter as a source, and the SRM-3006 meter. These measurements were performed at close range with the Smart Meter approximately six inches behind the mesh and the SRM-3006 probe approximately the same distance on the other side of the mesh. These measurements resulted in values for insertion loss ranging from 4.1 dB to 19.1 dB in the 900 MHz band and from 1.2 dB to 11.4 dB in the 2.4 GHz band, depending on mesh opening size. Additional insertion loss measurements were performed on a simulated stucco wall in Colville, WA resulting in values of 6.1 dB and 2.5 dB for the 900 MHz and 2.4 GHz bands respectively.

Since human RF exposure standards are based on spatial averages, spatially averaged values of RF fields were obtained along a vertical line at approximately one foot in front of a Smart Meter. It was found that over a six-foot vertical span, the spatially averaged RF field in the 900 MHz band corresponded to a value 23% of the measured peak value found near the height of the meter. In the 2.4 GHz band, the spatially averaged field was 18% of the spatial peak.

Using the detailed pattern measurement data described earlier, theoretical calculations of RF fields that could be associated with each of the transmitters in either end point meters or cell relays were made. A detailed analysis was developed to investigate the effect that ground reflected fields could have on the resultant field and what factors would be appropriate for including the effect of ground reflections in theoretical RF field calculations.

Human exposure to RF fields is judged by comparison to applicable exposure limits or standards. For the United States, and in regard to Smart Meters, the most applicable limits are those promulgated by the FCC, a spatially averaged and time averaged value of 610 microwatts per square centimeter ($\mu W/cm^2$) in the 900 MHz band and 1000 $\mu W/cm^2$ in the 2.4 GHz band. A proper comparison of Smart Meter produced RF fields to these limits should involve a determination of the time-averaged value where the averaging time is specified as any 30-minute period. To arrive at time-averaged values, the measurements or calculated fields reported above must be corrected for the operational duty cycle of the transmitters. This is the most complex issue connected with Smart Meter RF evaluations since transmitter activity is semi-random in nature, with only brief transmissions occurring throughout a day. The maximum value of duty cycle for end point meters has been estimated by Itron to be in the range of 5%. Actual measurements, however, tend to result in substantially smaller values, typically less than 1%. Because of the variable nature of transmitter activity, even accurate measurements of a specific meter or meters need to be repeated for some days and, possibly, weeks to obtain reliable estimates of typical duty cycles. Rather than measurements, Itron developed special software implemented by the two companies to collect transmit data gathered and reported on in this report. Such an approach represents a practical way for bracketing realistic values of meter duty cycles since it can be implemented in software and extended to a very large sample size, something that would be impractical to do via physical measurements of RF fields at the meters. Using this approach, SCE generated data were examined to identify what fraction of meters in the sample exhibited transmit durations over a range of times which are related directly to the transmitter duty cycle. This exercise, for example, supported 99th and 99.9th percentile duty cycles of 0.11% and 4.7% for the RF LAN component of end point meters. A complimentary analysis conducted by SDG&E but using a more accurate determination of transmitter activity revealed smaller duty cycles. Similarly small duty cycle values are associated with the HAN and cellular transmitters. Figure 1-1 illustrates the estimated maximum likely time-averaged RF fields that would be produced by both end point and cell relay meters.

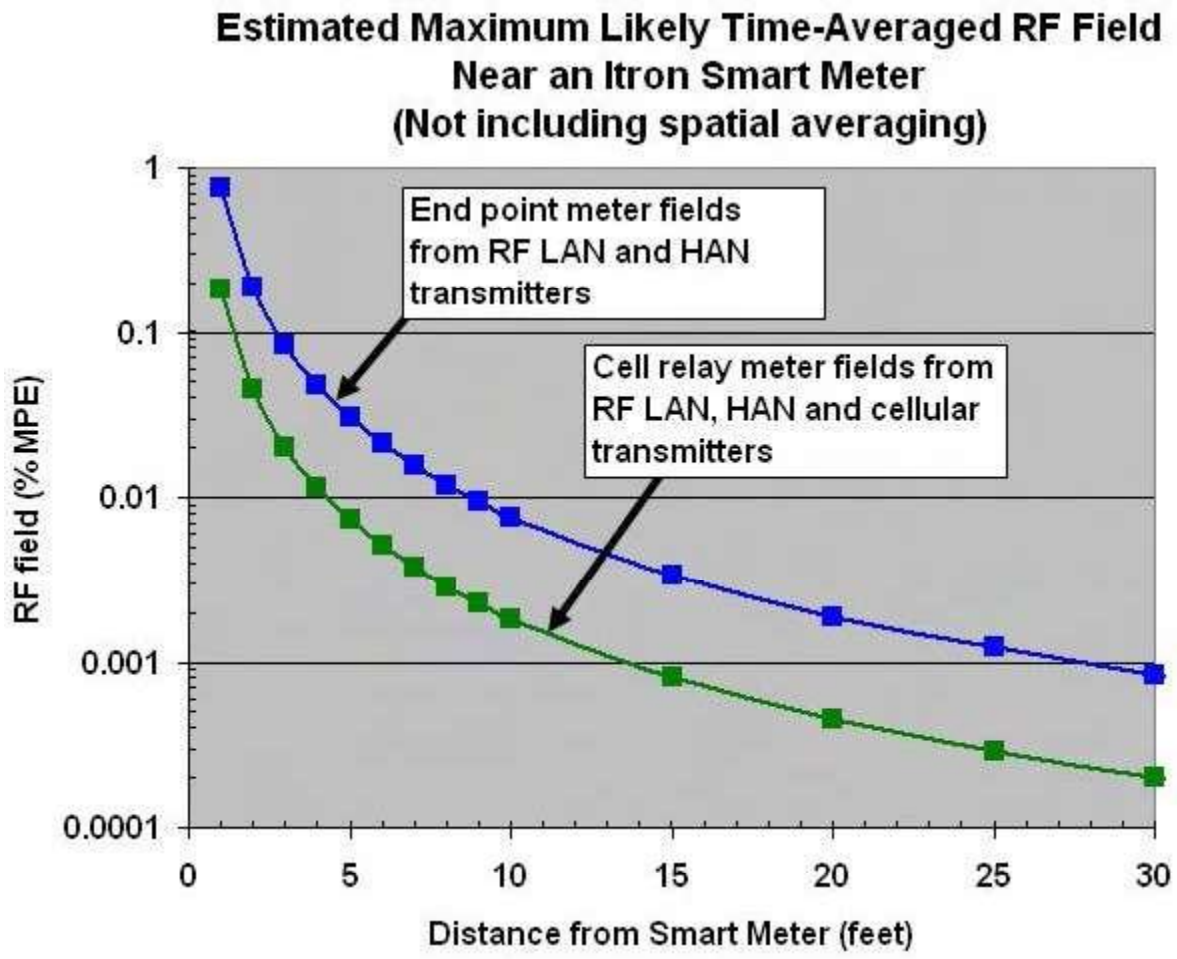

Figure 1-1
Calculated RF fields near Itron end point and cell relay meters based on 99th percentile transmitter power values, main beam exposure (point of maximum RF field), inclusion of the possibility of ground reflected fields and assumed 99th percentile duty cycles.

These data, when taken collectively, indicate that the RF emissions produced by the Itron Smart Meters evaluated in this study result in RF fields <0.06 mW/cm^2 (at least 10-fold below the FCC limit at 900 MHz). For instance, at one foot, the RF field from an end point meter would be expected to not exceed 0.8% of the MPE. For the cell relay, the RF field would not exceed 0.2% of the MPE. Even at very close distances, such as one foot directly in front of the meter, with an unrealistic assumption that the transmitters operate at 100% duty cycle (at which point the mesh network would not function) the resulting exposure is less than the FCC MPE. When viewed in the context of a typical, realistic exposure distance of 10 feet, the RF fields are much smaller, about 0.008% for the end point meter and about 0.002% of MPE for the cell relay. Spatial averaging of these "spatial maximum" fields brings the estimated values down to approximately one-fourth of these magnitudes.

For potential exposure of occupants of a home equipped with a Smart Meter, interior RF fields would be expected to be at least ten times less intense simply due to the directional properties of the meter. When the attenuation afforded by a stucco home's construction is included, a realistic value of the interior RF field would be about 0.023% of the MPE for an end point meter and about 0.065% for a cell relay meter. The WWAN operates at a far greater data throughput than the RF LAN within the mesh. Therefore, the duty cycle is correspondingly less for the cellular modem within the cell relay, despite the fact that it transmits all of the data collected from the relevant meters of its mesh network.

The most uncertainty in determining realistic time-averaged exposure from Smart Meters is associated with transmitter duty cycles. Hence, the most potentially useful avenue of future RF exposure assessment would include extensive statistical analyses of Smart Meter transmitter activity.

A detailed evaluation of possible RF fields produced by the Itron meters included in this study shows that regardless of duty cycle values for end point and cell relay meters, typical exposures that result from the operation of Smart Meters are very low and comply with scientifically based human exposure limits by a wide margin.

Section 2: Introduction and Background

As the electric utility industry in the United States moves toward implementing a "smart grid", one of the key components consists of so-called Smart Meters. These new technology electric power meters represent a part of the advanced metering infrastructure (AMI) that provides for automatic meter reading (AMR) and sophisticated control over the use of electric energy by consumers in their homes and businesses. When AMI technology is fully implemented, an enhanced balancing of power distribution throughout the various electrical grids of the country will exist and utility customers will be able to, among other things, determine when certain electrically operated appliances may operate, based on time-of-day pricing of electricity. Such advanced capability requires close to real-time data acquisition on electric energy usage and such data requirements mean that the existing, traditional electric power meters that employ manual energy consumption readings, for example, once a month, can't provide such timely data.

The modern technology of Smart Meters provides for an ability to almost instantly interrogate specific power meters as to electric energy usage. For the Smart Meters investigated in this study, this capability is accomplished via the use of data communications between the electric utility company and individual power meters through the medium of radio signals. This report is focused on the radiofrequency (RF) aspects of Smart Meters and in particular, the strength of the transmitted RF fields that may be produced by the meters from a human exposure perspective.

Smart Meters as RF Sources

A wireless Smart Meter makes use of miniature, low power (typically less than one watt) radio transceivers inside the meter to wirelessly communicate with the electric utility company. The transceivers (transmitter and receiver) allow both transmission of data as well as reception of data and instructions from the utility. These transmitters are contained within the housing of the electric meter but are not necessarily visually obvious to an observer. Antennas used for the transmitters are commonly created as slots on the various printed circuit boards that constitute the electronic makeup of the meter. A common transmitter configuration of Smart Meters includes two or three transmitters in the meter. Figure 2-1 shows a Smart Meter with its digital display that is used to indicate electric energy usage.

Figure 2-1
Photo of Itron Smart Meter.

How Smart Meters are Deployed

Radio communication by Smart Meters makes use of wireless networks whereby each Smart Meter can both transmit and receive data to and from the electric utility company. The wireless network is configured as a so-called mesh network. Mesh networks are characterized by providing a means for routing data and instructions between nodes. A mesh network allows for continuous connections and reconfiguration around broken or blocked data paths by "hopping" from node to node until the destination is reached. In the context of how Smart Meters are deployed, end-point meters are installed throughout neighborhoods, replacing existing electromechanical meters. The transceivers[4] within the Smart Meters act as wireless routers, identifying and, then, connecting with available transmission paths between themselves and a cell relay meter that collects data from the many, various meters in the region.[5] If communication between a given end-point meter and the associated cell relay cannot be achieved due to inadequate signal strength, an alternative end-point meter is used to establish communications onward toward the cell relay meter. In this sense, the mesh network is said to be self-healing in that should a particular transmission path becomes blocked, the network finds another way to get its data through the system. A simple example of this process could be that at some particular moment, a moving van travels down a street and temporarily blocks the previously preferred path from an end-point meter to the cell relay meter. In

[4] The RF devices inside the Smart Meter function as transceivers since they both transmit and receive radio signals. In this report, the term transmitter is often used in place of transceiver since the primary characteristic of the meters of interest in this study is the meter's ability to transmit radio signals.

this case, the data is rerouted via other end-point meters that act as alternative paths for the meter to initiate the data communications. This very powerful networking approach provides for good data communication reliability and can even allow communications for end-point meters that are outside the line-of-sight range to their cell relay meter. Additional end-point meters, therefore, have the ability to expand the geographical extent of a network. Figure 2-2 illustrates the concept behind a wireless mesh network implemented for a Smart Meter equipped neighborhood. Each meter communicates either directly with the cell relay meter or via multiple "hops" of the signals through other meters.

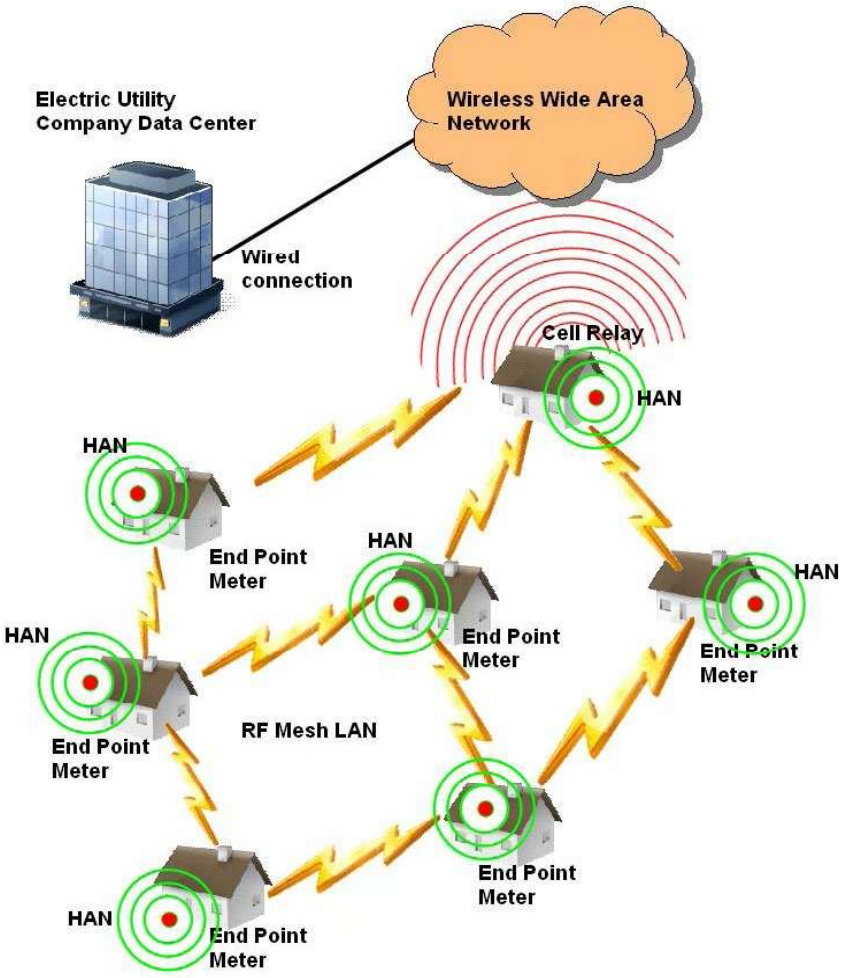

Figure 2-2
Simplistic illustrative diagram of an RF mesh network. Each end point also provides a Home Area Network (HAN) feature. The cell relay acts as a collector point for multiple meters distributed in a neighborhood and transmits received data onto a cellular wireless wide area network (WWAN).

[5] Southern California Edison Electric Company is deploying Smart Meters as part of their SmartConnect™ program with one access point for approximately every 500 end-point meters on residences. In the case of San Diego Gas and Electric Company, each access point serves for data collection from approximately every 750 end-point meters.

For the Itron equipment that was the subject of this investigation, two separate transmitters are contained in the end-point meters. The wireless mesh network can be referred to as an RF LAN (radio frequency local area network). The Itron RF LAN operates in the 902-928 MHz license free band using spread spectrum transmitting technology. A second, separate transmitter that operates in the 2.4 GHz frequency range (2405 MHz to 2483 MHz) uses direct sequence spread spectrum technology that is referred to as a Zigbee radio[6]. This second transmitter is included for use with Home Area Networks (HANs) allowing customers, for example, to control certain electric appliances or systems within the home. When fully implemented, the customers will be able to connect wirelessly with the HAN radio and set times at which various appliances and/or electrical systems may operate, thereby taking advantage of those times during which electricity rates are lowest.

The RF LAN provides data communications among the various end-point meters and an associated cell relay meter. Cell relays are end-point meters that contain yet a third transceiver that is designed for wireless connection to the cellular WWAN, i.e., relaying of the data received from the various end-point meters over a private connection to the electric utility company. The transceivers use the same frequency bands used by cell phones. Two different frequency bands are used by these cell-relay transceivers, either the 850 MHz band or the 1900 MHz band.[7] Figure 2-3 shows a cell relay with the flexible dual band antenna located on the inside surface of the meter cover.

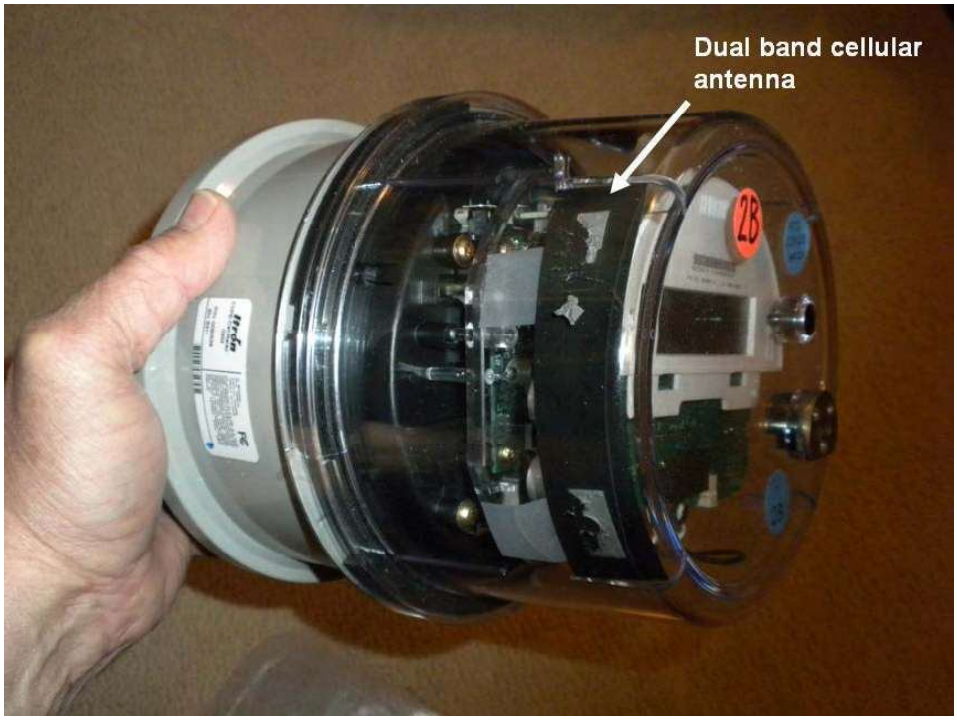

Figure 2-3
Cell relay meter with flexible, dual band (850 MHz and 1900 MHz) antenna affixed to interior surface of the meter cover.

[6]Zigbee is a name for a particular data communications protocol used in the HAN system.

[7]These frequency designations indicate the nominal frequencies used for the wireless WAN for Internet connectivity.

An important characteristic of this wireless mesh network technology is the fact that the RF emissions produced by Smart Meters, i.e., the signals that represent the data being transmitted, are not continuous but very intermittent in nature. For example, an electric utility company may interrogate the Smart Meters multiple number of times a day to acquire electric energy usage by the customer. While the Smart Meter may remain in stand-by in terms of transmissions at other times of the day, when an instruction is received to transmit energy consumption data, the meter transmits and proceeds to deliver the requested data to the cell relay meter. Hence, for the most part, Smart Meter transmissions are relatively infrequent during the day and may only consist of emissions for a few milliseconds during each of the interrogations throughout the day. This means that while the transceivers stand ready to transmit, there may be very little or no activity during most of the time. In addition to those periods during which data on electricity usage has been requested, however, Smart Meters must insure that they have a mesh network connection with at least one other Smart Meter so that, when necessary, it can deliver the data requested. Maintaining this connectivity within the mesh network requires periodic transmissions to alert the cell relay meter and other meters to its availability to be interrogated for data. So, Smart Meters spend part of their time in a so-called stand-by mode in which they issue beacon signals[8] to signify their identity to other nodes of the network with the objective of establishing a connection with the network. These beacon signals last for very brief periods of, nominally, 7.5 milliseconds and occur at various intervals. Finally, there are other instances during which certain network maintenance activities are accomplished and during which, again, various, very short duration and intermittent emissions exist. The cumulative effect of these transmissions is that while the total time spent transmitting signals from a Smart Meter is generally very modest within a day, the signals are very intermittent. They are not continuous in the same sense as the signal received from an FM radio broadcast station but, rather, exist as very short duration signals scattered throughout the day. This intermittency contributes to the difficulty in accurately measuring the strength of the emissions.

In practice, homes in a Smart Meter equipped neighborhood will have end-point Smart Meters installed that communicate with a cell relay meter either directly or though the medium of multiple end-point meter radio signal hops. Approximately every 500th (in the case of SCE) or 750th (in the case of SDG&E) residence may be equipped with a cell relay that not only handles the normal RF LAN communications but, also, relays these data onward, wirelessly, to the electric utility. All of these data communications proceed intermittently throughout each day.

The fact that the Itron Smart Meters studied here contain RF transmitters, albeit low power transmitters, means that relatively weak ambient RF fields exist in the vicinity of the meters. At the surface of the meter, the RF field strengths will be greatest with rapidly decreasing field strengths with increasing distance from the meter. While these low power transmitters cannot produce extremely intense RF fields, nonetheless, the issue of potential human exposure to these RF fields has, in some areas, become a question by the public.[9] A concern expressed by some has been the potential for adverse health effects that might be caused by exposure to the weak RF fields produced by Smart Meters. This report documents an investigation of the characteristics of RF fields associated with the Itron wireless Smart Meter that can assist in a better understanding of possible public exposure to the RF emissions produced by Smart Meters. Throughout this report, the term Smart Meter is intended to refer to the wireless type represented by the Itron meters discussed in this report.

[8] During the initial installation of an Itron Smart Meter, the meter enters a "discovery phase" in which it seeks to establish a link with the mesh network. During this discovery phase, beacon signals are emitted during approximately 3.5 second intervals until the meter becomes synchronized with the network or until a total time of about 6 minutes is reached after which beacons are emitted once about every 34 seconds until linked with the network or for up to 1½ hours. After this period, if a meter does not establish a link, it issues beacons once every hour during which it attempts to connect with the network. After 104 attempts, if still not linked with the network, the meter resets itself and begins the discovery sequence again. Once the meter becomes synchronized with the network, a beacon signal is emitted once every 94 seconds to 30 minutes depending on the level of other data traffic.

[9] Newspaper accounts of public reaction to Smart Meters

Section 3: Objective of Investigation

The work described in this report was focused on understanding the physical characteristics of the RF fields that are produced by Smart Meters such that an informed conclusion can be made as to the magnitude of possible human RF exposure caused by the meters. In this context, the objective of the work was to develop insight to the magnitude and spatial characteristics of Smart Meter RF fields including temporal aspects of the emissions that would allow a meaningful evaluation of possible exposures by reference to applicable RF human exposure limits.

Section 4: Technical Approach to Investigation

Characterizing RF fields produced by Smart Meters can be difficult. The intermittent nature of the emissions, addressed above, means that it is not a simple matter to simply bring instrumentation to an installed meter and be able to instantly detect the presence of the various emissions. The meter may or may not be in a transmit mode at the time when measurements are sought. Further, the spread spectrum characteristic of the emissions of the RF LAN and HAN transmitters leads to a further complication. For example, with the 900 MHz RF LAN transmitter, the emitted signal, at any particular instant in time, may be on any specific frequency within the 902 to 928 MHz band. When using narrow-band instrumentation, such as a frequency swept spectrum analyzer, the challenge is to have the analyzer on the specific frequency at the very instant in time that the emission is occurring to be able to measure its strength. Since the emissions are highly intermittent, this may take considerable time to insure that any such emissions have been captured by the instrumentation.

After careful consideration of the complexities associated with these kinds of measurements, it was decided that direct support of the testing by Itron, the manufacturer of the Smart Meter, could prove to be the most expedient approach to collecting the data useful to a complete exposure assessment study. As the manufacturer, Itron would have the knowledge and ability to control the Smart Meter to allow for meaningful measurements, avoiding the complications and uncertainties associated with working with already deployed meters.

Measurements at Itron

During the week of July 27, 2010, an extensive series of measurements was accomplished by the Principal Investigator at the Itron facility.

While at the Itron facility, detailed antenna pattern measurements were performed by the Principal Investigator on end point (Model CL200) and cell relay (Model C2SORD) meters. This included pattern measurements for the 900 MHz RF LAN transmitters in both the end point meter and as installed in a cell relay meter, pattern measurements of the 2.4 GHz Zigbee transmitter in both an end point meter and a cell relay meter and pattern measurements of the cell relay cellular transceiver operating in both the 850 MHz and 1900 MHz bands.

In addition to pattern measurements, Itron provided access to their Smart Meter farm, an area of some 20 acres in which approximately 7000 Smart Meters are installed. The ability to access this field provided insight to the cumulative RF field environment of multiple Smart Meters in close proximity with one another, and whether aggregate exposure produced by a multiplicity of Smart Meters concentrated in one area raises exposure risks.

Measurements in residential locations

Beyond the on-site measurements performed at the Itron facility, additional Smart Meter measurements were performed in a variety of residential environments. Using two Smart Meters that had been specifically programmed by Itron to operate continuously, to facilitate the measurements of field strength, measurements were performed at two residences in Downey, CA. These specially programmed meters were temporarily installed in the electrical service panel at each home and RF measurements were accomplished in the near vicinity of the meter and throughout the interior of each home. This procedure allowed for characterizing the RF fields that might exist inside of residences equipped with a Smart Meter. As a part of the residential measurements, a brief evaluation of the insertion loss afforded by three different metallic meshes, similar to what might be used in the construction of residential stucco walls, was conducted.

In addition to residence specific measurements with pre-programmed meters, RF fields were also measured adjacent to two separate apartment buildings wherein groups of 9 and 11 Smart Meters were grouped tightly together. Finally, a general area survey was conducted by driving throughout an established route within Downey, CA representative of a Smart Meter deployed neighborhood to form general observations of the ability to detect the presence of Smart Meter emissions. The residential measurements aspect of the work reported

here was concluded with a driving survey through Santa Monica, CA within which, at the time, there had been no deployment of Smart Meters.

Measurements in Colville, WA

Separate from the measurements at the Itron facility and the residential measurements in Downey, California, some limited measurements were conducted at the author's location in Colville, WA. These measurements included an evaluation of the comparative readings of RF field obtained by both the broadband field probe and the spectrum analyzer (selective radiation meter) used in the project measurements as well as an evaluation of the attenuation effect on Smart Meter signal propagation through a simulated, residential stucco wall.

Section 5: Transmitter Powers

A crucial aspect of any RF source, relative to its ability to produce RF fields, is the power of the transmitter. At the beginning of interactions with Itron, measurement data were sought on transmitter power levels. Historically, Itron has determined the power level of every transmitter used for the 900 MHz RF LAN and the 2.4 GHz Zigbee radios. These are transmitter devices on Itron manufactured printed circuit boards. All of the transmitters used in the Itron Smart Meters operate with low power, regardless of the frequency band used, nominally one watt or less. The 900 MHz RF LAN transmitter operates at a nominal power of 24 dBm (251 mW). Using Itron test data obtained from power measurements on a sample of 200,000 RF LAN transmitters, Figure 5-1 illustrates the accumulative fraction of transmitters having output powers across a range of power.

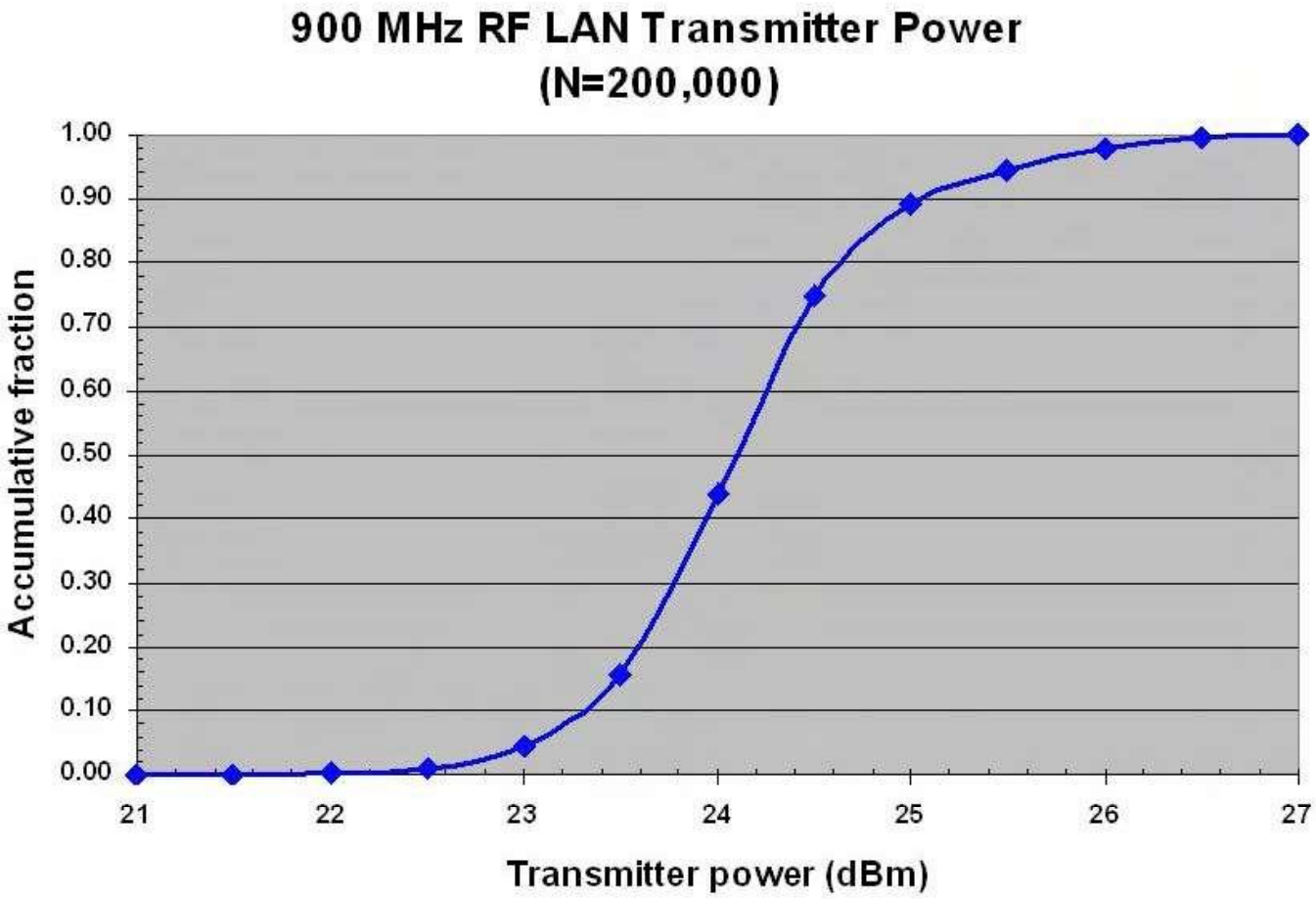

Figure 5-1
Accumulative fraction of 900 MHz RF LAN transmitter output power vs. transmitter power for a sample of 200,000 units. The median transmitter power is approximately 24.1 dBm (257 mW).

Based on a separate sample of 65,536 transmitters, used in end point meters, an average power output of 23.95 dBm (248 mW) was obtained with a standard deviation of 0.695 dBm.. Using these data, the 95% confidence interval would correspond to a range of transmitter power from 22.6 dBm (182 mW) to 25.3 dBm (339 mW) and the 99% confidence interval would correspond to a power range from 22.2 dBm (166 mW) to 25.7 dBm (372 mW).

Using the 200,000 transmitter sample, the median power level corresponds to approximately 24.1 dBm (257). The number of transmitters with power values in selected ranges is shown in Figure 5-2. The mode of transmitter power is approximately 24.5 dBm (282 mW).

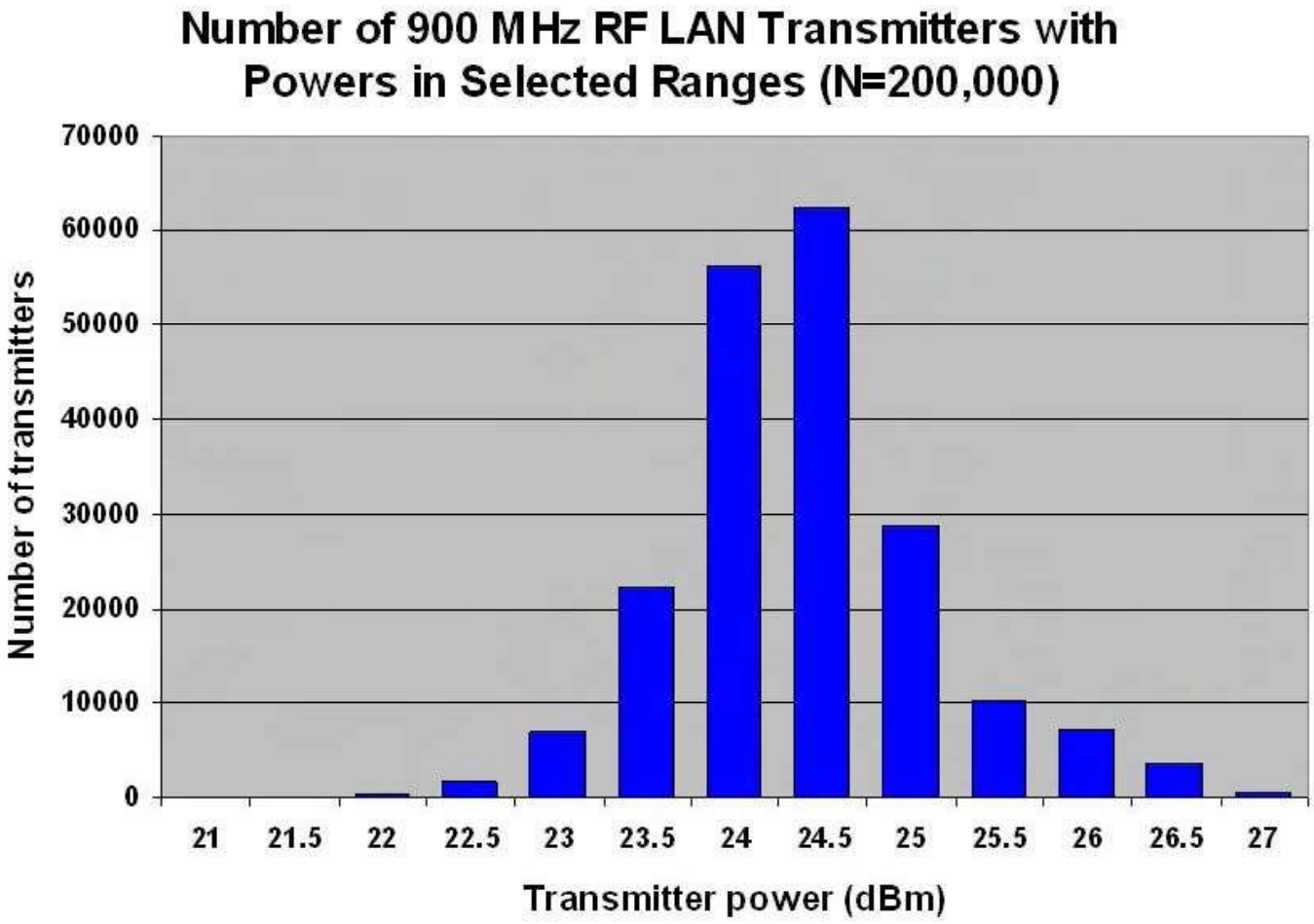

Figure 5-2
Number of 900 MHz RF LAN transmitters with powers within selected ranges. The transmitter power mode is approximately 24.5 dBm (282 mW).

These statistical data on the 900 MHz RF LAN transmitter powers indicate that the most likely power is 24.5 dBm (282 mW); an upper value of 26.0 dBm (398 mW), a value 41% greater than the most likely power, would include 99% of all transmitters.

In the case of the 2.4 GHz Zigbee transmitters, in a sample of 65,535 units used in end point meters, the mean value was found to be 18.31 dBm (67.6 mW) with a standard deviation of 0.76 dBm. This distribution would represent a 95% confidence interval of transmitter power from 16.8 dBm (47.9 mW) to 19.8 dBm (95.5 mW) and the 99% confidence interval would correspond to a power range from 16.4 dBm (43.7 mW) to 20.3 dBm (107.2 mW).

Figure 5-3 shows the accumulative fraction of transmitters having output powers across a range of power. Figure 8 illustrates the number of 2.4 GHz transmitters with powers within selected ranges. The transmitter power mode is approximately 18.5 dBm (70.8 mW).

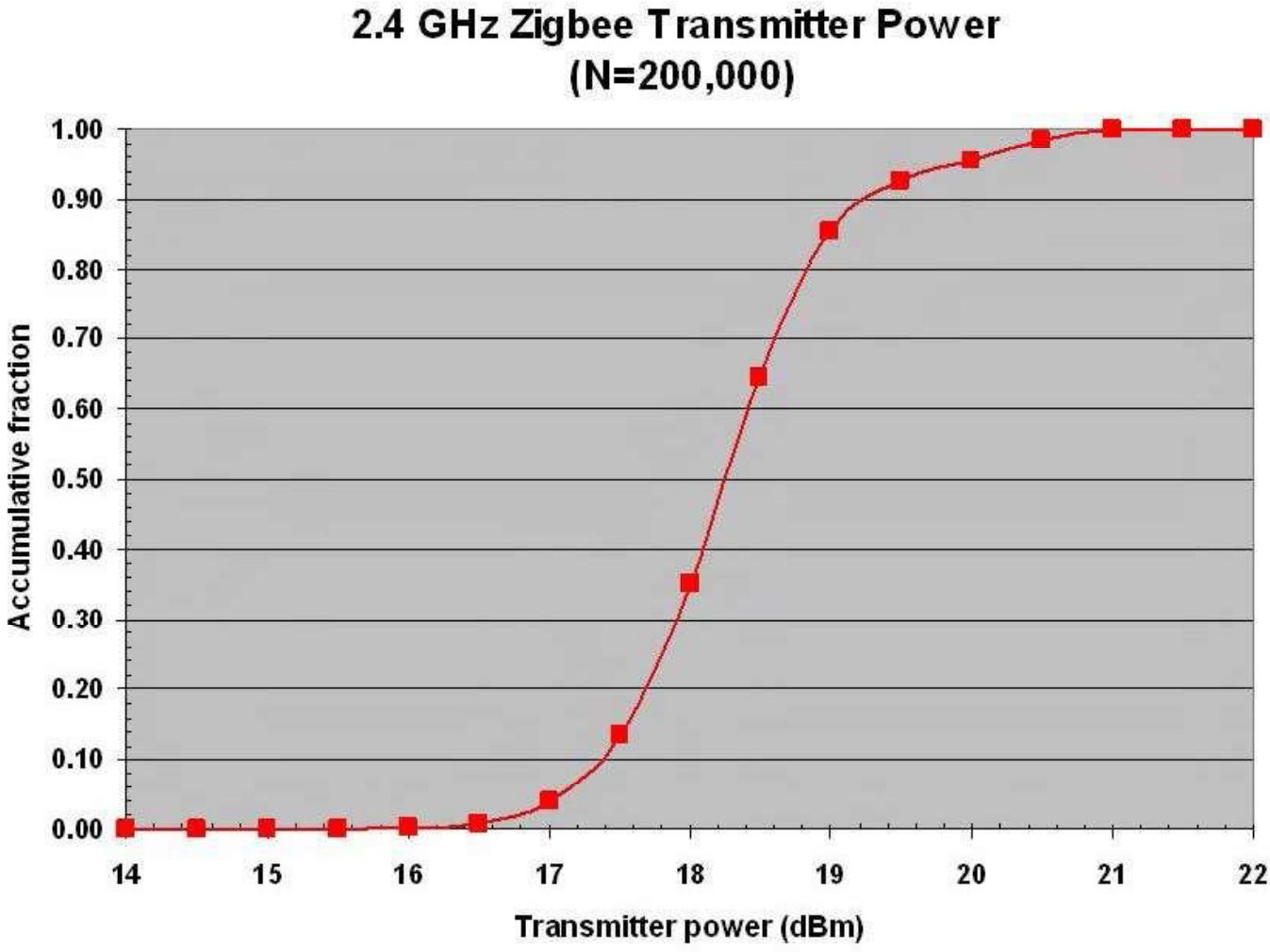

Figure 5-3
Accumulative fraction of 2.4 GHz Zigbee transmitter output power vs. transmitter power for a sample of 200,000 units. The median transmitter power is approximately 18.2 dBm (66.1 mW).

These statistical data on the 2.4 GHz Zigbee transmitter powers indicate that the most likely power is 18.5 dBm (70.8 mW); an upper value of 20.6 dBm (114.8 mW), a value 62% greater than the most likely power, would include 99% of all transmitters.

Cell relay meters contain the additional transceiver used for cellular WWAN connectivity in either the 850 MHz cellular band or 1900 MHz PCS band (personal communications service). Because these transceiver boards are produced by a different company and the units are specified to operate with specific powers and the fact that these units are separately certified by independent test labs for compliance with those specifications, Itron does not carry out additional power measurements. The transceivers, produced by Sierra Wireless operate with the following maximum powers:

Table 5-1
Sierra Wireless Transceivers Operation Maximum Powers

	GSM Modem Model MC8790 FCC ID: N7NMC8790	**CDMA Modem Model MC5725 FCC ID: N7N-MC5725**
Frequency Band (MHz)	Maximum power output (dBm) (mW)	
850	31.8 (1,514)	25.13 (326)
1900	28.7 (741)	24.84 (305)

Cell relays operate at the highest power of any of the meters due to their cellular/PCS modems but, similar to cellular telephones, the output power of the cellular modem is dynamically controlled by the applicable WWAN base station. This means that the actual operating power of the cellular radio in a cell relay will, generally, be less than the maximum power but will be determined by the signal strength it produces at whatever base station it is communicating with. Only one of the two modems would be active in a given deployment of Smart Meters in a neighborhood; the modem of choice is determined by the cellular wireless network service available and selected by the electric utility company.

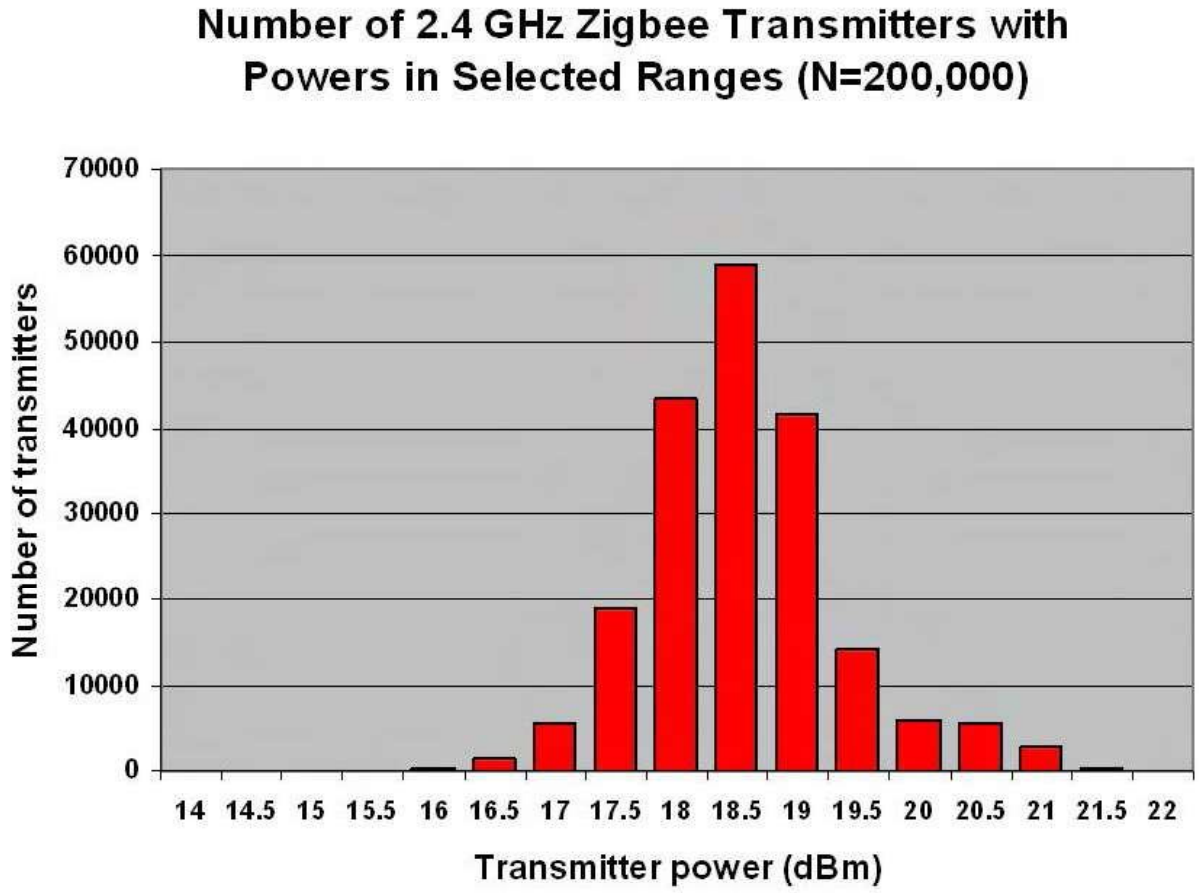

Figure 5-4
Number of 2.4 GHz Zigbee transmitters with powers within selected ranges. The transmitter power mode is approximately 18.5 dBm (70.8 mW).

Section 6: The Measurement Challenge Presented by Smart Meters

The difficulty of accurate RF field measurements near Smart Meters was discussed earlier. Low transmitted power levels in conjunction with intermittent emissions place considerable constraints on the measurement process. While a broadband measurement probe can eliminate the problem of the RF emissions occurring randomly on many different frequencies within the band, the relatively low sensitivity of broadband instruments places considerable restrictions on performing field strength measurements except within extremely close proximity of the meter. Intermittent emissions with very short duration, even if detectable, mean that it is difficult to observe when a transmission occurred. Generally, the desired measure of RF fields, from a human exposure perspective, is a measure of the average (root mean square - rms) value of the field strength or incident power density. The ratio of the average power density to the peak power density, for most Smart Meters is such that trying to measure the average field magnitude for a normally operating meter is very challenging. This can change if there exists a large aggregation of Smart Meters such that with their random on-off transmissions, much greater opportunity to "see" the emissions is possible.

Because of the rapid changes of frequency associated with the spread spectrum nature of the RF LAN and Zigbee radios in the Itron Smart Meters, an alternative approach is used to facilitate any antenna pattern and field measurements. This approach involves programming the relevant radios to transmit continuously, rather than their normal intermittent operation, and to transmit on a specific frequency within the relevant band as opposed to hopping across more than 50 channels within the 900 MHz band. Through this programming of the radios, the average signal level is now at its maximum, making it much easier to detect the RF field, and the fact that the emitted signal is now fixed on a specific and known frequency allows for ready confirmation that the measurement is of the intended signal. Since measurements under this scenario will indicate the peak value of RF field, other information is required to translate the peak field into what the equivalent average field would be. This requires a knowledge of the duty cycle of the emissions from the Smart Meter. The duty cycle can be thought of as the ratio of the amount of time that the transmitter is transmitting its signal to the total observation period. For example, if the Smart Meter were to typically transmit as much as 10 seconds during an hour (3600 seconds), the duty cycle would be 0.28%. In other words, the time-averaged power density of the RF field would be just 0.28% of the peak power density measured. The issue of Smart Meter duty cycles will be addressed later in this report.

Section 7: Measurement Methods and Instrumentation

Several different methods were applied during the course of this investigation to measure Smart Meter RF fields. These included detailed antenna radiation pattern measurements of both end point and cell relay Smart Meters in the Itron anechoic chamber facility, survey type measurements used at close and far distances from single meters and groups of meters, such as in the Itron meter farm and at residences in California, drive-through type surveys in neighborhoods in which Smart Meters had been deployed, instrumentation comparison measurements using special Smart Meters programmed for the occasion and measurements of the attenuation provided by various forms of metal lath (commonly used in construction of stucco homes).

In the Itron facility, pattern measurements were accomplished using a sophisticated system that permits orientation of a Smart Meter in 15 degree increments in all possible directions using a dual axis rotating system as shown in Figure 7-1. Associated instrumentation included a spectrum analyzer (Agilent Model E4405B (SN US40240612)) as the detector connected to a sense antenna (ETS Model 3115 double ridge guide horn (SN 0005-6166)) inside the anechoic chamber with instrumentation interfaced with a systems controller (Sunol Sciences Model SC104V). Data acquisition and analysis software provided for analysis and graphic display of measured antenna patterns (MI-Technologies Model MI-3000 workstation). Figure 7-2 shows the interior of the anechoic chamber with the reception horn antenna used to receive the signal emitted by the meter.

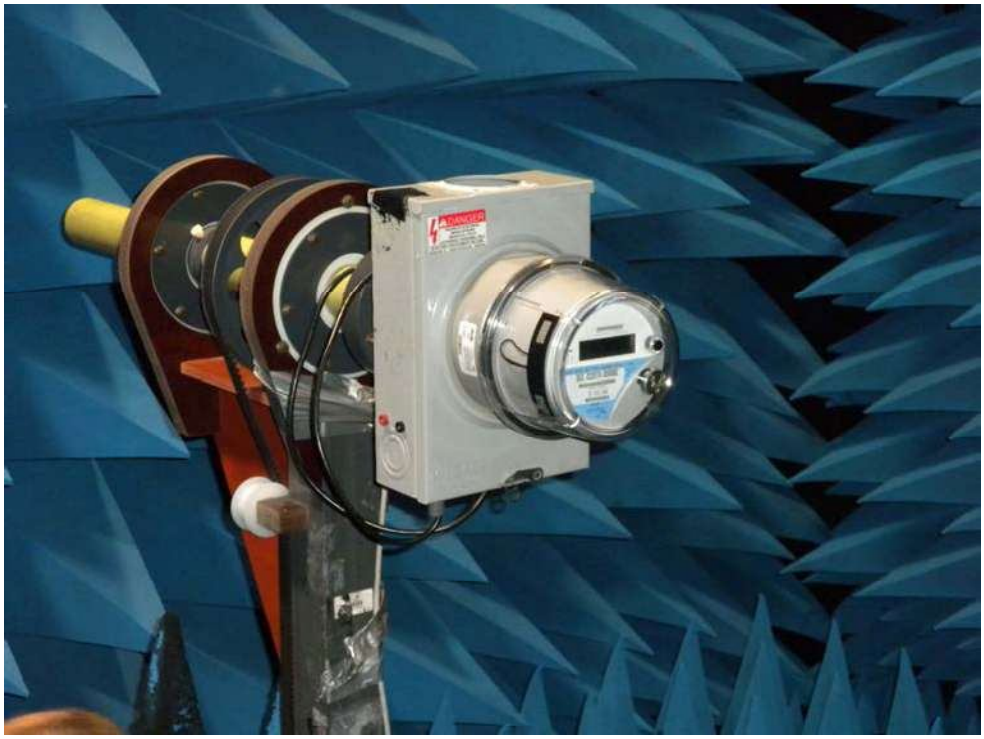

Figure 7-1
Close up view of dual axis antenna positioner system used to obtain antenna patterns of Smart Meter transmitters.

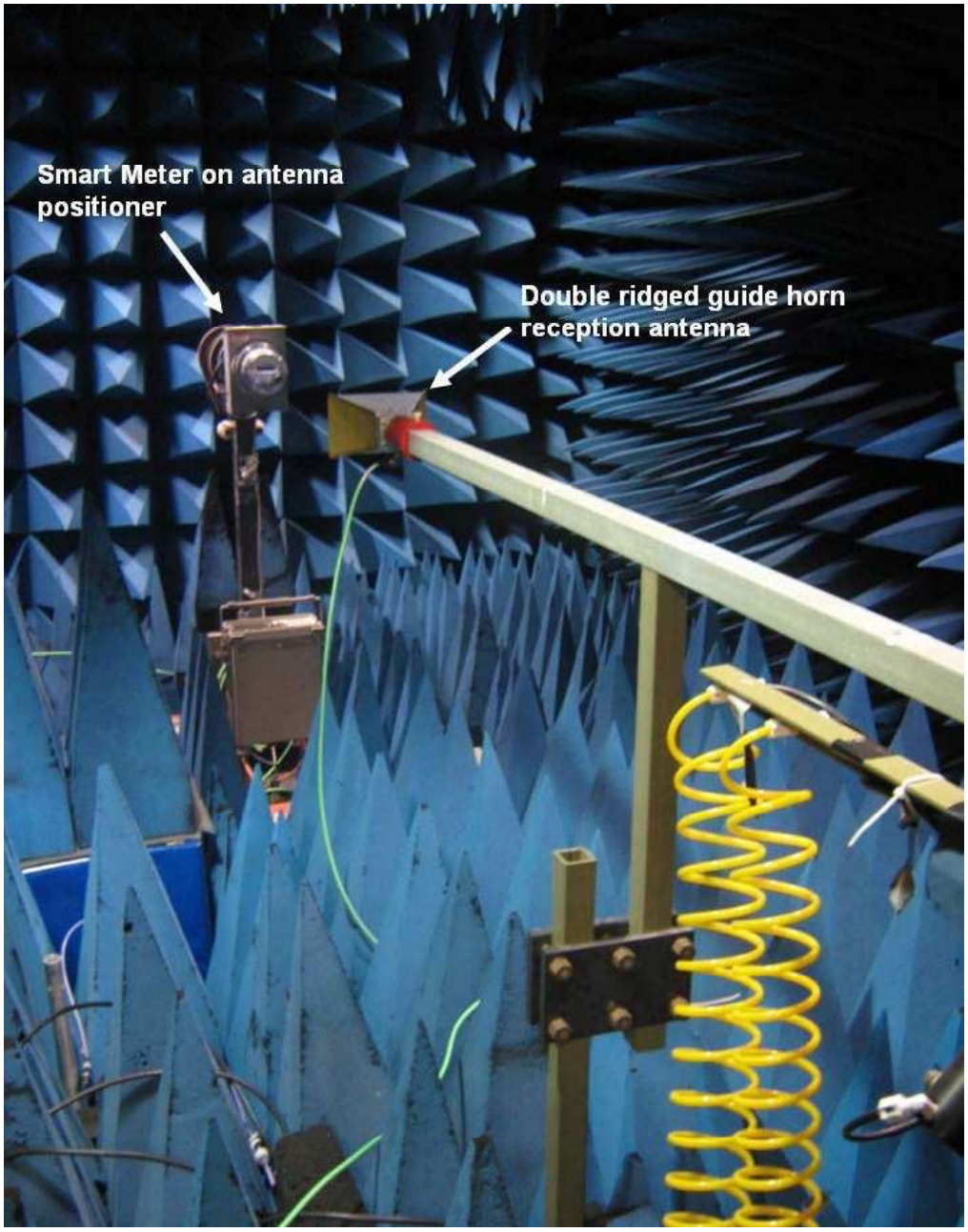

Figure 7-2
Interior of anechoic chamber showing reception horn antenna with Smart Meter on antenna positioner in background. During pattern measurements, the spectrum analyzer shown below the Smart Meter is removed.

Figure 7-3 shows the measurement instrumentation used for collecting and analyzing antenna pattern data. Smart Meters, when measured in the anechoic chamber, were installed in a metal meter box (Milbank Type 3R meter enclosure) supported on a dual axis rotator system (see Figure 7-1 for a close-up photo of the dual axis rotator system and meter box). Calibration signals could be injected into the spectrum analyzer with a separate signal generator (Agilent Model E4432B).

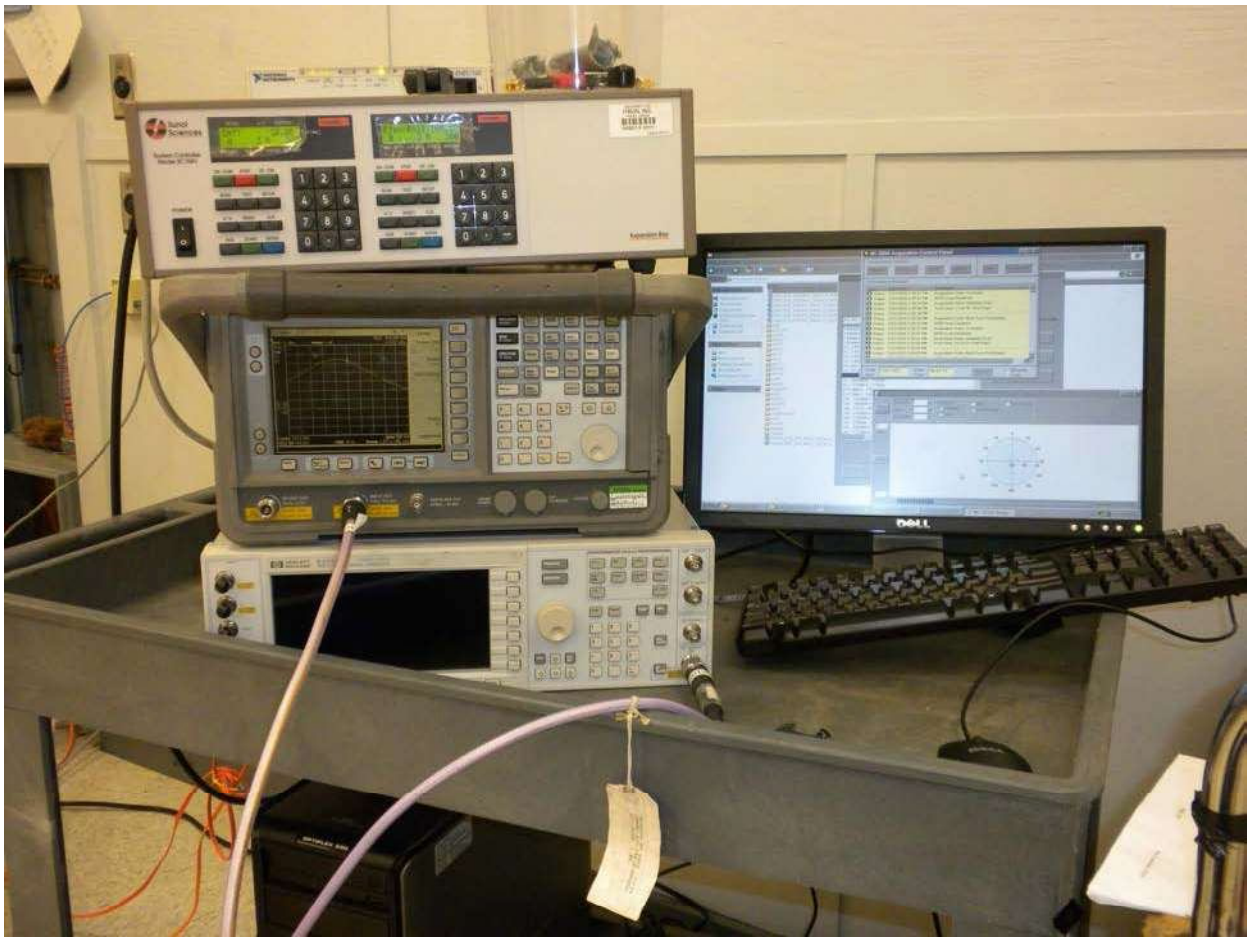

Figure 7-3
Instrumentation system for acquiring antenna pattern data.

All Smart Meter pattern measurements were performed in the Itron anechoic chamber. The interior of the shielded (0.2" metal) chamber measures 16 feet wide, 25 feet long and 12 feet high and is lined with anechoic material. The anechoic nature of the chamber provides for a very low level of reflection of RF fields from the floor, walls and ceiling, minimizing any perturbation that such reflections could have on the measured pattern of the Smart Meter transmitter.

Other instrumentation used in field measurements included use of a broadband, frequency conformal, isotropic, electric field probe (Narda Model B8742D, SN 03002) used with a readout meter (Narda Model 8715, SN 01028). This probe exhibits a frequency shaped response that follows the shape of the maximum permissible exposure (MPE) limit established by the Federal Communications Commission (FCC).[10] Such a shaped response allows the meter to read out directly in terms of a percentage of the MPE, regardless of the frequency or frequencies of the incident RF field(s). The B8742D is designed for response across the spectrum from 300 kHz to 3 GHz and is specified to yield reliable readings as low as 0.6% of the FCC general public MPE. Under optimum conditions (low ambient RF noise and a thermally stable environment), the meter can be used to read even lower RF field levels. The broadband probe consists of three, small mutually orthogonal elements combined electrically to yield an output on the meter that represents the resultant RF field magnitude. The isotropic nature of the probe

[10] FCC rules

produces an output that is independent of the orientation of the probe within the field being measured, thereby accounting for all field components of any polarization. The meter and probe, shown in Figure 7-4, had been calibrated at the factory within the previous twelve months of its application in this project as recommended by the manufacturer. Appendix A provides the calibration certificates for the meter and probe.

Figure 7-4
Frequency shaped, isotropic, electric field probe and meter (Narda B8742D and Narda 8715 meter).

Besides the use of the broadband meter, a special spectrum analyzer system was also used. This instrument (Narda Model SRM-3006 selective radiation meter, SN A-0077) combines a spectrum analyzer with an isotropic antenna (Narda three-axis-antenna, E-Field, SN H-0100) such that spectral scans may be performed with the resultant RF field value displayed on the analyzer's screen. The resultant field value is represented as the vector sum of the three orthogonal polarization components of the field, similar to how the broadband probe works. Built into the spectrum analyzer is a digital representation of the frequency dependence of the FCC MPE values. The system automatically corrects the measured fields for this frequency dependence so that the indicated spectrum observed on its screen is expressed, again, as a percentage of the FCC MPE. Further, the instrument can be instructed to integrate across a desired frequency range so that the overall, equivalent RF field as a percentage of the MPE can be displayed. Since this instrument can also display both the peak and average value of the RF fields being measured, it can provide insight to the duty cycle of the Smart Meter emissions. The SRM-3006 is shown in Figure 7-5. Calibration certificates for the SRM-3006 are provided in Appendix A.

Figure 7-5
Selective radiation meter (Narda Model SRM-3006).

One other piece of equipment used in the project is a tiny band-specific spectrum analyzer designed for the 902-928 MHz band. This analyzer (Metageek Model Wi-Spy 900X) is a USB based instrument that is connected to a portable computer such as a laptop or notebook computer.[11] The instrument, shown in Figure 7-6, is designed for investigating RF signals in the 900 MHz range from an interference perspective. A similar instrument is available for measurements in the 2.4 GHz wireless network band. The use of this device was aimed at exploring its potential utility in measurement of Smart Meter emissions in view of its low cost. An associated software program (Chanalyzer version 3.4) creates displays of the measured spectrum and provides for analysis of the measured RF signals. Using the software, for example, allows for retention of the maximum detected field at any given moment as well as the average value over whatever observation period is desired. A unique aspect of this spectrum analyzer is that, in conjunction with the connected computer, it records the result of each individual spectrum scan on the computer's hard disc drive. These recorded spectra can then be "replayed" at a later time to observe what the spectrum looked like at any previous point in time. Further, the stored, accumulated scans can be converted to a spreadsheet format for subsequent, custom analysis of the measurement data. Figure 7-6 shows a yagi antenna that was used with the Wi-Spy analyzer to achieve a higher level of system sensitivity and provide directionality for identifying the location of specific RF sources. The spectrum displayed on the notebook computer in Figure 7-6 is that acquired in the Itron laboratory with a Smart Meter operating on a fixed

[11] Metageek, LLC, 423 N. Ancestor Place, Suite 180, Boise, ID 83704 www.metageek.net.

frequency with lower level 900 MHz signals in the background from other meters in the vicinity.

The Wi-Spy unit was used for measurements of the insertion loss of a simulated wall in the Colville measurements. It was also employed in measurements of Smart Meter transmission activity in California. The Wi-Spy 900X has a detection sensitivity of approximately -105 dBm in the 900 MHz band, an amazing achievement for a device costing less than $200US.

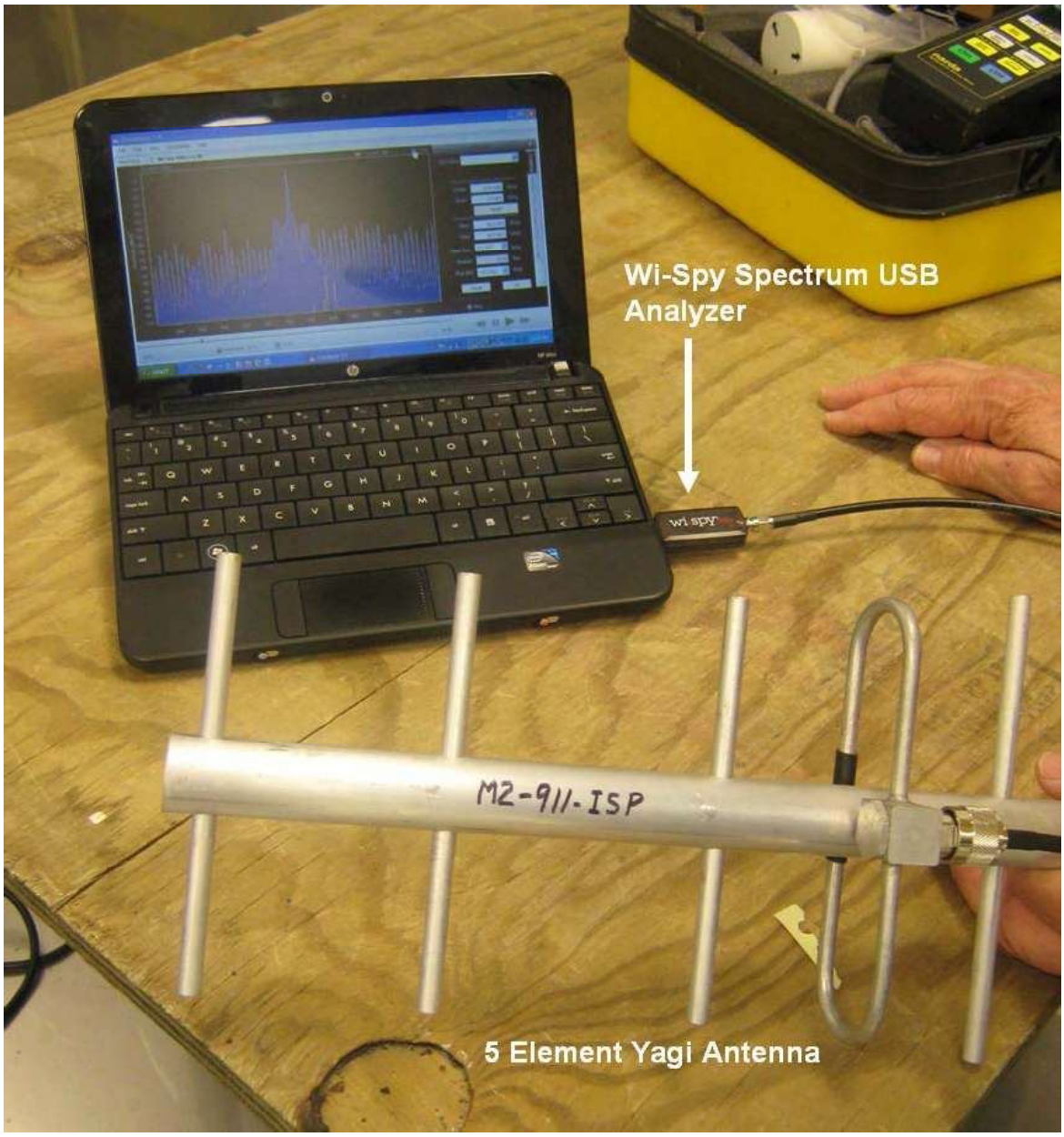

Figure 7-6
Wi-Spy USB spectrum analyzer connected to a notebook computer running software to operate the analyzer (Chanalyzer version 3.4) and an external yagi antenna.

Section 8: Laboratory Pattern Measurements

The radiation patterns for each antenna contained in the end point and access point Smart Meters were measured in the Itron anechoic chamber. This represented a total of six sets of patterns including: 900 MHz RF LAN in an end point meter and in a cell relay meter (access point), 2.4 GHz Zigbee radio in an end point meter and in a cell relay meter and the pattern of a cell relay meter using the GSM band (850 MHz) or the PCS band (1900 MHz). The 900 MHz RF LAN and 2.4 GHz Zigbee radios each have their own quarter-wave slot antennas that are etched on printed circuit boards inside the meter. The 900 MHz antenna is horizontal and located approximately 2.1 cm behind the front surface of the meter enclosure. When contained in a cell relay, the 900 MHz antenna is located approximately 15.1 cm from the front surface of the metal meter box in which it may be installed. The 2.4 GHz antenna is vertically oriented on its circuit board and is located approximately 2.5 cm behind the front surface of the meter enclosure; when installed in a cell relay, the antenna is approximately 14.7 cm in front of the metal meter box. A flexible dual band antenna is used for the cell relay function and it is adhered to the interior surface of the clear meter enclosure at a nominal nine o'clock position. The dual band antenna (AMR Under Glass Mount Antenna produced by WP Wireless)[12], shown in Figure 2-3, is approximately 2.5 cm wide with the front edge located approximately 0.4 cm from the front surface of the meter enclosure.

A feature of the Itron antenna pattern analysis system is the determination of the maximum isotropic effective radiated power (EIRP) for a particular amount of power being delivered to the antenna by the relevant transmitter. Because the Itron 900 MHz and 2.4 GHz transmitters are not designed for continuous operation (normal application in the Itron Smart Meters corresponds to a rather low duty cycle), all pattern measurements were obtained with the transmitters programmed to operate at a power level lower than their normal, maximum average power. This methodology helped avoid a slight decrease in transmitter output power after prolonged periods of transmitter activity during which the transmitters can heat up, insuring that the measured data was representative of the peak power that is achieved under normal operating conditions. Knowing the EIRP of each transmitter system in a meter, relative to the particular transmitter output power during the test, allowed subsequent RF field calculations to be scaled to actual maximum transmitter power levels. Acquiring a complete three dimensional antenna pattern requires almost two hours of measurement. The meter is repositioned every 15 degrees in both azimuth and elevation and measurements are made of both the horizontal and vertical polarization components of the emitted field. Through examination of the entire data set after the pattern has been measured, the single maximum value of field is converted to EIRP. This single value was used in most of the subsequent analyses in this report since it represents the EIRP that is associated with the strongest RF fields in the vicinity of the Smart Meter.

Measured radiation patterns for the 900 MHz RF LAN transmitter configured in an end point meter, Model CL200 (Itron #62_305_199, SCE #222010-273721) are shown in Figures 8-1 – 8-4. In these figures, a drawing representing the Smart Meter as mounted in a meter box is shown for reference to the meter orientation. These patterns were determined at near-mid-band frequency of 914.8 MHz. Figure 8-1 represents the azimuth plane pattern of the 900 MHz emissions. This particular pattern is for a horizontal plane running through the meter and as viewed from the bottom of the meter. The pattern data are referenced to 0 dB at the point of maximum field, close to 0°, with each dotted line, in this particular pattern, representing a 20 dB variation in signal level. Three curves are shown in the figure; one representing the pattern for the horizontally polarized component of the field (the black curve), one representing the vertical polarization component of the field (the blue curve) and one representing the total field produced by the composite sum of both the horizontal and vertical components (the red curve). From an exposure

[12] WP Wireless - A Division of World Products Inc., 19654 Eight Street East, Sonoma, CA 95476 www.wp-wireless.com.

assessment perspective, the total pattern is of more relevance for evaluating RF fields relative to human exposure limits. The pattern shows a reduction of radiated field, generally, to the rear of the meter being between 10 and 20 dB less than the values to the front of the meter. A colorized picture in Figure 8-4 illustrates the azimuth plane representation of total EIRP of the 900 MHz RF LAN transmitter.

Elevation plane patterns for the 900 MHz RF LAN transmitter are shown in Figures 8-3 and 8-4 with Figure 8-3 representing the patterns of the horizontal and vertical polarization components and the composite field (total), similar to the figure for the azimuth plane. Figure 8-4 illustrates the elevation plane representation of total EIRP of the 900 MHz RF LAN transmitter in an end point meter. In the elevation plane pattern, it can be seen that the maximum field is directed slightly upwards at about 30º rather than perfectly straight out toward the front of the meter.

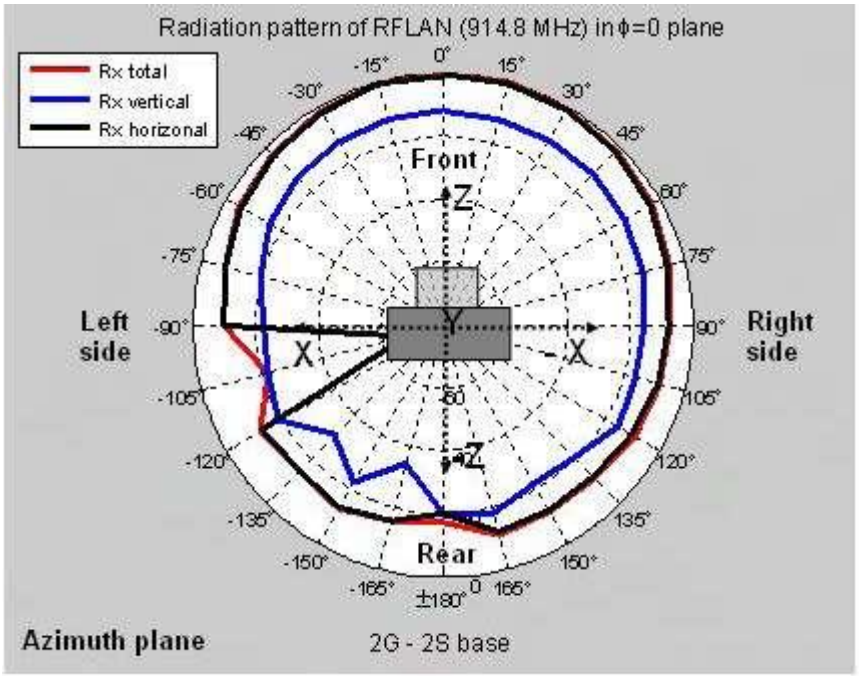

Figure 8-1
Azimuth plane pattern of the 900 MHz RF LAN transmitter configured in an end point meter showing the horizontal, vertical and total pattern as viewed from bottom of meter. The scale is in dB with the maximum field at the outer edge of the pattern circle.

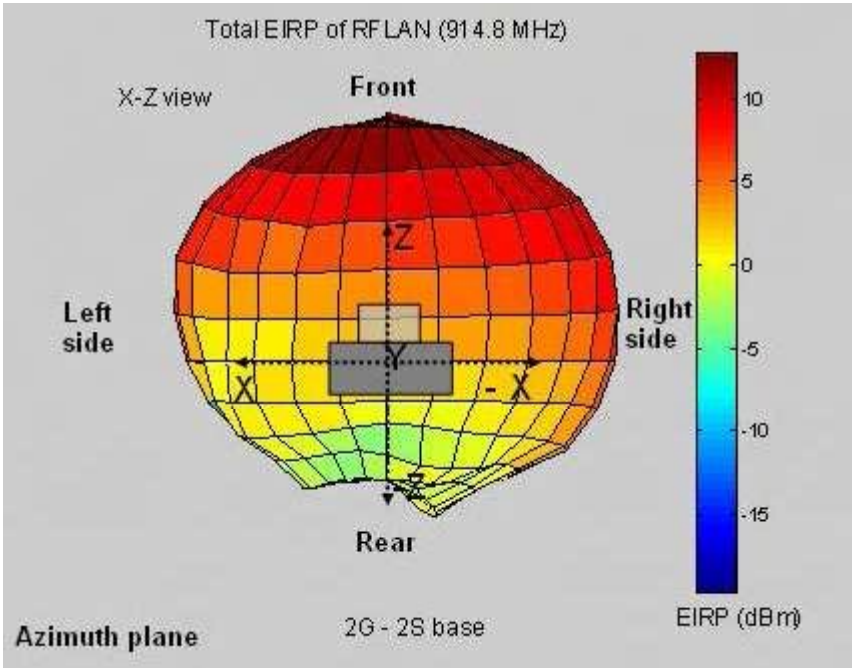

Figure 8-2
Azimuth plane view of the total EIRP of the 900 MHz RF LAN transmitter configured in an end point meter.

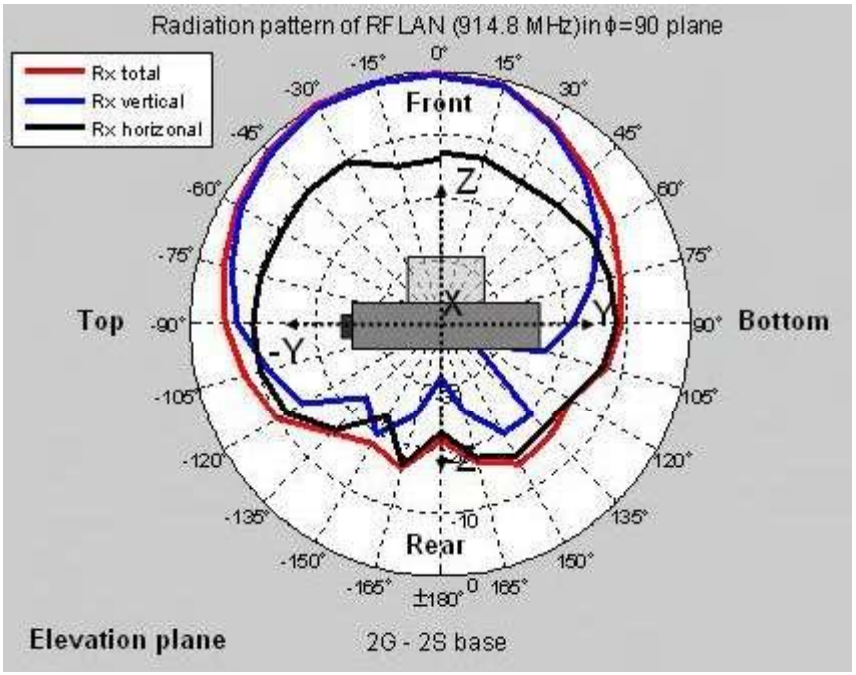

Figure 8-3
Elevation plane pattern of the 900 MHz RF LAN transmitter in an end point meter showing the horizontal, vertical and total pattern. The scale is in dB with the maximum field at the outer edge of the pattern circle.

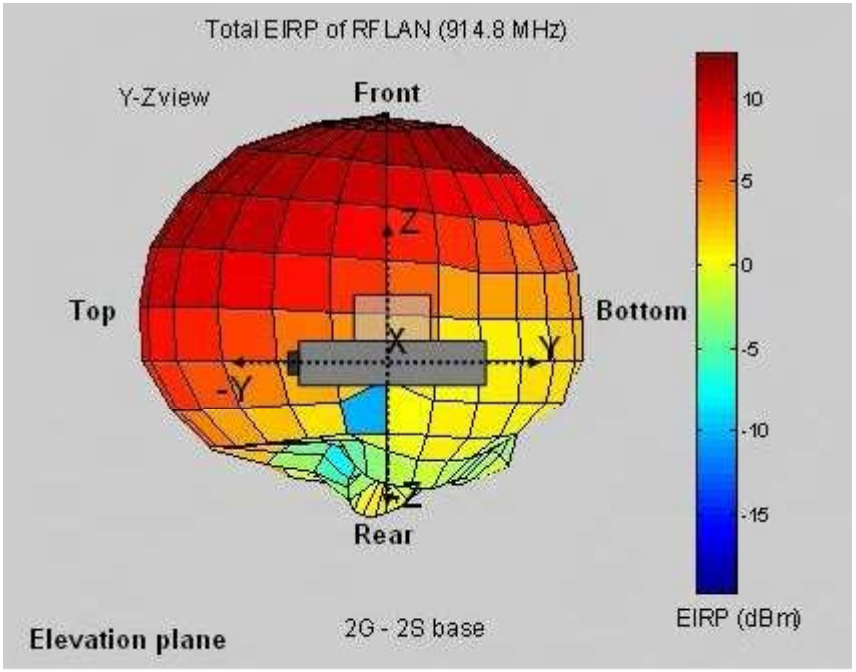

Figure 8-4
Elevation plane view of the total EIRP of the 900 MHz RF LAN transmitter in an end point meter.

Similar to the pattern measurements for the 900 MHz RF LAN transmitter in an end point meter, measured radiation patterns for the 900 MHz RF LAN transmitter configured in a cell relay meter, Model C2SORD, (Itron #661_912_646, SCE #222070-000082) are shown in Figures 8-5 – 8-8. The rationale behind documenting the pattern of the same transmitter and antenna type, but when installed in a cell relay, was to examine any differences that might be apparent that could be caused by the slightly different distance that the antenna would be relative to the front of the metal meter box.

Figures 8-5 and 8-6 represent the azimuth plane patterns of the 900 MHz cell relay emissions and total EIRP respectively. The pattern in Figure 8-5 shows a reduction of radiated field, generally, to the rear of the meter being between 10 and 20 dB less than the values to the front of the meter, similar to the 900 MHz RF LAN transmitter in an end point meter. A colorized picture in Figure 8-6 illustrates the azimuth plane representation of total EIRP of the 900 MHz RF LAN transmitter.

Elevation plane patterns for the 900 MHz RF LAN transmitter in a cell relay are shown in Figures 8-7 and 8-8 with Figure 8-7 representing the patterns of the horizontal and vertical polarization components and the composite field (total), similar to the figure for the azimuth plane. Figure 8-8 illustrates the elevation plane representation of total EIRP of the 900 MHz RF LAN transmitter in a cell relay. Similar to the upward maximum radiation direction for the end point 900 MHz RF LAN transmitter, it can be seen that the maximum field is directed slightly upwards at about 30° rather than perfectly straight out toward the front of the meter.

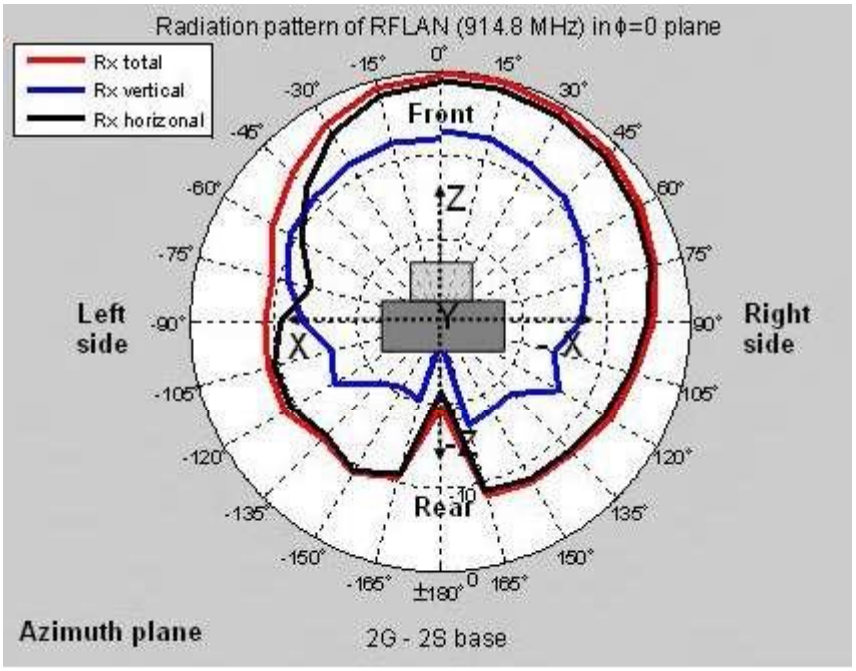

Figure 8-5
Azimuth plane pattern of the 900 MHz RF LAN transmitter in a cell relay showing the horizontal, vertical and total pattern as viewed from bottom of meter. The scale is in dB with the maximum field at the outer edge of the pattern circle.

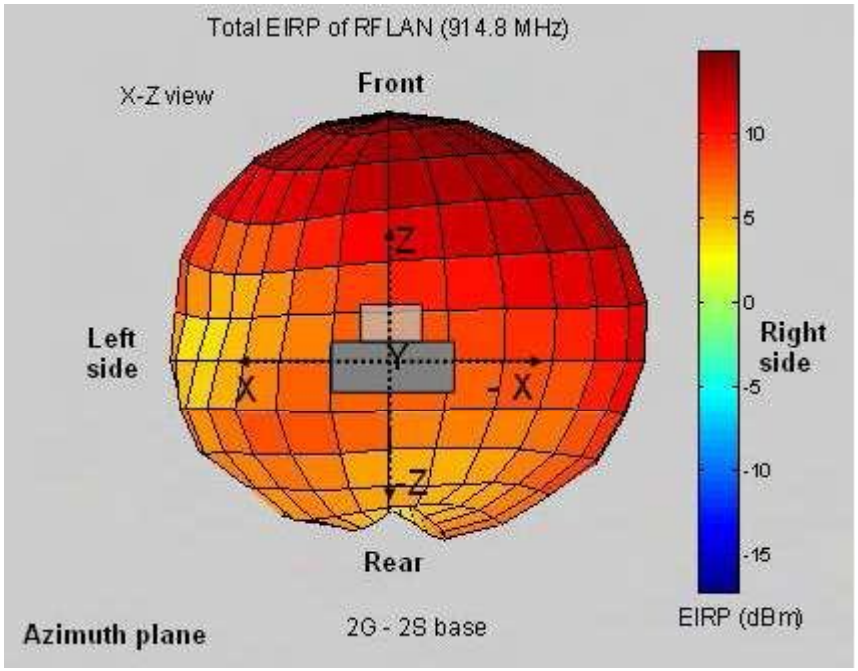

Figure 8-6
Azimuth plane view of the total EIRP of the 900 MHz RF LAN transmitter configured in a cell relay.

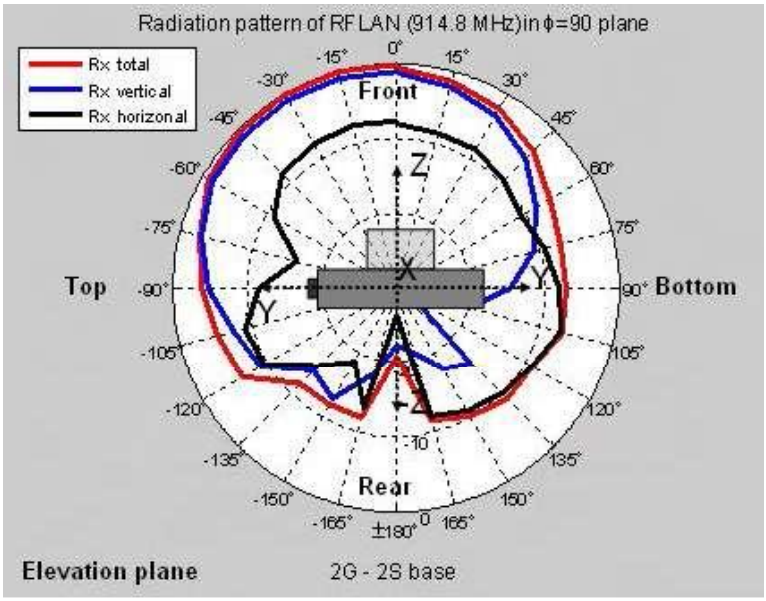

Figure 8-7
Elevation plane pattern of the 900 MHz RF LAN transmitter in a cell relay meter showing the horizontal, vertical and total pattern. The scale is in dB with the maximum field at the outer edge of the pattern circle.

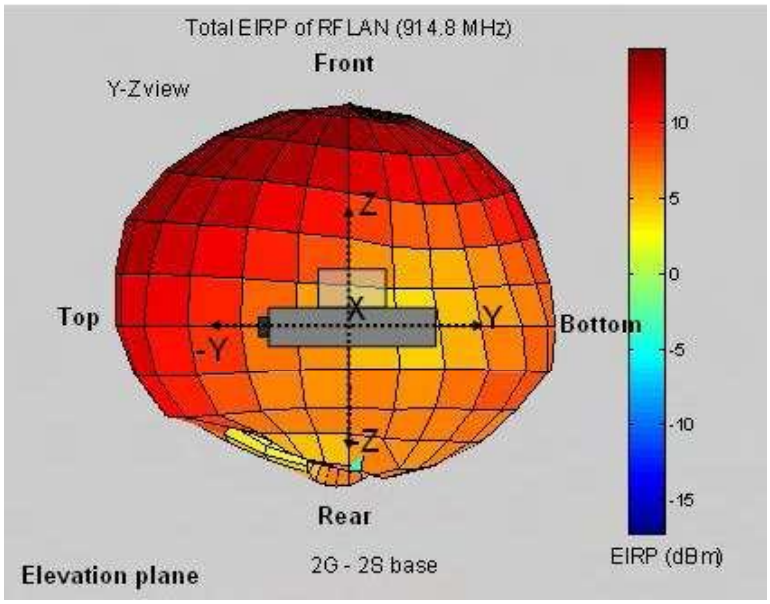

Figure 8-8
Elevation plane view of the total EIRP of the 900 MHz RF LAN transmitter in a cell relay meter.

A series of similar pattern measurements of the 2.4 GHz Zigbee radio configured in an end point meter, Model CL200 (Itron #62_305_199, SCE #222010-273721) are shown in Figures 8-9 – 8-12. Figure 8-9 shows the azimuth plane pattern; the azimuth plane pattern total EIRP is shown in Figure 8-10. Figure 8-9 shows that the direction of the maximum radiated field is very slightly canted to the right side of the meter, as viewed from the front, at about 15°.

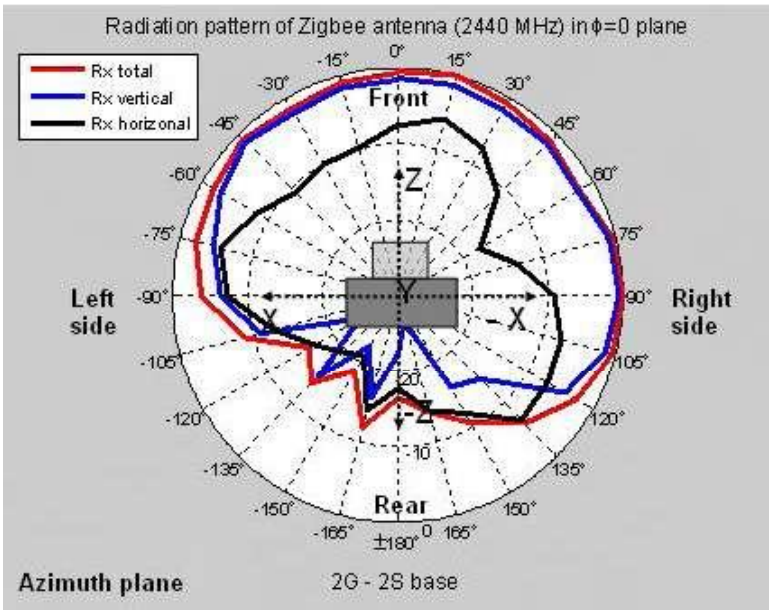

Figure 8-9
Azimuth plane pattern of the 2.4 GHz Zigbee transmitter configured in an end point meter showing the horizontal, vertical and total pattern as viewed from bottom of meter. The scale is in dB with the maximum field at the outer edge of the pattern circle.

The elevation plane pattern seen in Figure 8-11 reveals a tendency for the 2.4 GHz emission in the end point meter to be directed upwards, above the midline of the meter with a maximum field at approximately 45 to 60º.

Similar patterns were measured for the 2.4 GHz Zigbee radio in a cell relay meter (Model C2SORD, Itron # 61_912_646, SEC # 222070-000082). These patterns are shown in Figures 8-13 – 8-16 for the azimuth and elevation planes for relative field and total EIRP respectively. Figure 8-15 shows the tendency for an upward direction for emitted fields at approximately 45º.

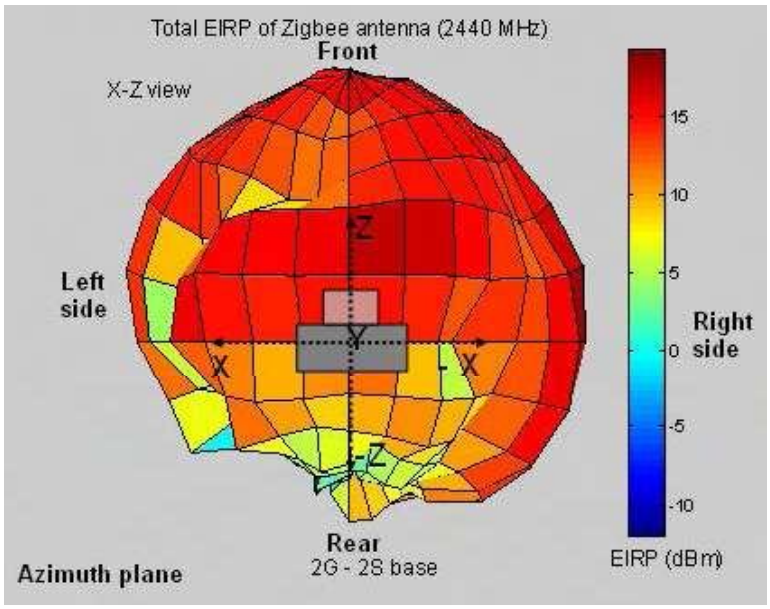

Figure 8-10
Azimuth plane view of the total EIRP of the 2.4 GHz Zigbee transmitter configured in an end point meter.

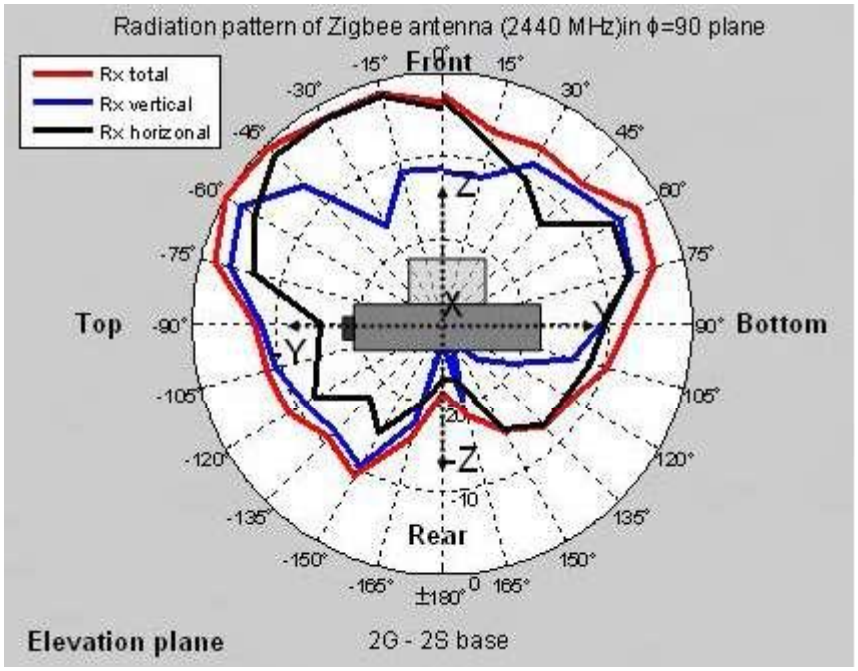

Figure 8-11
Elevation plane pattern of the 2.4 GHz Zigbee radio in an end point meter showing the horizontal, vertical and total pattern. The scale is in dB with the maximum field at the outer edge of the pattern circle.

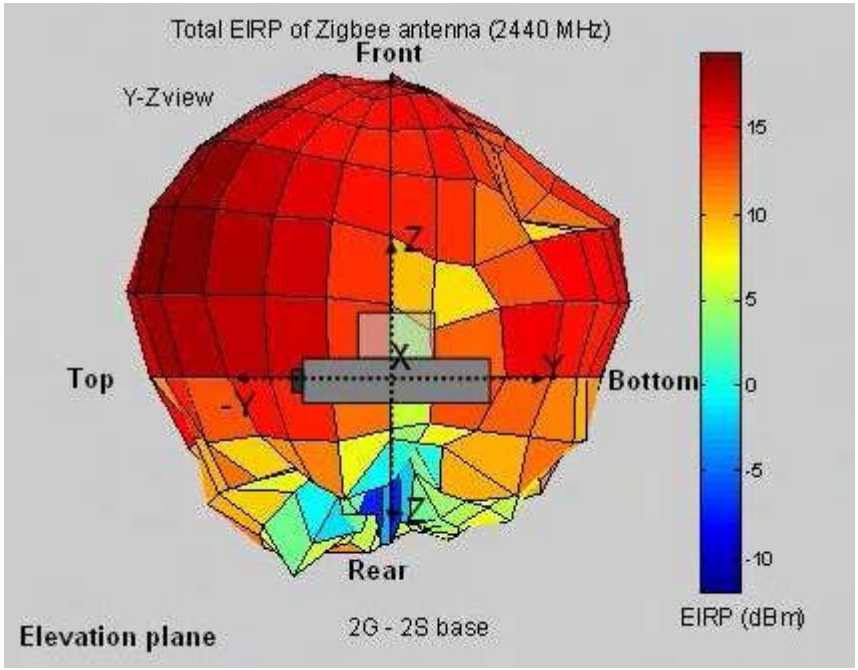

Figure 8-12
Elevation plane view of the total EIRP of the 2.4 GHz Zigbee radio in an end point meter.

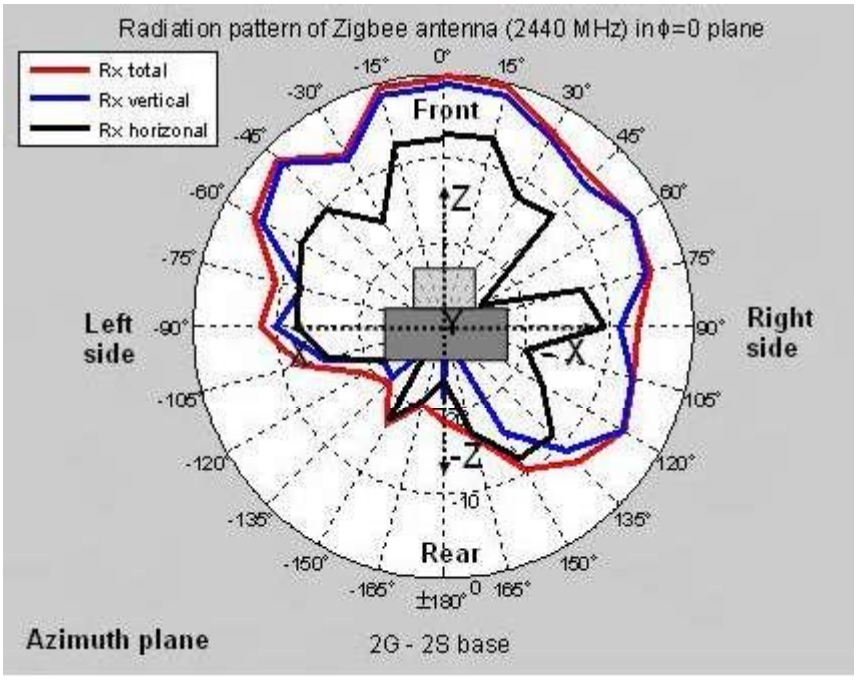

Figure 8-13
Azimuth plane pattern of the 2.4 GHz Zigbee transmitter configured in a cell relay meter showing the horizontal, vertical and total pattern as viewed from bottom of meter. The scale is in dB with the maximum field at the outer edge of the pattern circle.

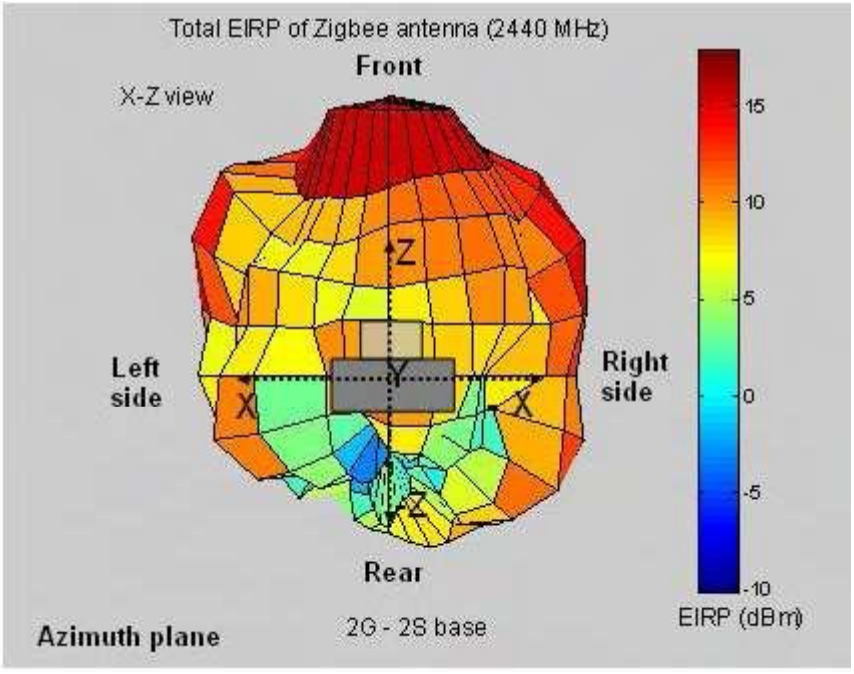

Figure 8-14
Azimuth plane view of the total EIRP of the 2.4 GHz Zigbee transmitter configured in a cell relay meter.

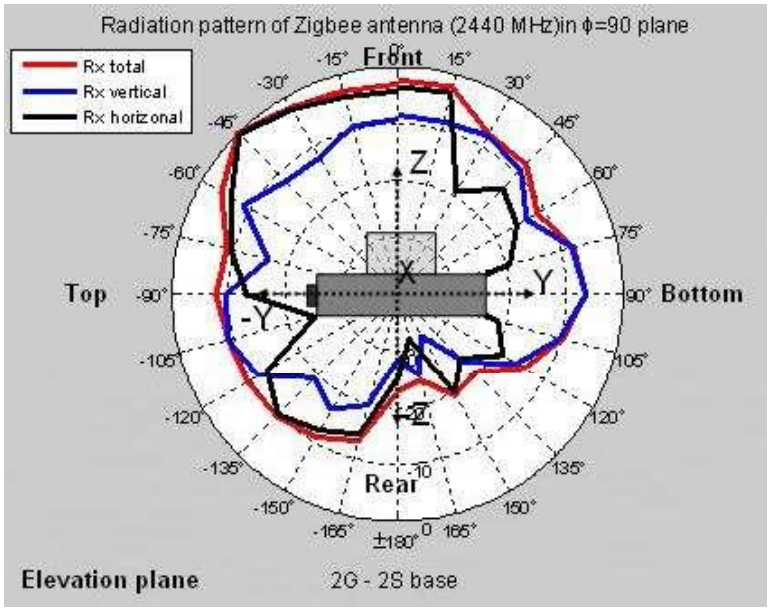

Figure 8-15
Elevation plane pattern of the 2.4 GHz Zigbee radio in a cell relay meter showing the horizontal, vertical and total pattern. The scale is in dB with the maximum field at the outer edge of the pattern circle.

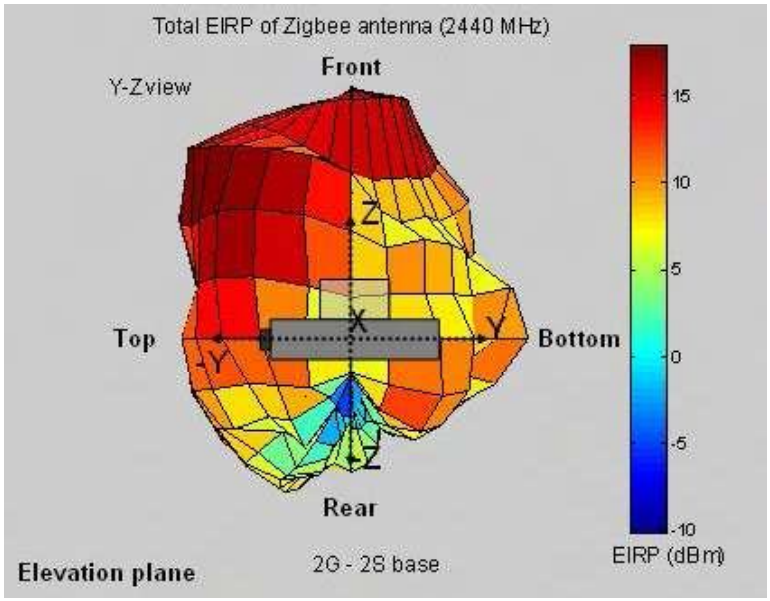

Figure 8-16
Elevation plane view of the total EIRP of the 2.4 GHz Zigbee radio in a cell relay meter.

For completeness, patterns of the cell relay cellular and PCS band antennas were also determined during this documentation. Figures 8-17 and 8-18, based on measurements at a frequency of 836.6 MHz for the cell relay Model C2SORD (Itron # 61_912_646, SCE # 222070-00082, GSM # 12460), show azimuth patterns for the relative field and total EIRP. Elevation patterns for the 836.6 MHz GSM transmitter are shown in Figure 8-19 and 8-20.

Antenna patterns were measured for the GSM radio operated in the 1900 MHz band as well. Figures 8-21 and 8-22 represent the azimuth patterns and Figures 8-23 and 8-24, the elevation patterns. The patterns were measured with the transmitter operating on a frequency of 1880 MHz.

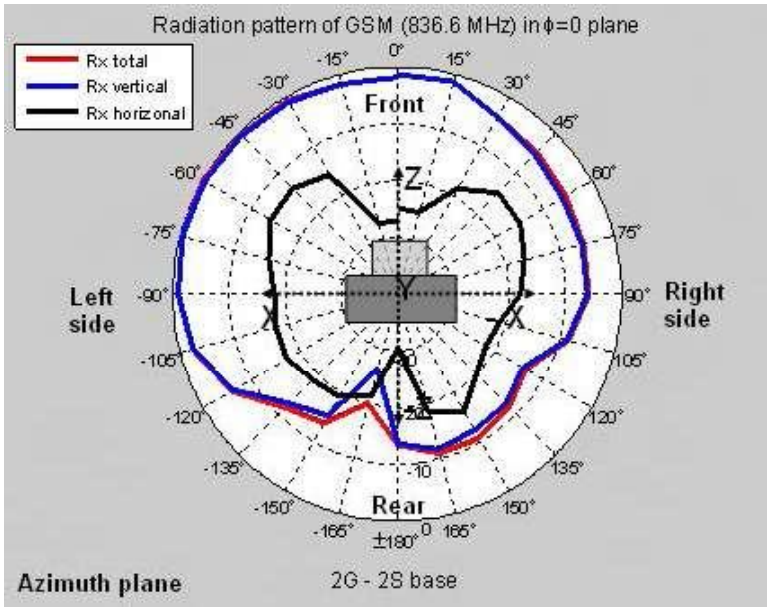

Figure 8-17
Azimuth plane pattern of the 836.6 MHz GSM cellular transmitter in a cell relay meter showing the horizontal, vertical and total pattern as viewed from bottom of meter. The scale is in dB with the maximum field at the outer edge of the pattern circle.

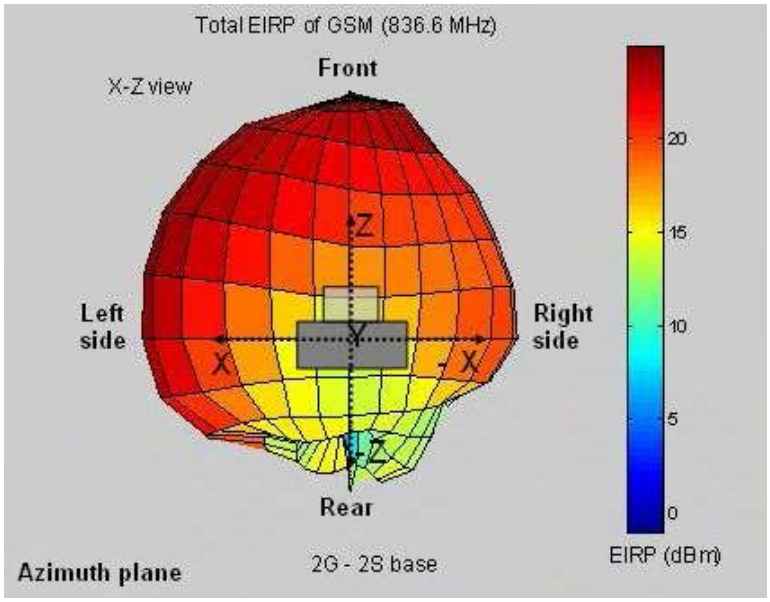

Figure 8-18
Azimuth plane view of the total EIRP of a GSM 836.6 MHz cellular radio in a cell relay meter.

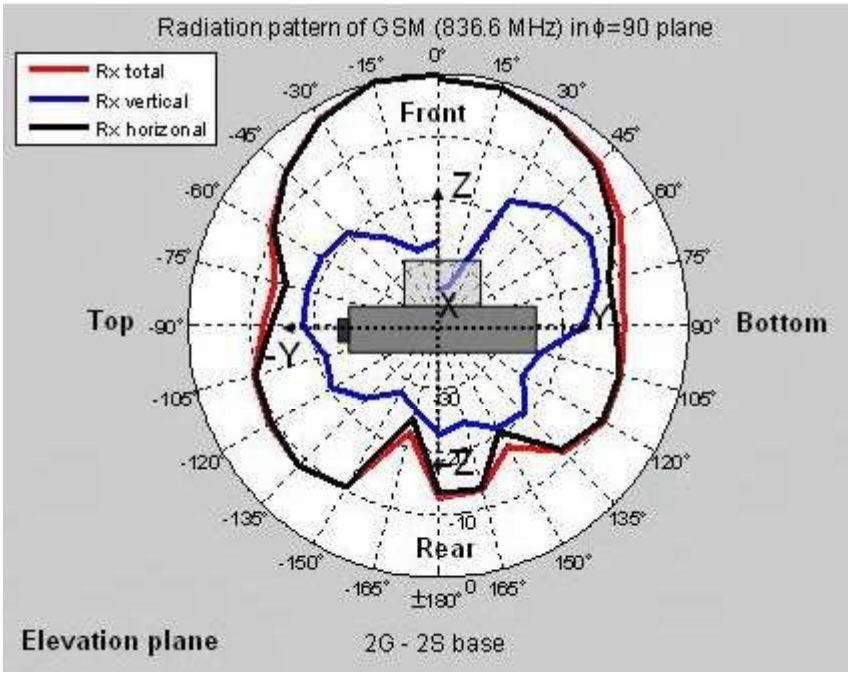

Figure 8-19
Elevation plane pattern of the 836.6 MHz GSM cellular transmitter in a cell relay meter showing the horizontal, vertical and total pattern as viewed from bottom of meter. The scale is in dB with the maximum field at the outer edge of the pattern circle.

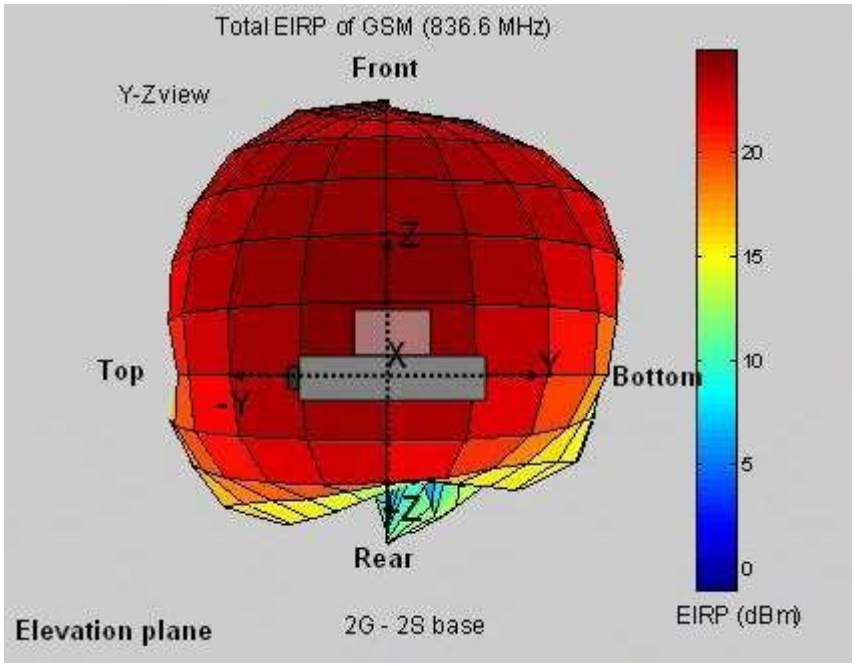

Figure 8-20
Elevation plane view of the total EIRP of a GSM 836.6 MHz cellular radio in a cell relay meter.

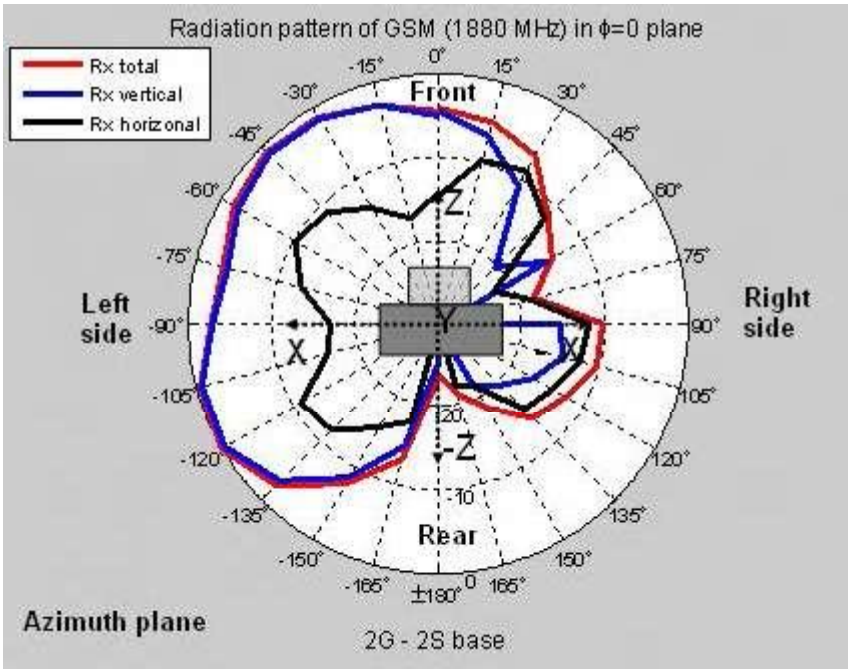

Figure 8-21
Azimuth plane pattern of the 1880 MHz GSM PCS transmitter in a cell relay meter showing the horizontal, vertical and total pattern as viewed from bottom of meter. The scale is in dB with the maximum field at the outer edge of the pattern circle.

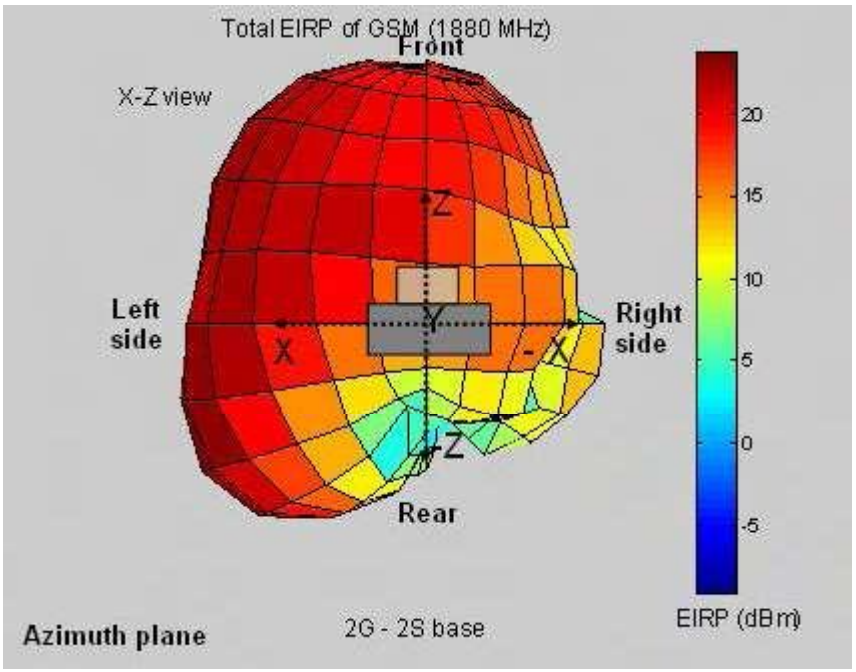

Figure 8-22
Azimuth plane view of the total EIRP of a GSM 1880 MHz PCS radio in a cell relay meter.

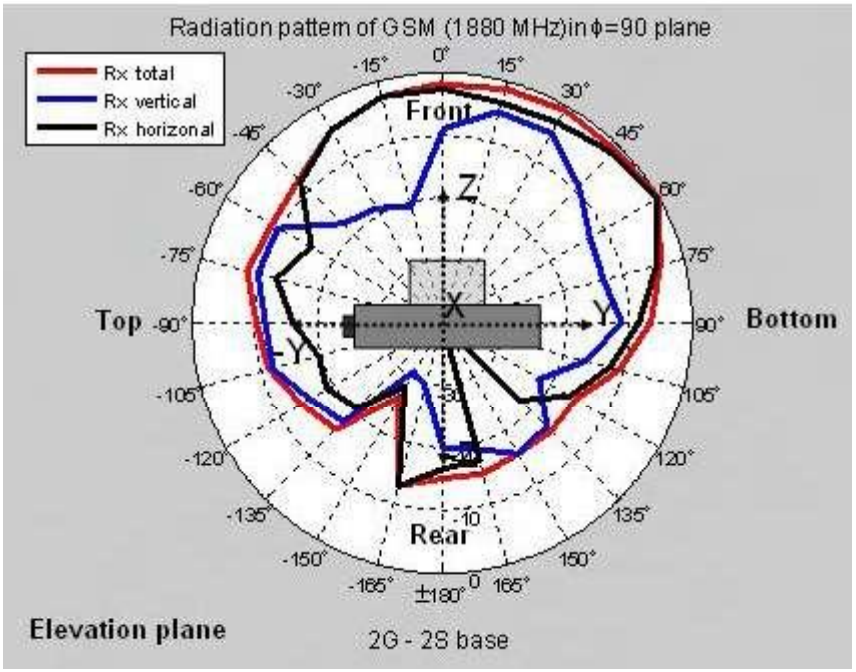

Figure 8-23
Elevation plane pattern of the 1880 MHz GSM PCS transmitter in a cell relay meter showing the horizontal, vertical and total pattern as viewed from bottom of meter. The scale is in dB with the maximum field at the outer edge of the pattern circle.

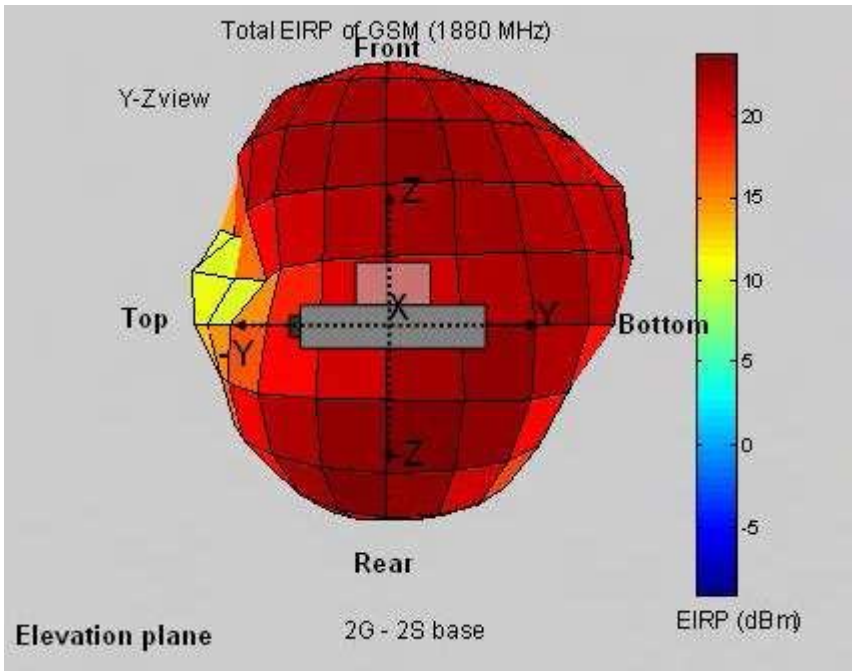

Figure 8-24
Elevation plane view of the total EIRP of a GSM 1880 MHz PCS radio in a cell relay meter.

From analysis of each set of pattern measurement data, the EIRP was determined for a given transmitter output power delivered to the antenna. Table 8-1 summarizes the maximum EIRP found for each of the different measurement conditions described above. Maximum EIRP is the absolute greatest value of EIRP found from all of the pattern measurements at any angle. As observed from the pattern data shown above, the maximum EIRP may not be aligned with a line directly normal to the face of the Smart Meter. In each case, the maximum EIRP has been referenced to one milliwatt. Hence, subsequent analyses making use of the maximum EIRP simply require adjusting the EIRP value for the actual transmitter power expected under normal operating conditions. In Table 8-1, the nominal specified transmitter power values are given in the next to last column and the maximum transmitter EIRP, referencing the nominal specified transmitter power, is given in the last column.

Table 8-1
Summary of antenna measurement data

Transmitter description	Max test EIRP (dBm)	Test power (dBm)	Gain (dBi)	Max TX P[a] (dBm)	Max TX EIRP[b] (dBm)
End point RF LAN, 914.8 MHz	12.8	9.9	2.9	24.0	26.9
Cell Relay RF LAN, 914.8 MHz	15.0	14.1	0.9	24.0	24.9
End point Zigbee, 2440 MHz	19.4	15.2	4.2	18.3	22.5
Cell Relay Zigbee, 2440 MHz	17.9	12.8	5.1	18.3	23.4
Cell Relay GSM, 836.6 MHz	24.9	23.1	1.8	31.8	33.6
Cell Relay GSM, 1880 MHz	23.9	22.3	1.6	28.7	30.3

[a]Nominal specified transmitter power

[b]The maximum transmitter EIRP assumes the nominal specified transmitter power.

Section 9: Smart Meter Field Measurements

The following narrative describes measurements of RF fields produced by Smart Meters that were obtained at the Itron meter farm in West Union, South Carolina, at residential settings in California and instrumentation comparisons and attenuation measurements for some selected materials including a simulated residential wall.

Meter farm measurements

A major feature of the Itron facility is a large "Smart Meter farm". An aerial view of the geographic layout of the meter farm is seen in Figure 9-1. Approximately 20 acres comprise the installation of some 7000 Smart Meters for evaluating the performance of Itron's meters in mesh networks. For the most part, Smart Meters are organized in groups of ten meters on wooden racks as shown in Figure 9-2. The meters are arranged in two rows of five meters each, one above the other. The rack is 48 inches wide with the meters mounted so that there is a 16 inch vertical spacing of the two rows of meters, center to center. The bottom row of meters is nominally 48 inches above the ground. In one area in which area survey measurements were performed, the meter racks were found to be 16 feet apart, side to side, with the rows of racks 20.5 feet apart. Broadband probe and spectrum analyzer field measurements were performed on both individual Smart Meters and groups of ten meters comprising a rack.

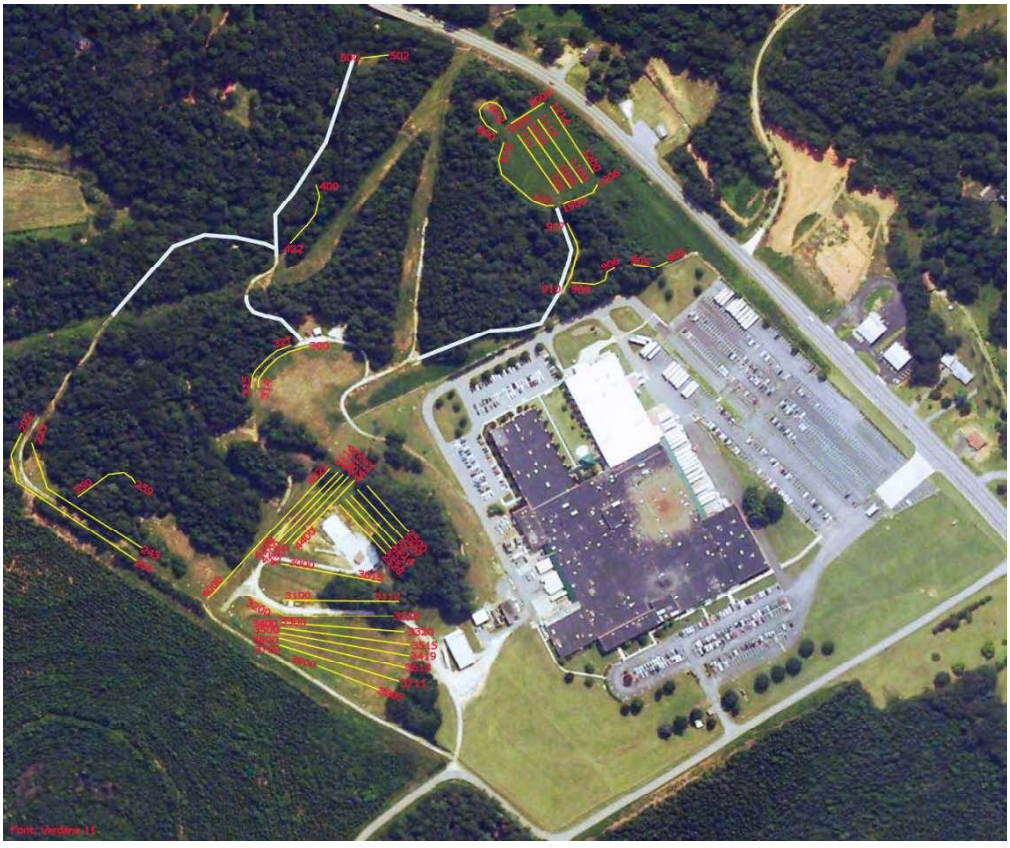

Figure 9-1
Aerial view of the Itron meter farm in West Union, SC. Yellow lines represent rows of Smart Meters grouped, generally, as racks of ten meters each. Photo courtesy of Itron.

Figure 9-2
Typical rack of ten meters shown in the western part of the Itron meter farm.

Individual meters

Initial field measurements in the meter farm were made using the broadband field probe (Narda Model B8742D). An objective of the broadband measurements was to assess what effect multiple Smart Meters might have on the measured RF field magnitude. The measurement approach sought to, first, examine the uniformity of measured field strengths among ten end point meters. To accomplish this, Itron programmed each of the ten meters to enter the continuous transmit mode of operation with three of the ten meters programmed to operate on the lowest frequency (L) in the 900 MHz RF LAN band (902.25 MHz), three meters to operate at the mid-band (M) frequency (914.75 MHz) and the remaining four meters to operate at the upper most channel (H) in the band (927.75 MHz). Measurements were performed over a period of time during which the transmitter power was not expected to diminish due to transmitter heating. RF sources on precisely the same frequency and physically coincident with one another could lead to the possibility of phase addition or phase cancellation of the resultant RF field at specific points. In the measurement method used, the ten meters were physically distributed over a distance of up to 48 inches (this being equivalent to approximately four wavelengths in the 900 MHz band and approximately ten wavelengths in the 2.4 GHz band). Further, while individual meter frequencies on specific channels are very close to one another, they are not exactly the same due to crystal drift in the oscillator circuitry. Hence, the likelihood of RF fields from various meters actually being perfectly coherent is extremely small. Further, because of the measurement technique of scanning a planar area for the maximum,

peak RF field at each distance from the rack of meters, whether constructive or destructive phase addition may have existed, become irrelevant.

Each of the ten Smart Meter locations within a rack were identified with a letter from A to J and a location for each of these meters was determined as shown in Figure 9-3. The rationale behind this arrangement was to try to group meters in such a way as to enhance the potential for RF field contribution from adjacent meters to the extent feasible when all ten meters were installed in the rack and actively transmitting. Initially, however, measurements were started with one meter only in the A position and successively replacing it with each of the other meters so that, ultimately, each of the ten meters had been installed in the A meter socket and the RF field was measured with the broadband probe. Each of the meter positions is also labeled as to the frequency of the associated meter as L, M or H, designating its frequency.

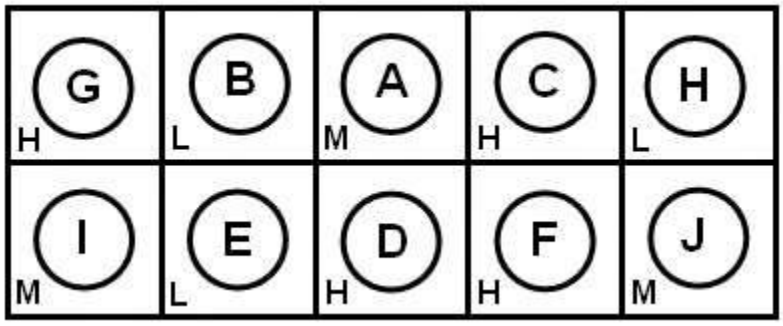

Figure 9-3
Layout of Smart Meter rack showing designated meter locations and frequency of various meters (L, M and H - see text) for the 900 MHz RF LAN and 2.4 GHz Zigbee transmitters.

Broadband field probe measurements were taken with the probe touching the surface of the meter face, with the probe at 20 cm from the meter face, with the probe at 30 cm from the meter face and, finally, with the probe behind the meter rack with the probe in contact with the rear of the rack, immediately behind meter position A. Use of the broadband field probe with a cardboard spacer affixed to the probe at the rack of ten meters is illustrated in Figure 9-4. These measurement results are given in Table 9-1. Each of the indicated values was obtained by multiplying the meter reading by the manufacturer's calibration correction factor applicable at 915 MHz of 0.67. Surface field measurements with an isotropic probe must be interpreted with care due to the potential for erroneous readings. Nonetheless, because others may apply such probes in this fashion, it was deemed relevant to examine what kind of response would be exhibited when contacting the probe to the Smart Meter.

Figure 9-4
Use of the broadband field probe with a cardboard spacer attached to the probe near meters in a rack of ten meters.

Table 9-1
Measurements of 900 MHz RF LAN emissions of individual Smart Meters installed in meter position A in the meter rack with the broadband field probe.

	RF field measured (% of FCC public MPE)				
Meter	Frequency	Surface	20 cm	30 cm	Rear surface
A	M	45.9	4.2	2.2	0.0
B	L	65.3	6.9	5.5	0.0
C	H	18.5	1.6	1.1	0.0
D	H	16.8	2.3	1.6	0.0
E	L	53.7	5.6	4.2	0.0
F	H	19.1	2.1	0.7	0.0
G	H	20.5	1.9	0.7	0.0
H	L	48.6	4.9	3.5	0.0
I	M	45.7	4.6	2.8	0.0
J	M	29.6	3.1	1.9	0.0

In examining the results, two issues are immediately apparent. First the instrument readings appear to be related to the channel to which the 900 MHz RF LAN transmitter was programmed. This can be more easily seen in Figure 9-5. Clearly, the indicated field magnitude is related to the frequency of the 900 MHz RF LAN transmitter; the highest readings are correlated with the lowest frequency and the lowest readings are correlated with the highest transmitter frequency. From data in Table 9-1 at 20 cm, the mean value of readings of the L meters is 5.8% while the mean value of the readings of the H meters is 2.0%; this corresponds to a ratio of 2.9 or a total range of about 4.6 dB from the lowest to the highest readings, i.e., a variation of ±2.3 dB relative to the band center frequency.

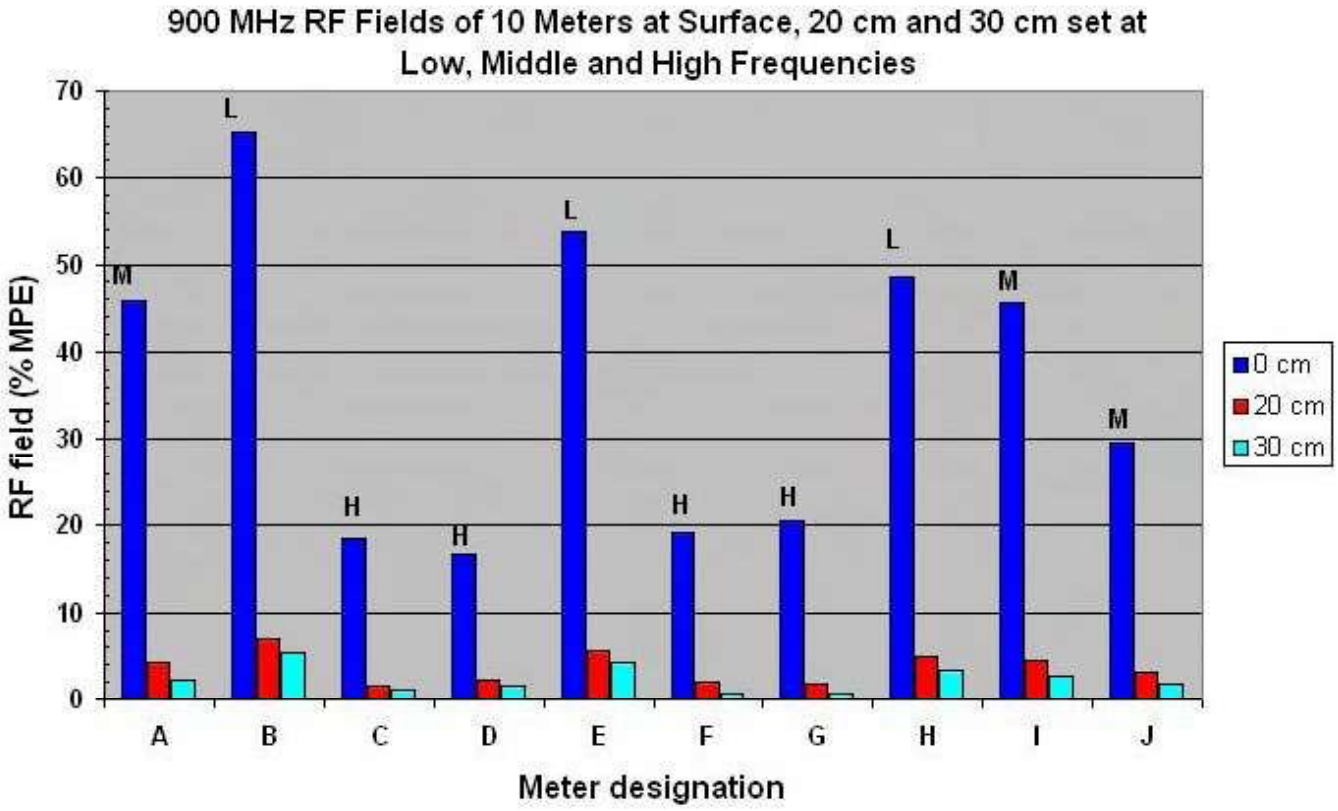

Figure 9-5
Corrected broadband probe RF field readings of the 900 MHz RF LAN transmitters from ten Smart Meters at the surface and at 20 cm and 30 cm from the meter.

A second observation is that the surface field strength readings are significantly greater than those at 20 cm. Why might this be the case? The probe protective shell surface is being placed in contact with the face of the Smart Meter, bringing the probe elements very significantly closer such that the probe is within the reactive near field region of the source antenna. The 900 MHz RF LAN antenna is only about 2.1 cm behind the meter envelope face; this is comparable to about only 0.06 wavelengths. Under these conditions, the probe may couple to the field source leading to erroneously high readings. Generally, field probes should not be used in such close proximity to the source because of this very issue. For example, IEEE Standard C95.3-2002[13] recommends a minimum measurement distance

[13] IEEE Standard C95.3-2002. IEEE Recommended Practice for Measurements and Computations of Radio Frequency

of 20 cm to minimize nearfield coupling and field gradient effects when using common broadband field probes. Measurement data can also be distorted when using an isotropic probe to measure steep spatial gradients close to a radiating element of the Smart Meter. These gradients can lead to considerable variation of the indicated amplitude of the field being measured over the volume of space occupied by the measurement probe elements. This is particularly true when employing field probes in the reactive near field that are comparable to the size of the source antenna. It should be noted that the elements inside the Narda B8742D probe are approximately 8 cm long; this is approximately the same length as the slot antenna of the 900 MHz RF LAN antenna that is approximately 6.3 cm long. Based on the potential for significant probe coupling with the Smart Meter internal transmitting antenna, the measured values indicated for surface contact of the probe with the Smart Meter should be considered suspect and, likely, substantial over estimates of the true field. Measurements at 20 cm and 30 cm, however, are deemed to be reliable since they are substantial fractions of the 900 MHz wavelength (20 cm is equivalent to 0.6 wavelengths and 30 cm is equivalent to 0.9 wavelengths.

Following measurement of the fields produced by the ten individual meters, measurements of the maximum indicated RF field were conducted in front of and behind the rack as individual meters were successively installed into their respective meter sockets. The objective was to observe for any increase in cumulative RF field caused by the contribution of an increasing aggregate of actively transmitting Smart Meters. These measurement results are summarized in Table 9-2. Due to technical problems associated with programming of two of the meters at the time (meters D and F), not all meters were included in each collection of active meters. However, at the end of the process, all meters were included when all ten meters were active.

Table 9-2
Summary of measurements of the 900 MHz composite RF field produced by an increasing number of closely spaced, collocated Smart Meters (meters A - J).[14]

Meters active	RF field at 20 cm	RF field at 30 cm	RF field behind rack
A	4.2	3.3	0.0
AB	7.9	5.0	0.0
ABC	7.4	5.3	0.1
ABCE	7.9	5.9	0.1
ABCEG	8.6	6.1	0.1
ABCEGH	9.0	6.2	0.3
ABCEGHI	8.7	6.8	0.7
ABCEGHIJ	9.2	6.7	0.9
ABCDEGHIJ	9.1	7.2	0.7
ABCDEFGHIJ	8.1	7.5	0.8

[14] During the testing, meter D exhibited a problem that was subsequently fixed but was left out of some of the test rows in Table 3.

In each case of added meters, the entire surface of the meter rack was scanned with the broadband probe with 20 and 30 cm spacers attached to the probe to search for the greatest meter reading. The location of maximum reading was not necessarily the same in each case and the data strongly suggest that for a given distance from the front of the meter rack, a finite maximum value of field is developed that will not be exceeded with the addition of more meters. Beyond three or four meters, the aggregate field does not materially increase with additional meters. The data indicate a maximum observed, composite field of 9.2% of the general public MPE at 20 cm and a maximum of 7.5% of the MPE at 30 cm (almost one foot). Immediately behind the meter rack, a maximum composite field equivalent to 0.9% of the MPE was measured.

A somewhat similar approach was used to measure the collective composite RF field produced by multiple Smart Meters with the 2.4 GHz Zigbee transmitters activated for transmission. Itron programmed each of the ten meters to enter the continuous transmit mode of operation with three of the ten meters programmed to operate on the lowest frequency (L) in the 2.4 GHz band (2405 MHz), three meters to operate at the mid-band (M) frequency (2440 MHz) and the remaining four meters to operate at the upper most channel (H) in the band (2475 MHz). In this case, the individual meters were measured with each meter being placed in position A but the overall composite field, with all meters active, was performed by inserting all meters into their designated positions without sequentially adding active meters as was done with the 900 MHz RF LAN tests. Table 9-3 summarizes the results of these measurements. All readings of the 2.4 GHz emissions were corrected by applying the manufacturer's determined correction factor for 2.45 GHz of 0.97.

Table 9-3
Summary of corrected measurement data on RF fields of individual 2.4 GHz Zigbee transmitters installed in meter position A and of the collection of all ten meters.

A	**M**	**10.2**		
B	L	11.8	1.7	0.0
C	H	5.1	1.4	0.0
D	H	5.5	1.0	0.0
E	L	11.8	0.9	0.0
F	H	6.3	1.5	0.0
G	H	5.4	0.9	0.0
H	L	8.8	1.0	0.0
I	M	7.0	1.0	0.0
J	M	5.1	0.7	0.0
All on		14.3	1.0	0.0
A	M	10.2	2.5	0.0

These data support the contention that the Zigbee transmitters operating at the lowest frequency within the 2.4 GHz band tended to produced the greatest measured field strength, similar to the finding for the 900 MHz RF LAN transmitters. Figure 9-6 illustrates the data in Table 9-3 graphically.

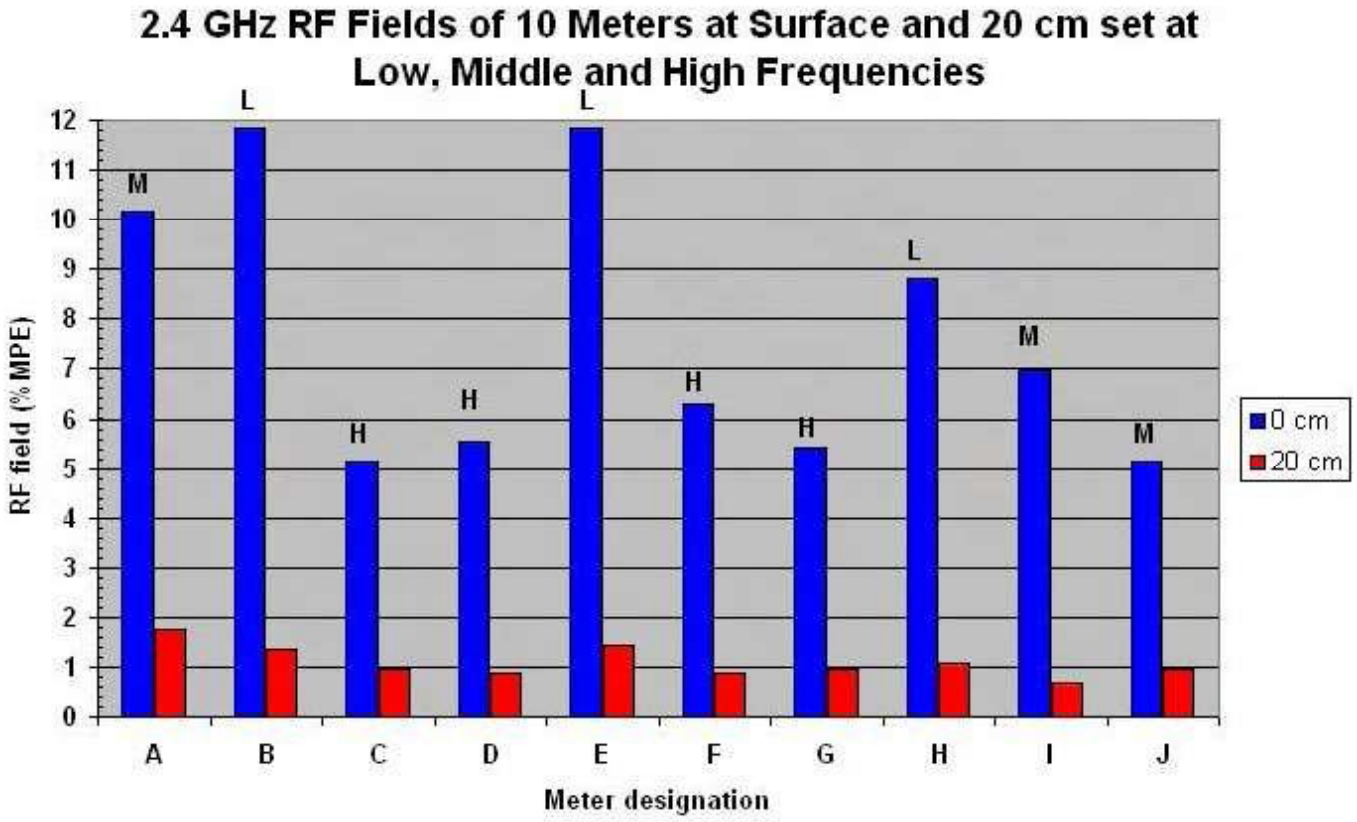

Figure 9-6
Corrected broadband probe RF field readings of the 2.4 GHz Zigbee transmitters from ten Smart Meters at the surface and at 20 cm from the meter.

Table 9-3 also indicates that the composite RF field associated with simultaneous operation of all ten Smart Meters with their Zigbee radios active provided a maximum reading of 14.3% of the FCC general public MPE with the probe in surface contact with the meters and a maximum of 2.5% of the MPE at 20 cm from the surface. The surface readings must also be considered suspect as is the case with the 900 MHz band measurements. The RF field behind the rack of ten active meters was not detectable with the broadband field probe.

Groups of meters

Through use of the Narda SRM-3006 instrument, measurements of RF fields could be made at much greater distances from the meter rack due to the significantly greater sensitivity of the narrowband device when compared to the broadband field probe. The aggregate RF field produced by a meter rack of ten Smart Meters was examined with the SRM-3006 by making measurements at successively greater distances from the front of the rack and observing the spectral display of the measurement result. The measurement process consisted of holding the SRM-3006 at the approximate mid-height of the rack at different distances from the frontal plane of the meters in the rack as shown in Figure 9-7

Figure 9-7
Using the Narda SRM-3006 to measure aggregate RF fields near a rack of ten meters programmed for fixed frequency, continuous transmission in the meter farm.

The instrument was used to acquire a "max hold" spectrum over a period of approximately one minute while slowly moving the probe in a planar area measuring approximately 2 feet by 2 feet. Figure 9-8 shows the result of the measurement with the SRM-3006 probe/antenna positioned at 1 foot from the front of the meter rack. In this display, the continuously operating 900 MHz RF LAN transmitters are clearly seen on their respective frequencies (902.25 MHz, 914.75 MHz and 927.95 MHz). A resolution bandwidth of 100 kHz was used in these spectral measurements within the frequency band of 902 MHz to 928 MHz. The SRM-3006 was set to display the maximum measured field at each distance directly as a percentage of the FCC general public MPE as seen on the vertical axis of the display.

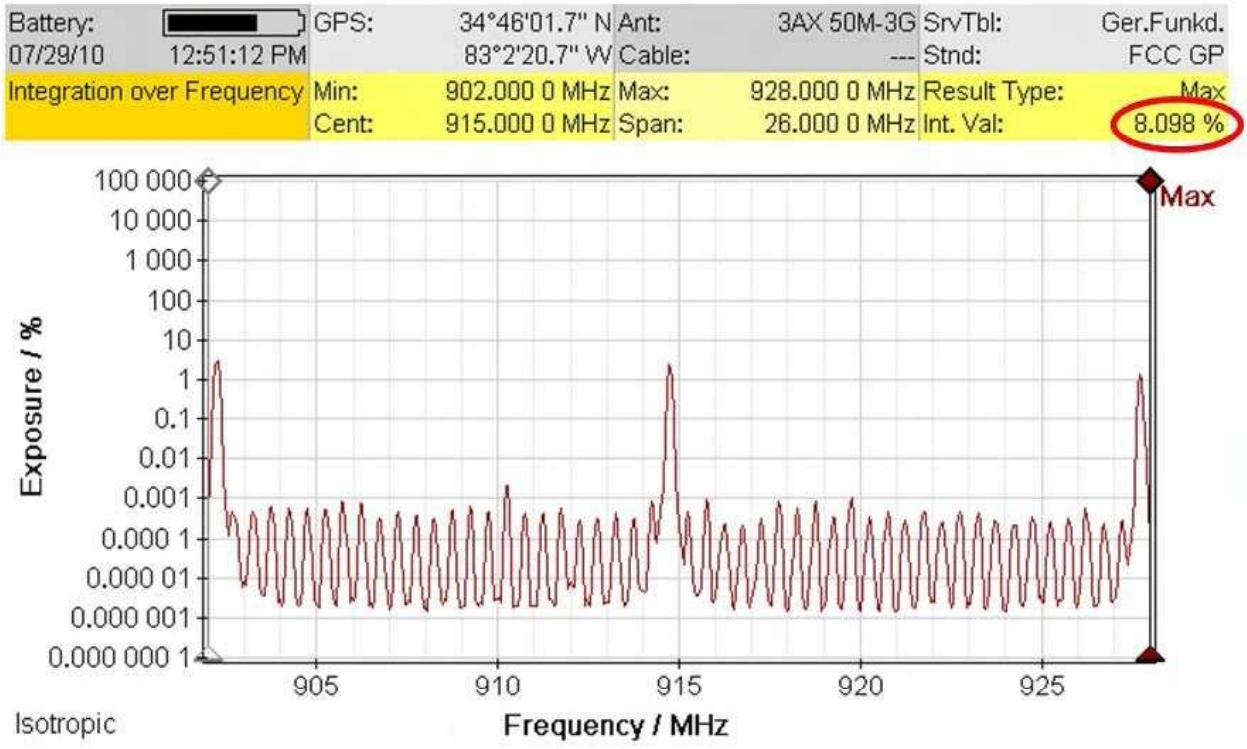

Figure 9-8
900 MHz band composite RF field from rack of 10 SmartMeters at 1 foot.

The many lower level spectral peaks were caused by the multiplicity of Smart Meters within the meter farm; in practice, it was not possible to completely remove oneself from the ambient background of RF fields present in the meter farm since moving away from one rack of meters meant that one was getting closer to another rack in some location. While the signals from the other thousands of Smart Meters were randomly occurring across the band, because of the number of meters simultaneously operating, the presence of signals on each frequency was evident. Had only one Smart Meter been operating, this would not have been the case, as discussed earlier. Using the internal integration feature of the SRM-3006, the total equivalent RF field power density was reported by the instrument as a percentage of the general public MPE in the upper right region of the spectral plot (see circled area). For the measurement at 1 foot in front of the meter rack, a total integrated RF field equivalent to 8.1% of the MPE was determined.

Using the spectrum analysis method describe above, measurements were made at successively greater distances from 1 foot to 100 feet from the Smart Meter rack. These spectrum scans obtained from the SRM-3006 are shown in Appendix B. Referring to Appendix B, it can be seen that as the distance between the rack and the measuring instrument was increased, the signal level of the programmed meters decreased until, at approximately 50 feet from the rack, the signal levels of the meter rack being investigated blended into the background of all of the other ambient RF fields from other meters within that area of the meter farm. In other words, the emitted signals became indistinguishable from the ambient environment of RF fields and could not be identified as being contributed by a specific meter rack or collection of Smart Meters. Field measurements taken to the rear of the meter rack are provided in Appendix C. Figure 9-9 shows measurements being performed behind the rack of specially programmed Smart Meters. The presence of other racks of active meters are evident in the background. As distance from the back side of the subject rack was increased, the distance to the other meter racks located behind the subject rack decreased meaning that the ambient, but intermittent, RF fields of other meters in the farm could be detected.

Figure 9-9
Field measurements at successively greater distances behind the subject meter rack resulted in closer proximity to other meter racks with the probability of detecting stronger, but intermittent, signals due to the ambient background.

Another set of field measurements in front of the meter rack was performed with the Zigbee radios in the meters programmed for continuous transmit operation on 2405 MHz, 2440 MHz and 2475 MHz. The SRM-3006 was set for a resolution bandwidth of 200 kHz over the band of 2400 to 2483 MHz for these measurements. A similar pattern of decreasing field magnitude with increasing distance was observed for the Zigbee radio emissions. Figure 9-10 shows the spectrum plot obtained at 1 foot from the front of the meter rack with all ten radios operating with an integrated RF field equivalent to 4.5% of the general public MPE. Appendix D provides each of the spectrum measurements of the ten Zigbee transmitters at distances from 1 foot to 100 feet from the front of the meter rack. Appendix E provides similar spectrum plots taken behind the meter rack as well as the result of a lateral walk at three feet in front of and across the face of the meter rack extending from a few feet beyond the edge of the rack to an equivalent distance beyond the opposite edge.

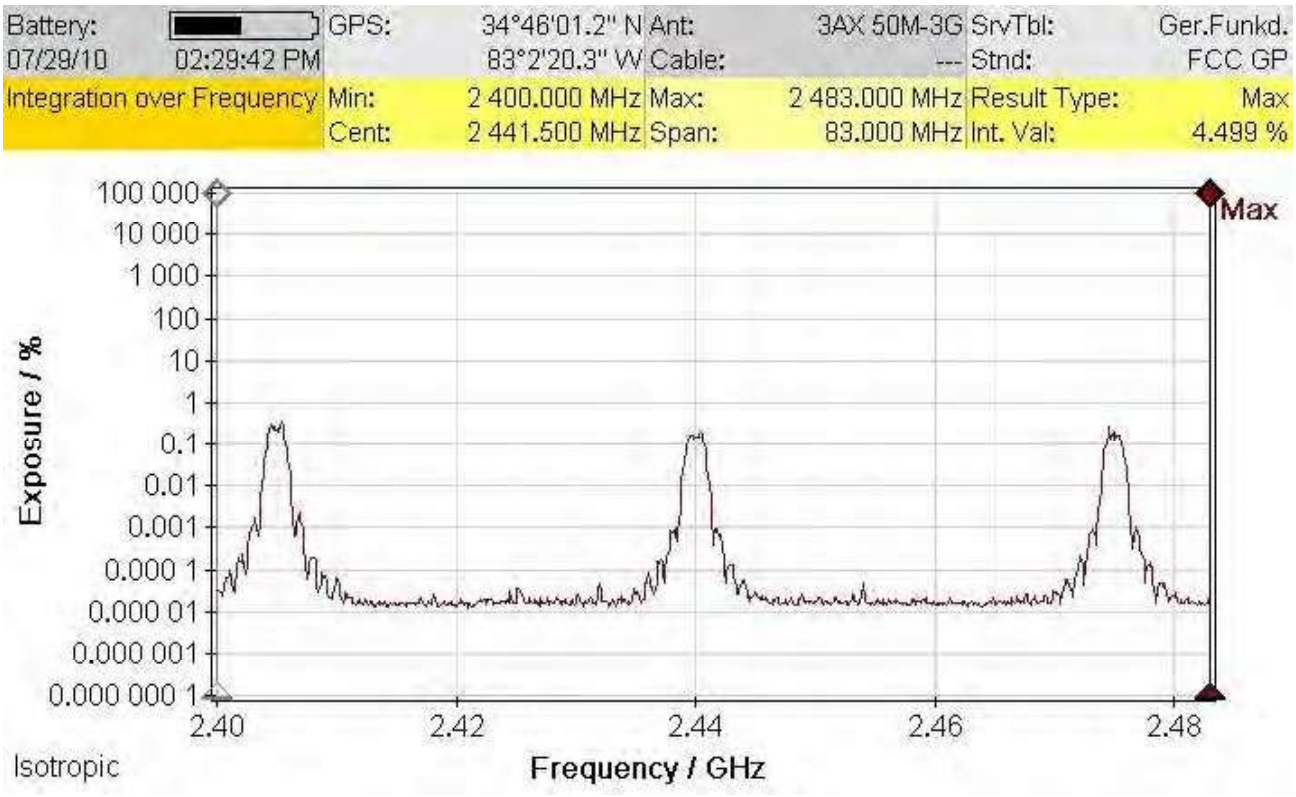

Figure 9-10
Spectrum measurement of 2.4 GHz RF fields from ten simultaneously transmitting Smart Meters.

Table 9-4 lists the integrated RF fields determined for both the 900 MHz RF LAN and 2.4 GHz Zigbee transmitters over the range of distances used. The data in Table 9-4 are plotted in both linear (Figure 9-11) and logarithmic (Figure 9-12) formats to illustrate graphically the decrease in RF field with distance from the meter farm rack of ten meters.

Table 9-4
Summary of composite RF field values (% general public MPE) determined with the SRM-3006 at various distances in front of a meter rack of 10 simultaneously operating Smart Meters.

Distance (ft)	900 MHz	2.4 GHz
1	8.098	4.499
2	3.898	2.459
3	2.471	1.021
4	1.827	0.587
5	1.382	0.457
6	1.157	0.348
7	0.722	0.258
8	0.655	0.187
9	0.681	0.163
10	0.536	0.134
15	0.356	0.076
20	0.177	0.044
25	0.152	0.033
30	0.144	0.02
40	0.113	0.014
50	0.107	0.013
75	0.073	0.0091
100	0.092	0.00852
Distance (ft)	900 MHz	2.4 GHz

From Table 9-4 and Figures 9-11 and 9-12, it is evident that the peak RF field measured for the group of ten active Smart Meters drops to less than 1% of the FCC general public MPE at a distance of approximately seven feet where the combined RF field from both frequency bands are summed. This peak value is not representative of the time averaged field that would be present during normal operation of the Smart Meter since the typical duty cycle of the meters would cause the composite time-averaged field to be substantially less.

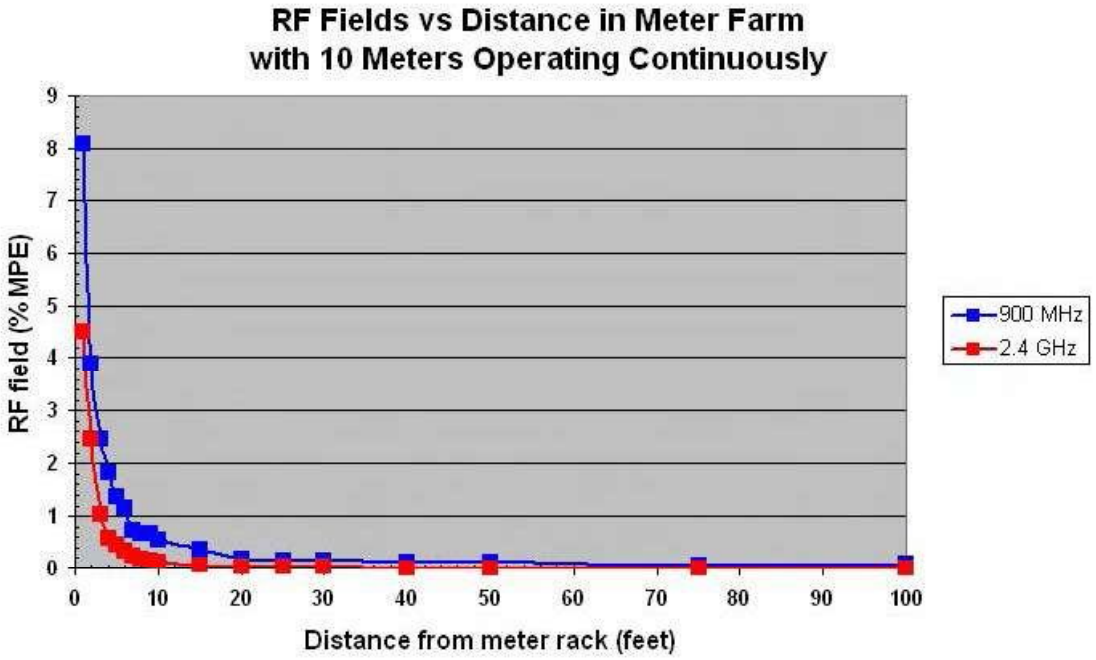

Figure 9-11
Integrated, total composite RF field obtained in meter farm for emissions from the 900 MHz RF LAN and 2.4 GHz Zigbee transmitters operating simultaneously in the vicinity of a meter rack (linear plot).

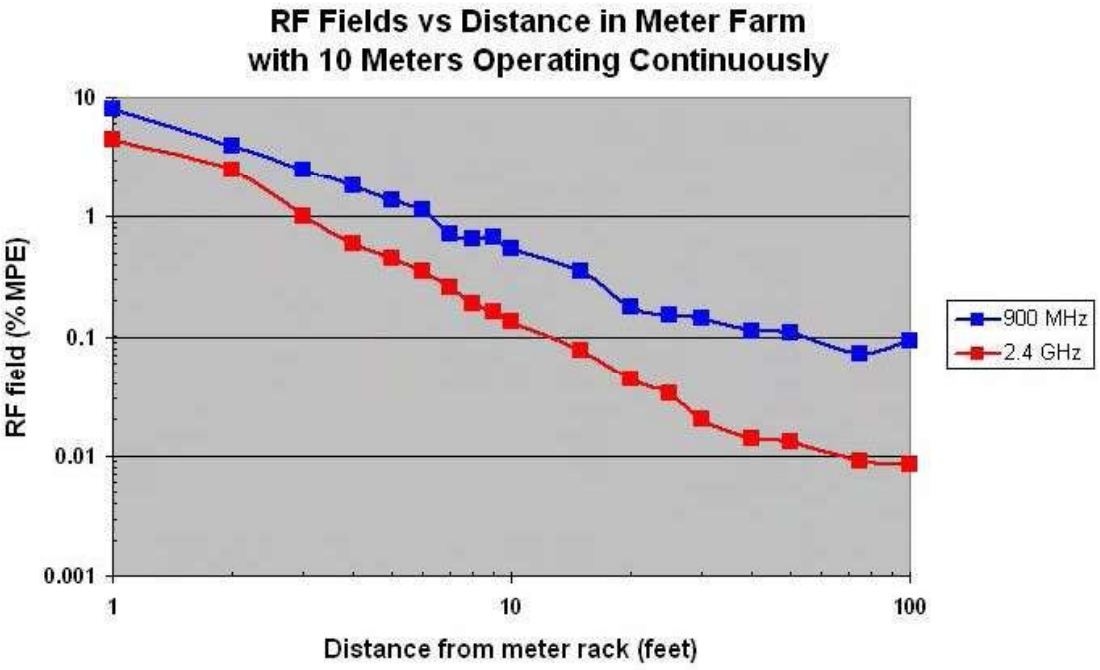

Figure 9-12
Integrated, total composite RF field obtained in meter farm for emissions from the 900 MHz RF LAN and 2.4 GHz Zigbee transmitters operating simultaneously in the vicinity of a meter rack (logarithmic plot).

Residential settings

Homes

In the interest of gathering data on RF fields from Smart Meters in a realistic residential environment, additional measurements were performed in a Downey, California neighborhood. On August 19, 2010, measurements were conducted at two different residences at which SCE Smart Meters had been previously deployed. Two different Smart Meters were used to facilitate the measurements, one that had been programmed to operate in continuous transmit mode on the lowest frequency in the 900 MHz band and the other programmed to operate in continuous transmit mode on the lowest frequency in the 2.4 GHz band. Each meter was temporarily installed at each of the two homes during which a series of RF measurements were taken at the meter service box on the home, within the front, side and backyards, and throughout the home in all rooms of the home. Figure 9-13 shows an SCE meter technician in the process of installing one of the special "test" meters in place of the meter normally present.

At the first residence, designated residence A, measurements of the maximum, instantaneous peak RF field were conducted by scanning a planar region at 1, 2, 3, and 5 feet in front of the meter. The measurements are summarized in Table 9-5. The region near the service box for residence A was somewhat cramped and measurements were not possible beyond five feet from the face of the Smart Meter.

Table 9-5
Summary of planar area scans performed with the SRM-3006 in front of residential meter installation at residence A, Downey, CA, with transmitters operating continuously.

	900 MHz RF LAN		2.4 GHz Zigbee	
Location relative to meter (feet)	**RF field (% public MPE)**	**Time of measurement (PDT)**	**RF field (% public MPE)**	**Time of measurement (PDT)**
Surface	9.67	9:49	7.93	11:19
1	0.875	9:54	0.615	11:22
2	0.361	9.56	0.258	11:22
3	0.186	9:58	0.142	11:23
5	0.096	10:00	0.071	11:25

With each of the two specially programmed Smart Meters installed in the home's service box meter socket, spectrum measurements were performed throughout the home including some outside areas. Figure 9-14 shows the measurement of a planar scan at the residence. Procedurally, the 900 MHz band measurements were performed first, followed by the 2.4 GHz measurements. Table 9-6 summarizes the measurements taken at residence A including a few outdoor measurements. The RF field reading recorded for each room or area represents the overall peak value of field obtained through a spatial scan of the room or area. It is noted that directly behind the service box with the Smart Meter, inside bedroom 1, the greatest field detected corresponded to 0.01% of the FCC general public MPE. Overall, the greatest RF fields found were in the home office area, where a wireless router was installed for Internet connectivity and in the kitchen when the microwave oven was operating.

Figure 9-13
SCE meter technician replacing existing Smart Meter with specially programmed meter for residential measurements.

Figure 9-14
Planar scans were performed at several distances in front of a residential Smart Meter by slowly moving the SRM-3006 within a plane at a fixed distance.

Table 9-6
Spectrum scan measurements of Smart Meter fields in the 900 MHz and 2.4 GHz bands in residence A, Downey, CA. RF field is peak value obtained from a spatial scan of the room interior or area in percent of FCC general public MPE.

	RF field (% MPE)	
Location at residence	**900 MHz**	**2.4 GHz**
Front yard	0.00014	0.00611
Bedroom 1	0.00355	0.00876
Bedroom 1 (directly behind meter)	0.010	
Bath	0.009	0.00941
Bedroom 2	0.00909	0.00637
Master bedroom	0.00056	0.00644
Family room	0.00055	0.00627
Dining room/Living room	0.00057	0.00651
Kitchen	0.00057	0.00616
Kitchen (microwave at 6.5 feet)		0.016
Kitchen (microwave at 2 feet)		22.04
Laundry room	0.00053	0.00588
Bath	0.00054	0.00723
Office (Wi-Fi on)	0.00052	0.0288
Garage	0.00055	0.00622
Back side yard	0.00053	0.00653
Backyard	0.00059	0.00658
Pool	0.00058	0.00647

Figure 9-15 shows an interior measurement taken at residence A in a bedroom directly opposite to the mounting location of the Smart Meter on the outside of the house. Outdoor measurements at residence A are shown in Figures 9-16 and 9-17.

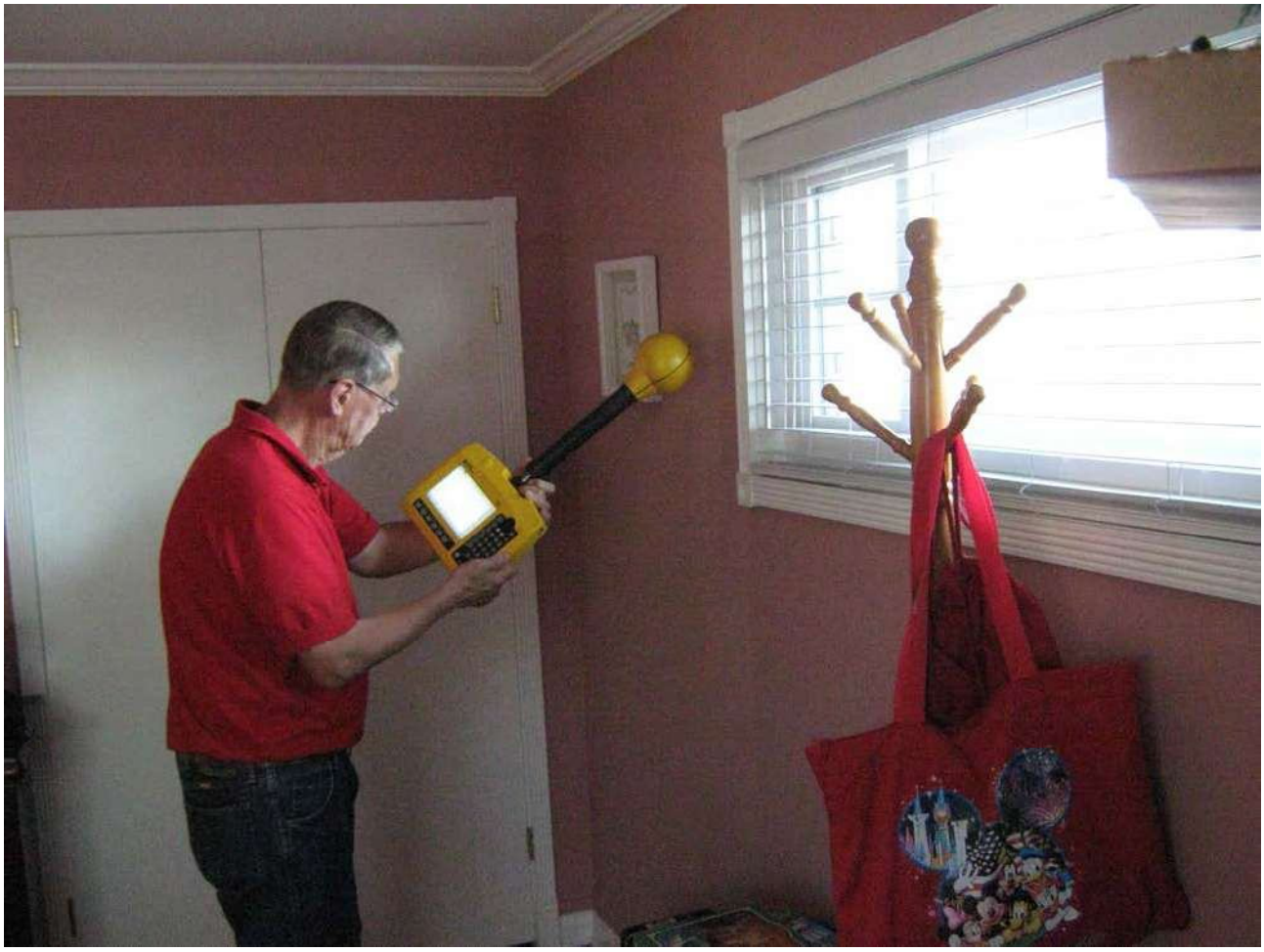

Figure 9-15
Interior residential measurements included measurements on the opposite side of the wall where the Smart Meter was installed at the home.

Figure 9-16
Residential measurements at residence A included both outdoors and indoors measurements of RF fields in both the 900 MHz and 2.4 GHz bands.

Figure 9-17
Measurements at residence A included exterior locations in the backyard.

At the second residence (Figure 9-18), designated residence B, also in the same neighborhood of Downey, CA as residence A, the measurement process again included scanning a planar region at 1, 2, 3, 4, 5, 6 and 10 feet in front of the meter. The measurements for residence B are summarized in Table 9-7. The specially programmed Smart Meters were used to facilitate measurements throughout the property. Figure 9-19 shows the Smart Meter installed at residence B and a planar scan being performed. Within residence B, similar to residence A, a wireless router for Internet connectivity was found in a home office (Figure 9-20). Field measurements acquired within and around the residence are listed in Table 9-8.

Figure 9-18
Residence B in Downey, CA where indoor and outdoor measurements were made with specially programmed Smart Meters installed to facilitate measurements.

Figure 9-19
Performing a planar scan of RF fields adjacent to a Smart Meter at residence B.

Figure 9-20
Measured RF fields inside residence B, similar to residence A, tended to be predominated by signals produced by wireless routers used for Internet connectivity throughout the home.

Table 9-7
Summary of planar area scans performed with the SRM-3006 in front of residential meter installation at residence B, Downey, CA.

	900 MHz RF LAN		2.4 GHz Zigbee	
Location relative to meter	**RF field (% public MPE)**	**Time of measurement (PDT)**	**RF field (% public MPE)**	**Time of measurement (PDT)**
Surface	10.84	1:34	10.02	1:34
1	1.386	1:35	0.985	1:35
2	0.351	1:36	0.290	1:36
3	0.159	1:37	0.160	1:37
4	0.104	1:38	0.117	1:38
6	0.048	1:40	0.053	1:40
10	0.020	1:41	0.026	1:41

Table 9-8
Spectrum scan measurements of Smart Meter fields in the 900 MHz and 2.4 GHz bands in residence B, Downey, CA. RF field is peak value obtained from a spatial scan of the room interior or area in percent of FCC general public MPE.

	RF field (% MPE)	
Location at residence	**900 MHz**	**2.4 GHz**
Front yard	0.00056	0.00659
Bedroom 1	0.00053	0.0063
Bath	0.00056	0.00597
Bedroom 2	0.00052	0.00618
Bedroom 3	0.0015	0.00638
Bedroom 3 (closet behind meter)	0.00872	0.00755
Master bedroom	0.00060	0.00643
Family room	0.00057	0.00753
Dining room/Living room	0.00063	0.00641
Kitchen	0.00056	0.012
Study (with Wi-Fi)	0.00055	0.015
Bath	0.0011	0.00596
Backyard	0.0051	0.015

Residential apartment setting

The use of the specially programmed Smart Meters allowed for relatively quick and definitive measurements of the peak RF fields produced by the internal 900 MHz and 2.45 GHz transmitters. In an effort to acquire data on neighborhood RF fields that might be produced by existing in-place meters, a Downey, CA neighborhood was explored to identify apartment complexes that had obvious groups of meters installed in easily accessible areas for measurement. Measurements with the SRM-3006 instrument were subsequently accomplished in front of meter banks at two apartment buildings on August 20, 2010. Figures 9-21 and 9-22 show the two apartment meter banks with nine and eleven Smart Meters respectively.

At each of the two meter banks, the SRM-3006 was held at a distance of 1 foot from the frontal plane of the meters and moved back and forth and up and down in this plane to maximize the probability of capturing the peak value of any meter emissions. This process was continued for a period of five minutes at each of the two meter banks. Captured spectra were integrated to obtain the composite RF fields in terms of the instantaneous peak values and average values over the five minute monitoring period. Figure 9-23 shows the resulting observed spectrum of peak values at the nine meter bank with the integrated value of 4.6% of the public MPE. The corresponding integral of the average field was 0.00105% of the MPE.

Figure 9-21
A nine-meter bank at an apartment house in Downey, CA.

Figure 9-22
An eleven-meter bank at an apartment house in Downey, CA.

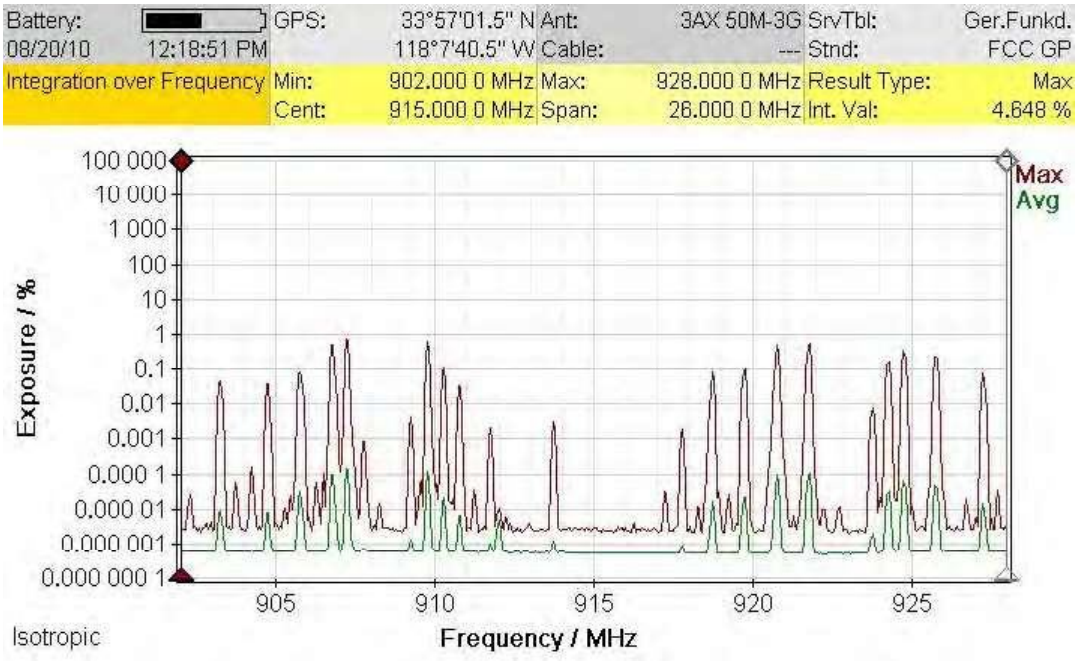

Figure 9-23
Measured maximum (peak) and average RF fields in the 900 MHz band at one foot in front of a nine-meter bank of Smart Meters.

Figure 9-24 provides the measured spectrum of fields at the second meter bank consisting of eleven meters. In this case the integrated composite peak field was 4.9% of the MPE with an integrated composite average field of 0.00124% of MPE.

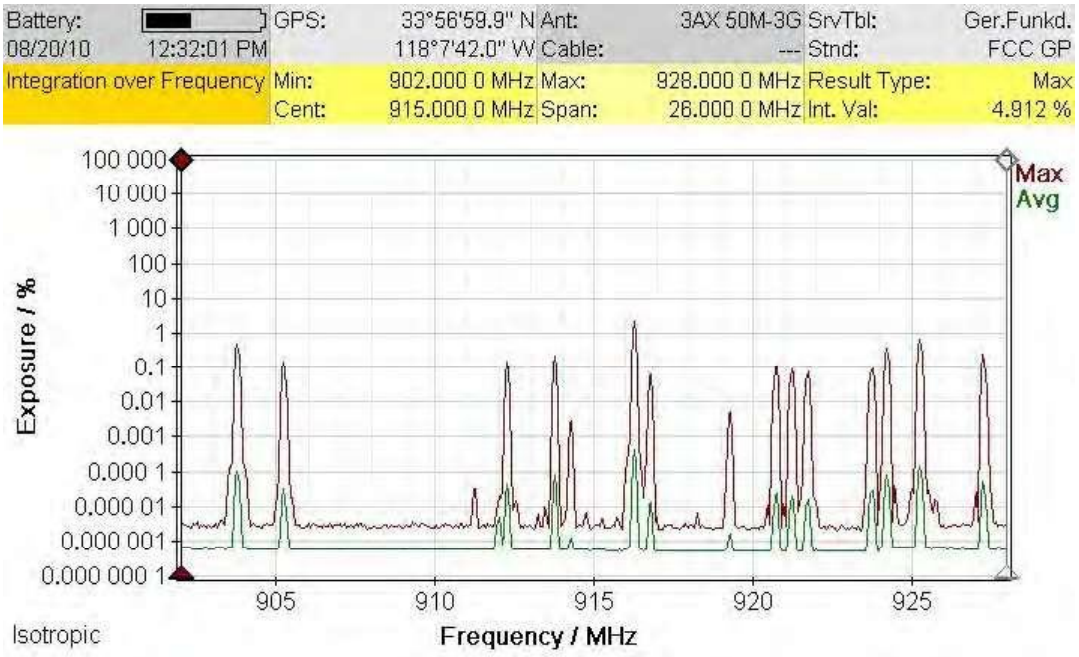

Figure 9-24
Measured maximum (peak) and average RF fields in the 900 MHz band at one foot in front of an eleven-meter bank of Smart Meters.

Neighborhoods with and without Smart Meters

A driving survey of a Downey neighborhood where SCE had deployed Smart Meters was also conducted. Figure 9-25 illustrates the route followed by slowly driving in an automobile with the SRM-3006 probe held out of the front passenger window (see Figure 9-26). The objective of this exercise was to see if Smart Meter RF fields could be detected with the instrumentation used under these conditions and to see if a neighborhood with installed Smart Meters could be distinguished from another neighborhood in which Smart Meters had not been deployed. The resulting accumulative spectrum of peak fields is shown in Figure 9-27. It must be noted that these integrated values of RF field represent the instantaneous peak values of RF fields that were observed on the spectrum analyzer, even if the field existed for a fraction of a second; for proper comparison to RF exposure standards, time-averaged values of RF fields must be used. Hence, the indicated integrated values are extremely conservative estimates of actual time-averaged exposure. A peak value corresponding to 0.00686% of the FCC MPE was found from the 34 minute drive. Because of the intermittent nature of the Smart Meter signals, peaks would appear from time to time but quickly vanished. While a large number of apparent Smart Meter signals could be observed, the average value of these signals is nearly vanishingly small. This activity was somewhat similar to walking through the Itron meter farm in that signals from the many meters occur randomly, making it difficult to definitely identify any particular meter's emission but relatively easy to observe signals when originating from many different meters.

Figure 9-25
Route in Downey, CA neighborhood with SCE deployed Smart Meters over which a driving survey was conducted to test the ability to detect RF signals associated with residential meter installations.

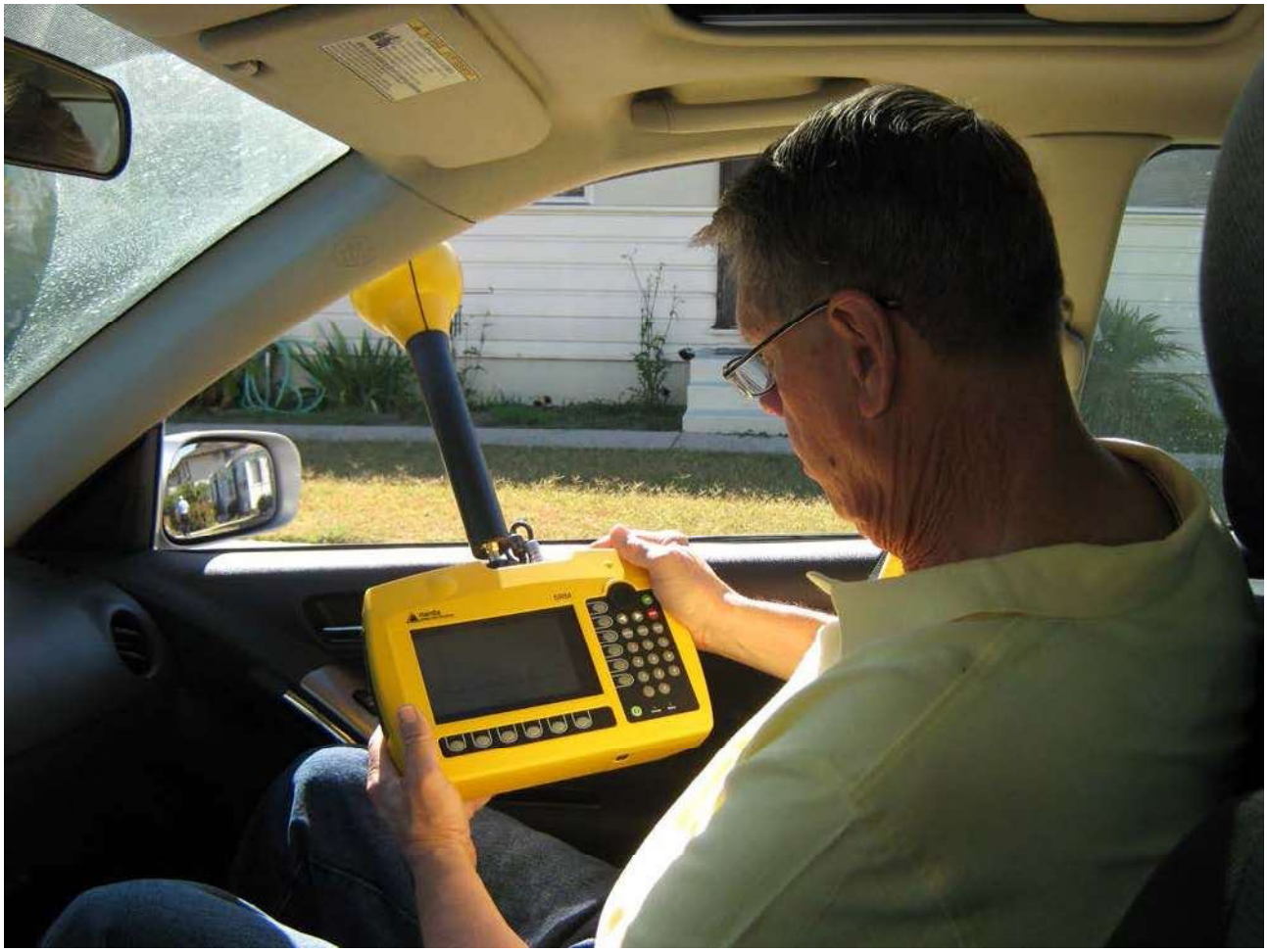

Figure 9-26
Conducting a "driving survey" of a Smart Meter deployed neighborhood.

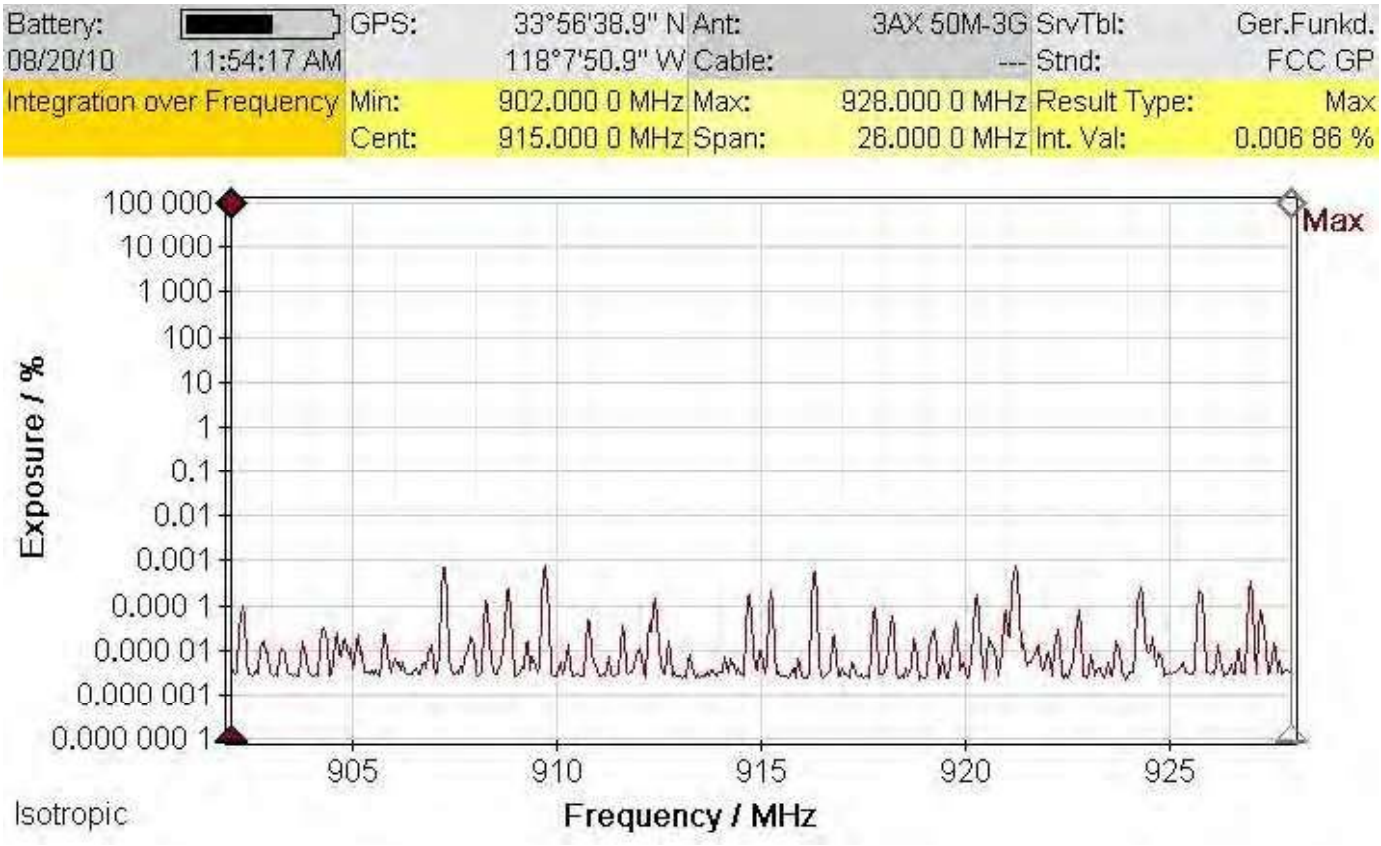

Figure 9-27
Measured peak spectrum of RF fields detected with the SRM-3006 during traveling the route mapped in Figure 9-25. Fields were monitored for a total of approximately 34 minutes.

To develop a comparative view of neighborhood ambient RF fields in the 900 MHz band, a driving survey through a portion of Santa Monica was conducted in the afternoon of August 20, 2010 (see route map in Figure 9-28). SCE had not yet deployed Smart Meters in Santa Monica at that time. The results of two spectrum scans in which the instantaneous peak fields were monitored are shown in Figures 9-29 and 9-30. These scans reveal a lack of 900 MHz signal activity other than for an occasional emission, perhaps related to cordless telephones. While the residential neighborhood areas were essentially absent of 900 MHz activity, such was not the case for a commercial district in Santa Monica, as shown in Figure 9-31. As the survey vehicle turned onto a main street in the commercial district, signals were almost immediately noted to appear on the spectrum analyzer. None of these signals were exceptionally strong but they were plentiful during the few minutes spent within the commercial district.

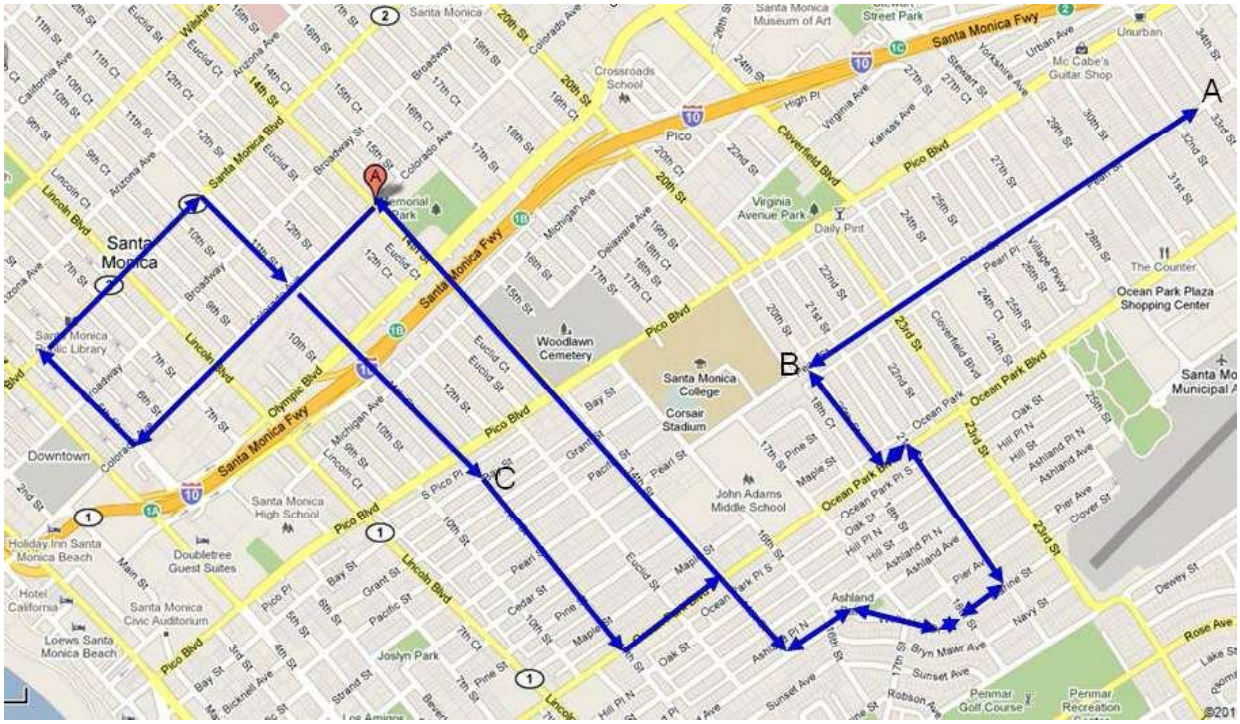

Figure 9-28
Route in Santa Monica, CA neighborhood where SCE Smart Meters have not been deployed over which a driving survey was conducted to test the ability to detect RF signals that might exist in the 900 MHz band.

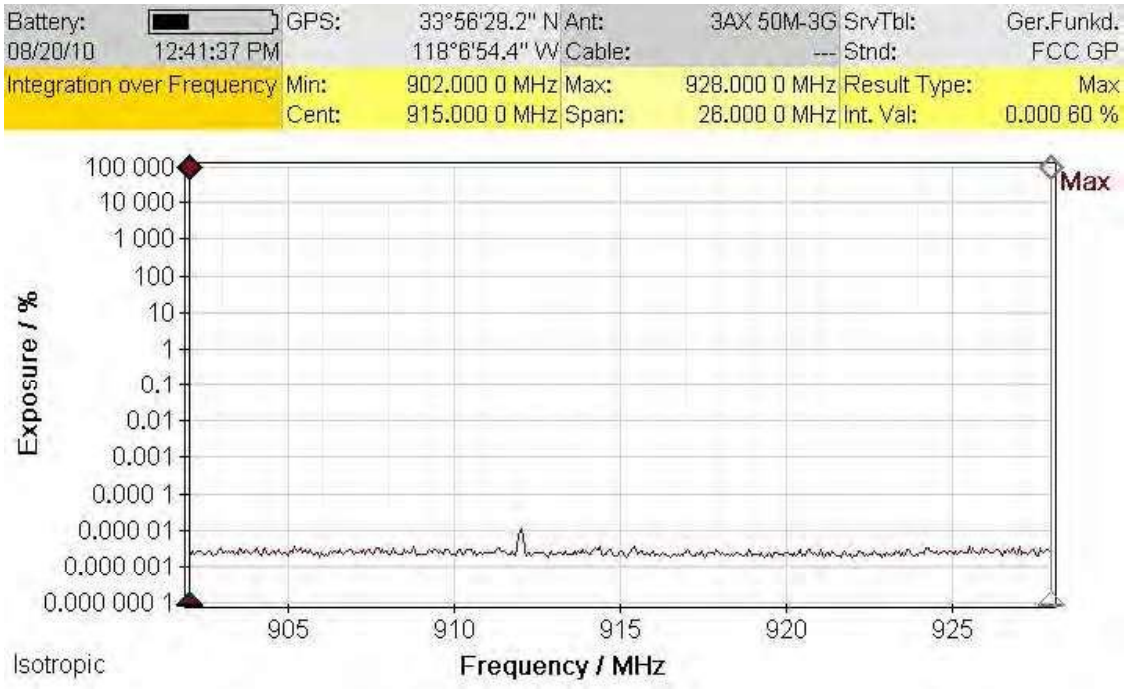

Figure 9-29
Spectrum scan in residential neighborhood of Santa Monica, CA consisting of an approximately 20 minute drive.

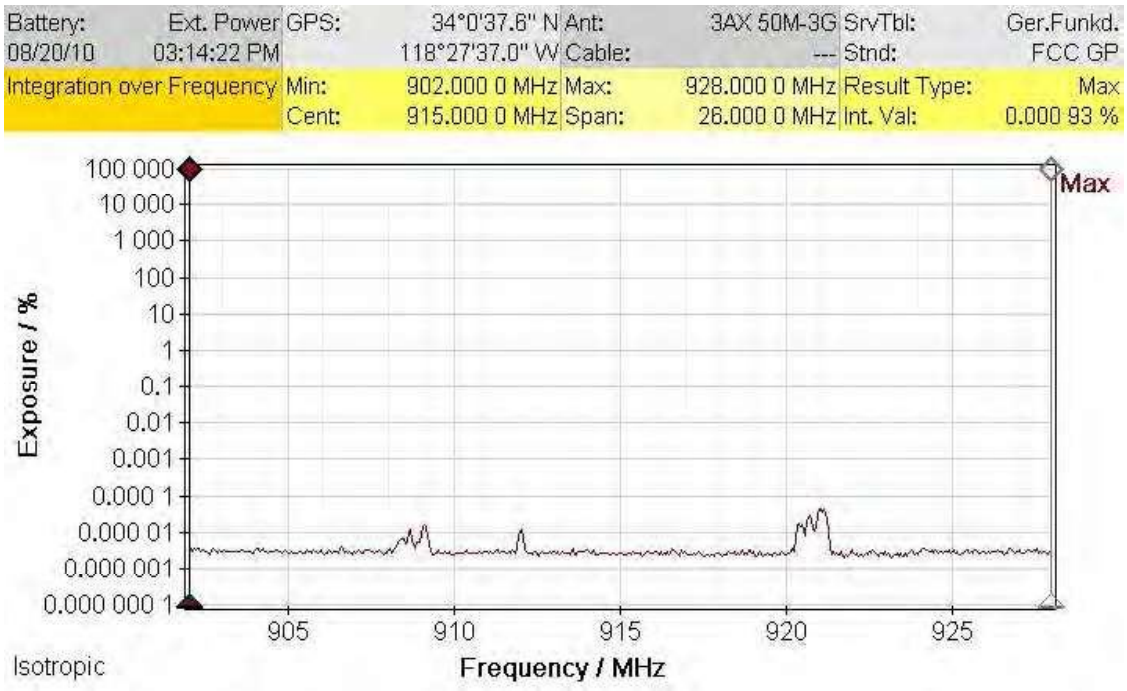

Figure 9-30
Spectrum scan in residential neighborhood of Santa Monica, CA showing weak signals, apparently caused by 900 MHz cordless telephones.

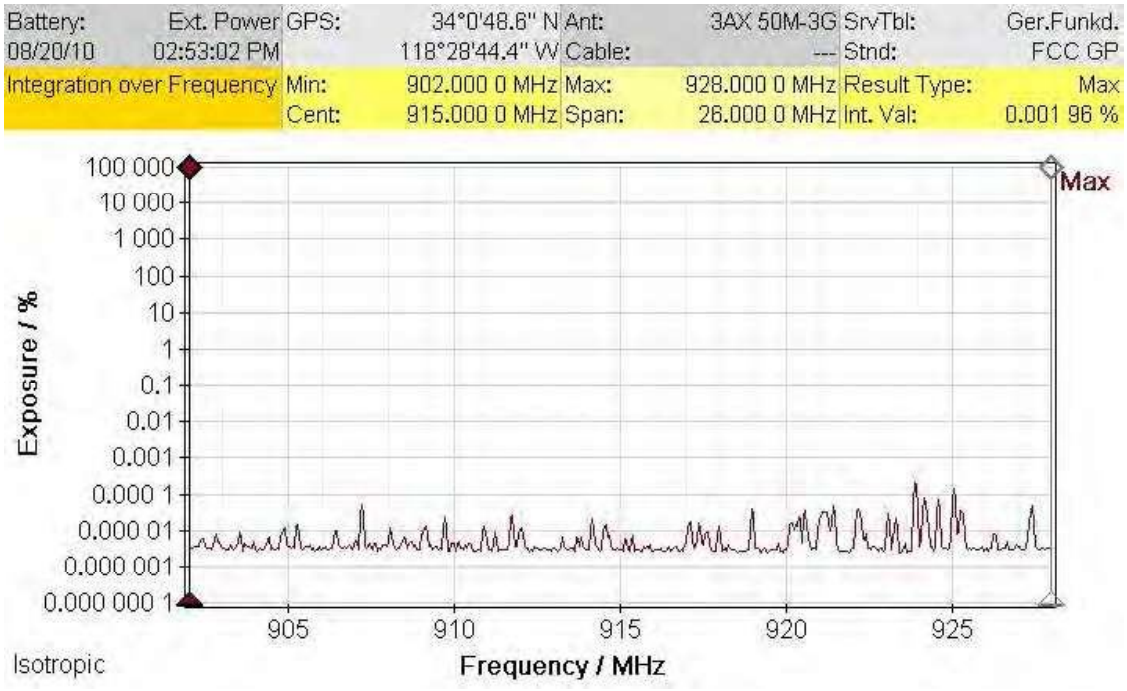

Figure 9-31
Spectrum scan in a commercial district of Santa Monica, CA showing noticeable activity from devices other than Smart Meters.

A second SRM-3006 was use to measure RF fields in several other frequency bands in Santa Monica, CA (The calibration certificates are contained in Appendix A). Measurements included the:

- FM radio broadcast band of 88-108 MHz (Figure 9-32),
- spectrum of 800 to 900 MHz band (Figure 9-33),
- PCS band from 1.9 to 2.0 GHz (Figure 9-34).
- VHF spectrum of 50 MHz to 216 MHz (Figure 9-35), and
- 2.4 to 2.5 GHz band which includes Wi-Fi and microwave ovens
- (Figure 9-36)

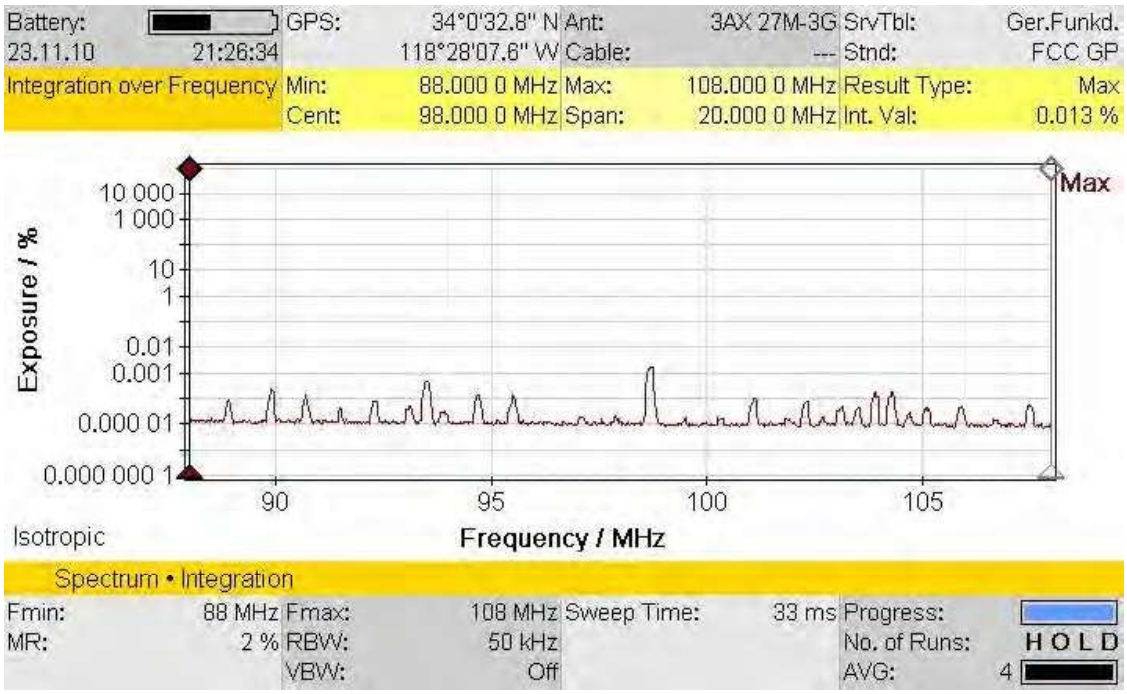

Figure 9-32
Spectrum scan of the FM radio broadcast band in Santa Monica, CA with a band integrated RF field equivalent to 0.013% of the FCC MPE for the public.

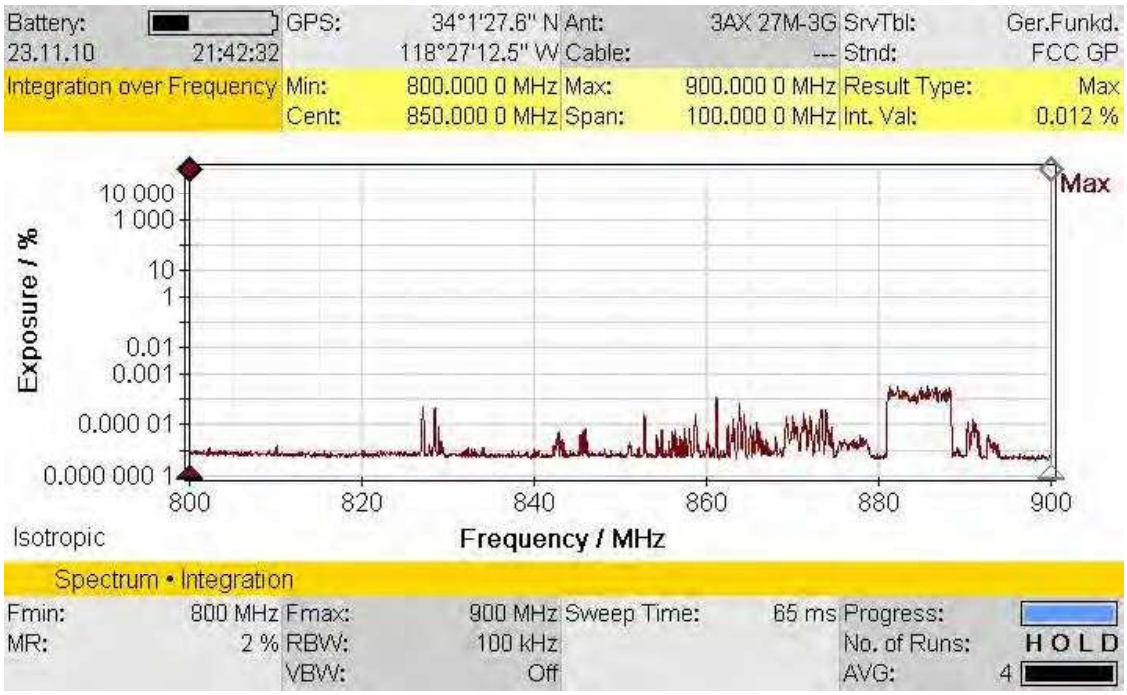

Figure 9-33
Spectrum scan of the 800 MHz to 900 MHz band in Santa Monica, CA, with a band integrated RF field equivalent to 0.012% of the FCC MPE for the public.

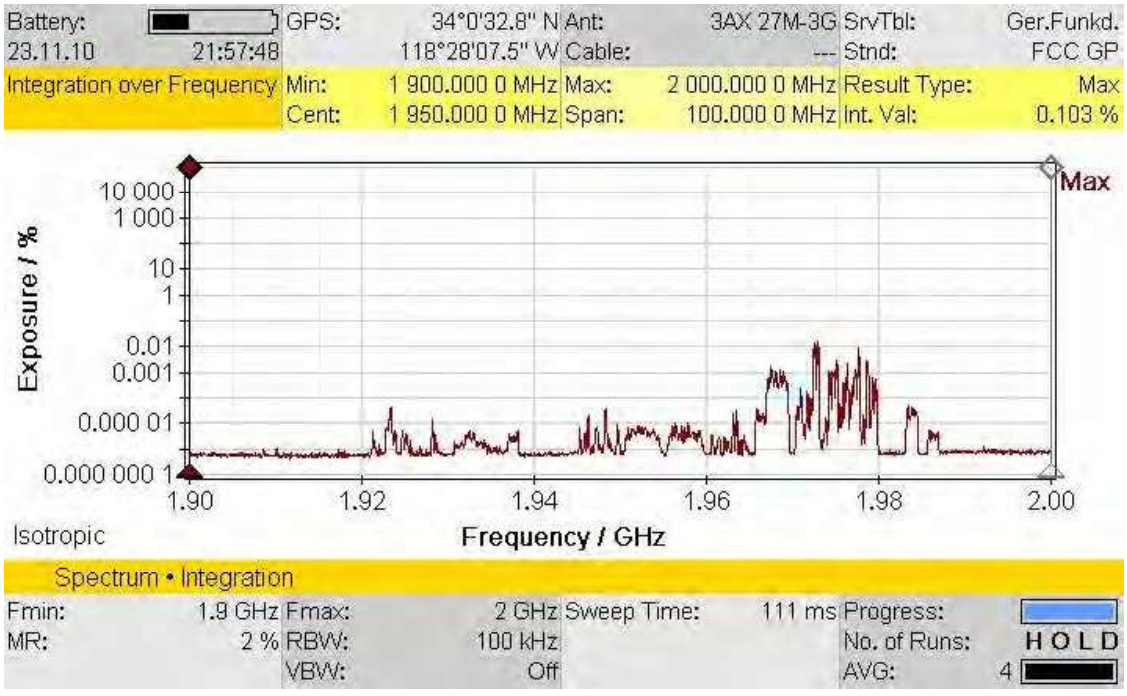

Figure 9-34
Spectrum scan of the 1.9 GHz to 2.0 GHz band in Santa Monica, CA, with a band integrated RF field equivalent to 0.103% of the FCC MPE for the public.

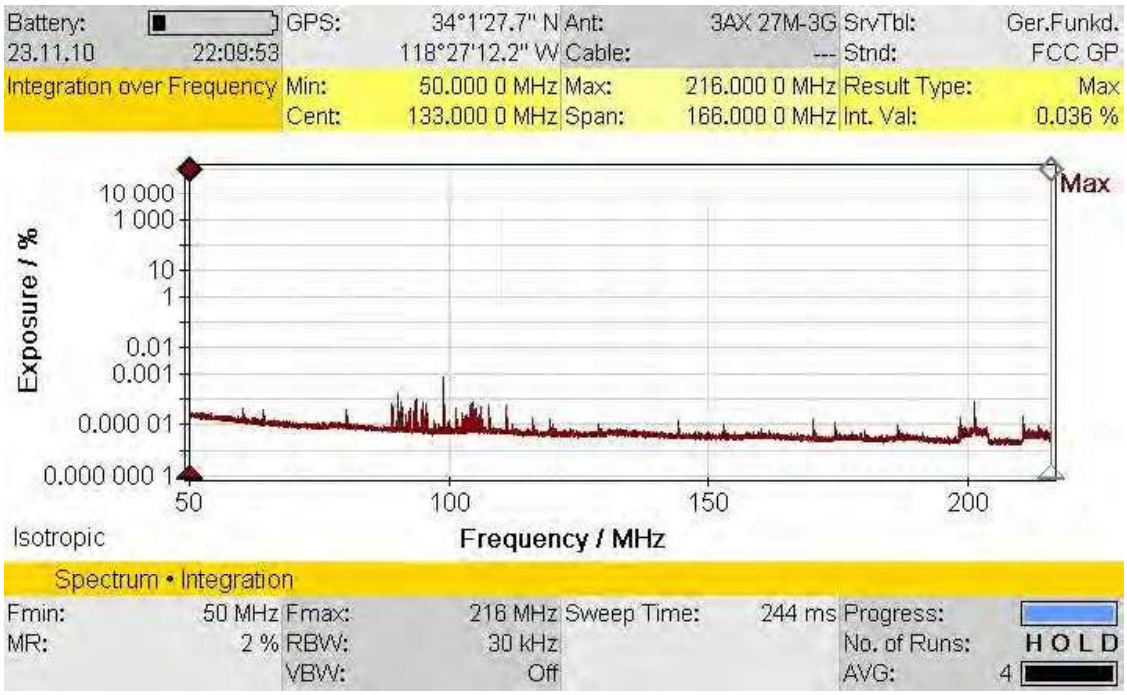

Figure 9-35
Spectrum scan of the 50 MHz to 216 MHz band in Santa Monica, CA, with a band integrated RF field equivalent to 0.036% of the FCC MPE for the public.

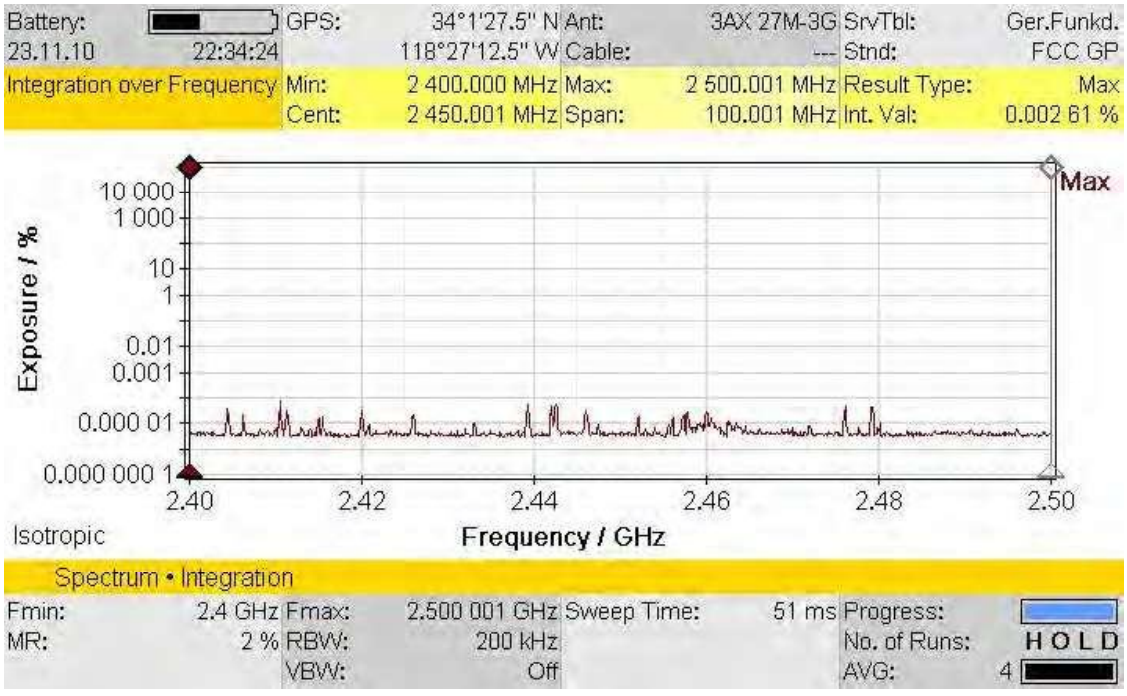

Figure 9-36
Spectrum scan of the 2.4 GHz to 2.5 GHz band in Santa Monica, CA, with a band integrated RF field equivalent to 0.0026% of the FCC MPE for the public.

The spectrum measurements represented in Figures 9-32-9-36 provide some perspective on environmental levels of RF from sources other than Smart Meters. The FM radio broadcast band has, historically, been determined to be a primary contributor to ambient RF fields[15]. Because most of the FM broadcasting within the LA region originates from atop Mt. Wilson, a considerable distance from many parts of the metropolitan area, median RF fields, in 1980, were found to be somewhat lower than in some other large cities. However, with the introduction of cellular telephone base stations, ambient RF fields have likely increased somewhat simply due to the density of cellular and PCS base stations and their distribution among the population. Interestingly, Figure 9-34, which illustrates activity in the PCS band (cellular telephones), indicated the greatest value of peak RF field contribution of the several bands measured in this neighborhood set of measurements with a value equivalent to 0.1% of the FCC MPE.

[15] Tell, R. A. and E. D. Mantiply (1980). Population exposure to VHF and UHF broadcast radiation in the United States. *Proceedings of the IEEE*, Vol. 68, No. 1, January, pp. 6-12.

Section 10: Shielding Effectiveness Measurements

RF fields can be reduced in strength by introducing conductive materials between the field source and the area to be shielding from the emissions. For example, the metallic meter box within which all electric power meters are installed will attenuate RF fields that may be directed to the back of the meter. This is partially responsible for the reduction in radiated power directly behind Smart Meters tested found in this investigation. While performing the measurements of RF fields associated with Smart Meters in California, a series of measurements of insertion loss afforded by three different types of metal lath was conducted. Metal lath is commonly used in construction of stucco homes, typical of southern California and it was of interest to examine what influence such material might have on Smart Meter RF fields.

Shielding effectiveness of different metal meshes

An impromptu measurement of three different metallic meshes was conducted by setting up the specially programmed Smart Meters, one for the 900 MHz band and the other for the 2.4 GHz Zigbee band, measuring the RF field with the SRM-3006 instrument and then placing a sheet of the different meshes between the Smart Meter and the measurement probe. Figure 10-1 shows this process where the Smart Meter is installed in a specially designed socket to allow convenient operation of the meter in different locations.

Three different forms of metal lath (mesh/netting) were evaluated at both the 900 MHz and 2.4 GHz frequencies associated with the Smart Meter emissions. Figure 10-2 shows these lath samples. The lath shown in panel A consisted of a square shape, measuring 2 inches on a side. The lath in panel B is what is commonly known as "chicken wire" consisting of hexagonal shaped openings approximately one inch by one inch. The lath in panel C was comparatively, significantly smaller in dimension, measuring approximately one-quarter inch by one-half inch for the openings. The lath in panel C is more commonly used in plaster work as opposed to exterior stucco application but is also used in application of exterior rock surfacing in some areas of the country. Insertion loss measurement results for the different types of metal lath are given in Table 10-1. Insertion loss is expressed in linear units as a reduction factor and logarithmically as decibels (dB). These data are presented graphically in Figure 10-3.

Figure 10-1
Measurement setup to determine the insertion loss presented by a conductive mesh (chicken wire in this case).

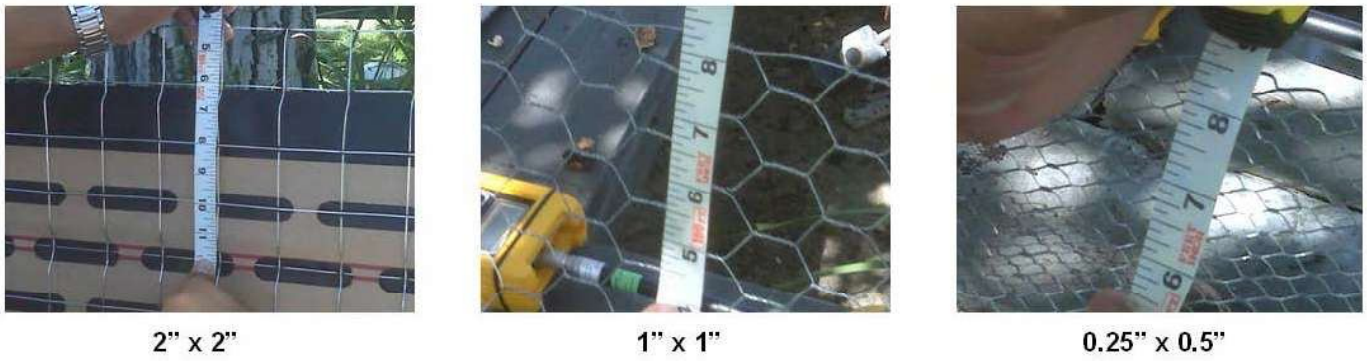

Figure 10-2
Measurement setup to determine the insertion loss presented by a conductive mesh (chicken wire in this case).

Table 10-1
Insertion loss measurement results for three different types of metal lath expressed as a reduction factor (F) and in decibels (dB).

	Panel A lath		**Panel B lath**		**Panel C lath**	
Frequency band	F	dB	F	dB	F	dB
900 MHz	2.5	4.1	8.9	9.5	82	19.1
2.4 GHz	1.3	1.2	2.6	4.2	14	11.4

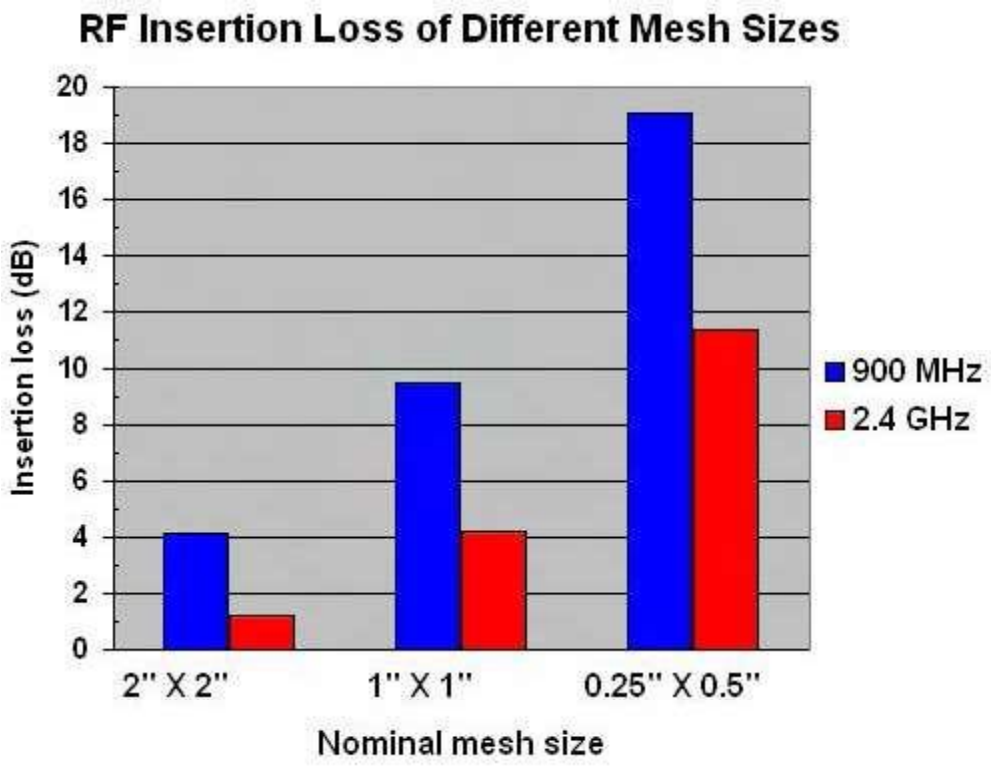

Figure 10-3
Insertion loss of three different metal mesh sizes.

The data given in Table 10-1 and shown in Figure 10-3 show an expected increase in insertion loss (attenuation) of RF fields with decreasing mesh size and a decrease in insertion loss with the higher frequency band. With the relatively large 2" by 2" mesh, the least insertion loss was associated with the 2.4 GHz band. The greatest insertion loss was for the longer wavelength emission in the 900 MHz band and the finest mesh size. The data suggest that the chicken-wire type netting commonly used in stucco home construction can afford significant reductions in RF fields that may enter the home ranging from 4.2 dB to 9.5 dB. This range corresponds to mesh transmissions of 38% in the 2.4 GHz band and 11% in the 900 MHz band.

Shielding effectiveness of a simulated stucco wall

To more completely evaluate the attenuation exhibited by a typical stucco wall, as might be found in southern California homes, a simulated wall section was constructed in Colville, WA. The wall was built as a 4 feet wide by 8 feet tall section on a support with casters to allow mobility. The wall was constructed with 2x4 dimensional lumber with studs on 16 inch centers with the "outside" wall sheathed with 7/16" OSB (oriented strand board). This sheathing was then covered with one layer of underlayment paper (Davis Wire All Purpose Super Kraft Asphalt Sheathing Paper) followed with the "chicken wire" lath being stapled onto the wall section (Davis Wire "self-furr" stucco netting, woven 17 gauge, 1.5 inch by 2.25 inch mesh opening). Two coats of stucco (Spec Mix® Scratch & Brown Preblended Stucco), including a base scratch coat followed with a brown coat, were applied to the lath. Each coat was allowed to set for several days. R-13 fiberglass insulation (Owens Corning with Kraft facing) was placed between the studs and the "interior" side of the wall was then covered with half-inch sheetrock (dry wall). Appendix F shows the wall during construction and Figures 10-4 and 10-5 show it set up with a Smart Meter positioned on a shelf on the "outside" wall surface with the two different measurement antennas supported on a tripod.

Measurements were conducted using the Wi-Spy spectrum analyzer device described earlier. The Wi-Spy unit was evaluated prior to its use by injecting swept signals from a communications monitor (IFR Model 2975) and using the Wi-Spy to acquire a large number of scans of the 900 MHz and 2.4 GHz bands. The Wi-Spy was determined to exhibit a response across each band that was within the range of ±0.5 dB. A yagi antenna was established on a support and directed toward a Smart Meter programmed for continuous operation without the wall in place. The Smart Meters[16] were positioned approximately 1 inch from the stucco surface of the wall to the rear surface of the meter and 58 inches from the ground to the center of the meter. RF field strength was then measured without the wall and with the wall in place to assess insertion loss. The measurement antenna and the Smart Meter was not moved during this process; only the wall was removed and replaced for the measurements with and without the wall. For the 900 MHz band, the five element yagi antenna was an M^2 Antenna Systems, Inc. Model 911-ISP (with 11 dBi gain) set 37 inches from the backside of the simulated wall to the end of the antenna boom and at the same height as the center of the Smart Meter. For the 2.4 GHz band, the yagi antenna was an Air802, LLC Model ANYA2412 (with 12 dBi gain) set 47 inches from the backside of the simulated wall to the end of the antenna boom. Figure 10-4 shows the 900 MHz yagi antenna in place for measuring the RF field behind the simulated wall. The 2.4 GHz yagi is shown during measurements of the attenuation of the wall in the higher frequency band in Figure 10-5.

For each frequency band, a series of five repeated measurements were performed of the received signal strength, measured in dBm, for horizontal and vertical polarizations. Table 10-5 summarizes the measurement data from which insertion loss values for the 900 MHz band and 2.4 GHz bands were determined. A representative view of the Wi-Spy spectrum analyzer display is seen in Figure 10-6.

[16] Model CL200, 902.25 MHz, SCE# 222010-273722. Model CL200, 2405 MHz, SCE# 222010-273720.

Figure 10-4
Measurement setup for determining insertion loss of simulate stucco wall shown with the 900 MHz yagi antenna.

The data in Table 10-2 show that, under the measurement conditions used, the simulated stucco wall offered an overall average insertion loss (attenuation) of the Smart Meter RF fields of 6.1 dB in the 900 MHz RF LAN band and 2.5 dB in the 2.4 GHz Zigbee band. These data are consistent with the earlier measurements of insertion loss of different dimension metallic lath materials using the isotropic probe of the SRM-3006 instrument. Differences between the two sets of measurements are likely due to the different dimensions of the wire netting (1.5 inches in this case), inclusion of the wall building materials and the different measurement distances used. In the case of the earlier described measurements, the measurement probe was located 6 inches from the various mesh materials compared to between 37 and 47 inches in the simulated wall measurements. Further, the isotropic measurements were made with the Smart Meter source placed 6 inches from the mesh and directed toward the mesh; some degree of interaction with the RF transmitters could be expected that could also contribute to differences in measured values of insertion loss. In the simulated wall measurements, the back of the meter was placed within 1 inch of the stucco with its underlying metallic netting.

Figure 10-5
Measurement setup for determining insertion loss of simulate stucco wall shown with the 2.4 GHz yagi antenna.

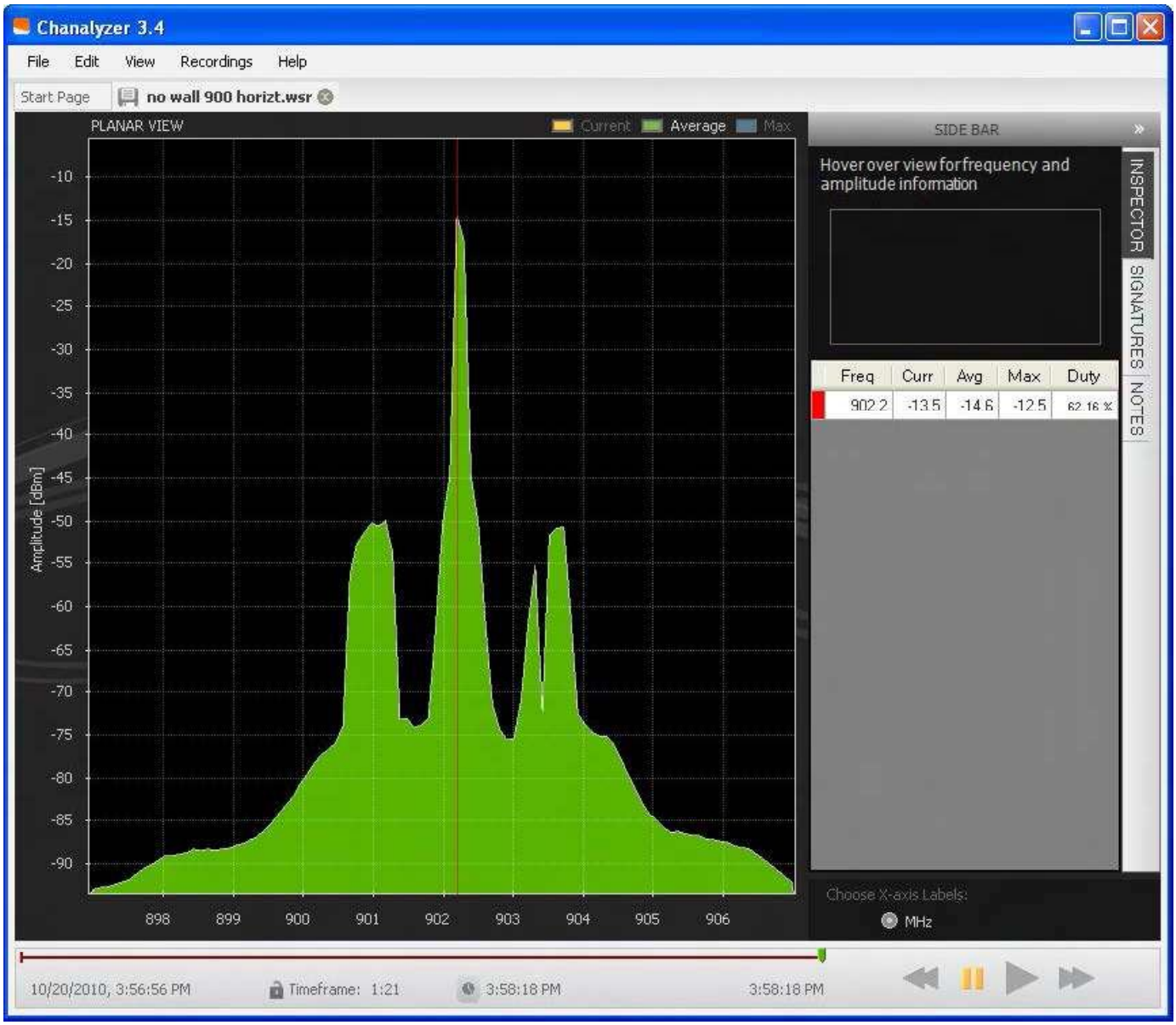

Figure 10-6
Illustrative display of the Wi-Spy spectrum analyzer display showing the captured average signal measured over a period of 81 seconds from a 900 MHz RF LAN Smart Meter transmitter. In this measurement, the simulated wall was not present. Marker readout data are shown on the right side of the display.

Table 10-2
Insertion loss measurement data for simulated wall in 900 MHz and 2.4 GHz bands.

		Signal level (dBm)				
Frequency (MHz)	Trial	No wall		With wall		Insertion loss (dB)
		Horizontal	Vertical	Horizontal	Vertical	
902.25	1	-14.5	-18.2	-22.5	-21.8	6.2
902.25	2	-14.5	-18.0	-22.5	-21.5	6.1
902.25	3	-14.5	-18.3	-22.6	-21.4	6.0
902.25	4	-14.5	-18.0	-22.5	-21.6	6.1
902.25	5	-14.5	-18.1	-22.5	-21.6	6.1
					Mean±SD	**6.1±0.1**
2405	1	-30.7	-34.7	-34.7	-35.6	2.9
2405	2	-30.5	-35.0	-34.8	-35.2	2.8
2405	3	-30.7	-35.3	-34.5	-34.5	2.1
2405	4	-30.3	-35.2	-34.6	-33.8	2.1
2405	5	-30.3	-35.2	-34.4	-34.6	2.4
					Mean±SD	**2.5±0.4**

Section 11: Spatial Variation of RF Fields

During the time that the simulated wall was being constructed, the variation of RF field along a vertical line adjacent to a Smart Meter was determined for the 900 MHz RF LAN and the 2.4 GHz Zigbee transmitters. This measurement was to provide practical insight to the spatial distribution of exposure for someone standing near a Smart Meter. Such is relevant since RF exposure limits are in terms of not only time averaged values of fields but the spatial average over the dimensions of the body. In practice, to evaluate the spatial average for purposes of demonstrating compliance with the IEEE exposure limits, an average of the RF field power density along a vertical line is recommended in IEEE C95.1-2005.

The SRM-3006 was used to measure the RF fields and log repetitive values as the SRM-3006 probe was moved slowly from the floor to a height of six feet above the floor with the Smart Meter positioned on a nonconductive table at a height of three feet. Figures 11-1 and 11-2 illustrate the measured results for this evaluation for the 900 MHz RF LAN and 2.4 GHz Zigbee bands.

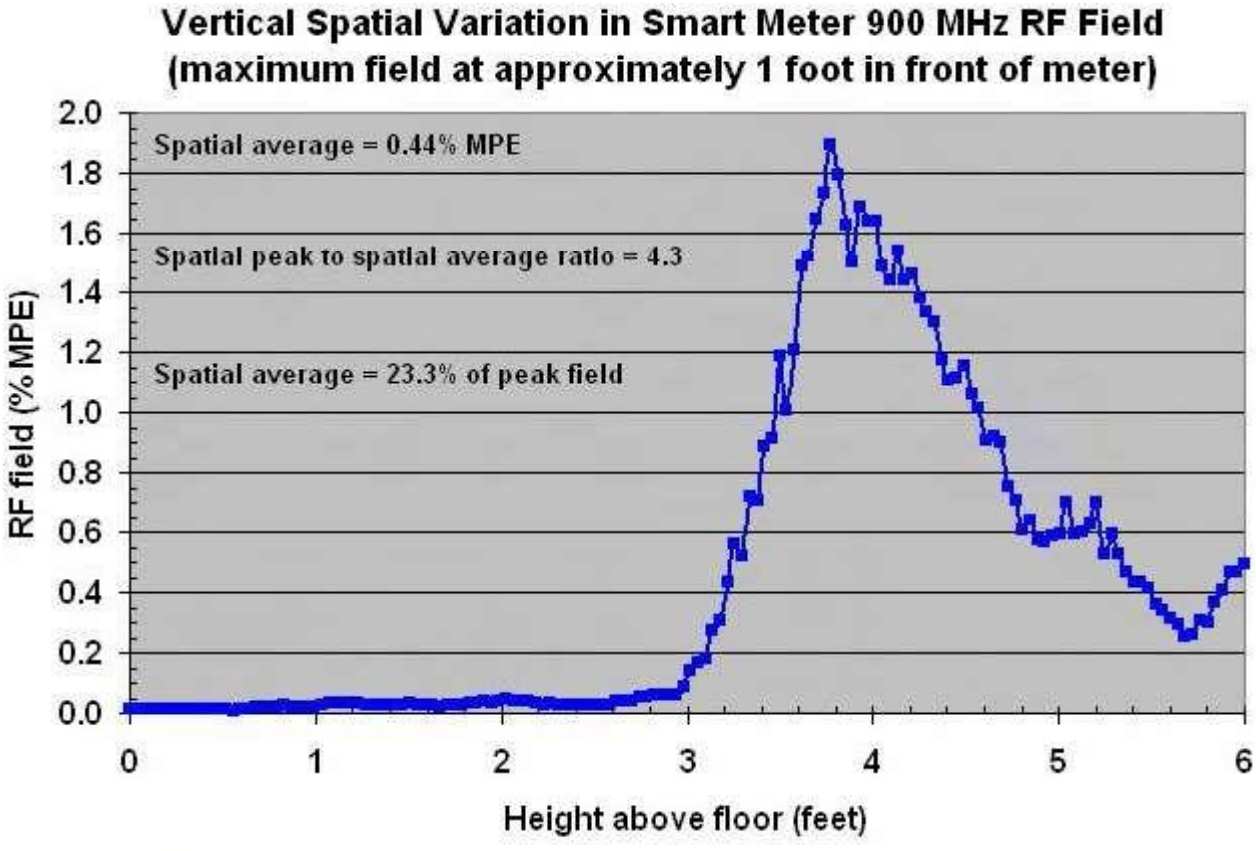

Figure 11-1
Vertical spatial variation in Smart Meter 900 MHz RF LAN field from 0 to 6 feet above the floor at a lateral distance from the Smart Meter of approximately 1 foot.

When the 252 readings obtained for the 900 MHz band over the six foot vertical height were analyzed, a spatial average equivalent to 0.44% of the MPE was obtained. The peak value was equivalent to 1.9% of the MPE. Hence, the six-foot spatial average was 23.3% of the overall peak value.

The result of similar measurements for the 2.4 GHz Zigbee transmitter with the Smart Meter at the same height is shown in Figure 11-2. A total of 136 measurements were made along the vertical span of six feet. Analysis of the values resulted in a peak value equivalent to 1.3% of the public MPE with a spatial average of 0.24% making the spatial average 17.8% of the overall peak value. These results provide insight to how the spatially averaged exposure is related to the maximum (peak) value of field for someone standing immediately near a Smart Meter.

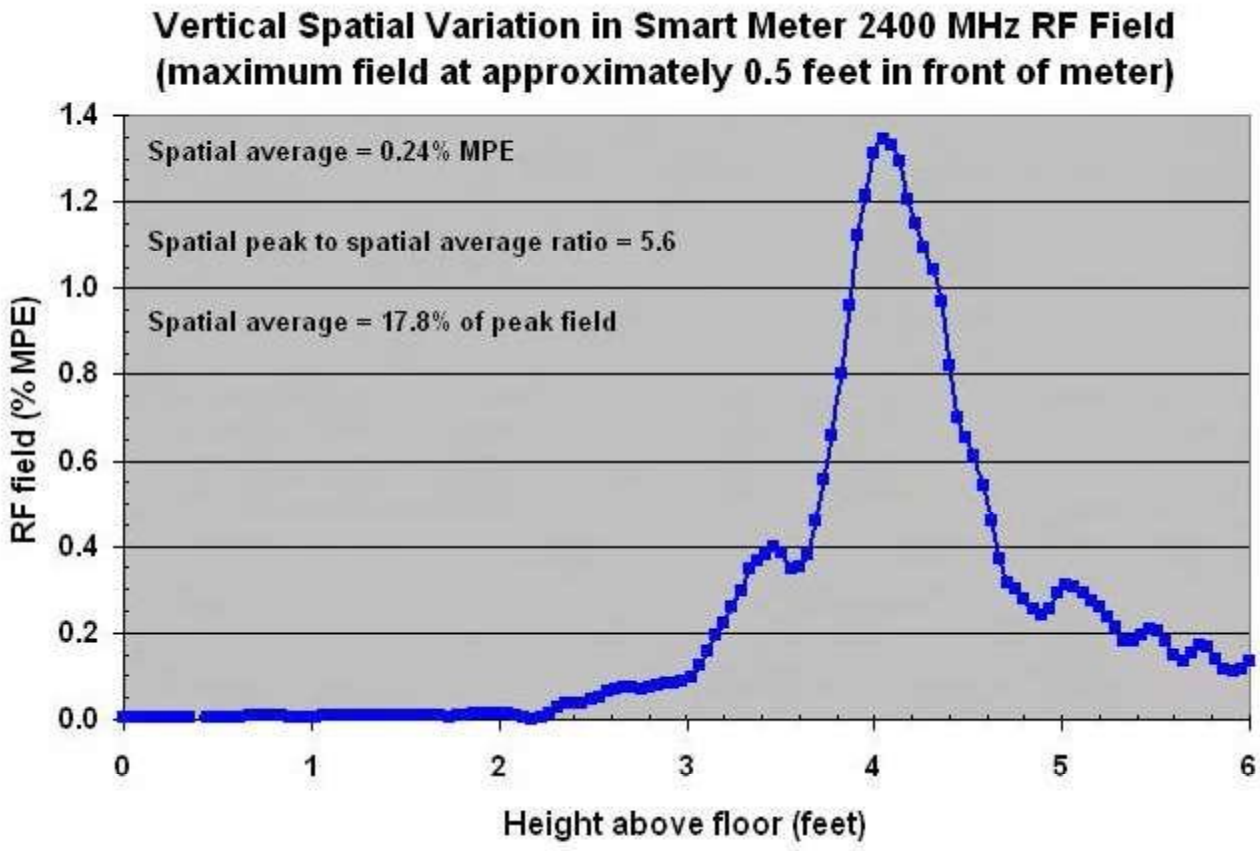

Figure 11-2
Vertical spatial variation in Smart Meter 2.4 GHz Zigbee transmitter field from 0 to 6 feet above the floor at a lateral distance from the Smart Meter of approximately 0.5 feet.

Section 12: Operational Duty Cycle of Meter Transmitters

While the utility of using continuous transmissions for performing measurements to characterize the magnitude of RF fields from Smart Meters has been described in this report, all of the scientifically based human exposure standards for RF exposure are in terms of time-averaged values of the RF fields, typically expressed in power density units. Attempting to make measurements of the actual time-averaged field magnitude, however, can be very difficult and time consuming. Further, the transmitter activity of a given Smart Meter is expected to vary from hour to hour and day to day. Hence, a single determination of duty cycle may provide insight to the likelihood of the value being rather large or very small but without much statistical power. Ideally, the duty cycle should be characterized over a sufficiently long time period to provide confidence in what the maximum expected duty cycle during any 30-minute period would be[17]. Clearly, making such determinations from on-site RF field measurements is fraught with significant challenge since an extended monitoring program would be needed. The limited measurements discussed below represent on a very preliminary approach to dealing with this challenge. A more comprehensive technical approach is warranted that is beyond the scope of what this project represented.

SRM-3006 measurements of peak and average values

Two different opportunities was pursued that provided limited insight to Smart Meter duty cycles. First, in the Itron meter farm, the SRM-3006 was taken on a stroll down two lanes of a part of the meter farm (western part), walking approximately mid-distance between the rows of meter racks (approximately 10-11 feet from either row of meter racks as shown in Figure 12-1. The instrument was set for measurement of both the peak field and average field. After completion of the walking survey, the recorded spectrum appeared as in Figure 12-2. When the peak and average spectra were integrated, the overall composite peak field was indicated as 0.114% of the MPE and the average value was indicated as 0.00023% of the MPE. These values may provide insight to what might be found in Smart Meter deployed neighborhoods and should represent conservative estimates of the magnitude of RF fields since is it difficult to envision a residential neighborhood with such a dense distribution of Smart Meters.

[17] Thirty minutes is the averaging time for the FCC MPE for general public exposure.

Figure 12-1
Measurements of aggregate maximum and average RF fields found along two rows of Smart Meters in the Itron meter farm during normal operation of the meters.

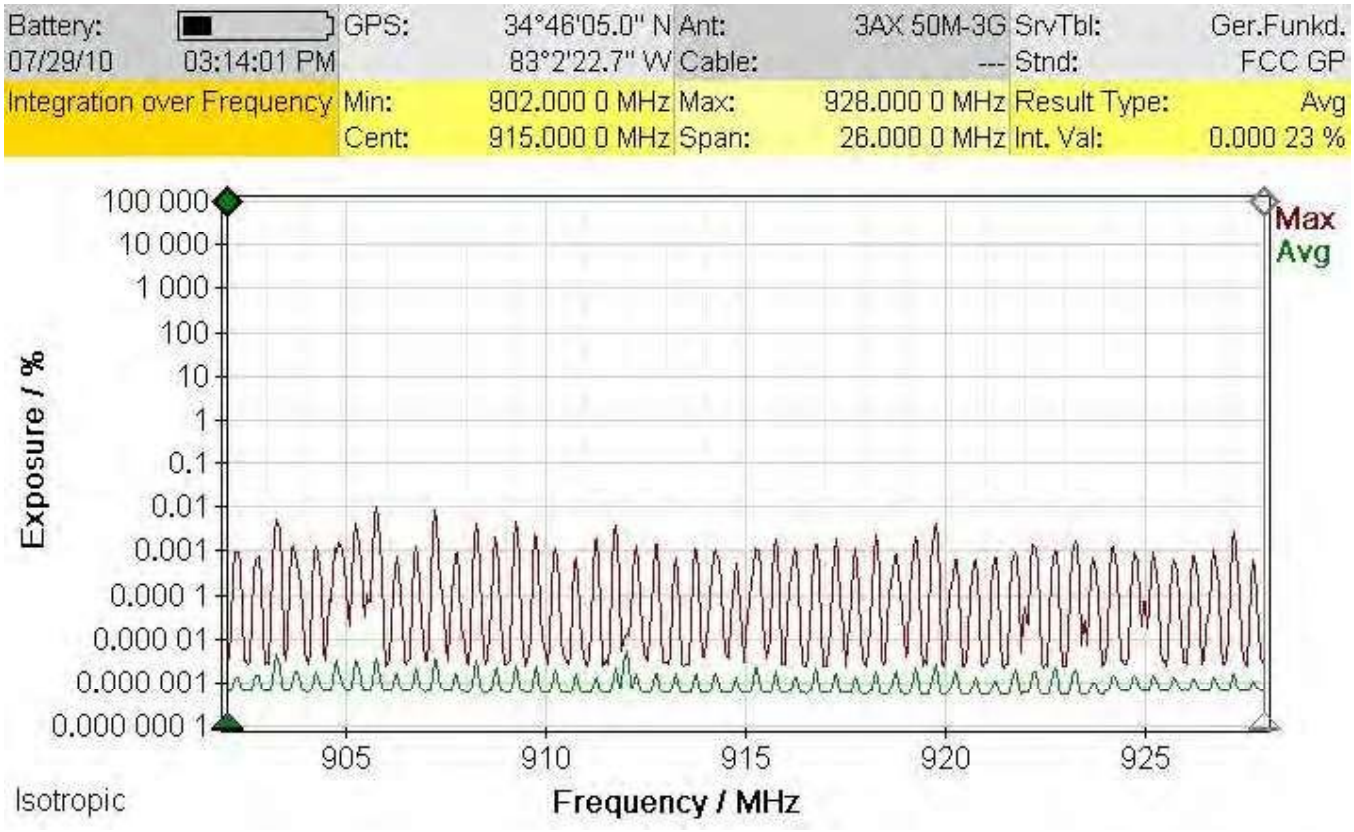

Figure 12-2
Spectrum analysis measurement of RF fields during walk through section of the Itron meter farm in which both the maximum (peak) field and the average field was measured.

An estimate of the duty cycle associated with operation of multiple Smart Meters is provided by the ratio of the average to peak integrated values. In this case, the ratio was equivalent to an approximate duty cycle of 0.2%. This singular value provides no insight to what the duty cycle might be of an individual, residential meter since measurements over a much longer period would be required.

Secondly, measurements taken at two apartment buildings in Downey, California during which measurements were performed over five-minute periods in front of banks of nine and eleven Smart Meters. The ratio of the integrated average to integrated peak spectra yielded apparent duty cycles of approximately 0.023% at the nine meter bank and 0.025% at the eleven meter bank. These values are substantially less than that found in the vicinity of thousands of meters at the Itron meter farm despite that fact that transmitter activity was easily noticeable during even the short duration of measurement.

Wi-Spy spectrum analyzer measurements

A Wi-Spy spectrum analyzer, described earlier in the instrumentation section, was applied to the measurement of the momentary emissions of a residential Smart Meter. A Wi-Spy feature that records the result of each spectrum scan by the analyzer was exploited to evaluate whether such a low cost instrument could be effective in revealing Smart Meter duty cycles. The device was place close to a residential Smart Meter and allowed to acquire scans of the 902-928 MHz spectrum over a period of an hour. Each scan took approximately 0.37 seconds so during one hour of monitoring, approximately 10,657 scans of the spectrum were accumulated and stored to the hard disc drive of the laptop computer. This measurement was made during a time when the mesh network was being queried and the meter was expected to be transmitting. In essence, the frequency spectrum is captured as 255 signal amplitude values uniformly distributed over the frequency band. The resolution bandwidth of the Wi-Spy analyzer was 125 kHz. The stored data file was opened and converted to a spreadsheet compatible format to allow inspection of the individual signal levels

at each frequency for each of the 10,657 scans to provide insight about transmitter activity during the monitoring period. Hence, a spreadsheet containing a number of rows equal to the number of scans accomplished by the Wi-Spy analyzer with 255 columns resulted. A primary objective was to determine whether the recorded data in the Wi-Spy file could be interpreted in terms of transmitter activity.

The strategy used in inspecting the data was to count how many rows of data contained at least one instance of a signal being present on any frequency within the spectrum. This would signify that an emission from the meter was captured by the analyzer. Since the analyzer can only record the amplitude of a given frequency once every 0.37 seconds, it cannot accurately indicate how long any particular emission lasted, only that one occurred during a scan. Thus, the analysis method applied in this case was that if an emission was detected during any one scan, it was assumed to last for the duration of the scan. In this way, a count of the total number of scans in which a signal was detected when multiplied by the scan time was assumed to conservatively represent (over estimate) the total amount of time that the transmitter was active. Duty cycle was then obtained by simply dividing the total transmit time by the total observation time.

Figure 12-3 shows the appearance of one of the measured "max hold" spectra of signals observed at a residential Smart Meter during an approximately 66 minute monitoring period.

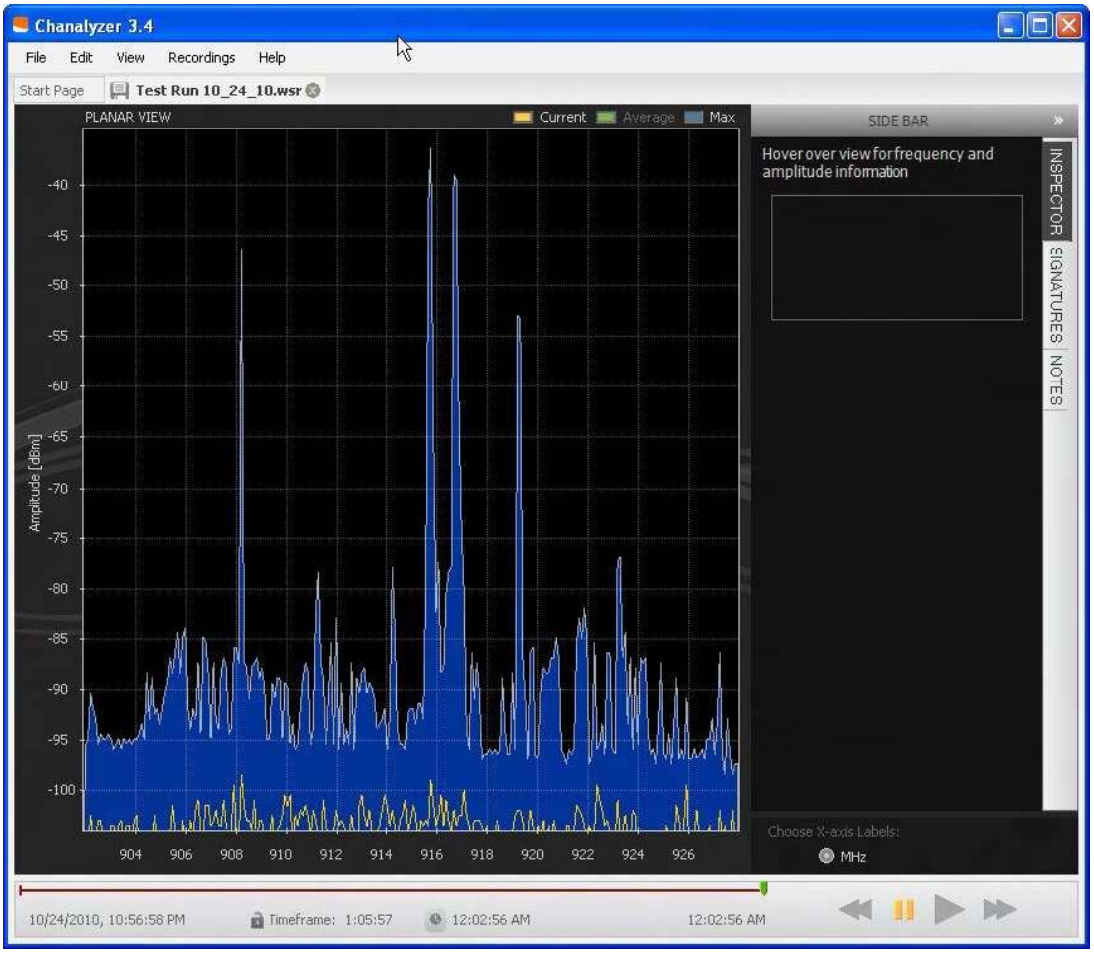

Figure 12-3
Wi-Spy display of Smart Meter RF fields observed during 66 minute monitoring period beginning at 10:56 P.M. local time. The blue spectrum is the "max hold" spectrum showing the maximum received signal power detected during the entire monitoring period. The four peaks that stand out from all of the rest of the detected signals, because of close proximity of the Wi-Spy to the meter, represent the momentary emissions detected from the Smart Meter being studied. Lower level signal peaks (typically 20 to 30 dB lower) are from other smart meters in the neighborhood.

Upon examination of the data file, it was found that four emission events occurred during the approximately one hour observation period that were apparently associated with the Smart Meter of interest. This means that the four major peaks shown in Figure 12-3 corresponded to those four events with no prior or subsequent signals occurring on or near the frequencies of the peaks. Following the conservative assumption that the detected emissions lasted for the duration of a single scan, i.e., 0.37 seconds, a total transmission time of 4 × 0.37 seconds or 1.48 seconds. This total emission duration corresponds to a duty cycle, during the nominal one hour monitoring period, of 0.037%.

A subsequent Wi-Spy measurement was performed near the same residential Smart Meter on the next evening, this time almost two hours (1:55:25) between 9:25 P.M. and 11:20 P.M. as shown in Figure 12-4.

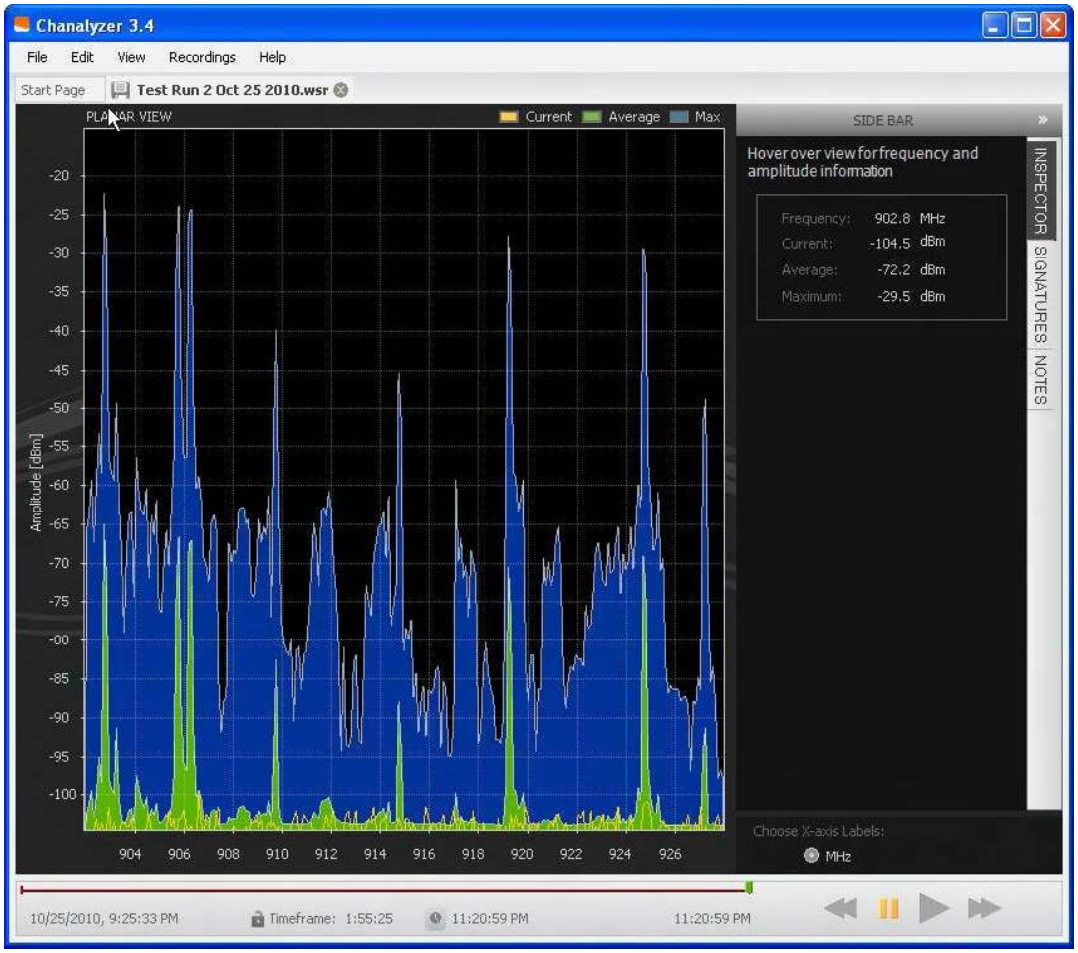

Figure 12-4
Wi-Spy display of Smart Meter RF fields observed during 115 minute monitoring period beginning at 9:25 P.M. local time.

Using the same analysis method, the measurement results were contained in 18632 scans of the band and suggested at least eight emission events were captured during the monitoring period from the meter of interest. Assuming that this represented the actual number of transmissions, with the same sweep time as in Figure 12-3, the apparent duty cycle would have been 0.0427%. This value is not significantly different from the first measurement on the previous day during approximately the same time.

On November 4, 2010, a Wi-Spy unit was set up at a fixed location in the Itron meter farm to capture a sample of RF fields in which the peak signal power and

average signal power values were measured over a 63 minute period. During this period, the Wi-Spy acquired a total of 15879 scans of the 902 - 928 MHz band emissions. This corresponds to a sweep period of 238 ms. Signal level measurements were made every 157 kHz across the 26 MHz of the band resulting in a total of 166 power values representing the spectrum. The result showed two clearly defined spectra, one of the peak signal level and the other of the average signal level. No attempt was made to translate these measured signal power values into equivalent field strength or power density of the RF fields. A small, omnidirectional antenna was attached to the Wi-Spy analyzer at the time. The data file captured by the Wi-Spy software was separately analyzed to find the mean values of the peak and average power spectra. This result in shown in Figure 12-5.

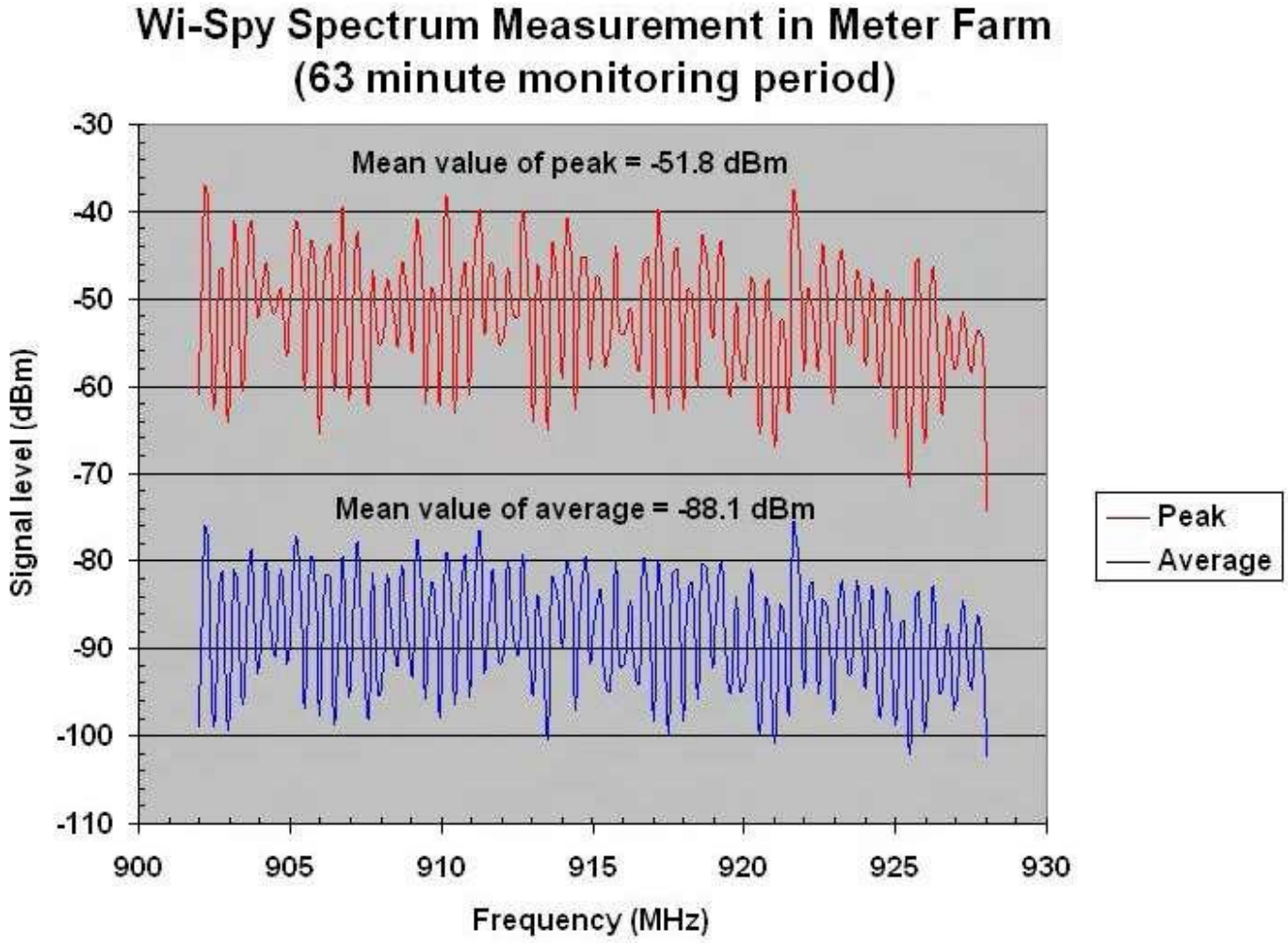

Figure 12-5
Processed signal power values obtained with the Wi-Spy spectrum analyzer on November 4, 2010, in the Itron meter farm. The average values of the peak and average spectra were -51.8 dBm and the -88.1 dBm respectively.

The difference between the mean values of the average and peak power spectra corresponds to 36.3 dB or an apparent duty cycle of 0.023%.

These exercises clearly indicate that the Wi-Spy unit is capable of detecting the Smart Meter emissions in terms of sensitivity. However, it is not clear that all transmissions from the meter are captured due to the sweep time and display update produced by the Chanalyzer software. The analyzer sweep time as set up for the 902 MHz to 928 MHz frequency band is 370 ms. Hence, any specific frequency within the band is

observed once every 370 ms. This suggests that for emissions lasting less than 370 ms, the probability of detection is decreased but will also depend on when during a particular scan that the emission occurs.

SCE Smart Meter Network Management System

A preliminary investigation was made to examine the potential utility of the SCE Smart Meter Network Management software for remotely determining the operational duty cycle of specific Smart Meters. Acquiring measurement data at specific meters from which an assessment of transmitter duty cycle can be made is technically demanding because of the highly intermittent nature of the Smart Meter signals, the pseudorandom frequencies of the signals across the spectrum and the general variability of Smart Meter mesh network activity throughout a day, week, month or year. Because of the self-healing character of mesh networks, wherein alternative data transmission paths can be invoked on a moment-to-moment basis, Smart Meter transmitter activity is more meaningfully defined through a statistical description. A Smart Meter's transmitter activity on one day may not be the same as on another day despite the periodic transmission of beacon signals to alert other meters of its presence in the network or of regularly scheduled data dumps of electric energy consumption; activity during a particular hour of the day may not be replicated during the same hour on another day. Further, depending on the topology of the mesh network, the duty cycle of more distant meters within a given network could be expected to be less than that of meters closer to the associated cell relay meter. Smart Meter duty cycles are, therefore, not fixed and can be dissimilar from one another and vary over time. Consequently, a full characterization of a particular Smart Meter duty cycle requires collection of transmitter activity over a prolonged period of days if not weeks and months. Added to this complexity is the fact that the network consists of a large number of meters and a full understanding of duty cycle means that a relatively long term data collection effort across many meters is necessary. The advantages of exploiting automated software based methods for obtaining such data are obvious. Finally, once the HAN function is implemented, the cumulative RF field caused by both the RF LAN and HAN transmitters and their effective duty cycles for a particular Smart Meter location may change.

During the period July 30 through October 26, 2010, SCE collected information pertaining to the number of data packets associated with either downlink or uplink communications from approximately 47,000 Smart Meters in part of SCE's territory. Downlink activity relates to data being propagated away from a cell relay meter while uplink activity is related to the transmission of data toward a cell relay meter. A presumption was made that both downlink and uplink traffic resulted in activity of the 900 MHz RF LAN transmitters in the meters. Using an estimate for packet length provided by Itron of 150 bytes for end point meters, with 8 bits per byte, and a data transmission rate for the 900 MHz RF LAN radio of 19.2 kbps (kilobits per second), the amount of transmitter activity was estimated for each of the meters on a daily basis for a total of 89 consecutive days. These transmitter duration data represented a total of 4,156,164 values. Each value of time (seconds per day) was then expressed as an estimated, average daily duty cycle. During the 89 days of data acquisition, some meters were found to not respond for various reasons or the data was corrupted resulting in an average number of meters from which valid data were obtained of 46,698 meters. Ultimately, these daily average duty cycles were then evaluated by examining their distribution in a percentile analysis. Figure 12-6 illustrates the result of this analysis where the average daily duty cycles are plotted across a range of percentiles.

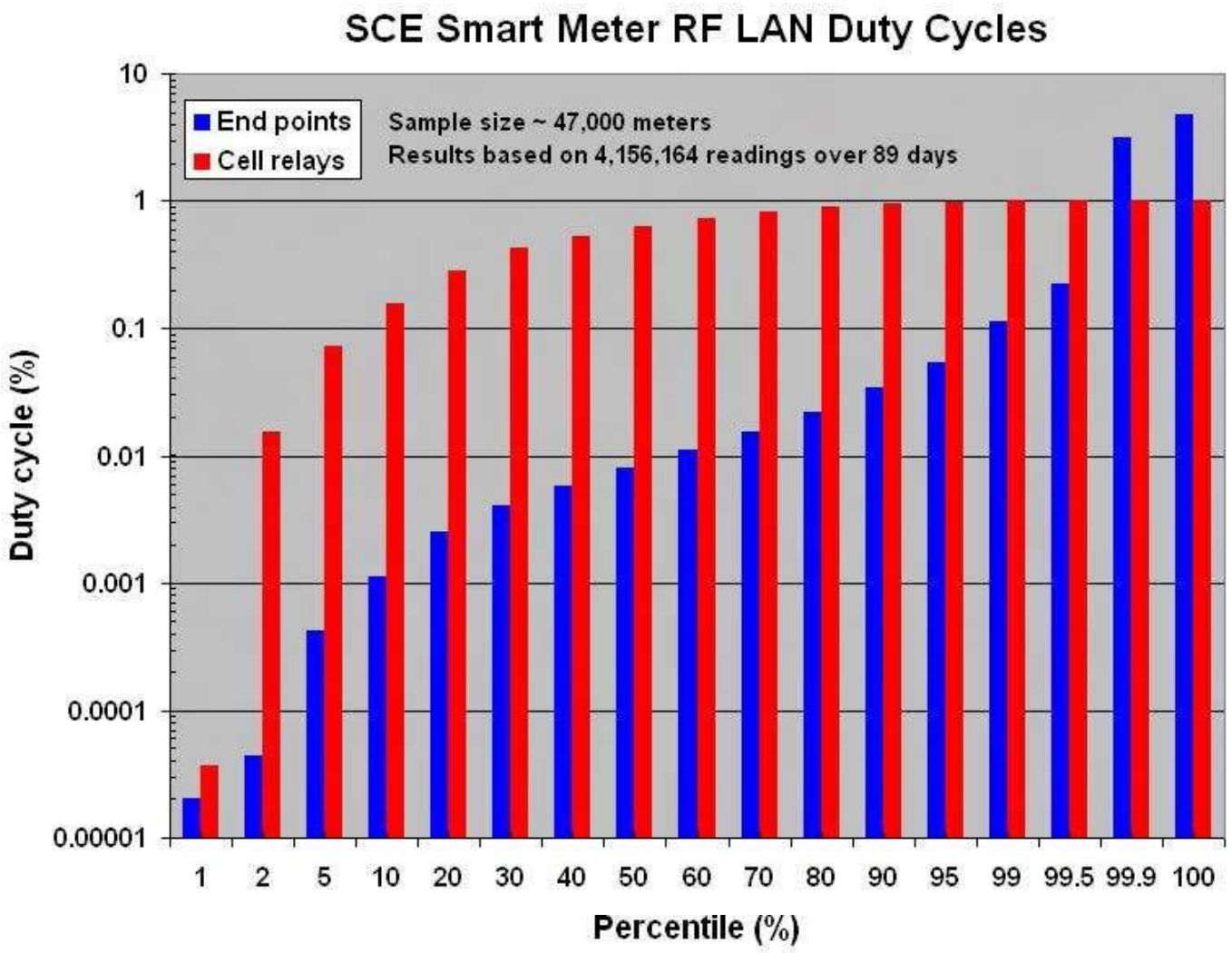

Figure 12-6
Analysis of SCE daily average duty cycle distribution for different percentiles based on 4,156,164 readings of transmitter activity from an average of 46,698 Itron Smart Meters over a period of 89 consecutive days. Analysis based on estimated transmitter activity during a day (see text).

RF LAN duty cycle data are presented in Figure 12-6 for both end point meters as well as cell relay meters. It was found that the sample size included some 111 cell relays. Based on information from Itron, the nominal packet size for data being transmitted down to the mesh network by a cell relay meter is 32 bytes. Using this figure for cell relays only, their estimated duty cycles were also determined and are shown in the figure.

Figure 12-6 tends to support the generally conservative estimate of approximately 5% for the duty cycle of some meters within a mesh network stated by Itron.[18] The maximum duty cycle obtained in the study was 4.74%. These data show that the upper duty cycle values pertain to only a very small percentage of meters; for example, while the duty cycle at the 99.9th percentile level (i.e., 99.9% of meters have smaller duty cycles) is 4.62%, dropping to the 99th percentile results in an average duty cycle of only 0.11%. Across the range of the 10th to 99th percentile, the duty cycle ranges from approximately 0.001% up to 0.1%. Figure 12-6 indicates

[18] *Analysis of Radio Frequency Exposure Associated with Itron OpenWay Communications Equipment. Itron publication, undated.*

that most meters, most of the time operate with very small duty cycles. The data presented in Figure 12-6 must be recognized as what is likely a conservative approach insofar as the total data packets (up link and down link) passing through a Smart Meter were tallied by the SCE data collection effort with the same assumed packet size assigned to each (i.e., 150 bytes).

Using results from the SCE duty cycle study relative to uplink data for the cell relay meters, an estimate of the maximum cellular transmitter activity was made. In this analysis, the greatest uplink data passing through the cell relay was assumed to be transmitted to the WWAN by the cellular transceiver in the cell relay with a throughput of 1.536 Mbps[19] with a 1/3 encoding overhead. Under this condition, an average duty cycle for the cellular transceiver in a cell relay is estimated to be approximately 0.088%. This very small value is due to the high data rate provided by the CDMA EVDO technology. This very low duty cycle for the CDMA EVDO implemented cell relays means that the time-averaged RF fields produced by cell relays will be correspondingly low. So, while the cellular transmitter is rated at a power near one watt, the effective duty cycle will reduce time-averaged RF fields to small values.

SDG&E Smart Meter Network Management System

Similar efforts to characterize typical Smart Meter duty cycles were made by SDG&E, with support from Itron, that provide additional insight to Smart Meter transmitter activity[20]. In the SDG&E study, 6,865 end point and cell relay meters were monitored for the number of bytes of data transmitted over an observation period of one day ending December 2, 2010. The data is for meters distributed across ten cells of approximately 600 meters per cell. In this study, while substantially smaller in size than the SCE study, a more accurate and direct assessment of the transmitter activity was made by interrogating the number of bytes of data transmitted by the transmitter. This approach does not rely on any assumption of the data packet size as was done in the SCE data collection and, hence, minimizes uncertainty in the assessment of duty cycles. Figure 12-7 illustrates an analysis of the distribution of Smart Meter RF LAN activity for the SDG&E data.

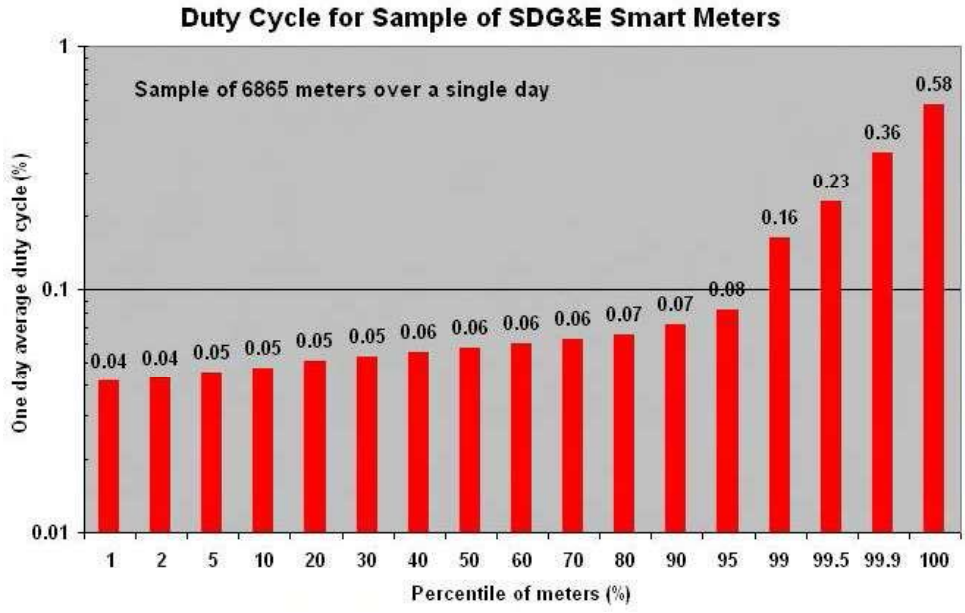

Figure 12-7
Results of an analysis of duty cycles for a sample of 6865 Itron Smart Meters deployed by SDG&E based on transmit duration during a single day of observation.

[19] Data rate for the CDMA EVDO Rev A cell relay modems ranges from 1536 kbps to 3072 kbps.

[20] Provided by Jim Turman, Safety and Emergency Services, San Diego Gas and Electric.

Figure 12-7 reveals a lower maximum duty cycle for the highest activity meters than observed from the SCE data but when the vast majority of meters are considered as a whole, the duty cycles are roughly in the same range. For instance, half of the SDG&E meters exhibited duty cycles of approximately 0.06%. From the SCE data, the 50th percentile of duty cycles was found to be approximately 0.01% rising to a value of about 0.06% at the 95th percentile. The differences in these two data sets is confounded by the fact that the data were collected in different ways, using different parameters for assessing transmitter activity, and represent substantially different sample sizes and sample collection periods. Nonetheless, because of uncertainties associated with data packet sizes in the down link and up link streams within the Itron mesh network, the SDG&E approach should yield more accurate values for meter duty cycles. Of further relevance, during this data collection period, a day light savings time update was performed as well as a meter firmware download (which would require a large number of up link acknowledgements). These factors would tend to drive the apparent duty cycle of meters upward when compared to other times of the year. Importantly, any differences between these preliminary studies of Itron Smart Meter duty cycles should not be viewed as differences in how the two networks, one deployed by SCE and the other by SDG&E, are designed to operate but, rather, as the result of the differences in how data were collected. In the case of SCE, only indirect measures of transmitter activity were obtained that require an assumption as to data packet sizes whereas in the case of SDG&E, a direct assessment of data traffic through the meters was obtained. Future software based studies of duty cycles of Smart Meters should include measures of both data packet counts as well as the number of bytes of data transmitted; such studies can provide insight to average packet sizes should other studies be performed in a similar fashion.

Section 13: Ancillary Measurements

Microwave Oven

During the residential surveys, when a microwave oven was encountered in the kitchen, measurements were made with a cup of water placed inside the oven. Leakage fields, as documented in Table 9-8, were detected at two feet from the oven as great as 22% of the public MPE. Subsequently, additional measurements were performed at the author's home using the SRM-3006. These data for a measurement at 1 foot from the oven are shown in Figure 13-1. The spectral distribution of the detected signal in Figure 13-1 is characteristic of microwave ovens; the frequency of the microwave oven drifts across a part of the spectrum and is essentially continuous in nature. Loading on the microwave oven due to temperature increases in the material being heated will cause the frequency to vary. Measurements taken at additional distances from the oven demonstrate a rapid decrease in signal with increasing distance as shown in Figure 13-2.

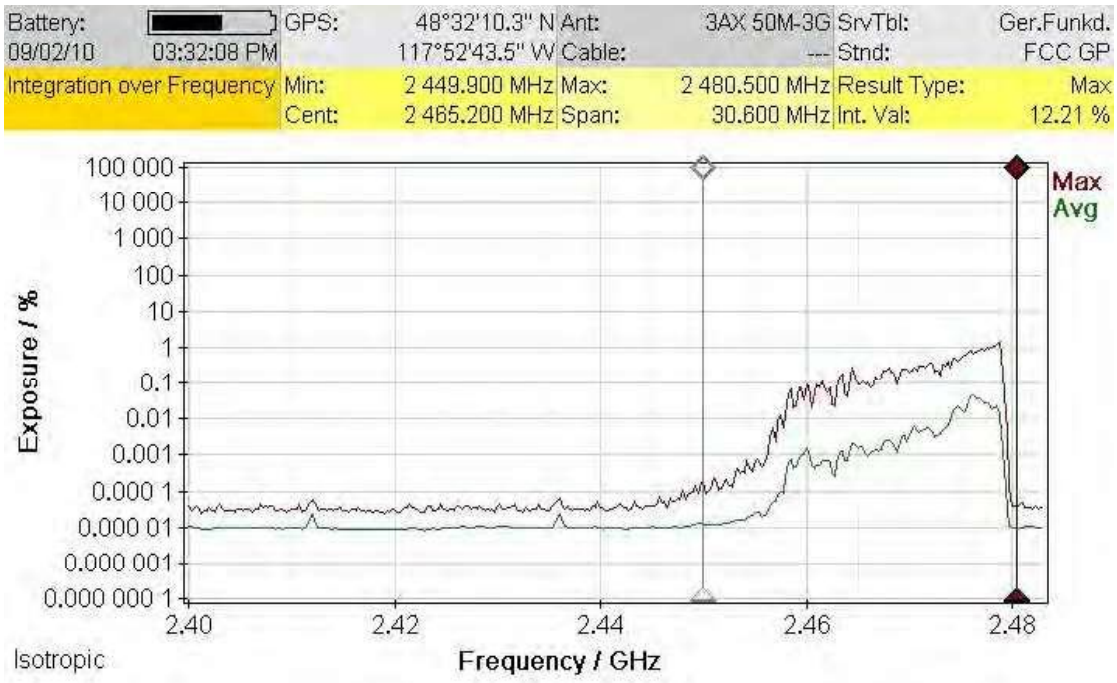

Figure 13-1
Measurement of microwave oven leakage at 1 foot from oven door seal.

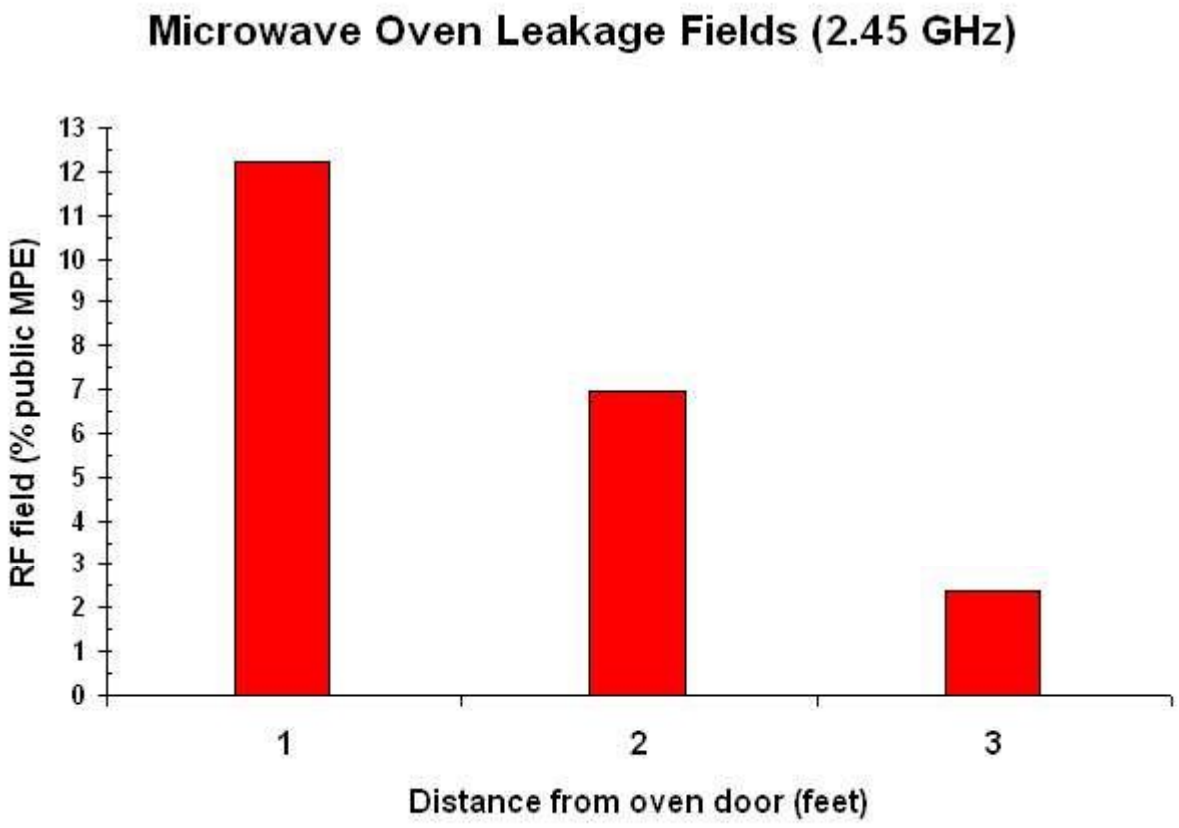

Figure 13-2
Microwave oven leakage vs. distance.

Microwave ovens must comply with regulations promulgated by the Food and Drug Administration (FDA) on leakage levels.[21] These regulations specify that the maximum leakage from a new oven, at the time of manufacture, shall not exceed a power density of 1 mW/cm^2. Once put into operation, the limit is set at a maximum leakage value of 5 mW/cm^2 at 5 cm from the oven surface. This is a product performance standard, not an exposure standard.[22] In practice, modern microwave ovens typically comply easily with the regulations by a wide margin. Since the FCC MPE for whole body exposure at the microwave oven frequency, 2.45 GHz, is 1 mW/cm^2, a measurement value of 12% of the MPE corresponds to only 1/5 of 12%, or about 2.4% of the 5 mW/cm^2 product performance standard.

Cordless telephone

Cordless telephones operate in a number of different frequency bands including 49 MHz, 900 MHz, 2.4 GHz and 5.8 GHz. Figure 13-3 shows a spectrum obtained from a 900 MHz band cordless phone with the base unit emitting signals near the upper end of the spectrum and the portable receiver unit emitting near the bottom of the spectrum. This figure is applicable to a measurement at one foot from the base unit and receiver.

[21] Performance Standards for Microwave and Radio Frequency Emitting Products, 21CFR1030.10. Food and Drug Administration, Department of Health and Human Services.

[22] It should be noted that the local RF field leakage limit of 5 mW/cm^2 is five-fold greater than the MPE for whole body exposure applied by the FCC.

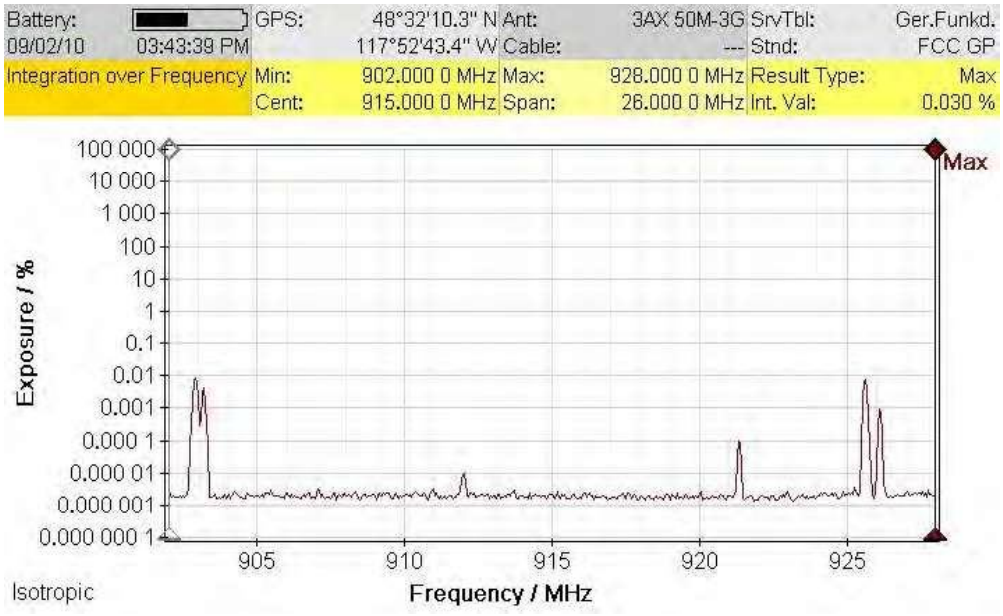

Figure 13-3
900 MHz cordless telephone RF fields from the base unit near the upper end of the spectrum and from the portable receiver unit near the lower end of the spectrum, measured at 1 foot from the base and receiver.

Wireless router

An observation also made during the residential measurements was the presence of a wireless router in a home office operating in the 2.4 GHz band. Figure 13-4 illustrates a measurement of the RF field produced by a wireless router in the author's office at approximately one foot from the router. A maximum field of 0.24% of the MPE was measured.

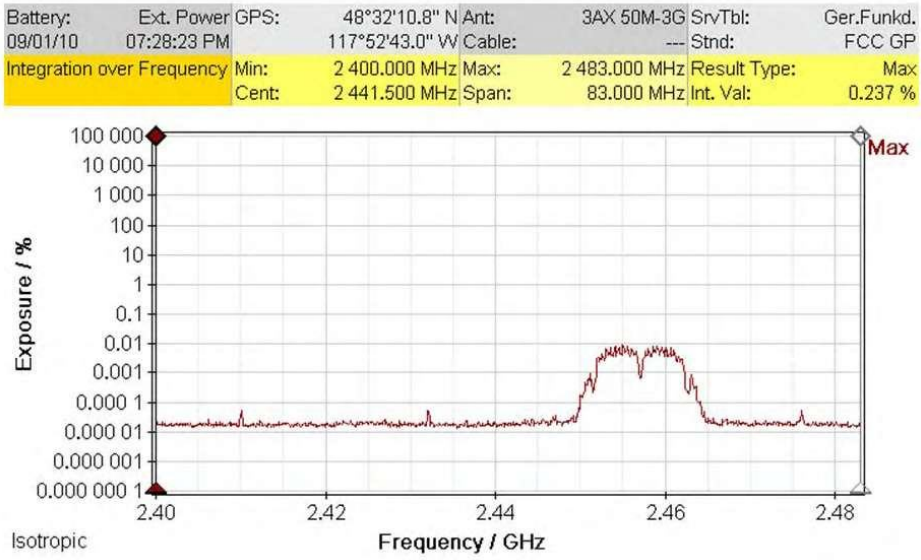

Figure 13-4
Measured RF emission spectrum of a wireless router at one foot from the router. The router was set to operate on Wi-Fi channel 10.

Measurement comparison (Model B8742D probe and SRM-3006)

Using the two specially programmed Smart Meters previously employed in the simulated stucco wall attenuation measurements, a comparison was made between the response of the broadband probe and the SRM meter. RF fields were measured by placing either of the two probes with their centers at 20 cm from the face of either Smart Meter and recording the readings. Each broadband probe reading was multiplied by the factory determined correction factor for the appropriate frequency (0.67 at 915 MHz and 0.97 at 2450 MHz). The results are presented below.

Table 13-1
Comparison of RF field probe readings at 902.25 MHz and 2405 MHz (%MPE)

	Frequency (MHz)	
Probe	902.25	2405
B8742D	2.01	1.21
SRM-3006	2.29	1.37
Percentage difference (%)	13.0	12.3
Difference (±dB)	+0.53, -0.57	+0.54, -0.54

These data indicate that the two instruments yielded readings of the RF fields that were within the manufacturer's specified uncertainties of calibration.

Section 14: Theoretical Analysis of RF Fields

Assessing RF fields associated with Smart Meters through theoretical analysis is an expedient approach to evaluating potential exposure of individuals who may be near the meters. This section describes the method used to obtain theoretical estimates of RF fields in the vicinity of the Itron Smart Meters studied in this project.

RF fields that might be associated with emissions from the various transmitting components of the Itron system, consisting of the Model CL200 end point meter and the Model C2SORD cell relay meter, were calculated following the methodology described here. This method includes a conservative approach of accounting for the possibility of ground reflections that can enhance the local RF field strength at any given location. The intensities of RF fields (expressed as power density) are calculated using conventional field calculation methods but with the inclusion of a ground reflection factor as recommended by the FCC[23]. Power densities were calculated according to the following relationship:

$$S(W/m^2) = \frac{P_t \times G_{\max} \times \delta \times \Gamma}{4\pi R^2} \qquad \textit{Equation 14-1}$$

Where,

S is plane-wave equivalent power density (W/m^2)

P_t is maximum transmitter output power (W)

G_{max} is the maximum possible antenna power gain (a dimensionless factor)

δ is the duty cycle of the transmitter (percentage of time that the transmitter actually transmits over time). More specifically, δ is the maximum duty cycle as found over any 30 minute period. This is because the averaging time for the MPE in the FCC rules and the IEEE standard (C95.1-2005) for the general public and applicable to the frequencies used by the Itron Smart Meters is 30 minutes. Determining the value of δ is challenging as described above. In most cases, estimates of δ are used based on understanding of the mesh network characteristics. In any event, δ is generally a very small value since the Smart Meters do not transmit most of the time.

R is the radial distance between the transmitter and the point of interest (meters)

Γ is a factor that accounts for possible in-phase ground reflections that could enhance the resultant power density. Under ideal reflective conditions, such as with a metallic ground plane, a field reflected from the ground could add constructively (in phase) with the field directly incident from the source to cause a maximum two-fold increase of the field strength at the reception point. Were this to happen, the phenomenon could lead to an increase of $(2)^2$ or 4 fold in the power density since the electric field is proportional to the square of field strength. In this case, the value of Γ in equation 1 would be 4. Under more realistic environmental conditions, where perfectly reflective surfaces are rare, an electric field strength enhancement of 60% has been recommended by the FCC. This corresponds to an enhanced electric field strength of 1.6 times the field arriving from the source without reflection or a power density enhancement factor of $(1.6)^2$ or 2.56 for use in equation 1. Application of a ground reflection factor in exposure assessment calculations becomes less meaningful at locations that are very close to the Smart Meter for two reasons. Since the emissions are directional in the elevation plane with generally relatively a smaller magnitude of RF energy being propagated downward at steep angles, the RF field strength striking the a reflective ground will be small. The reflected magnitude will be similarly small. Further, for close exposure conditions, within a few feet of the Smart Meter, the difference in propagation path length between that of the directly incident field and the reflected field usually be substantial. Hence, any reflected field component at the location of a person

[23] FCC (1997). *Evaluating Compliance with FCC Guidelines for Human Exposure to Radiofrequency Electromagnetic Fields*. Federal Communications Commission, Office of Engineering & Technology, OET Bulletin 65, Edition 97-01, August.

standing immediately next to a Smart Meter will be a very small fraction of the directly incident RF field leading to, essentially, no material enhancement of the field. For this reason, calculation of RF fields for the situation of very close proximity to the meter, wherein the greatest intensity of the field will exist, will more accurately estimate the actual field if the value of Γ is set to unity (1). As the distance from the Smart Meter increases beyond some value such that the body may be more uniformly illuminated by the incident fields, ground reflected fields have the potential of much more significant enhancement of the radiated field but at such distances, the magnitude of the field has become extremely small in comparison with that exhibited within a few feet of the meter.

For exposure distances of 1 to 3 feet in front of a meter mounted at 5 feet above the ground, the elevation angles to the ground that would result in an in-phase field addition range between nominally 78 degrees and 60 degrees. An examination of the elevation plane radiation patterns presented above reveals the following approximate values in Table 10-1 for the reduction in field for elevation angles of 60 to 90 degrees below the horizontal to the meter.

Table 14-1
Approximate RF field reductions (dB) caused by Smart Meter elevation plane patterns in the 60° to 90° range below a horizontal to the meter.

	Field reduction (dB)					
Angle (°)	900 MHz RF LAN end point meter	900 MHz RF LAN cell relay meter	2.4 GHz Zigbee end point meter	2.4 GHz Zigbee cell relay	850 MHz cellular cell relay	1880 PCS cell relay
60	-8	-7	-3	-11	-7	0
75	-10	-8	-4	-8	-10	-4
90	-11	-8	-8	-8	-10	-7

These data show that the typical range of RF field reduction is in the range of -3 to -10 dB with one exception associated with the 1880 MHz PCS transmitter in a cell relay meter at an angle of 60 °. This range is equivalent to between 50% and 10% of the field power density at the same distance but within the main beam of the transmitter. The maximum possible combined RF field occurs if both the directly incident plus ground reflected waves add in phase with one another; the contribution provided by the ground reflected component suffers degradation simply due to the weaker field directed toward the ground and the greater distance from the source to the point of interest. Through examination of the detailed pattern data represented by, for example, Figure 8-11 for the elevation plane pattern of a 900 MHz RF LAN end point transmitter, the relative EIRP along a vertical line located 1 foot adjacent to the Smart Meter from ground level to a height of 6 feet would be expected to be similar to Figure 14-2. Note the approximate qualitative similarity to the measured pattern shown in Figure 11-2 for the same type of transmitter.

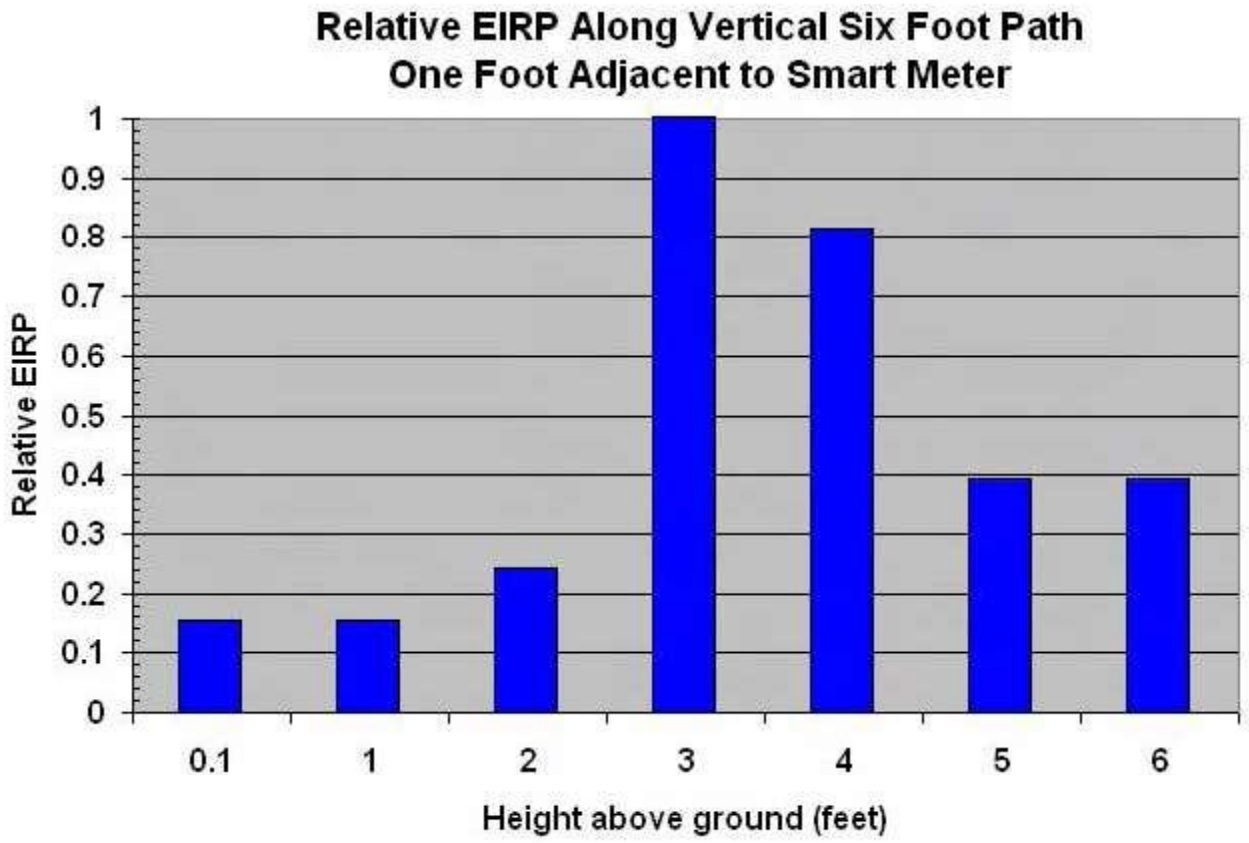

Figure 14-1
Relative EIRP of a 900 MHz RF LAN transmitter as observed along a six foot vertical line located one foot adjacent to the Smart Meter.

To illustrate the results of applying equation 1, calculations were performed for distances from 1 foot to 100 feet from each of the Smart Meter transmitters included in this study with the results shown in Figure 14-3. For the initial set of calculations, the duty cycle was assumed to be 100%, the same as the unique conditions under which RF fields were measured in this project. Such a value of duty cycle is not possible under normal operation of the mesh network. In fact, the mesh network is incapable of operation once the duty cycle were to reach approximately 30%[24]. As discussed above, realistic duty cycle values are in the range of a few percent at most with typical values well less than one percent. The results displayed may be adjusted by multiplying the indicated value by the duty cycle. Thus, the displayed field values are in terms of the instantaneous peak values of RF field. The calculated values also are for the maximum possible field based on the detailed pattern measurement data presented earlier; i.e., the calculated field magnitude is relevant only to that specific direction away from the meter at which the greatest RF field exists as represented by the transmitter powers summarized in Table 10-2. Further, the calculations plotted in Figures 14-3 and 14-4 include a value of 1.00 for Γ, i.e., no ground reflection enhancement.

[24] Personal communication from Itron.

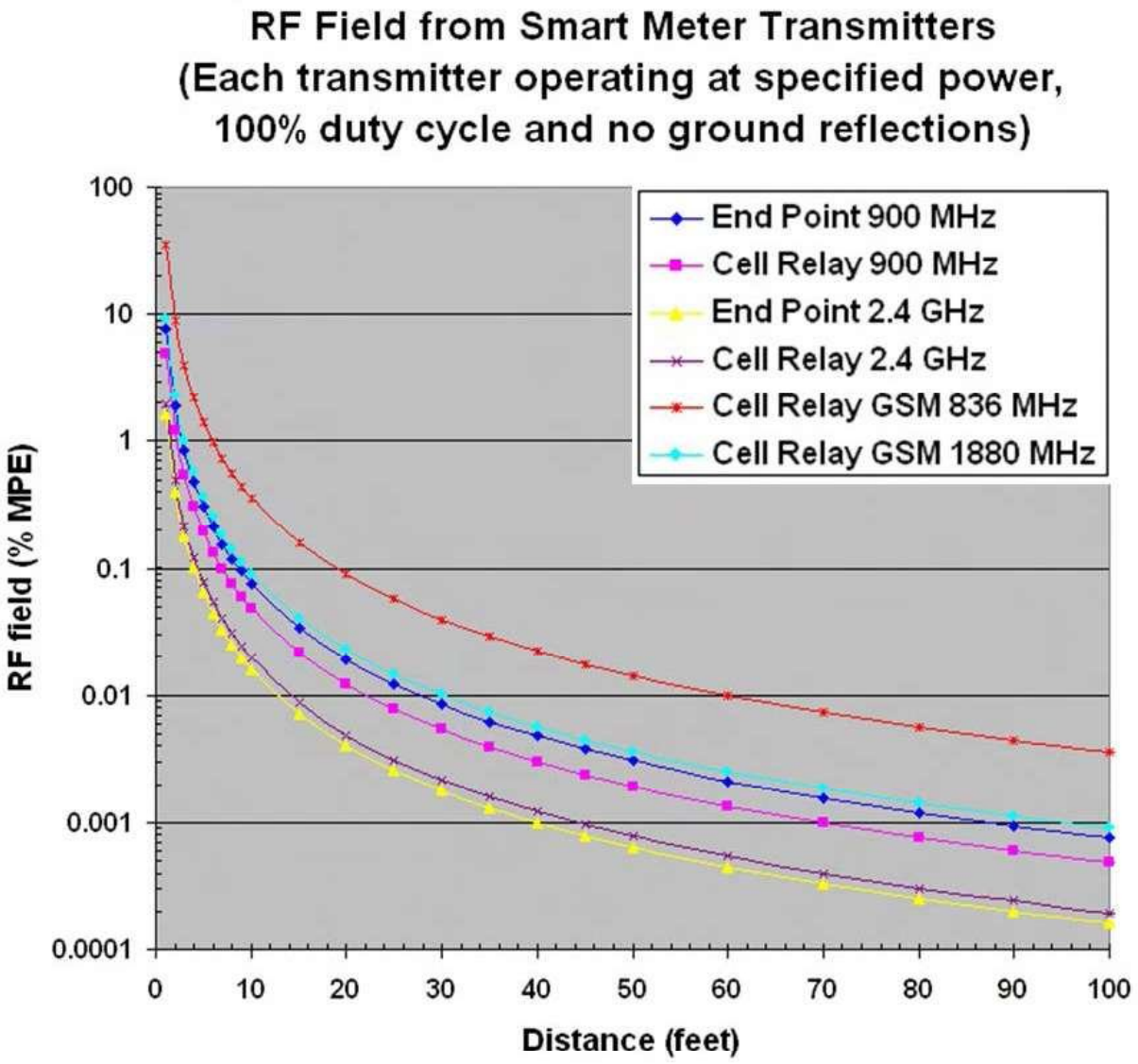

Figure 14-2
Calculated RF fields up to 100 feet at point of maximum intensity in main beam expressed as percentage of FCC general public MPE as function of distance in maximum beam for Itron Smart Meter components investigated in this study. No ground reflections have been included in this analysis, the field values are relevant to the peak value of field during the time that the transmitter is actually transmitting (no duty cycle correction) and spatial averaging has not been applied to the values. Normal operation of the Smart Meter will significantly reduce the actual field found in practice near operating meters.

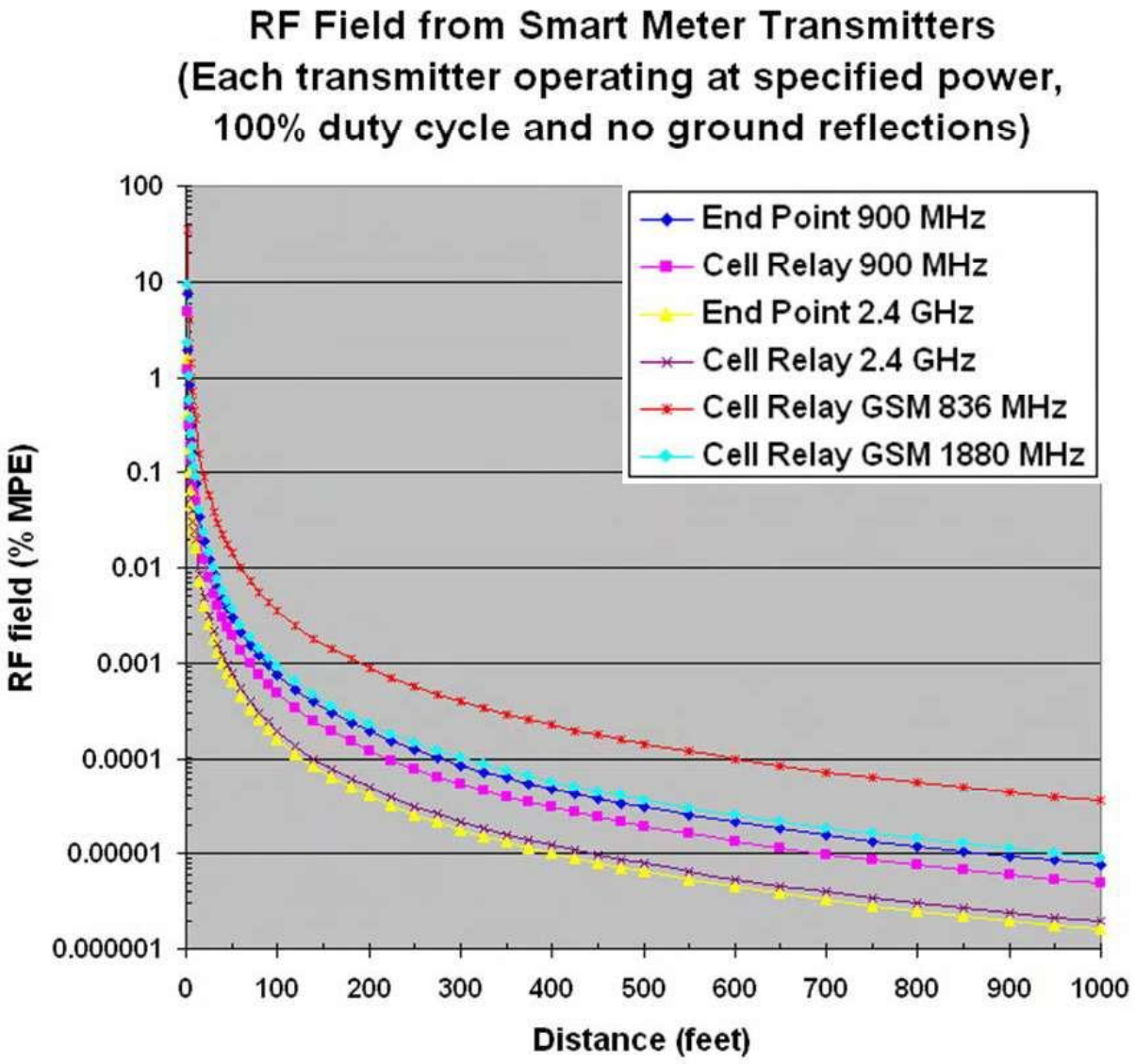

Figure 14-3
Calculated RF fields up to 1000 feet associated with Smart Meters included in this study with the assumption that the transmitters operate continuously (not possible in actual operation in a mesh network) and no ground reflections occur that might add constructively to enhance the field at the point of interest.

Table 14-2
Summary of nominal transmitter peak powers and 99th percentile powers for Itron Smart Meters studied in this investigation.[a]

Transmitter/ Meter	Frequency (MHz)	Gain (dBi)	Power gain	Most likely power (dBm)	99th percentile power (dBm)
RF LAN end point	914.8	2.85	1.93	24.5	26.0
RF LAN cell relay	914.8	0.88	1.22	24.5	26.0
Zigbee end point	2440	4.24	2.65	18.5	20.6
Zigbee cell relay	2440	5.08	3.22	18.5	20.6
Cell relay GSM	836.6	1.85	1.53	31.8	
Cell relay GSM	1880	1.56	1.43	28.7	

[a]Under FCC rules at 47 CFR 15.247, the peak output power of frequency hopping (using more than 50 channels) and direct sequence spread spectrum transmitters is limited to 1 watt.

Figure 14-2 shows the calculated peak value of RF field for the most likely transmitter powers without the presumed presence of possible ground reflections for distances up to 100 feet. To emphasize, the values shown do not account for typical transmitter duty cycles or spatial averaging. And, it's relevant to recognize that the peak calculated field applies to a single point in space that corresponds to the specific direction in space occupied by the main beam emerging from the Smart Meter. This is not necessarily a direction perpendicular to the case of the meter. Hence, measured values of field taken along directions geometrically normal to the meter surface will not represent the maximum possible field value. Figure 14-3 is a similar plot of calculated field values over a distance up to 1000 feet.

Figures 14-2 and 14-3 illustrate the most likely maximum RF field adjacent to the Itron Smart Meter components evaluated in this study, before adjustment for transmitter duty cycles. To provide conservative estimates of maximum possible peak RF fields, calculations were prepared using the 99th percentile values of transmitter powers discussed earlier and listed in Table 10-2. These results are shown in Figures 14-4 and 98 for distances up to 30 feet from the meter for a 900 MHz RF LAN end point transmitter and a 2.4 GHz Zigbee cell relay transmitter, respectively, these two transmitters yielding the greatest measured gain as listed in Table 10-2. In each of the figures, the solid curve represents the most likely transmitter power and the assumption of no ground reflections with the upper bars showing the RF field value is the 99th percentile transmitter power with the worst case ground reflections. For the upper bars, an assumption was made that ground reflections could exist that would enhance the RF field at the calculation point following procedures outlined in FCC OET Bulletin 65 (Γ = 2.56). Such an assumption of ground reflections, however, is not realistic since the point of maximum field magnitude near the Smart Meter may not correspond to the point in space at which any ground reflected wave happens to add constructively to the directly incident wave (in phase field addition). RF field values comprising the plotted values in Figures 14-4 and 98 and a similar maximum possible value for the 836 MHz cellular transmitter of the cell relay are given in Table 14-3 for the distance range up to 30 feet.

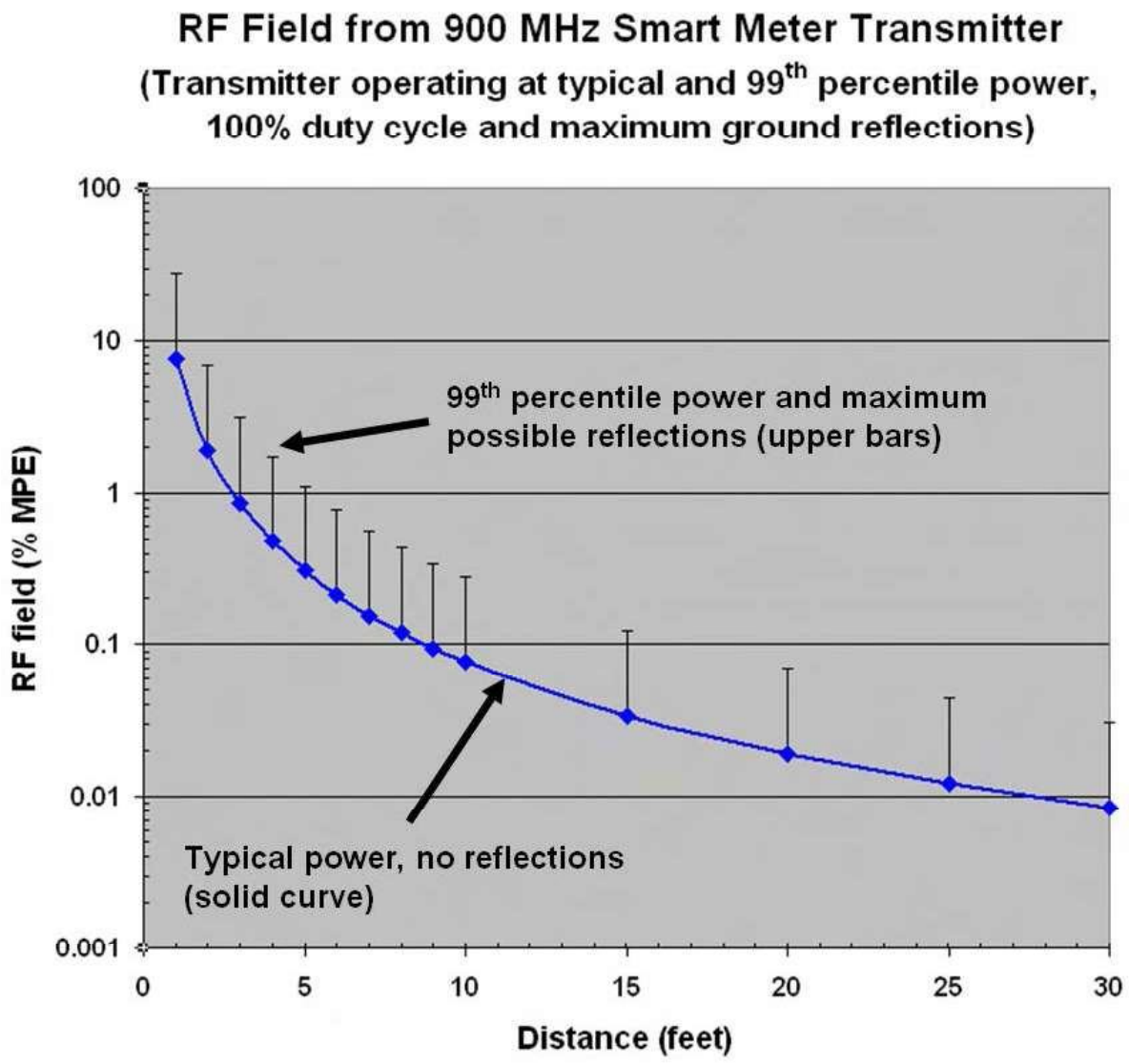

Figure 14-4
Calculated RF field produced by Smart Meter 900 MHz RF LAN transmitter using most likely transmitter power (solid curve) and 99th percentile transmitter power (bars above solid curve). Calculated maximum possible values are based on the 99th percentile transmitter power but assume the possibility of ground reflections (= 2.56) that would enhance the RF field at the calculation point (point of maximum RF field emission near the meter). Such ground reflections are not realistic but follow guidance in FCC OET 65.

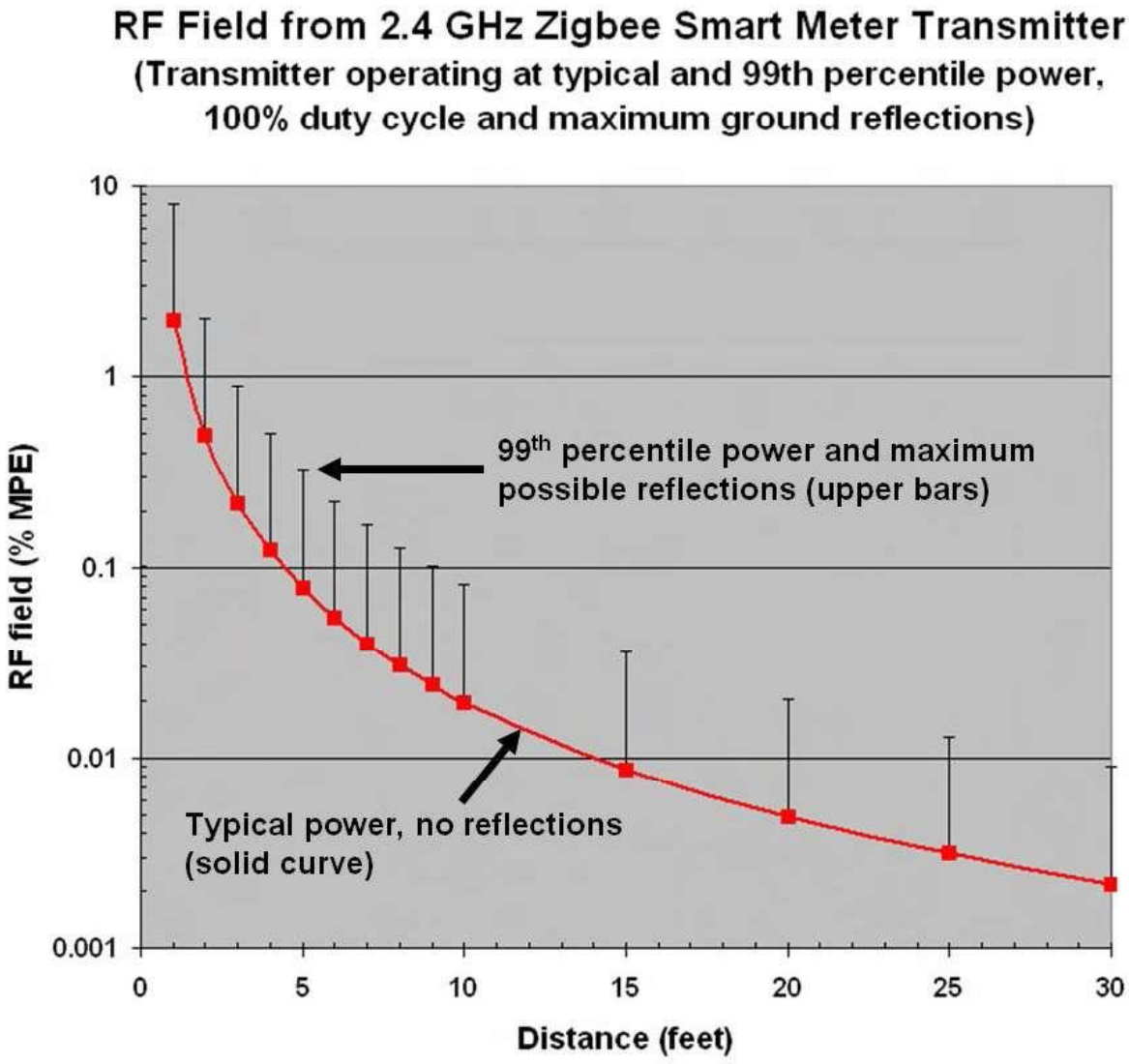

Figure 14-5
Calculated RF field produced by Smart Meter 2.4 GHz Zigbee transmitter using most likely transmitter power (solid curve) and 99th percentile transmitter power (bars above solid curve). Calculated maximum possible values are based on the 99th percentile transmitter power but assume the possibility of ground reflections ($\Gamma = 2.56$) that would enhance the RF field at the calculation point (point of maximum RF field emission near the meter). Such ground reflections are not realistic but follow guidance in FCC OET 65.

Table 14-3
Calculated upper range of possible RF fields associated with the 900 MHz RF LAN, 2.4 GHz Zigbee and cellular cell relay Smart Meter transmitters. The 99th percentile powers for the RF LAN and Zigbee transmitters, main beam exposure, 100% duty cycle and presence of ground reflections (using a ground reflection factor of 2.56) to enhance fields were assumed. However, no spatial averaging was assumed.

	RF field (% public MPE)			
Distance(ft)	900 MHz end point	2.4 GHz cell relay	836 MHz cellular (cell relay)	Cumulative value of peak fieldsa
1	27.6	8.11	91.1	126.8
2	6.91	2.03	22.8	31.7
3	3.07	0.901	10.1	14.1
4	1.73	0.507	5.69	7.93
5	1.11	0.324	3.64	5.07
6	0.767	0.225	2.53	3.52
7	0.564	0.165	1.86	2.59
8	0.432	0.127	1.42	1.98
9	0.341	0.100	1.12	1.57
10	0.276	0.081	0.911	1.27
15	0.123	0.036	0.405	0.564
20	0.069	0.020	0.228	0.317
25	0.044	0.013	0.146	0.203
30	0.031	0.009	0.101	0.141

[a] This column represents a highly conservative estimate of the single point in space RF field and is the simple sum of values of maximum calculated fields from the three transmitters in the Smart Meter. Because of the difference in wavelengths of the three RF emissions, it is not possible for the three ground reflected field components to be a maximum at the exact same point in space. Hence, the values in this column are highly conservative estimates (over estimates) of the actual fields that would exist near the meter. Further, this analysis presumes that each transmitter is transmitting continuously; this is not realistic nor would the mesh network function if the 900 MHz RF LAN transmitter were to transmit continuously.

The matter of including a factor to account for ground reflections in Equation 1 was examined by applying a method of moments computation[25] to model the field produced by a horizontally oriented 900 MHz half-wave dipole antenna located five feet above the ground. RF field strengths were computed along a vertical line path of six feet, at one foot adjacent to the dipole antenna, with a vertical distance increment of one inch. Electric field strengths were converted to plane wave equivalent power density at each of 73 points and normalized to the greatest value along the vertical line. The model was run for realistic ground constants[26] and assuming free space conditions with no ground reflections. Figure 14-6 presents the results of this analysis.

Values of spatially averaged power densities were computed for both cases, free space and ground reflections possible. The ratio of the spatially averaged resultant field with ground reflected field components

[25] EZNEC+ version 5.0.36 developed by Roy W. Lewallen www.eznec.com

[26] Ground conductivity of 0.005 siemens per meter and dielectric constant of 13.

to the spatially averaged free space field was found to be 1.032 meaning that inclusions of possible ground reflections resulted in a spatially averaged field that was 3.2% greater than under free space assumptions (no reflections accounted for). At the point of maximum field, near the height of the dipole, the ratio of power densities was 1.127 showing that the ground reflections enhanced the local field, at that specific point, by 12.7% over the free space value. While this does represent a slight enhancement, it is substantially less than if an assumption of a ground reflection factor of 2.56 were to be used. Such an assumption would imply that the RF field (power density) all along the vertical line would be 256% greater than the free space modeled value (no reflections). For this specific comparison (mounting height, ground conditions, lateral distance to the antenna, etc.), inclusion of a ground reflection factor of 2.56 for Γ in Equation 1 would result in RF fields being as much as 20 fold greater (227%) than the actual value with ground reflections. The magnitude of Γ affects the magnitude of the RF field at all points along the vertical line, resulting in spatially averaged values that are also 227% greater than that produced by the actual resultant field caused by ground reflections.

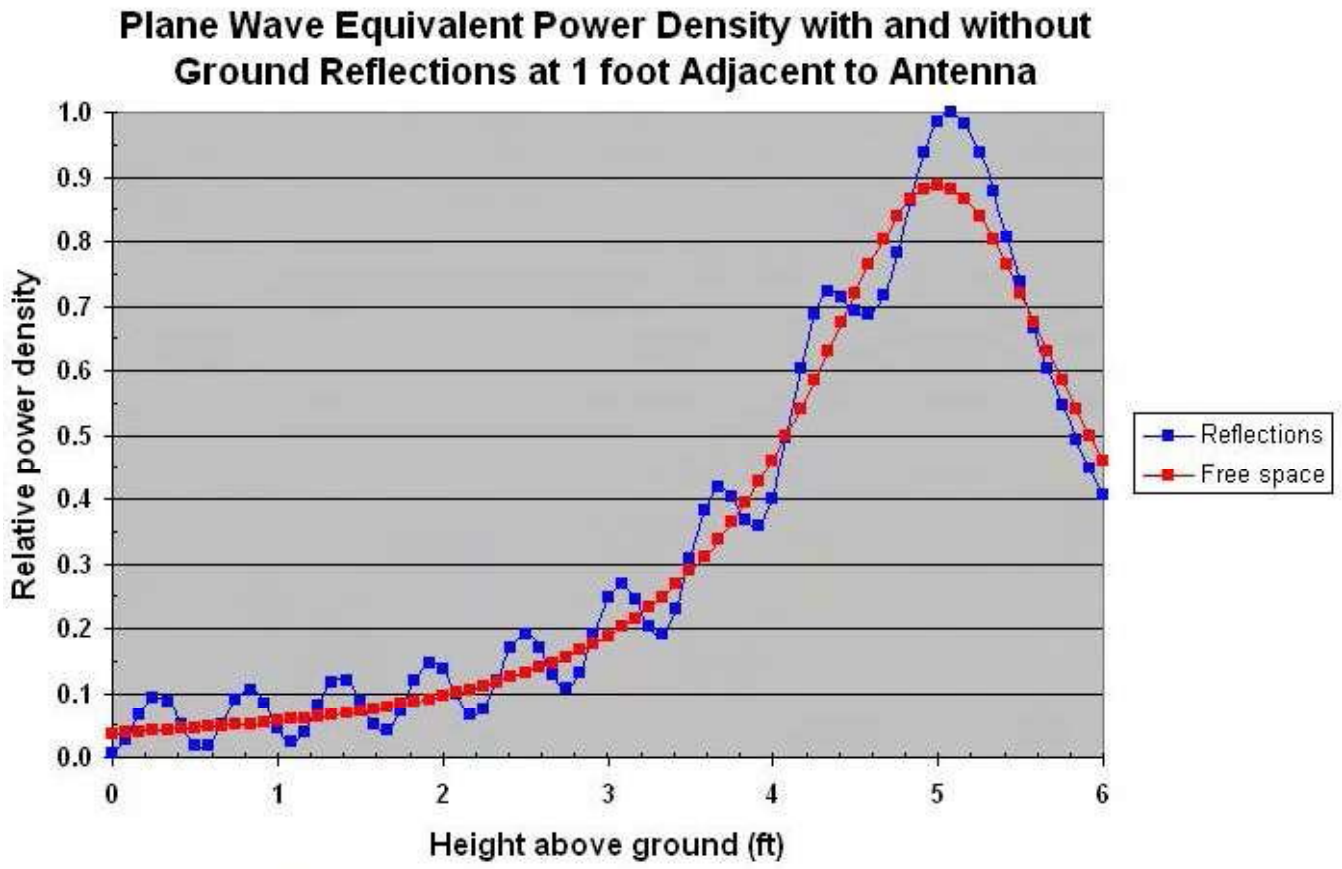

Figure 14-6
Relative calculated plane wave equivalent power density along a six-foot vertical path, one foot adjacent from a 900 MHz half-wave dipole positioned at five feet above the ground. Power density values are compared with and without ground reflections.

Similar calculated values of spatially averaged power densities, with and without the presence of ground reflections, are illustrated in Appendix G for vertical paths displaced 1 foot, 3 feet, 6 feet, 10 feet, 15 feet and 20 feet from a horizontally oriented 900 MHz dipole mounted five feet above the ground. These results are summarized in Figure 14-7 which displays the impact of ground reflections on calculated six-foot spatially averaged values of exposure to RF fields.

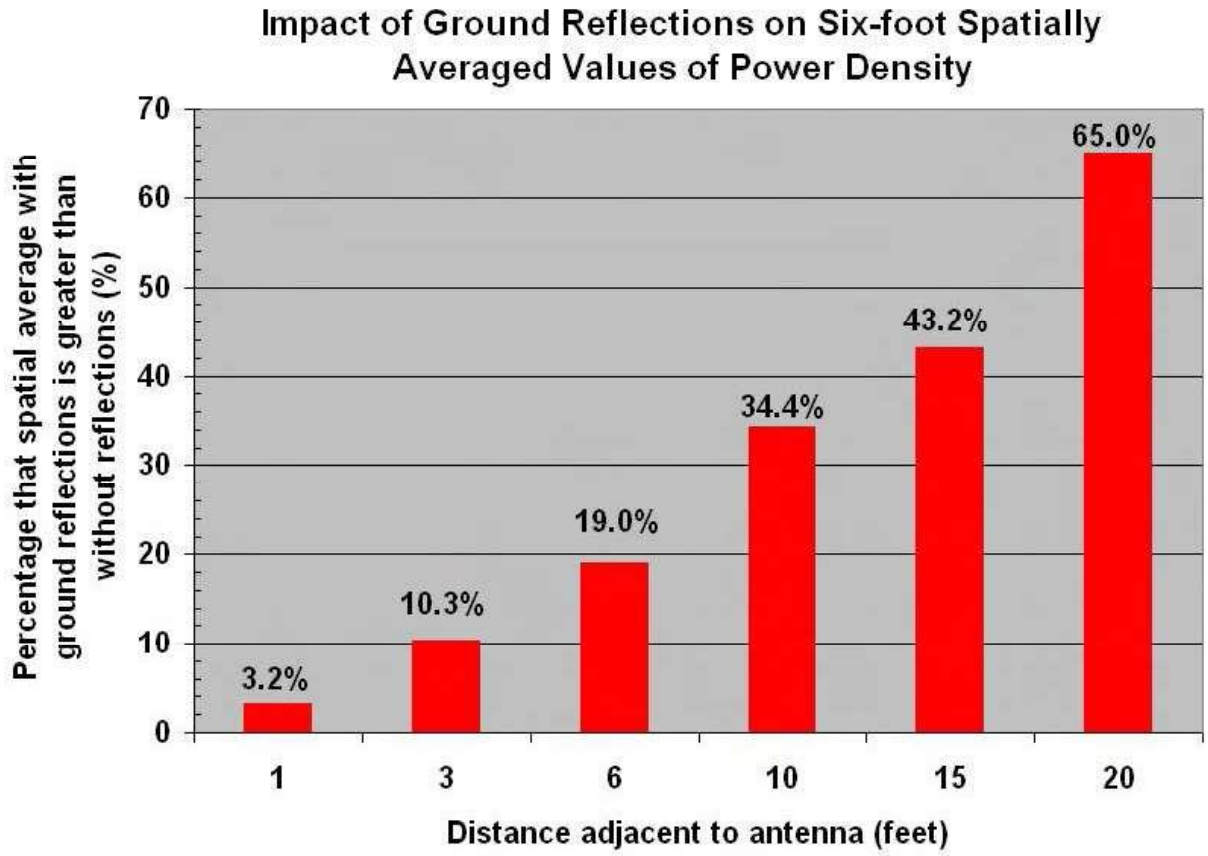

Figure 14-7
Impact of ground reflections on six-foot spatial average of power density for different distances lateral to a 900 MHz dipole antenna mounted at five feet above ground. Vertical axis is represents the percentage that the spatially averaged power density that includes any ground reflected fields is greater than the spatially averaged power density in free space (without any ground reflected fields).

The significance of the above analysis is that use of a fixed value for Γ can significantly over estimate spatially averaged values of RF fields in an attempt to account for possible ground reflection enhancement of the resultant field. Figure 14-7 illustrates that very close to the Smart Meter, ground reflections only account for approximately a 3.2% increase in the spatially averaged value of power density compared to assuming that reflections cannot occur. The contribution of constructive interference between the ground reflected field and that directly incident from the source results in greater values of spatial averages as the distance from the antenna increases. However, even at 20 feet from the source, the enhanced value comparable to being 65% greater than that without inclusion of ground reflections is still substantially less than that obtained from application of a Γ value of 2.56, i.e., a 256% increase in apparent power density! It is also significant to note that at the closest distances, where the RF field will be greatest simply due to proximity, theoretical estimates of RF power density will have the greatest error due to over estimation if the ground reflection factor is more than a few percent. This finding suggests that theoretical estimates of Smart Meter RF fields very near the meters will be most accurate if Γ is approximately unity in value.

Calculated RF fields can often be greater than the actual fields that might be measured when occupying areas near the Smart Meters evaluated in this study. This can, among other things, be a result of not identifying the precise point in space wherein the maximum EIRP exists from the meter during measurements. This situation can also manifest itself when attempting to measure the composite RF fields associated with a large number of Smart Meters that are located close to one

another. For example, when positioned very close to a group of meters, such as represented by the meter racks in the Itron meter farm or adjacent to apartment houses with banks of meters, it is not possible to find any specific point that will be subject to the main beam of transmission from all of the collective meters simply due to geometric considerations. The close measurement may capture the main beam of one of the meters in the group but cannot capture the main beams of all of the meters. This can result in a lower field strength at points very close to the group of meters than might be anticipated. However, as the distance from the group of meters increases, there is a greater likelihood that the patterns of each meter will overlap in such a way as to make each meter a more equal contributor to the overall combined RF field. Simply increasing distance results in a less spatially critical relationship between RF fields emitted by a given meter and specific location in the vicinity of the group of meters. Conceivably, with sufficient distance, albeit the combined RF fields will be very weak, the various meters will become essentially equal contributors to exposure.

This phenomenon is evident when the calculated maximum possible RF field of a single meter is compared to the measured composite field of many meters. In Figure 14-8, the measured values of the collective RF field of meters in the Itron meter farm were compared to the theoretical calculation of maximum possible field of a single 900 MHz RF LAN transmitter. In this figure, the ratio of the measured collective field to the single meter calculated field is plotted.

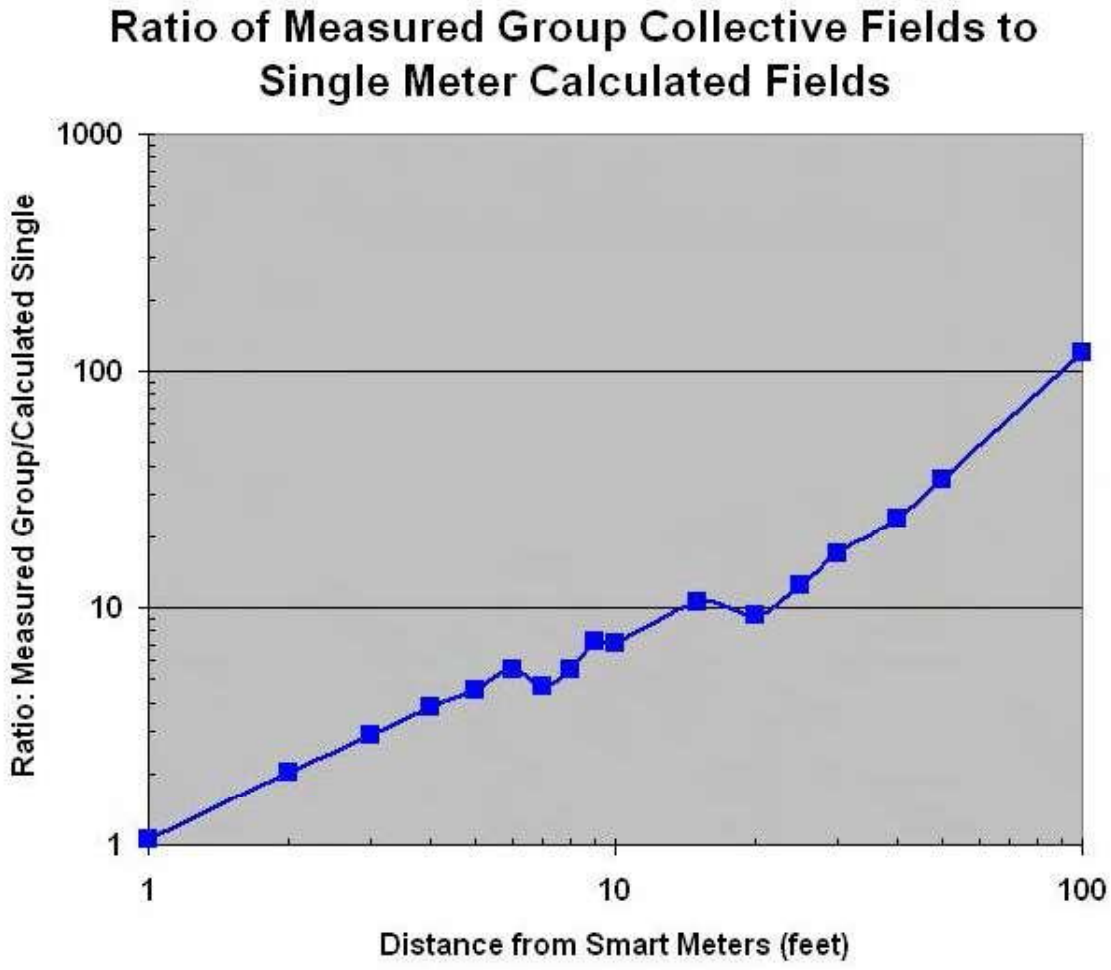

Figure 14-8
Ratio of magnitude of measured RF fields of group of ten meters in the meter farm to magnitude of calculated RF field of a single meter from 1 to 100 feet. At greater distances from the group of ten meters, the contribution of RF fields from other meters within the meter farm become proportionally more significant.

In this figure, it is evident that near the group of multiple meters (ten meters in a rack) the measured value of the group is similar to that calculated for a single meter. However, as the distance from the group of meters increases, the ratio becomes greater, reflecting the capture of more energy from multiple meters. This supports the contention that when located very near a large group of meters, exposure is not necessarily simply additive due to the number of meters present. However, at large distances from the collection of meters, it is expected that exposure becomes more a result of the collective transmissions of all of the meters. Nonetheless, while this phenomenon is evident, the actual magnitude of the aggregate field becomes significantly less than when located near the meters such that the resulting field may be irrelevant when assessing compliance with applicable RF exposure limits. The contributions of the many Smart Meters in the Itron meter farm likely impact the rate of field reduction with distance such that the decrease is not simply a function proportional to of the inverse square of distance.[27] One additional factor that relates to the observed decrease in measured field with distance from the rack of meters is that the measurement also included the contribution provided by the some 7000 meters operating within the meter farm; these signals were a part of the ambient field being measured since the detection probe of the SRM-3006 has an isotropic response.

[27] It is relevant to note that measurements made very close to a group of Smart Meters is somewhat similar to measurements in the near field of a large aperture antenna; when close to the group, the source does not appear as a point source but, rather, a spatially distributed source.

Section 15: RF Exposure Limits

FCC

Recommendations for safe human exposure levels to RF fields have been developed by a number of organizations nationally and internationally. The underlying common basis for all of the present scientifically derived limits on RF exposure is a whole-body averaged energy absorption rate of 4 watts per kilogram of body mass. This specific absorption rate (SAR) is that value found to apply across multiple species and frequencies and modulation types that result in a reliable indication of behavioral disruption which is correlated with a body core temperature increase of nominally 1°C. With application of safety factors of 10 for occupational exposure and 50 for general public exposure, limits on the strengths of external RF fields are derived. While these limits are often referred to as thermal standards, they are, in fact, based on the most sensitive indicator of a potentially adverse biological effect. Of most notable significance in the context of Smart Meters are the regulations of the Federal Communications Commission (FCC). The FCC exposure limits (MPEs) represent a hybrid of recommendations from an earlier IEEE standard[29]

Frequency Range (MHz)	Electric Field Strength (E) (V/m)	Magnetic Field Strength (H) (A/m)	Power Density (S) (mW/cm^2)	Averaging Time $\|E\|^2$, $\|H\|^2$ or S (minutes)
0.3-1.34	614	1.63	(100)*	30
1.34-30	824/f	2.19/f	($180/f^2$)*	30
30-300	27.5	0.073	0.2	30
300-1500	--	--	f/1500	30
1500-100,000	--	--	1.0	30

f = frequency in MHz *Plane-wave equivalent power density

Figure 15-1
Chart of FCC MPEs applicable to members of the general public.

For the 902-928 MHz RF LAN emissions, the FCC regulations specify a power density limit (MPE) of 0.601 mW/cm^2 or 601 $\mu W/cm^2$. This value is applicable at 902 MHz with a slightly greater value (0.618 mW/cm^2 or 618 $\mu W/cm^2$) at the upper end of the band. At the mid-band frequency of 915 MHz, the MPE is 0.610 mW/cm^2 or 610 $\mu W/cm^2$. For the 2.4 GHz Zigbee transmitter emissions, the IEEE standard calls for a limit of 1 mW/cm^2 or 1,000 $\mu W/cm^2$ which applies across the frequency range of 2 GHz to 100

GHz. The FCC MPEs are in terms of 30-minute time averaged values as averaged over the body dimensions. It is important to note, from an FCC compliance perspective, that the use of the time averaging provision of the regulations relying on a particular behavior or action of an exposed person to achieve the necessary averaging of exposure is not acceptable. However, "source based" time-averaging based on an inherent property or duty-cycle of a device is allowed.[28,29] Hence, the intermittent but routine transmissions of Smart Meters means that the time averaged value of exposure during any 30-minute period is to be used for assessing compliance with the FCC rules. To properly evaluate exposure relative to the FCC rules, the spatially averaged value of field is to be determined for comparison to the MPEs.

The above described FCC rules on RF exposure apply to FCC licensees. Electric utilities who may deploy many Smart Meters are not FCC licensees in respect to their use of Smart Meters with internal transmitters operating in the license free bands. However, besides the FCC rules applicable to its licensees, the FCC's equipment authorization program oversees authorization of equipment using the radio frequency spectrum. These devices may not be imported and/or marketed until they have shown compliance with the technical standards which have been specified by the Commission. For many of the devices subject to the equipment authorization program, including those covered by Part 15 of the FCC's rules, the FCC has included a requirement that the device manufacturer determine and represent that it meets the RF rules on exposure. This includes Smart Meters. In fact, the low power transmitters used in the Itron Smart Meters must be tested for compliance with the FCC RF rules before the Commission can issue a certification allowing the device to be marketed and used in commerce. An examination of the FCC's equipment authorization database of certification reports reveals the application of the FCC MPEs for assessing compliance with the FCC RF exposure rules[30]. Smart Meters have been designated as mobile or fixed mount devices for which proximity to humans, once the device is installed, is specified as being 20 cm or greater. The FCC applies a 20 cm distance criterion to the intended use of a transmitting device relative to the body surface for determining whether the exposure must be evaluated through specific absorption rate (SAR) measurements. Because of the way that Smart Meters are intended to be used, SAR measurements are not called for, simply an assessment of RF fields in comparison to the MPEs.[31]

IEEE

The Institute of Electrical and Electronics Engineers (IEEE) in their IEEE Standard C95.1-2005 recommend maximum permissible exposure (MPE) values across the 3 kHz to 300 GHz spectrum that are frequency dependent. This frequency dependency feature is common to virtually all of the present standards or guidelines on RF exposure, taking into account the variation in RF energy absorption rates within the human body due to dimensional aspects of the body relative to wavelength. Figure 15-2 illustrates this frequency dependence for the IEEE standard for members of the general public. In the IEEE standard, these values are recommended as action levels. If the ambient RF field exceeds the action level, an RF safety program should be implemented to insure that exposures do not exceed the upper tier of the standard, or the MPE. When no RF safety program exists, the action levels may be used as MPEs for the general public. The MPEs are those values of RF field strength, or power density, that have been averaged over any 30-minute period (time averaging) and averaged over the dimensions of the body (spatial averaging).

[28] IEEE Standard C95.1-1991.

[29] Evaluating Compliance with FCC Guidelines for Human Exposure to Radiofrequency Electromagnetic Fields (1997). Office of Engineering and Technology Bulletin 65, Edition 97-01, Federal Communications Commission, August, p. 76. See also: 47 CFR 2.1093 (d)(5).

[30] See for example: http://www.fcc.gov/oet/ea/fccid/ with an FCC device ID of SK9AMI-4.

[31] When SAR is required, for devices intended to be used within 20 cm (about 8 inches) of the body, the localized SAR limit is 1.6 walls per kilogram of tissue, as averaged over any one gram of tissue.

Frequency range (MHz)	RMS electric field strength (E)[a] (V/m)	RMS magnetic field strength (H)[a] (A/m)	RMS power density (S) E-field, H-field (W/m^2)	Averaging time[b] $\|E\|^2$, $\|H\|^2$ or S (min)	
0.1–1.34	614	$16.3/f_M$	$(1000, 100\,000/f_M^2)$[c]	6	6
1.34–3	$823.8/f_M$	$16.3/f_M$	$(1800/f_M^2, 100\,000/f_M^2)$	$f_M^2/0.3$	6
3–30	$823.8/f_M$	$16.3/f_M$	$(1800/f_M^2, 100\,000/f_M^2)$	30	6
30–100	27.5	$158.3/f_M^{1.668}$	$(2, 9\,400\,000/f_M^{3.336})$	30	$0.0636\,f_M^{1.337}$
100–400	27.5	0.0729	2	30	30
400–2000	–	–	$f_M/200$	30	
2000–5000	–	–	10	30	
5000–30 000	–	–	10	$150/f_G$	
30 000–100 000	–	–	10	$25.24/f_G^{0.476}$	
100 000–300 000	–	–	$(90f_G-7000)/200$	$5048/[(9f_G-700)f_G^{0.476}]$	

NOTE—f_M is the frequency in MHz, f_G is the frequency in GHz.

[a]For exposures that are uniform over the dimensions of the body, such as certain far-field plane-wave exposures, the exposure field strengths and power densities are compared with the MPEs in the Table. For non-uniform exposures, the mean values of the exposure fields, as obtained by spatially averaging the squares of the field strengths or averaging the power densities over an area equivalent to the vertical cross section of the human body (projected area) or a smaller area depending on the frequency (see NOTES to Table 8 and Table 9 below), are compared with the MPEs in the Table.

[b]The left column is the averaging time for $|E|^2$, the right column is the averaging time for $|H|^2$. For frequencies greater than 400 MHz, the averaging time is for power density S

[c]These plane-wave equivalent power density values are commonly used as a convenient comparison with MPEs at higher frequencies and are displayed on some instruments in use.

Figure 15-2
Summary of the IEEE C95.1-2005 action levels or MPEs for the lower tier (applicable to members of the general public if uninformed about RF exposure and not able to reduce their exposure if necessary).

For the 902-928 MHz RF LAN emissions, the IEEE standard specifies an action level (MPE) of 0.451 mW/cm^2 or 451 $\mu W/cm^2$. This value is applicable at 902 MHz with a slightly greater value (0.464 mW/cm^2 or 464 $\mu W/cm^2$) at the upper end of the band. At the mid-band frequency of 915 MHz, the MPE is 0.458 mW/cm^2 or 458 $\mu W/cm^2$. For the 2.4 GHz Zigbee transmitter emissions, the IEEE standard calls for a limit of 1 mW/cm^2 or 1,000 $\mu W/cm^2$ which applies across the frequency range of 2 GHz to 100 GHz.

ICNIRP

Internationally, the most widely recognized recommendations on RF exposure are guidelines of the International Commission on Non-Ionizing Radiation Protection (ICNIRP)[32]. The guidelines for public exposure, reproduced in Figure 15-3 (Table 9-6 from the ICNIRP guidelines), are similar to the MPEs of the FCC. A difference, however, is in the averaging time (see footnote 3). The ICNIRP specifies an averaging time of six-minutes for assessing compliance with the "reference levels" for general public exposure to RF fields. This contrasts with the half-hour period stated in both the FCC rules and IEEE limits.

[32] Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Physics, Vol. 74, No. 4, April 1998, pp. 494-522.

Table 7 Reference levels for general public exposure to time-varying electric and magnetic fields (unperturbed rms values)

Frequency range	E-field strength ($V\ m^{-1}$)	H-field strength ($A\ m^{-1}$)	B-field (μT)	Equivalent plane wave power density S_{eq} ($W\ m^{-2}$)
up to 1 Hz	—	3.2×10^4	4×10^4	—
1–8 Hz	10,000	$3.2 \times 10^4 / f^2$	$4 \times 10^4 / f^2$	—
8–25 Hz	10,000	$4{,}000/f$	$5{,}000/f$	—
0.025–0.8 kHz	$250/f$	$4/f$	$5/f$	—
0.8–3 kHz	$250/f$	5	6.25	—
3–150 kHz	87	5	6.25	—
0.15–1 MHz	87	$0.73/f$	$0.92/f$	—
1–10 MHz	$87/f^{1/2}$	$0.73/f$	$0.92/f$	—
10–400 MHz	28	0.073	0.092	2
400–2000 MHz	$1.375f^{1/2}$	$0.0037f^{1/2}$	$0.0046f^{1/2}$	$f/200$
2–300 GHz	61	0.16	0.20	10

Notes:

1. f as indicated in the frequency range column.
2. Provided that basic restrictions are met and adverse indirect effects can be excluded, field strength values can be exceeded.
3. For frequencies between 100 kHz and 10 GHz, S_{eq}, E^2, H^2, and B^2 are to averaged over any 6-minute period.
4. For peak values at frequencies up to 100 kHz see Table 4, note 3.
5. For peak values at frequencies exceeding100 kHz see Figures 1 and 2. Between 100 kHz and 10 MHz, peak values for the field strengths are obtained by interpolation from the 1.5-fold peak at 100 kHz to the 32-fold peak at 10 MHz. For frequencies exceeding 10 MHz it is suggested that the peak equivalent plane wave power density, as averaged over the pulse width, does not exceed 1000 times the S_{eq} restrictions, or that the field strength does not exceed 32 times the field strength exposure levels given in the table.
6. For frequencies exceeding 10 GHz, S_{eq}, E^2, H^2, and B^2 are to be averaged over any $68/f^{1.05}$-minute period (f in GHz).
7. No E-field value is provided for frequencies <1 Hz, which are effectively static electric fields. For most people the annoying perception of surface electric charges will not occur at field strengths less than 25 kVm^{-1}. Spark discharges causing stress or annoyance should be avoided.

Figure 15-3
Summary of ICNIRP guidelines on RF exposure limits for the general public.

With regard to the Smart Meter fields associated with the transmitters evaluated in this study, i.e., in the 900 MHz and 2.4 GHz bands), the ICNIRP guidelines are identical to those of the FCC in terms of incident power densities except for averaging time.

Section 16: Discussion of Results and Insights

The issue of potential Smart Meter RF exposure of individuals can be addressed through an exposure assessment based on either direct measurements or theoretical calculations. This investigation sought to do both by collecting information on the physical transmitting characteristics of Itron Smart Meters at Itron's facility in South Carolina and at residential locations in southern California supplemented with some measurements in Colville, Washington. From detailed measurements of the transmitting pattern of the different RF sources within the meters, RF fields can be modeled through knowledge of the transmitter powers. A comprehensive exposure assessment for Smart Meters includes an evaluation of the maximum magnitude of the instantaneous peak field strength or power density in the area of interest and, then, adjustment of this value to account for the duty cycle of RF emissions from the meter and spatial averaging over typical body dimensions. Because the Smart Meters investigated in this study must meet certain technical specifications required by the FCC, application of the FCC MPE rules are most appropriate for such exposure assessments and, in fact, have become a requirement of the FCC as part of their equipment authorization program.

The low power nature of Smart Meter transmitters, typically less than one watt, means that any RF fields produced by them will be relatively weak. For example, even when the peak value of transmitter output power is assumed to apply continuously, which it does not and cannot when used in the mesh networks of Smart Meter deployed regions, the resulting RF fields are well below the applicable MPE limits. But, when typical transmitter duty cycles are applied, the resulting RF fields are reduced, commonly, by a hundred times or more to even lower values. Finally, if the potential exposure is interpreted as a spatially averaged value over the body, the final result is yet further reduced. Ultimately, indoor exposures will be even further reduced due to the RF field attenuation characteristics of common building materials.

Smart Meters are devices that are significantly different from portable radio transmitters, such as cellular telephones and handi-talkies that are held against the head. In the latter case, RF exposure can be significantly greater simply due to the influence of device proximity relative to the body. Smart Meters are mounted in fixed locations and are not designed to be held against the body for proper use. Rather, typical exposure to Smart Meter fields will generally always be at some considerable distance as opposed to the use of devices like cell phones, cordless telephones, microwave ovens, wireless routers, etc. Nonetheless, for an exposure assessment that might be deemed a "worst case" scenario, it could be assumed that under relatively rare conditions, Smart Meters might be viewed from a close distance rather than from afar. For a person who may have a desire to approach the meter for a close-up view, while it would likely be a rare event, RF exposure will be near a maximum value. Such an exposure will be characterized by considerable spatial variability with the greatest RF fields near the surface of the meter.

Table 14-3 presents the results of an analysis that includes an upper range potential exposure scenario. The conservative estimates of duty cycle from the SCE data were used to convert peak values of RF fields to appropriate time-averaged values. A time-averaged duty cycle of 5% (maximum for end point meter RF LAN radios from the SCE data) has been applied to the calculated upper range values of fields for the 900 MHz RF LAN component of end point meters in Table 14-2. Consideration of ground reflections was included by application of a ground reflection factor of 1.344 at all distances, this being a conservative estimate of the actual value for distances up to ten feet from the meter (a ground reflection factor applicable at the closest analysis distance of one foot from the meter is actually smaller, being 1.032). An assumed upper range duty cycle of 1% has been applied to the 2.4 GHz Zigbee radio based on Itron information[33]. In the case of cell relay meters, a duty cycle of 1% (based on the 99.9th percentile value of duty cycles) has been applied to the

[33] Analysis of Radio Frequency Exposure Associated with Itron OpenWay Communications Equipment. Itron publication, undated.

900 MHz RF LAN radio. An assumed duty cycle of 0.1% (based on uplink data traffic and the cellular modem data transmission rate) was applied to the cellular transmitter emissions of the cell relay meter. Table 14-3 provides values of RF fields expressed as a percentage of the MPE for two conditions: an assumption that each transmitter could operate at its maximum possible peak power (100% duty cycle) and the maximum expected operational duty cycle as given above. These values represent the estimated maximum likely time-averaged total RF field that could exist near the Itron end point and cell relay Smart Meters studied in this report. The data tabulated in Table 14-3 for the maximum operational duty cycles are illustrated graphically in Figure 16-1. These results should be viewed as highly conservative estimates with actual RF fields being, in some cases, substantially less based on actual duty cycles.

Table 16-1

Estimated upper range of RF fields as a percentage of the FCC MPE for the public associated with Itron end point meters and cell relay meters including all RF components (900 MHz RF LAN, 2.4 GHz Zigbee and 850 MHz cellular transmitters). The 99th percentile powers for the RF LAN and Zigbee transmitters, main beam exposure and possibility of ground reflections to enhance fields was assumed.

	End point Smart Meter						**Cell relay Smart Meter**							
	RF LAN		HAN		Total		RF LAN		HAN		Cellular		Total	
DC (%)	100	5	100	1	100	Max	100	1	100	1	100	0.1	100	Max
Distance (ft)														
1	14.5	0.724	3.51	0.0351	18.0	0.759	9.20	0.092	4.26	0.0426	47.8	0.0478	61.3	0.182
2	3.62	0.181	0.876	0.00876	4.50	0.19	2.30	0.023	1.06	0.0106	12.0	0.012	15.4	0.0456
3	1.61	0.0805	0.389	0.00389	2.00	0.0844	1.02	0.0102	0.473	0.00473	5.31	0.00531	6.80	0.0202
4	0.906	0.0453	0.219	0.00219	1.12	0.0475	0.575	0.00575	0.266	0.00266	2.99	0.00299	3.83	0.0114
5	0.580	0.029	0.140	0.0014	0.72	0.0304	0.368	0.00368	0.170	0.0017	1.91	0.00191	2.45	0.00729
6	0.402	0.0201	0.0974	0.000974	0.499	0.0211	0.256	0.00256	0.118	0.00118	1.33	0.00133	1.70	0.00507
7	0.296	0.0148	0.0715	0.000715	0.368	0.0155	0.188	0.00188	0.0869	0.000869	0.976	0.000976	1.25	0.00372
8	0.226	0.0113	0.0548	0.000548	0.281	0.0118	0.144	0.00144	0.0665	0.000665	0.747	0.000747	0.958	0.00285
9	0.179	0.00894	0.0433	0.000433	0.222	0.00937	0.114	0.00114	0.0526	0.000526	0.590	0.00059	0.757	0.00226
10	0.145	0.00724	0.0351	0.000351	0.18	0.00759	0.092	0.00092	0.0426	0.000426	0.478	0.000478	0.613	0.00182
15	0.0644	0.00322	0.0156	0.000156	0.08	0.00338	0.0409	0.000409	0.0189	0.000189	0.212	0.000212	0.272	0.00081
20	0.0362	0.00181	0.00876	8.76E-05	0.045	0.0019	0.023	0.00023	0.0106	0.000106	0.120	0.00012	0.154	0.000456
25	0.0232	0.00116	0.00561	5.61E-05	0.0288	0.00122	0.0147	0.000147	0.00681	6.81E-05	0.0765	0.0000765	0.098	0.000292
30	0.0161	0.000805	0.00389	3.89E-05	0.02	0.000844	0.0102	0.000102	0.00473	4.73E-05	0.0531	0.0000531	0.068	0.000202

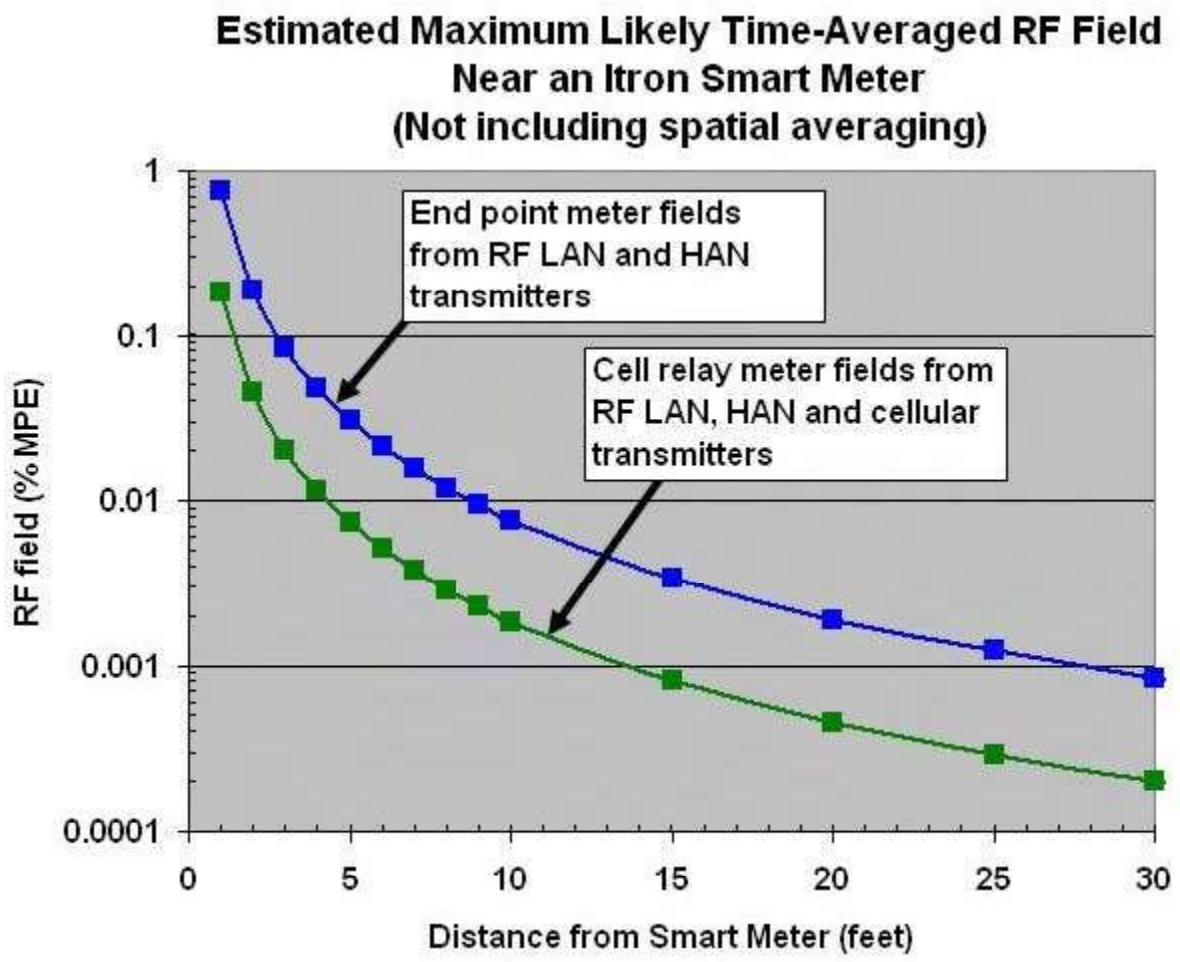

Figure 16-1
Estimated maximum likely time-averaged RF fields near Itron end point and cell relay Smart Meters included in this study. The plotted values are based on the 99th percentile values of transmitter powers, duty cycles given in Table 14-3 based on the conservative estimates from SCE data, main beam exposure and inclusion of realistic ground reflected fields (Γ= 1.344) that might add constructively to the resultant field. An assumption is made that the maximum RF field from all transmitters occurs at the same point in space. The graph pertains to a single end point meter and a single cell relay meter.

The RF field values shown in Figure 16-1 do not account for spatial averaging. Spatial averaging will be most significant when an individual is close to the meter such that there is considerable variation in the field over the body dimensions. As illustrated in Figures 11-2 and 11-3, when very close to a Smart Meter, the spatially averaged field will be substantially less than the spatial maximum value. For an end point meter, with exposure at approximately one foot from the meter, spatial averaging would be expected to reduce the maximum value shown in Figure 16-1 of about 0.8% of MPE to approximately 0.2% of MPE. For a cell relay, spatial averaging would result in reducing a spatial peak value of about an exposure of 0.2% of MPE to about 0.06% of MPE.

When the exposure assessment is for locations within a residence, generally the point of maximum RF field produced by Smart Meters is very close to where the meter is mounted on the outside of the house. For those interior areas located behind the meter, the antenna patterns obtained during this study show that relatively significant reductions in RF energy exist toward the back side of the meter as compared to the frontal region, outside the house. Inspection of the various antenna patterns discussed earlier reveals, in most cases,

very significant reduction of the RF field directly behind the meters, some times less than one percent of the forward directed value (more than 20 dB down from the forward value). Although significant notches in the pattern can exist behind the meters, these pattern notches are in some cases not very broad. In a more conservative view, however, the pattern data support a practical field reduction of nominally 10 dB, i.e., a factor of ten reduction in the field relative to the forward directed value. This factor means that the RF fields behind a Smart Meter mounted on the exterior wall of a house that are directed toward the house, will be about 1/10th of the RF field intensity at the same distance but in front of the meter, not taking into account any attenuation afforded by the wall construction itself. Peak values of Smart Meter RF fields found inside two residences equipped with continuously transmitting Smart Meters (to facilitate the measurements) are summarized in Table 16-1.

Table 16-2
Summary of interior residential RF field measurements on two residences equipped with Smart Meters operating in continuous transmit mode.

	Percent FCC MPE for the general public			
	Residence A		Residence B	
	900 MHz	2.4 GHz	900 MHz	2.4 GHz
Home maximum	0.0100	0.0288	0.00872	0.0150
Home average	0.00237	0.00825	0.00144	0.00779
Home minimum	0.00014	0.00588	0.00052	0.00596

These data pertain only to the interior room measurements but are exclusive of specific microwave oven measurements taken in residence A. The highest values in the 2.4 GHz band were both associated with the use of a wireless router within the room.

The effect that the structure of homes can have on Smart Meter fields inside the home is also significant. Based on the measurements reported here, RF fields directly behind a Smart Meter mounted on the exterior wall of a stucco home, but inside the house, would be expected to be attenuated by at least 6.1 dB in the 900 MHz RF LAN band and 2.5 dB in the 2.4 GHz Zigbee frequency band (these values represent the most conservative values of insertion loss found in the measurements conducted in California and Washington). For locations immediately near the surface of the wall, greater wall attenuation effects would be expected, similar to the values reported above in Table 10-1. Hence, it is expected that, for stucco type homes that have typical stucco netting on the outside, RF fields will be attenuated by a factor of 4 for the 900 MHz RF LAN transmitter and a factor of about 1.7 for the 2.4 GHz Zigbee transmitter. In the case of an end point meter, this would translate to a maximum indoor RF field of about 0.55% of the MPE, assuming that both the RF LAN and Zigbee transmitters are continuously active and the distance from the meter to the inside surface of the wall is 12 inches. With only the RF LAN transmitter active, as was the case during the residential measurements in California, a maximum value of 0.36% of the MPE would be expected. These values do not assume application of spatial averaging or time averaging. During the measurements in actual residences, a maximum interior field from the RF LAN transmitter of 0.01% was measured (residence A). Interestingly, the measured RF field in the home office of this residence was 0.03% of the MPE due to the presence of a wireless router located in the office.

Acquiring measurement data on the very intermittent emissions of Smart Meters that can accurately yield the duty cycle of the meters is challenging. Even measuring the number of times that a Smart Meter emits a signal over a 24-hour day is problematic. The measurements documented here show that in no case did the RF fields, even with continuous transmission (100% duty cycle), approach the FCC MPE for the general public - calculated exposure at one foot for 100% duty cycle when spatially averaged (61.3% MPE × 0.233 = 14.3% MPE). Generally, maximum measured values were small fractions of the MPE. Limited measurement data taken in the Itron meter farm, with thousands of Smart Meters operating, suggested very low duty cycles of

about 0.2%. This measurement involved contributions of many meter emissions so that the meter farm likely represents a much more dense Smart Meter environment than most U.S. neighborhoods wherein Smart Meters have been deployed.

An alternative view of the RF field data presented in Table 14-3 is to note that at a distance of one foot directly in front of the meter, even if all transmitter components in the end point meters or cell relays were to operate in a continuous mode which is unrealistic and which would inhibit the mesh network from performing, and no adjustment for spatial averaging of the fields were to take place, the resulting RF fields are still less than the FCC MPE for public exposure.

The Wi-Spy measurements taken at a single residence and in the Itron meter farm also suggested that the duty cycle of Smart Meters, during the observation time, was very small, in the range of less than one percent.

Likely, the most accurate assessment of Smart Meter duty cycles can be through use of the meter data management system associated with operation of an electric utility company network of meters. Determining the amount of data transferred across the mesh network by a given meter can be one approach to "remotely" determining the activity of the Smart Meter. Through such analyses of meter activity, duty cycles applicable over specific time periods can be ascertained. For example, the variation of duty cycle throughout a day could be examined given collection of meter data over sufficiently long times. In this way, maximum 30-minute duty cycles can be determined and applied to calculated peak field values to obtain time-averaged values for direct comparison to the FCC MPEs.

Section 17: Conclusions

The radio transmitters inside the Itron Smart Meters studied in this project typically produce RF fields substantially below the FCC limit at 900 MHz in their vicinity. Because of the low power, low antenna gains and highly intermittent emissions, time-averaged RF fields to which someone standing immediately next to the meter could be exposed are substantially below any of the current scientifically based human exposure limits.

RF exposure produced by Smart Meters is dependent on transmitter powers, frequencies (exposure limits are frequency dependent), meter installation details such as mounting height, the construction details of the structure on which the meter is installed (building materials attenuate the RF signals of the Smart Meters) and the activity of the wireless mesh network itself. The transmitters emit intermittent signals having instantaneous peak field strengths, or power densities, that are already generally low by comparison with exposure limits but any resulting spatial averaging over the body and temporal averaging, for determining compliance with FCC MPEs, further reduces the exposure magnitude.

The Itron end point Smart Meters included in this study contained two transmitters, one operating in the 900 MHz license free band (for the RF LAN function) and the other in the 2.4 GHz license free band (for the home area network, HAN, function). These transmitters operate with nominal powers of 24.5 dBm (282 mW) and 18.5 dBm (70.8 mW) respectively. The more rare cell relay meters contain the same two transmitters described above and an additional cellular transmitter that operates at a nominal output power of between 25.1 dBm and 31.8 dBm depending on whether it uses GSM or CDMA technology and the particular frequency band used.

Antenna pattern measurements show that the Smart Meters generally radiate RF signals preferentially away from the meter but with relatively broad patterns, producing a very rough approximation to an omnidirectional emitter. However, behind the meter, the RF field is nominally a factor of 10 less than at the same distance in front of the meter. Even with the broad pattern, RF exposure of a person standing immediately adjacent to a Smart Meter will be predominately of the portion of the body nearest the meter. Spatial averaging of the RF fields shows that the spatially averaged value of exposure, in terms of a percentage of the MPE, is on the order of one-fourth of the maximum value at any location on the body.

Measurements of Smart Meter fields present a challenge due to their highly intermittent nature and frequency hopping characteristic. The most effective approach to evaluating the magnitude of RF fields produced by Smart Meters with field measuring equipment is to cause the transmitters to operate in continuous mode, some times referred to as FCC mode, since this allows for much easier measurements. Such an approach results in determination of the instantaneous peak value of RF field to which must be applied appropriate duty cycle values to obtain time-averaged values for exposure. A valid and readily expedient approach to estimating potential exposure of Smart Meters is via calculation based on knowledge of the transmitting characteristics of the meter transmitters. The most common method of calculation is to invoke the maximum gain of the antennas in arriving at a conservative (typically an over estimate) value for the emitted field in the vicinity of the meter.

Both measurements and calculations of RF fields associated with the Itron meters included in this study were pursued in an effort to develop a solid basis for determining the peak fields that can exist near the meters. Both of these approaches yielded similar findings, namely that the RF fields produced by the Itron Smart Meters are compliant with applicable human exposure. Typical RF exposure near (at one foot) the Smart Meters evaluated are most likely less than 20% of the MPE in terms of instantaneous peak RF fields of an end point meter and less than 1% of the MPE in terms of time-averaged values (see Table 7-6). RF fields that occur as close as 1 foot from the cell relays (which occur in one in 500 to 750 residences) are less than 61% of the MPE in terms of instantaneous peak RF fields and less than 0.2% of the MPE in terms

of time-averaged values. At ten feet from an end point or cell relay meter, RF fields are likely less than 0.2% and 0.6% of the MPE in terms of instantaneous peak RF fields, respectively, and less than 0.01% and 0.002% of the MPE in terms of typical time-averaged RF fields, respectively.

To put these estimates into some practical perspective, spatially averaged values of fields will be substantially less (typically between 18%-24% of the peak values) and, for indoor locations such inside a residence, any resulting exposure will also be significantly reduced because of attenuation to the Smart Meter emissions caused by the construction materials of the wall separating the meter from the inside of the house. House attenuation effects could easily account for between 75%-89% reduction of the 900 MHz RF LAN emissions and between 44%-64% reduction of the 2.4 GHz Zigbee radio emissions indicated above.

Accurately determining time-averaged RF fields represents the most uncertainty in exposure assessments for the Smart Meters evaluated in this study. This is due to the dynamic signal transmissions of Smart Meters of varying length and the associated frequency hopping nature of the 900 MHz RF LAN transmitter making it difficult to properly capture every emission with "off the shelf" instrumentation. In reality, the transmitter activity of Smart Meters is best described in a statistical fashion since it is not possible to exactly define if and when a given Smart Meter will transmit and how often, with absolute accuracy. Given the nature of mesh networks, hundreds of meters are interacting with one another in a way to form connections between the various meters and, ultimately, a cell relay meter. The activity of this interaction leads to variability in the activity of each of the RF LAN transmitters and, hence, measurements at any particular time are not expected to necessarily be indicative of the same transmitter's activity during another time of day or on another day. Such measurements, even if properly made, can provide insight to a Smart Meter's transmitting characteristics but, unless conducted over an extended period, are unlikely to yield meaningful measures of maximum average duty cycles. The most meaningful determination of Smart Meter duty cycles over the long term so that maximum 30-minute values can be ascertained is most likely based on exploiting software approaches. Through examining meter data throughputs, over many meters within a Smart Meter deployed region, and over an extended period of time, good statistical representation of meter RF activity should be achievable. Future work to develop comprehensive statistical descriptions of Smart Meter transmissions remains to be done but, in any event, the real duty cycles of Smart Meters would appear to be very small percentages. Ambient RF fields associated with operation of cellular base stations, radio and TV broadcasting and the emissions produced by a variety of everyday activities that involve the use of electronic devices can be comparable to or even exceed common exposures resulting from the operation of Smart Meters.

Regardless of duty cycle values for end point and cell relay meters, common exposures of individuals that are likely to result from the operation of the Itron Smart Meters evaluated in this study are very low and comply with scientifically based human exposure limits by a wide margin.

Appendix A: Instrument Calibration Certificates

Page 1 of 1

L3 communications
Narda Microwave-East

Certificate of Calibration

L-3 Communications, Narda Microwave-East, hereby certifies that the referenced instrument has been calibrated by qualified personnel to Narda's approved test procedures.

Furthermore, the instrument meets, or exceeds, all published specifications and the calibration has been performed with test instrumentation that, where applicable, is traceable to the National Institute of Standards and Technology.

Narda's calibration measurements are traceable to the National Institute of Standards and Technology to the extent allowed by the bureau's calibration facilities.

Customer: RICHARD TELL ASSOCIATES INC
COLVILLE, WA 99114

Certificate #: 99298 1

Model #: 8715
Description: METER
Date Calibrated: 09/30/2009

Serial #: 01028
PO #: AMEX - TELL
R.O. #: 99298

Dan Beach
Manufacturing

Ken Peck
Quality Assurance

L-3 COMMUNICATIONS, NARDA MICROWAVE-EAST, 435 MORELAND ROAD, HAUPPAUGE, NEW YORK 11788, TEL: 631-231-1700, FAX: 631-231-1711

Figure A-1
Calibration certificate for the Narda Model 8715 digital meter for use with the Model B8742D probe.

Page 1 of 1

L3 communications
Narda Microwave-East

Certificate of Calibration

L-3 Communications, Narda Microwave-East, hereby certifies that the referenced instrument has been calibrated by qualified personnel to Narda's approved test procedures.

Furthermore, the instrument meets, or exceeds, all published specifications and the calibration has been performed with test instrumentation that, where applicable, is traceable to the National Institute of Standards and Technology.

Narda's calibration measurements are traceable to the National Institute of Standards and Technology to the extent allowed by the bureau's calibration facilities.

Customer: RICHARD TELL ASSOCIATES INC
COLVILLE, WA 99114

Certificate #: 99298 2

Model #: B8742D
Description: PROBE
Date Calibrated: 10/07/2009

Serial #: 03002
PO #: AMEX - TELL
R.O. #: 99298

Dan Beach
Manufacturing

Ken Peck
Quality Assurance

L-3 COMMUNICATIONS, NARDA MICROWAVE-EAST, 435 MORELAND ROAD, HAUPPAUGE, NEW YORK 11788, TEL: 631-231-1700, FAX: 631-231-1711

Figure A-2
Calibration certificate for the Narda Model B8742D broadband probe.

Calibration Certificate

Narda Safety Test Solutions hereby certifies that the object referred to in this certificate has been calibrated by qualified personnel using Narda's approved procedures. The calibration was carried out in accordance with a certified quality management system which conforms to ISO 9001

OBJECT	Selective Radiation Meter, Basic Unit, SRM-3006
MANUFACTURER	Narda Safety Test Solutions GmbH
PART NUMBER (P/N)	3006/01
SERIAL NUMBER (S/N)	A-0077
CUSTOMER	
CALIBRATION DATE	2009-11-06
RESULT ASSESSMENT	within specifications
AMBIENT CONDITIONS	Temperature: (23 ± 3)°C Relative humidity: (25 to 75)%
CALIBRATION PROCEDURE	3006-8701-00A

ISSUE DATE: 2009-12-10

MANAGEMENT SYSTEM

CALIBRATED BY:
Paul Geyer

AUTHORIZED SIGNATORY:
Norbert Moll

Certified by DQS against DIN EN ISO 9001:2000 (Reg.-No. 099379)

CERTIFICATE 300601-A0077-20091106-62

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Figure A-3
Calibration certificate sheet 1 for the Narda Model SRM-3006 SN A-0077.

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OBJECT

The spectrum analyzer is based on digital signal processing. Small frequency spans were measured at fixed local oscillator (1st LO) settings using discrete Fourier transformation (DFT). The LO was also swept for larger frequency spans.
A memory chip contains correction values for various frequencies and object settings. The stored values were taken into account automatically during the measurement.

METHOD OF MEASUREMENT

Calibration using the reference standard. The output power level of the synthesized CW generator was adjusted and calibrated using power sensors as reference standards.
The frequency of the generator was calibrated using a frequency counter.

The reflection of the object was measured directly using a vector network analyzer (VNA) calibrated by means of a calibration kit. The measuring equipment and the associated uncertainty were verified using a reference standard (traceability kit).

CALIBRATION PROCEDURE

The object was connected to the signal source instead of the power sensors in order to calibrate it.

Measurement of the RF frequency response was made with different settings of the measurement range. As a result, the measured values also include the effects due to the "input attenuator" and the "reference level accuracy".
The calibration factor was calculated for various frequencies and settings from a comparison between the "actual level" and the "indicated level".

All the selection filters are digital filters. No calibration of the filters is necessary.

TRACEABILITY

The calibration results are traceable to the International System of Units (SI) in accordance with ISO/IEC 17025. The measuring equipment used for calibration is traceable through the reference standards listed below.

STANDARD	MANUFAC-TURER	MODEL	SERIAL NUMBER	ID	CERTIFICATE	NEXT CAL. DATE	TRACE
HF-MILLIVOLTMETER	R&S	URV55	100143	913	0124 DKD-K-16101 2008-04	2010-04	DKD
DIODE POWER SENSOR	R&S	NRV Z4	100122	912	0171 DKD-K-16101 2008-11	2010-11	DKD
THERMAL POWER SENSOR	R&S	NRV Z51	101777	1635	0264 DKD-K-16101 2008-11	2010-11	DKD
MISMATCH VSWR 1,2 (f)	Rosenberger	--	01237	552-3	12996 DKD-K-00201 2008-05	2010-05	DKD
FREQUENCY COUNTER	Advantest	R5362	120700137	923	15137 DKD-K-00201 2009-09	2011-09	DKD

Figure A-3 (continued)
Calibration certificate sheet 2 for the Narda Model SRM-3006 SN A-0077.

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UNCERTAINTY

The reported expanded uncertainty U is based on a standard uncertainty multiplied by a coverage factor $k = 1.96$, providing a level of confidence of approximately 95 %. The uncertainty evaluation has been carried out in accordance with the "Guide to the Expression of Uncertainty in Measurement" (GUM). The reported measurement uncertainty is derived from the uncertainty of the calibration procedure and the object during calibration, and makes no allowance for drift or operation under other environmental conditions.

MEASURING CONDITIONS

The following results were obtained after adjustment of the object under calibration. These values are within the setting ranges defined by the manufacturer.

RESULTS

1	FREQUENCY RESPONSE (IF):	passed
2	FREQUENCY RESPONSE (RF):	passed
3	OUT-OF-BAND RESPONSE:	passed
4	FREQUENCY ACCURACY	passed
5	NOISE SIDEBAND (SSB):	passed
6	SPURIOUS (input related)	passed
7	SPURIOUS (residual)	passed
8	NOISE FLOOR:	passed
9	INTERMODULATION REJECTION (2^{nd} and 3^{rd} order):	passed
10	INPUT RETURN LOSS:	passed

Figure A-3 (continued)
Calibration certificate sheet 3 for the Narda Model SRM-3006 SN A-0077.

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APPENDIX

FREQUENCY RESPONSE (RF)

The generator was set to the *Fgen*. The object settings were *Fspan*, *RBW*, and *Fcent*.
The measurements were made at different settings of the measurement range *MR*. The nominal level of the generator was -7 dBm for $-32\text{dBm} \le MR \le -5\text{dBm}$ and -32 dBm for the remaining measurement ranges.

The frequency response *G* was calculated as the difference of the actual generator level L_{actual} and the indicated level $L_{indicated}$ according to the following equation:

$$G/\text{dB} = \left(L_{indicated} - L_{actual}\right)/\text{dBm}$$

Frequency in MHz	Fspan in MHz	RBW in kHz	Fcent in MHz	MR												U
				-30	-28	-25	-20	-15	-10	-5	0	5	10	15	20	
0.00901	0.002	0.01	0.01	0.04	0.03	0.02	0.01	0.01	0.04	0.02	0.02	0.01	0.01	0	0	0.2
0.012	0.006	0.5	0.012	0.04	0.03	0.02	0.01	0.01	0.04	0.02	0.02	0.01	0.01	0	-0.01	0.2
0.02	0.02	2	0.02	0.04	0.03	0.02	0.01	0.01	0.04	0.02	0.02	0.01	0.01	0	-0.01	0.2
0.04	0.02	2	0.04	0.04	0.03	0.02	0.01	0.01	0.04	0.02	0.02	0	0.01	0	-0.01	0.2
0.1	0.02	2	0.1	0.04	0.03	0.02	0.01	0.01	0.03	0.02	0.01	0	0.01	0	-0.01	0.2
0.5	0.02	2	0.5	0.04	0.03	0.02	0.01	0.01	0.04	0.02	0.02	0.01	0.01	0	-0.01	0.2
2	0.02	2	2	0.04	0.03	0.02	0.02	0.01	0.04	0.02	0.02	0.01	0.01	0	-0.01	0.2
10	0.02	2	10	0.05	0.05	0.04	0.03	0.02	0.05	0.04	0.03	0.02	0.02	0.01	0.01	0.2
20	0.02	2	20	0.05	0.05	0.04	0.03	0.02	0.06	0.04	0.04	0.03	0.03	0.02	0.02	0.2
30	0.02	2	30	0.05	0.04	0.04	0.03	0.02	0.04	0.04	0.03	0.02	0.02	0.01	-0.01	0.2
31.233	26.75	30	44.578	-0.28	-0.36	-0.27	-0.36	0.01	-0.23	-0.26	-0.36	0.01	-0.22	-0.25	-0.36	0.2
36.1	26.75	30	44.578	-0.13	-0.21	-0.16	-0.22	-0.04	-0.12	-0.16	-0.21	-0.03	-0.13	-0.16	-0.22	0.2
40	0.02	2	40	0.05	0.04	0.04	0.03	0.03	0.07	0.04	0.03	0.03	0.02	0.03	0.05	0.2
44.1	26.75	30	44.578	-0.01	-0.03	-0.03	-0.04	-0.05	-0.01	-0.04	-0.03	-0.06	-0.04	-0.03	-0.02	0.2
50	0.02	2	50	0.05	0.04	0.03	0.02	0.01	0.02	0.03	0.02	0.01	0.01	-0.01	-0.05	0.2
52.1	26.75	30	44.578	0.04	0.03	0.01	0.01	-0.1	0.05	-0.01	0.02	-0.1	-0.02	-0.02	0.06	0.2
57.9948	0.02	2	57.9868	0.04	0.03	0.03	0.02	0.01	0.07	0.03	0.02	0.01	0.01	0.02	0.05	0.2
58.344	26.75	30	44.999	0.03	0.03	-0.02	-0.01	-0.17	0.03	-0.04	0	-0.17	-0.07	-0.04	0.02	0.2
60.1	26.75	30	60.1	-0.01	-0.02	-0.02	-0.02	-0.02	0	-0.02	-0.02	-0.03	-0.02	-0.03	0	0.2
100.1	26.75	30	100.1	-0.02	-0.01	-0.01	-0.02	-0.03	-0.01	-0.02	-0.02	-0.03	-0.03	-0.03	-0.02	0.2
200.1	26.75	30	200.1	-0.02	-0.02	-0.02	-0.02	-0.03	0	-0.02	-0.03	-0.03	-0.03	-0.04	-0.01	0.2
300.1	26.75	30	300.1	-0.05	-0.05	-0.05	-0.05	-0.06	-0.05	-0.06	-0.06	-0.07	-0.07	-0.07	-0.06	0.2

Figure A-3 (continued)
Calibration certificate sheet 4 for the Narda Model SRM-3006 SN A-0077.

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Frequency in MHz	Fspan in MHz	RBW in kHz	Fcent in MHz	MR												U
				-30	-28	-25	-20	-15	-10	-5	0	5	10	15	20	
400.1	26.75	30	400.1	0.02	0.02	0.01	0.02	0.01	0.02	0	0	-0.01	0	0	0	0.2
500.1	26.75	30	500.1	0.02	0.02	0.01	0.02	0.01	0.02	0	0	-0.01	-0.01	-0.01	0	0.2
600.1	26.75	30	600.1	-0.01	-0.01	-0.02	-0.02	-0.02	-0.02	-0.04	-0.03	-0.05	-0.05	-0.04	-0.01	0.2
700.1	26.75	30	700.1	-0.02	-0.03	-0.03	-0.03	-0.03	-0.03	-0.04	-0.04	-0.05	-0.05	-0.05	-0.03	0.2
800.1	26.75	30	800.1	-0.02	-0.02	-0.02	-0.03	-0.03	-0.03	-0.04	-0.04	-0.06	-0.06	-0.05	-0.04	0.2
900.1	26.75	30	900.1	0	0	0	0	-0.01	-0.01	-0.02	-0.03	-0.04	-0.04	-0.03	-0.02	0.2
1000.1	26.75	30	1000.1	-0.01	-0.01	-0.01	-0.01	-0.02	-0.02	-0.03	-0.04	-0.04	-0.04	-0.04	-0.03	0.2
1100.1	26.75	30	1100.1	0	0	-0.01	0	-0.01	0	-0.03	-0.03	-0.03	-0.04	-0.04	0	0.2
1200.1	26.75	30	1200.1	-0.01	-0.01	-0.02	-0.02	-0.02	-0.02	-0.04	-0.05	-0.04	-0.04	-0.05	-0.03	0.2
1300.1	26.75	30	1300.1	-0.03	-0.03	-0.03	-0.04	-0.05	-0.05	-0.06	-0.07	-0.07	-0.07	-0.07	-0.06	0.2
1400.1	26.75	30	1400.1	0.02	0.01	0.01	0.01	0	0.01	-0.02	-0.02	-0.02	-0.03	-0.02	-0.02	0.2
1500.1	26.75	30	1500.1	0.07	0.07	0.06	0.06	0.06	0.05	0.03	0.02	0.03	0.02	0.01	0.03	0.2
1600.1	26.75	30	1600.1	0.05	0.03	0.03	0.04	0.03	0.03	0	0	0.01	-0.01	0	0.01	0.2
1700.1	26.75	30	1700.1	0.03	0.02	0.03	0.02	0.01	0.02	-0.01	-0.02	-0.01	-0.03	-0.02	-0.01	0.2
1800.1	26.75	30	1800.1	0.03	0.02	0.02	0.01	0.01	0.01	-0.02	-0.02	-0.01	-0.02	-0.03	-0.01	0.2
1900.1	26.75	30	1900.1	0.02	0.02	0.03	0.02	0.01	0.01	-0.02	-0.02	-0.01	-0.04	-0.04	-0.02	0.2
2000.1	26.75	30	2000.1	0.05	0.04	0.04	0.03	0.02	0.03	-0.01	-0.01	0	-0.02	-0.02	-0.01	0.2
2100.1	26.75	30	2100.1	0.04	0.04	0.04	0.04	0.03	0.03	-0.01	-0.02	-0.01	-0.03	-0.01	-0.02	0.2
2200.1	26.75	30	2200.1	0.05	0.05	0.05	0.05	0.03	0.03	-0.02	-0.01	-0.01	-0.03	-0.01	-0.02	0.2
2300.1	26.75	30	2300.1	0.05	0.05	0.05	0.05	0.03	0.02	0	-0.01	-0.02	-0.02	-0.02	0.01	0.2
2400.1	26.75	30	2400.1	0.02	0.02	0.02	0.01	0.02	-0.01	-0.03	-0.03	-0.06	-0.06	-0.03	-0.02	0.2
2500.1	26.75	30	2500.1	0.03	0.03	0.02	0.03	0.03	0	-0.03	-0.03	-0.05	-0.05	-0.04	-0.01	0.2
2600.1	26.75	30	2600.1	0.05	0.04	0.04	0.05	0.04	-0.02	-0.02	-0.01	-0.05	-0.02	-0.03	-0.01	0.2
2700.1	26.75	30	2700.1	0.06	0.06	0.05	0.05	0.04	-0.02	-0.02	-0.02	-0.04	-0.04	-0.04	-0.02	0.2
2800.1	26.75	30	2800.1	0.06	0.05	0.05	0.05	0.04	-0.01	-0.02	-0.01	-0.04	-0.05	-0.03	-0.01	0.2
2900.1	26.75	30	2900.1	0.06	0.06	0.03	0.06	0.06	-0.03	-0.02	-0.01	-0.05	-0.06	-0.03	0	0.2
2999.9	26.75	30	2999.9	0.08	0.06	0.08	0.05	0.05	-0.02	-0.02	-0.02	-0.06	-0.04	-0.04	0.01	0.2
3002.1	26.75	30	3002.1	0.01	0.01	0	0	0.02	-0.01	0.07	0.07	0.07	0.07	0.08	0.1	0.2
3100.1	26.75	30	3100.1	-0.02	-0.02	-0.02	-0.01	-0.03	-0.02	0.05	0.06	0.06	0.06	0.07	0.1	0.2
3200.1	26.75	30	3200.1	0.01	0.01	0	0.01	0.01	0.03	0.08	0.1	0.11	0.1	0.11	0.12	0.2
3300.1	26.75	30	3300.1	0.03	0.03	0.04	0.03	0.04	0.08	0.11	0.11	0.12	0.12	0.13	0.13	0.2
3400.1	26.75	30	3400.1	0.01	0.01	0.01	0.01	-0.02	0.1	0.09	0.1	0.11	0.1	0.11	0.12	0.2

CERTIFICATE 300601-A0077-20091106-62

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Figure A-3 (continued)
Calibration certificate sheet 5 for the Narda Model SRM-3006 SN A-0077.

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Frequency in MHz	Fspan in MHz	RBW in kHz	Fcent in MHz	MR												U
				-30	-28	-25	-20	-15	-10	-5	0	5	10	15	20	
3500.1	26.75	30	3500.1	0.02	0.01	0.01	0.01	0	0.12	0.12	0.11	0.13	0.13	0.13	0.13	0.2
3600.1	26.75	30	3600.1	0.02	0.02	0.01	0	0.03	0.14	0.13	0.12	0.15	0.14	0.13	0.13	0.2
3700.1	26.75	30	3700.1	0.02	0.02	0.03	0.03	0.02	0.16	0.13	0.13	0.17	0.14	0.15	0.17	0.2
3800.1	26.75	30	3800.1	0.01	0.02	0.01	0.01	0.01	0.16	0.14	0.13	0.16	0.14	0.18	0.16	0.2
3900.1	26.75	30	3900.1	0.01	0.01	0.01	0	-0.01	0.17	0.14	0.14	0.16	0.16	0.17	0.15	0.2
4000.1	26.75	30	4000.1	0	0.01	0	-0.02	-0.01	0.18	0.15	0.14	0.16	0.17	0.16	0.14	0.2
4100.1	26.75	30	4100.1	0.04	0.03	0.04	0.03	0.02	0.19	0.19	0.19	0.2	0.2	0.2	0.21	0.2
4200.1	26.75	30	4200.1	0.06	0.05	0.05	0.05	0.03	0.2	0.22	0.21	0.22	0.22	0.22	0.24	0.2
4300.1	26.75	30	4300.1	0.05	0.07	0.05	0.05	0.04	0.24	0.23	0.23	0.24	0.24	0.24	0.26	0.2
4400.1	26.75	30	4400.1	0.04	0.04	0.04	0.04	0.02	0.23	0.24	0.23	0.22	0.24	0.26	0.26	0.2
4500.1	26.75	30	4500.1	0.02	0.02	0.03	0.03	0.02	0.23	0.24	0.25	0.23	0.24	0.25	0.26	0.2
4600.1	26.75	30	4600.1	0.06	0.06	0.05	0.04	0.04	0.29	0.27	0.29	0.25	0.27	0.3	0.31	0.2
4700.1	26.75	30	4700.1	0.07	0.07	0.06	0.06	0.06	0.33	0.32	0.32	0.27	0.29	0.31	0.33	0.2
4800.1	26.75	30	4800.1	0.05	0.03	0.04	0.03	0.03	0.34	0.3	0.3	0.24	0.28	0.29	0.31	0.2
4900.1	26.75	30	4900.1	0.02	0.02	0.02	0.01	0.01	0.33	0.3	0.3	0.24	0.29	0.29	0.3	0.2
5000.1	26.75	30	5000.1	0.03	0.03	0.02	0.03	0.02	0.37	0.3	0.3	0.27	0.28	0.29	0.32	0.2
5100.1	26.75	30	5100.1	0.05	0.04	0.04	0.05	0.05	0.37	0.34	0.33	0.3	0.32	0.33	0.35	0.2
5200.1	26.75	30	5200.1	0.05	0.07	0.07	0.04	0.05	0.39	0.33	0.33	0.3	0.32	0.34	0.33	0.2
5300.1	26.75	30	5300.1	0.09	0.09	0.09	0.09	0.07	0.42	0.36	0.36	0.33	0.34	0.34	0.33	0.2
5400.1	26.75	30	5400.1	0.09	0.1	0.08	0.07	0.1	0.4	0.34	0.34	0.35	0.35	0.34	0.32	0.2
5500.1	26.75	30	5500.1	0.07	0.08	0.08	0.07	0.08	0.35	0.29	0.3	0.32	0.31	0.32	0.31	0.2
5600.1	26.75	30	5600.1	0.1	0.12	0.11	0.13	0.1	0.32	0.3	0.31	0.35	0.31	0.33	0.32	0.2
5700.1	26.75	30	5700.1	0.13	0.12	0.11	0.1	0.12	0.29	0.3	0.29	0.32	0.3	0.29	0.28	0.2
5800.1	26.75	30	5800.1	0.16	0.13	0.13	0.13	0.12	0.33	0.3	0.25	0.32	0.3	0.28	0.26	0.2
5900.1	26.75	30	5900.1	0.13	0.16	0.17	0.16	0.16	0.35	0.3	0.26	0.3	0.29	0.29	0.25	0.2
5986.1	26.75	30	5986.625	0.16	0.17	0.15	0.16	0.16	0.33	0.27	0.24	0.28	0.28	0.28	0.25	0.2

Frequency Response *G* and Uncertainty *U* in dB

Figure A-3 (continued)
Calibration certificate sheet 6 for the Narda Model SRM-3006 SN A-0077.

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Calibration Certificate

Narda Safety Test Solutions hereby certifies that the referenced equipment has been calibrated by qualified personnel to Narda's approved procedures. The calibration was carried out within a certified quality management system conforming to ISO 9001:2000.

The metrological confirmation system for test equipment complies with ISO 10012-1.

Object	**Three-Axis-Antenna, E-Field, 50 MHz to 3 GHz**
Type	**P/N 3501/02**
Serial Number	**H-0100**
Manufacturer	Narda Safety Test Solutions
Customer	
Date of Calibration	21-Nov-2008
Results of Calibration	Test results within specifications
Confirmation interval recommended	24 Months
Ambient conditions	23℃ +/-3℃ (20...60)% rel. humidity
Calibration procedure	3000-8702-00A

Pfullingen, 22-Nov-2006

MANAGEMENT SYSTEM

Person in charge
Yeter

Quality management representative
W. Kumbier

Certified by DQS according to DIN EN ISO 9001:2000 (Reg.-No. 99379-QM)

Certificate No. 350102-H0100-061121 Date of issue: 22-Nov-2006 Page 1 of 5

Figure A-4
Calibration certificate sheet 1 for the Narda Model SRM-3006 SN H-0100.

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Measurements

The calibration of RF field strength probes involves the generation of a calculable linearly polarized electromagnetic field, approximating to a plane wave, into which the device is placed.
The RSS value of three axis is used.

At each test frequency, the probe is orientated in the analytic angle (54.74 degrees between probe axis and electric field vector) and rotated 360 degrees. The noted indicated output voltage is calculated from the geometric mean of the minimum and maximum readings during rotation. The antenna factor is calculated from the ratio of the applied field strength to the output voltage (nominal impedance 50 Ohm). The minimum and maximum readings during rotation are further used to calculate the ellipse ratio.
A power meter head is connected by means of an ferrite beaded 50 Ohm coaxial cable.

A Crawford TEM cell is used to generate the known field at frequencies up to 100 MHz. The field strength is derived from the TEM cell's properties and from the output power of the cell.
Over the frequency range from 200 MHz to 1.6 GHz, the probe is positioned in front of a double balanced ridge horn antenna. The field strength is set to a known value by means of a calibrated E-field reference probe.
Above 1.7GHz the probe is positioned with the boresight of a linearly polarized horn antenna. The field strength is derived from the mechanical dimensions and the input power of the antenna.

The antenna factor is permanently stored in the antenna connector memory. When combined with the SRM basic unit (BN 3001 series) the frequency response of the antenna is automatically compensated.

Uncertainties

The measurement uncertainty stated in this document is the expanded uncertainty with a coverage factor of 2 (corresponding, in the case of normal distribution, to a confidence probability of 95%).

The uncertainty analysis for this calibration was done in accordance with the ISO-Guide (**G**uide to the expression of **U**ncertainty in **M**easurement). The measurement uncertainties are derived from contributions from the measurement of power, impedance, attenuation, mismatch, length, frequency, stability of instrumentation, repeatability of handling and field uniformity in the field generators (TEM cell and anechoic chamber).

This statement of uncertainty applies to the measured values only and does not make any implementation or include any estimation as to the long-term stability of the calibrated device.

Figure A-4 (continued)
Calibration certificate sheet 2 for the Narda Model SRM-3006 SN H-0100.

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Traceability of Measuring Equipment

The calibration results are traceable to National Standards, which are consistent with the recommendations of the General Conference on Weights and Measure (CGPM), or to standards derived from natural constants. Physical units, which are not included in the list of accredited measured quantities such as field strength or power density, are traced to the basic units via approved measurement and computational methods.

The equipment used for this calibration is traceable to the reference listed above and the traceability is guaranteed by ISO 9001 Narda internal procedure.

Ref.- / Working Standard	Type	Serial Number	Certificate Number	Cal Due Date	Traced Property
Network Analyser	ZVC	100032	0063 DKD-K-16101 06-04	Apr-07	Reflection
Calibration Kit, 50 Ohm N	ZV-Z21	100072	98553 DKD-K-19401 06-04	Apr-07	Reflection
Power Meter	URV 55	100213	0065 DKD-K-16101 06-04	Apr-08	Voltage
Power Sensor DC - 6GHz	NRV Z4	100199	0010 DKD-K-16101 06-05	Mai-08	Power
N Thru, 50 Ohm	0659.4666.00	79006	0067 DKD-K-16101 06-04	Apr-08	Attenuation
Calipor 0 - 600mm	-	310121016	649724 DKD-K-12001 06-05	Mai-07	Length
Set-Up "D" (50 MHz to 100 MHz)					
TEM Cell, DC - 120MHz	2-2250	-	MM 2-2250	Apr-2007	Length
Power Sensor 4.2GHz	8482A	2652A13544	04D265 DKD-K-02201 04-11	Nov-2006	Power
Power Meter, Two Channel	438A	2741U00723	2-119508307-1B	Nov-2006	Voltage
Attenuator 30dB	49-30-33	KCC 115	2824 DKD-K-C0501 05-04	Apr-2007	Attenuation
Set-Up "B" (200 MHz to 1600 MHz)					
E-Field Reference Probe	Type 9.2	V-0017	601C1734 / 51200637E	Dez-07	Field Strength
Radiation Meter	EMR-300	AG-0099	22443100-AG00990347	Nov-2007	Voltage
Set-Up "A" (1800 MHz to 3 GHz)					
Power Sensor 18GHz	8481A	2702A57612	05D131 DKD-K-02201 05-07	Jul-2007	Power
Power Sensor 18GHz	8481A	2702A75991	05D130 DKD-K-02201 05-07	Jul-2007	Power
Power Meter, Two Channel	E4419B	GB43311917	2-170204368-1B	Dez-2006	Voltage
Coupler 1-4GHz & 6dB Att.	3022 & 777C-6	77961/34425	00 996	Okt-2006	Attenuation
Coupler 1-4GHz & 10dB Att.	3022 & 777C-10	77960/38334	00 995	Okt-2006	Attenuation

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Figure A-4 (continued)
Calibration certificate sheet 3 for the Narda Model SRM-3006 SN H-0100.

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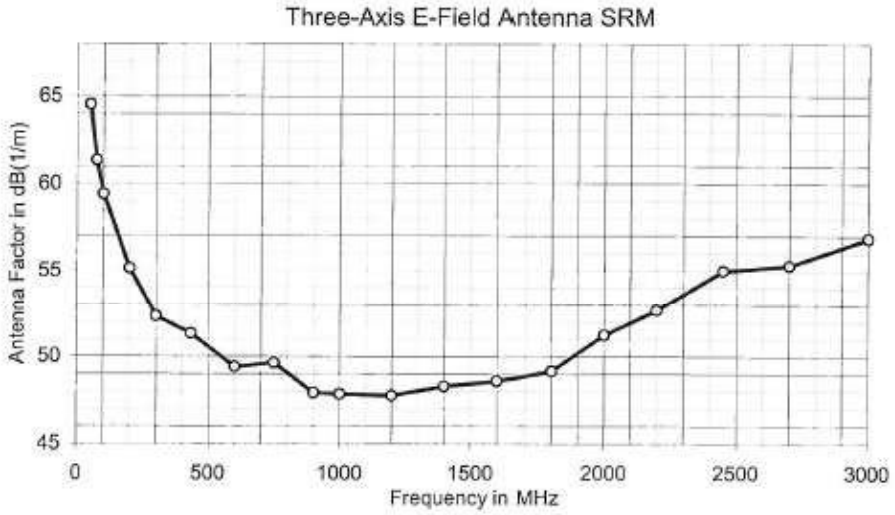
Results

Frequency Response **passed**

Frequency in MHz	E_applied in V/m	Output voltage in dB(µV)	Meas. Uncertainty in dB	Antenna Factor in dB(1/m)
50	10,0	75,47	1,0	64,53
75	10,0	78,67	1,0	61,33
100	10,0	80,61	1,0	59,39
200	10,0	84,88	1,0	55,12
300	10,0	87,66	1,0	52,34
433	10,0	88,68	1,5	51,32
600	10,0	90,60	1,5	49,40
750	10,0	90,36	1,5	49,64
900	10,0	92,09	1,5	47,91
1000	10,0	92,17	1,5	47,83
1200	10,0	92,25	1,5	47,75
1400	10,0	91,72	1,5	48,28
1600	10,0	91,41	1,5	48,59
1800	10,0	90,85	1,0	49,15
2000	10,0	88,77	1,0	51,23
2200	10,0	87,32	1,0	52,68
2450	10,0	85,01	1,0	54,99
2700	10,0	84,71	1,0	55,29
3000	10,0	83,13	1,0	56,87

Frequency Flatness (100 - 3000 MHz): 13,6 dB

The Antenna Factor data is permanently stored in the antenna connector memory.
The SRM basic unit uses this correction data to correct the display.



Certificate No. 350102-H0100-061121 Date of issue: 22-Nov-2006 Page 4 of 5

Figure A-4 (continued)
Calibration certificate sheet 4 for the Narda Model SRM-3006 SN H-0100.

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Rotational Ellipticity **passed**

Frequency in MHz	Ellipse Ratio in dB
50	+/-0,08
75	+/-0,05
100	+/-0,08
200	+/-0,08
300	+/-0,05
433	+/-0,13
600	+/-0,14
750	+/-0,20
900	+/-0,19
1000	+/-0,36
1200	+/-0,30
1400	+/-0,45
1600	+/-0,68
1800	+/-0,90
2000	+/-1,21
2200	+/-1,28
2450	+/-1,22
2700	+/-0,96
3000	+/-1,45

Output Return Loss **passed**

Figure A-4 (continued)
Calibration certificate sheet 5 for the Narda Model SRM-3006 SN H-0100.

Calibration Certificate

Narda Safety Test Solutions hereby certifies that the object referred to in this certificate has been calibrated by qualified personnel using Narda's approved procedures. The calibration was carried out in accordance with a certified quality management system which conforms to ISO 9001

OBJECT	Selective Radiation Meter, Basic Unit, SRM-3006
MANUFACTURER	Narda Safety Test Solutions GmbH
PART NUMBER (P/N)	3006/01
SERIAL NUMBER (S/N)	D-0070
CUSTOMER	
CALIBRATION DATE	2010-10-18
RESULT ASSESSMENT	within specifications
AMBIENT CONDITIONS	Temperature: (23 ± 3)°C Relative humidity: (25 to 75) %
CALIBRATION PROCEDURE	3006-8701-00A

ISSUE DATE: 2010-10-18

MANAGEMENT SYSTEM

CALIBRATED BY:
Paul Geyer

AUTHORIZED SIGNATORY:

Certified by DQS against ISO 9001:2008 (Reg.-No. 099379 QM08)

CERTIFICATE 300601-D0070-20101018-74

PAGE 1 OF 6

Figure A-5
Calibration certificate sheet 1 for the Narda Model SRM-3006 SN H-0100.

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OBJECT

The spectrum analyzer is based on digital signal processing. Small frequency spans were measured at fixed local oscillator (1st LO) settings using discrete Fourier transformation (DFT). The LO was also swept for larger frequency spans.
A memory chip contains correction values for various frequencies and object settings. The stored values were taken into account automatically during the measurement.

METHOD OF MEASUREMENT

Calibration using the reference standard. The output power level of the synthesized CW generator was adjusted and calibrated using power sensors as reference standards.
The frequency of the generator was calibrated using a frequency counter.

The reflection of the object was measured directly using a vector network analyzer (VNA) calibrated by means of a calibration kit. The measuring equipment and the associated uncertainty were verified using a reference standard (verification kit).

CALIBRATION PROCEDURE

The object was connected to the signal source instead of the power sensors in order to calibrate it.

Measurement of the RF frequency response was made with different settings of the measurement range. As a result, the measured values also include the effects due to the "input attenuator" and the "reference level accuracy".
The calibration factor was calculated for various frequencies and settings from a comparison between the "actual level" and the "indicated level".

All the selection filters are digital filters. No calibration of the filters is necessary.

TRACEABILITY

The calibration results are traceable to the International System of Units (SI) in accordance with ISO/IEC 17025. The measuring equipment used for calibration is traceable through the reference standards listed below.

STANDARD	MANUFAC-TURER	MODEL	SERIAL NUMBER	ID	CERTIFICATE	NEXT CAL. DATE	TRACE
HF-MILLIVOLTMETER	R&S	URV 55	100143	913	0116 DKD-K-16101 2010-05	2012-05	DKD
DIODE POWER SENSOR	R&S	NRV Z4	100199	956	0104 DKD-K-16101 2010-05	2012-05	DKD
THERMAL POWER SENSOR	R&S	NRV Z51	101777	1635	0264 DKD-K-16101 2008-11	2010-11	DKD
MISMATCH VSWR 1,2 (f)	Rosenberger	--	01237	552-3	12996 DKD-K-00201 2008-05	#	DKD
FREQUENCY COUNTER	Advantest	R5362B	120700137	923	15137 DKD-K-00201 2009-09	#	DKD

Reference standard; not used for routine calibration

Figure A-5 (continued)
Calibration certificate sheet 2 for the Narda Model SRM-3006 SN H-0100.

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UNCERTAINTY

The reported expanded uncertainty U is based on a standard uncertainty multiplied by a coverage factor $k = 1.96$, providing a level of confidence of approximately 95 %. The uncertainty evaluation has been carried out in accordance with the "Guide to the Expression of Uncertainty in Measurement" (GUM). The reported measurement uncertainty is derived from the uncertainty of the calibration procedure and the object during calibration, and makes no allowance for drift or operation under other environmental conditions.

MEASURING CONDITIONS

The following results were obtained after adjustment of the object under calibration. These values are within the setting ranges defined by the manufacturer.

RESULTS

1	FREQUENCY RESPONSE (IF):	passed
2	FREQUENCY RESPONSE (RF):	passed
3	OUT-OF-BAND RESPONSE:	passed
4	FREQUENCY ACCURACY	passed
5	NOISE SIDEBAND (SSB):	passed
6	SPURIOUS (input related)	passed
7	SPURIOUS (residual)	passed
8	NOISE FLOOR:	passed
9	INTERMODULATION REJECTION (2^{nd} and 3^{rd} order):	passed
10	INPUT RETURN LOSS:	passed

Figure A-5 (continued)
Calibration certificate sheet 3 for the Narda Model SRM-3006 SN H-0100.

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APPENDIX

FREQUENCY RESPONSE (RF)

The generator was set to the *Fgen*. The object settings were *Fspan*, *RBW*, and *Fcent*.
The measurements were made at different settings of the measurement range *MR*. The nominal level of the generator was -32 dBm (for *MR* < -5 dBm) and -7 dBm (for *MR* ≥ -5 dBm), respectively. The frequency response *G* was calculated as the difference of the actual generator level L_{actual} and the indicated level $L_{indicated}$ according to the following equation: $G/\mathrm{dB} = (L_{indicated} - L_{actual})/\mathrm{dBm}$

Frequency in MHz	Fspan in MHz	RBW in kHz	Fcent in MHz	MR												U
				-30	-28	-25	-20	-15	-10	-5	0	5	10	15	20	
0.00901	0.002	0.01	0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.02	-0.02	-0.02	-0.02	0.2
0.012	0.006	0.5	0.012	0	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	0.2
0.02	0.02	2	0.02	0	0	0	0	0	0	0	-0.01	-0.01	-0.01	-0.01	-0.01	0.2
0.04	0.02	2	0.04	0	0	0	0	0	0	0	0	-0.01	0	-0.01	-0.01	0.2
0.1	0.02	2	0.1	0.01	0.01	0.01	0	0	0.01	0	0	0	0	0	0	0.2
0.5	0.02	2	0.5	0.01	0.01	0.01	0	0	0.01	0	0	0	0	0	-0.01	0.2
2	0.02	2	2	0.01	0.01	0.01	0.01	0.01	0.01	0	0	0	0	0	-0.01	0.2
10	0.02	2	10	0.03	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.02	0.01	0.01	0.2
20	0.02	2	20	0.03	0.03	0.02	0.03	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.2
30	0.02	2	30	0.03	0.03	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.02	0.01	0.02	0.2
31.233	26.75	30	44.578	-0.06	-0.17	-0.13	-0.15	-0.27	-0.02	-0.15	-0.18	-0.28	0.02	-0.11	-0.13	0.2
36.1	26.75	30	44.578	0.03	-0.05	-0.03	-0.05	-0.09	0.09	-0.05	-0.06	-0.1	0.07	0.03	0.1	0.2
40	0.02	2	40	0.03	0.02	0.02	0.02	0.02	0.03	0.02	0.02	0.01	0.01	0	0.01	0.2
44.1	26.75	30	44.578	0.1	0.06	0.07	0.05	0.05	0.1	0.05	0.04	0.05	0.05	0.03	0.05	0.2
50	0.02	2	50	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.02	0.2
52.1	26.75	30	44.578	0.09	0.07	0.05	0.04	0.08	0.12	0.04	0.03	0.08	0	0.04	0.08	0.2
57.9948	0.02	2	57.9868	0.03	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.01	0.01	0.01	0.2
58.344	26.75	30	44.999	0.07	0.05	0.02	0	0.06	0.13	0.01	-0.01	0.05	-0.06	0.03	0.11	0.2
60.1	26.75	30	60.1	0.08	0.08	0.08	0.07	0.07	0.08	0.07	0.07	0.07	0.06	0.07	0.06	0.2
100.1	26.75	30	100.1	0.08	0.08	0.08	0.07	0.07	0.08	0.07	0.06	0.07	0.06	0.08	0.05	0.2
200.1	26.75	30	200.1	0.06	0.06	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.03	0.2
300.1	26.75	30	300.1	0.06	0.07	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.05	0.06	0.05	0.2
400.1	26.75	30	400.1	0.07	0.06	0.07	0.07	0.06	0.07	0.05	0.05	0.06	0.06	0.06	0.05	0.2

Figure A-5 (continued)
Calibration certificate sheet 4 for the Narda Model SRM-3006 SN H-0100.

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Frequency in MHz	Fspan in MHz	RBW in kHz	Fcent in MHz	MR												U
				-30	-28	-25	-20	-15	-10	-5	0	5	10	15	20	
500.1	26.75	30	500.1	0.06	0.06	0.06	0.07	0.06	0.07	0.06	0.06	0.05	0.05	0.05	0.06	0.2
600.1	26.75	30	600.1	0.08	0.08	0.08	0.08	0.08	0.07	0.08	0.07	0.07	0.07	0.06	0.07	0.2
700.1	26.75	30	700.1	0.08	0.08	0.08	0.07	0.07	0.08	0.07	0.08	0.07	0.07	0.06	0.06	0.2
800.1	26.75	30	800.1	0.07	0.07	0.07	0.07	0.07	0.06	0.06	0.07	0.07	0.06	0.06	0.06	0.2
900.1	26.75	30	900.1	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.05	0.06	0.05	0.05	0.04	0.2
1000.1	26.75	30	1000.1	0.06	0.06	0.07	0.05	0.05	0.05	0.06	0.06	0.06	0.06	0.05	0.04	0.2
1100.1	26.75	30	1100.1	0.07	0.07	0.07	0.07	0.06	0.07	0.07	0.06	0.06	0.06	0.06	0.05	0.2
1200.1	26.75	30	1200.1	0.07	0.06	0.06	0.06	0.06	0.07	0.06	0.06	0.06	0.06	0.06	0.06	0.2
1300.1	26.75	30	1300.1	0.06	0.06	0.06	0.06	0.06	0.06	0.05	0.05	0.05	0.05	0.05	0.04	0.2
1400.1	26.75	30	1400.1	0.09	0.09	0.08	0.08	0.08	0.08	0.08	0.08	0.07	0.08	0.08	0.07	0.2
1500.1	26.75	30	1500.1	0.11	0.1	0.11	0.1	0.1	0.1	0.09	0.1	0.09	0.1	0.09	0.1	0.2
1600.1	26.75	30	1600.1	0.08	0.09	0.08	0.09	0.08	0.08	0.07	0.08	0.07	0.08	0.06	0.07	0.2
1700.1	26.75	30	1700.1	0.12	0.12	0.12	0.13	0.12	0.12	0.12	0.12	0.12	0.11	0.11	0.12	0.2
1800.1	26.75	30	1800.1	0.08	0.08	0.08	0.08	0.08	0.08	0.08	0.08	0.07	0.06	0.07	0.05	0.2
1900.1	26.75	30	1900.1	0.07	0.07	0.07	0.07	0.06	0.06	0.06	0.05	0.06	0.06	0.06	0.07	0.2
2000.1	26.75	30	2000.1	0.08	0.08	0.08	0.06	0.07	0.07	0.06	0.06	0.06	0.07	0.06	0.06	0.2
2100.1	26.75	30	2100.1	0.09	0.09	0.08	0.09	0.07	0.1	0.09	0.09	0.08	0.07	0.08	0.07	0.2
2200.1	26.75	30	2200.1	0.1	0.08	0.08	0.09	0.08	0.08	0.09	0.08	0.08	0.08	0.08	0.07	0.2
2300.1	26.75	30	2300.1	0.09	0.08	0.09	0.08	0.08	0.1	0.09	0.08	0.08	0.08	0.06	0.07	0.2
2400.1	26.75	30	2400.1	0.09	0.08	0.08	0.1	0.09	0.09	0.1	0.08	0.08	0.09	0.07	0.06	0.2
2500.1	26.75	30	2500.1	0.07	0.05	0.05	0.05	0.04	0.05	0.06	0.04	0.04	0.05	0.04	0.04	0.2
2600.1	26.75	30	2600.1	0.08	0.07	0.07	0.08	0.07	0.06	0.07	0.07	0.06	0.07	0.07	0.06	0.2
2700.1	26.75	30	2700.1	0.11	0.11	0.12	0.12	0.11	0.12	0.12	0.11	0.12	0.1	0.1	0.1	0.2
2800.1	26.75	30	2800.1	0.11	0.12	0.1	0.09	0.09	0.09	0.09	0.09	0.09	0.1	0.09	0.08	0.2
2900.1	26.75	30	2900.1	0.08	0.08	0.1	0.09	0.08	0.08	0.09	0.1	0.08	0.08	0.08	0.08	0.2
2999.9	26.75	30	2999.9	0.07	0.09	0.06	0.07	0.08	0.07	0.09	0.08	0.07	0.09	0.07	0.07	0.2
3002.1	26.75	30	3002.1	0.06	0.07	0.07	0.06	0.07	0.06	0.07	0.07	0.06	0.07	0.05	0.05	0.2
3100.1	26.75	30	3100.1	0.08	0.07	0.07	0.06	0.07	0.07	0.06	0.05	0.06	0.06	0.05	0.06	0.2
3200.1	26.75	30	3200.1	0.08	0.09	0.08	0.08	0.08	0.09	0.09	0.09	0.09	0.08	0.08	0.08	0.2
3300.1	26.75	30	3300.1	0.09	0.09	0.1	0.09	0.09	0.08	0.09	0.09	0.08	0.08	0.09	0.08	0.2
3400.1	26.75	30	3400.1	0.1	0.08	0.09	0.09	0.08	0.09	0.09	0.09	0.09	0.09	0.09	0.09	0.2
3500.1	26.75	30	3500.1	0.11	0.1	0.1	0.09	0.08	0.1	0.1	0.1	0.09	0.1	0.09	0.1	0.2

Figure A-5 (continued)
Calibration certificate sheet 5 for the Narda Model SRM-3006 SN H-010 .

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Frequency in MHz	Fspan in MHz	RBW in kHz	Fcent in MHz	MR												U
				-30	-28	-25	-20	-15	-10	-5	0	5	10	15	20	
3600.1	26.75	30	3600.1	0.06	0.06	0.07	0.07	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.06	0.2
3700.1	26.75	30	3700.1	0.05	0.06	0.05	0.07	0.06	0.07	0.07	0.06	0.06	0.06	0.07	0.05	0.2
3800.1	26.75	30	3800.1	0.07	0.07	0.07	0.08	0.07	0.08	0.07	0.08	0.06	0.07	0.05	0.06	0.2
3900.1	26.75	30	3900.1	0.07	0.05	0.06	0.06	0.06	0.06	0.06	0.05	0.06	0.05	0.07	0.05	0.2
4000.1	26.75	30	4000.1	0.06	0.06	0.06	0.05	0.05	0.07	0.06	0.05	0.05	0.06	0.06	0.03	0.2
4100.1	26.75	30	4100.1	0.09	0.07	0.09	0.09	0.09	0.08	0.08	0.07	0.07	0.07	0.06	0.08	0.2
4200.1	26.75	30	4200.1	0.11	0.11	0.1	0.11	0.1	0.13	0.11	0.11	0.1	0.1	0.1	0.09	0.2
4300.1	26.75	30	4300.1	0.1	0.1	0.1	0.1	0.1	0.12	0.1	0.1	0.11	0.1	0.11	0.1	0.2
4400.1	26.75	30	4400.1	0.08	0.09	0.09	0.08	0.09	0.08	0.09	0.09	0.07	0.08	0.07	0.1	0.2
4500.1	26.75	30	4500.1	0.05	0.06	0.05	0.05	0.06	0.05	0.05	0.06	0.05	0.05	0.04	0.03	0.2
4600.1	26.75	30	4600.1	0.06	0.08	0.07	0.09	0.08	0.08	0.06	0.07	0.07	0.07	0.06	0.07	0.2
4700.1	26.75	30	4700.1	0.09	0.09	0.1	0.09	0.1	0.09	0.1	0.09	0.09	0.08	0.08	0.08	0.2
4800.1	26.75	30	4800.1	0.06	0.08	0.06	0.05	0.05	0.07	0.06	0.05	0.05	0.05	0.05	0.03	0.2
4900.1	26.75	30	4900.1	0.04	0.04	0.04	0.04	0.04	0.05	0.05	0.05	0.04	0.04	0.04	0.03	0.2
5000.1	26.75	30	5000.1	0.07	0.08	0.06	0.08	0.06	0.05	0.04	0.06	0.06	0.05	0.05	0.05	0.2
5100.1	26.75	30	5100.1	0.09	0.08	0.09	0.08	0.06	0.07	0.08	0.09	0.08	0.08	0.07	0.07	0.2
5200.1	26.75	30	5200.1	0.1	0.09	0.1	0.08	0.08	0.1	0.09	0.1	0.09	0.08	0.09	0.07	0.2
5300.1	26.75	30	5300.1	0.12	0.1	0.11	0.09	0.09	0.11	0.09	0.11	0.09	0.09	0.09	0.1	0.2
5400.1	26.75	30	5400.1	0.11	0.1	0.12	0.11	0.12	0.12	0.09	0.09	0.11	0.09	0.1	0.09	0.2
5500.1	26.75	30	5500.1	0.09	0.1	0.1	0.1	0.09	0.08	0.12	0.1	0.1	0.09	0.1	0.1	0.2
5600.1	26.75	30	5600.1	0.12	0.11	0.1	0.1	0.08	0.1	0.09	0.11	0.07	0.09	0.1	0.08	0.2
5700.1	26.75	30	5700.1	0.11	0.11	0.11	0.08	0.08	0.09	0.11	0.09	0.1	0.11	0.09	0.08	0.2
5800.1	26.75	30	5800.1	0.14	0.11	0.12	0.11	0.12	0.12	0.1	0.12	0.11	0.1	0.12	0.12	0.2
5900.1	26.75	30	5900.1	0.13	0.15	0.12	0.12	0.13	0.16	0.15	0.14	0.12	0.14	0.14	0.11	0.2
5986.1	26.75	30	5986.625	0.16	0.14	0.15	0.15	0.14	0.14	0.14	0.14	0.13	0.13	0.13	0.12	0.2

Frequency Response *G* and Uncertainty *U* in dB

Figure A-5 (continued)
Calibration certificate sheet 6 for the Narda Model SRM-3006 SN H-0100.

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Calibration Certificate

Narda Safety Test Solutions hereby certifies that the referenced equipment has been calibrated by qualified personnel to Narda's approved procedures. The calibration was carried out within a certified quality management system conforming to ISO 9001.

Object	**Antenna, Three-Axis, E-Field, 27 MHz to 3 GHz**
Part Number (P/N)	**3501/03**
Serial Number (S/N)	**K-0243**
Manufacturer	Narda Safety Test Solutions GmbH
Customer	
Date of Calibration	07-Okt-2010
Results of Calibration	Test results within specifications
Confirmation interval recommended	24 Months
Ambient conditions	Temperature: (23 ± 3) °C Relative humidity: (20 to 60) %
Calibration procedure	3000-8702-00A

Pfullingen, 07-Okt-2010

MANAGEMENT SYSTEM

Person in charge
Geyer

i.A.
Head of Laboratory
J. v. Freeden

Certified by DQS according to ISO 9001:2008
(Reg.-No. 099379 QM08)

Figure A-6
Calibration certificate sheet 1 for the Narda Model SRM-3006 SN H-0100.

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Measurements

The calibration of RF field strength probes involves the generation of a calculable linearly polarized electromagnetic field, approximating to a plane wave, into which the device is placed.
The RSS value of three axis is used.

At each test frequency, the probe is orientated in the analytic angle (54.74 degrees between probe axis and electric field vector) and rotated 360 degrees. The noted indicated output voltage is calculated from the geometric mean of the minimum and maximum readings during rotation. The antenna factor is calculated from the ratio of the applied field strength to the output voltage (nominal impedance 50 Ohm). The minimum and maximum readings during rotation are further used to calculate the ellipse ratio.
A power meter head is connected by means of an ferrite beaded 50 Ohm coaxial cable.

A Crawford TEM cell is used to generate the known field at frequencies up to 100 MHz. The field strength is derived from the TEM cell's properties and from the output power of the cell.
Over the frequency range from 200 MHz to 1.6 GHz, the probe is positioned in front of a double balanced ridge horn antenna. The field strength is set to a known value by means of a calibrated E-field reference probe.
Above 1.7GHz the probe is positioned with the boresight of a linearly polarized horn antenna. The field strength is derived from the mechanical dimensions and the input power of the antenna.

The antenna factor is permanently stored in the antenna connector memory. When combined with the SRM basic unit (BN 3001 series) the frequency response of the antenna is automatically compensated.

Uncertainties

The measurement uncertainty stated in this document is the expanded uncertainty with a coverage factor of 2 (corresponding, in the case of normal distribution, to a confidence probability of 95%).

The uncertainty analysis for this calibration was done in accordance with the ISO-Guide (**G**uide to the expression of **U**ncertainty in **M**easurement). The measurement uncertainties are derived from contributions from the measurement of power, impedance, attenuation, mismatch, length, frequency, stability of instrumentation, repeatability of handling and field uniformity in the field generators (TEM cell and anechoic chamber).

This statement of uncertainty applies to the measured values only and does not make any implementation or include any estimation as to the long-term stability of the calibrated device.

Figure A-6 (continued)
Calibration certificate sheet 2 for the Narda Model SRM-3006 SN H-0100.

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Traceability of Measuring Equipment

The calibration results are traceable to National Standards, which are consistent with the recommendations of the General Conference on Weights and Measure (CGPM), or to standards derived from natural constants. Physical units, which are not included in the list of accredited measured quantities such as field strength or power density, are traced to the basic units via approved measurement and computational methods.

The equipment used for this calibration is traceable to the reference listed above and the traceability is guaranteed by ISO 9001 Narda internal procedure.

Reference- / Working- Standard	Manu facturer	Model	Serial Number	Certificate Number	Cal Due Date	Trace
Power Sensor	R&S	NRV-Z4	100122	0171 DKD-K-16101 2008-11	2010-11	DKD
RF-Millivoltmeter	R&S	URV55	100213	0224 DKD-K-16101 2010-08	2012-08	DKD
Set-Up "A" (1800 MHz to 3 GHz)						
Calliper	Preisser	0-800mm	310121016	649724 DKD-K-12001 06-05	#	DKD
Power Sensor	agilent	8481A	US37299951	1-2217165994-1	2011-08	UKAS147
Power Sensor	agilent	8481A	US37299952	1-2217214152-1	2011-09	UKAS147
Power Meter	agilent	E4419A	MY40330449	1-2217141092-1A	2011-09	UKAS147
Set-Up "B" (200 MHz to 1600 MHz)						
E-Field Reference Probe	Narda	Type 9.2	V-0017	51200637E	#	SIT08
Power Sensor	agilent	8481A	US37299870	1-2217214643-1	2011-09	UKAS147
Power Sensor	agilent	8481A	2702A57611	1-2217165866-1	2011-09	UKAS147
Power Meter	agilent	E4419B	GB43311917	1-2295928041-1A	2011-11	UKAS147
Set-Up "D" (100 kHz to 100 MHz)						
Calliper	Preisser	0-800mm	310121016	649724 DKD-K-12001 06-05	#	DKD
Power Sensor	agilent	8482A	2652A13544	08D177 DKD-K-02201 2008-06	2010-12	DKD
Power Meter	agilent	438A	2741U00723	1-1321958613-1A	2010-12	UKAS147
Attenuator	Weinschel	49-30-33	KC115	3248 DKD-K-00501 2008-06	2011-06	DKD

Reference standard; not used for routine calibration

Figure A-6 (continued)
Calibration certificate sheet 3 for the Narda Model SRM-3006 SN H-0100.

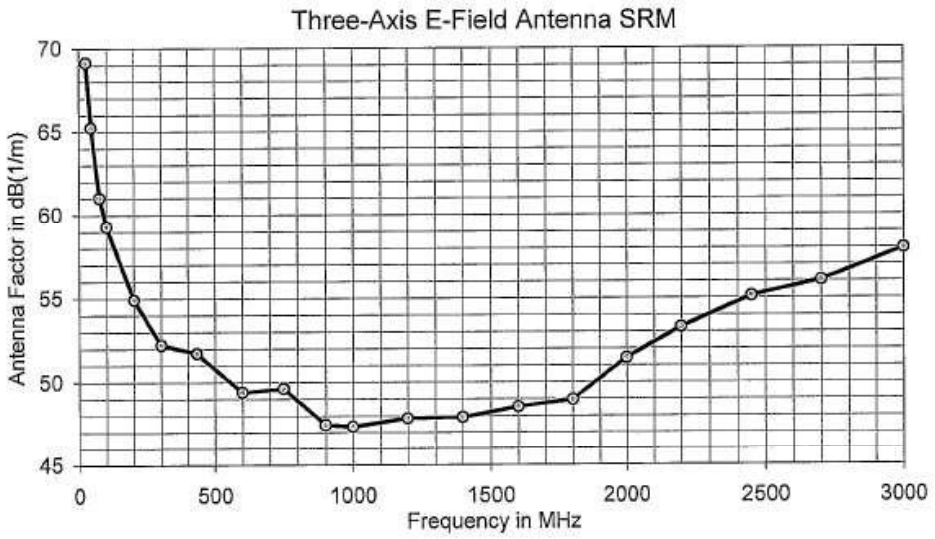
Results

Frequency Response **passed**

Frequency in MHz	E_applied in V/m	Output voltage in dB(µV)	Meas. Uncertainty in dB	Antenna Factor in dB(1/m)
26	10,0	70,85	1,0	69,15
45	10,0	74,77	1,0	65,23
75	10,0	78,97	1,0	61,03
100	10,0	80,73	1,0	59,27
200	10,0	85,08	1,0	54,92
300	10,0	87,79	1,0	52,21
433	10,0	88,28	1,5	51,72
600	10,0	90,61	1,5	49,39
750	10,0	90,39	1,5	49,61
900	10,0	92,56	1,5	47,44
1000	10,0	92,66	1,5	47,34
1200	10,0	92,17	1,5	47,83
1400	10,0	92,10	1,5	47,90
1600	10,0	91,48	1,5	48,52
1800	10,0	91,06	1,0	48,94
2000	10,0	88,57	1,0	51,43
2200	10,0	86,76	1,0	53,24
2450	10,0	84,91	1,0	55,09
2700	10,0	83,97	1,0	56,03
3000	10,0	82,07	1,0	57,93

Frequency Flatness (100 - 3000 MHz): 11,9 dB

The Antenna Factor data is permanently stored in the antenna connector memory.
The SRM basic unit uses this correction data to correct the display.



Certificate No. 350103-K0243-101007 Date of issue: 07-Okt-2010 Page 4 of 5

Figure A-6 (continued)
Calibration certificate sheet 4 for the Narda Model SRM-3006 SN H-0100.

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Rotational Ellipticity **passed**

Frequency in MHz	Ellipse Ratio in dB
26	+/-0,14
45	+/-0,17
75	+/-0,14
100	+/-0,14
200	+/-0,09
300	+/-0,06
433	+/-0,04
600	+/-0,08
750	+/-0,19
900	+/-0,13
1000	+/-0,36
1200	+/-0,46
1400	+/-0,41
1600	+/-0,86
1800	+/-0,68
2000	+/-1,22
2200	+/-1,55
2450	+/-1,45
2700	+/-1,66
3000	+/-1,99

Output Return Loss **passed**

Figure A-6 (continued)
Calibration certificate sheet 5 for the Narda Model SRM-3006 SN H-0100.

Appendix B: SRM-3006 900 MHz Spectrum Measurement Scans (meter farm)

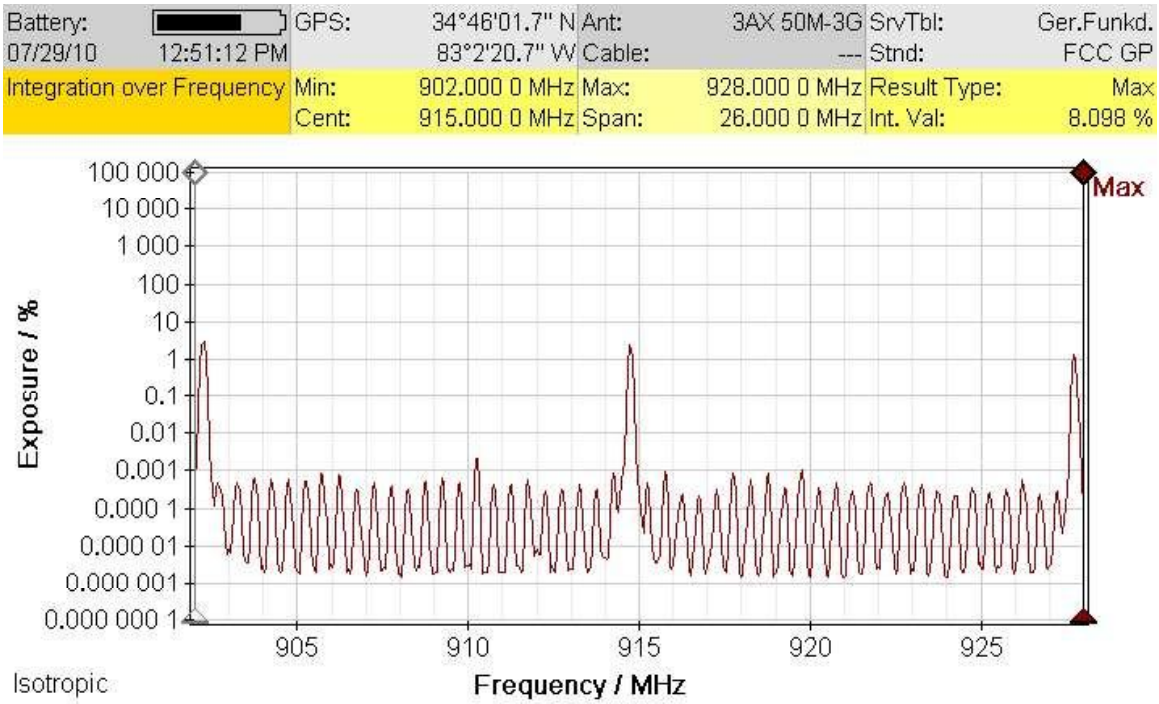

Figure B-1
900 MHz band composite RF field from rack of 10 SmartMeters at 1 foot.

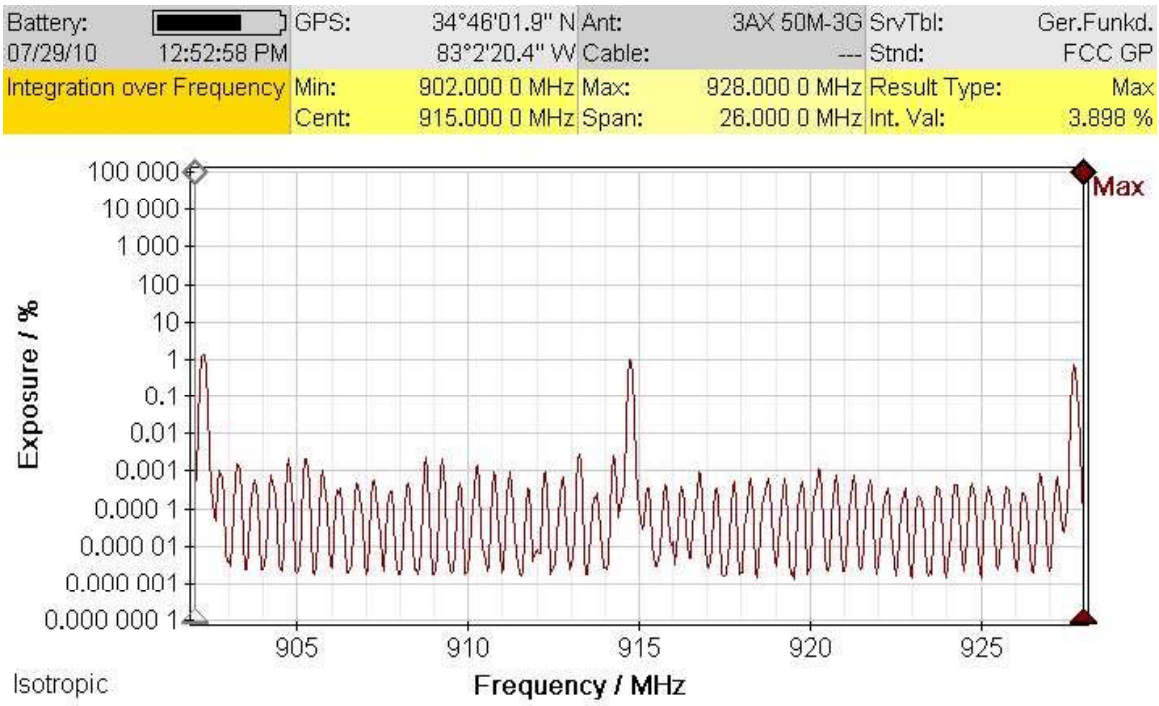

Figure B-2
900 MHz band composite RF field from rack of 10 SmartMeters at 2 feet.

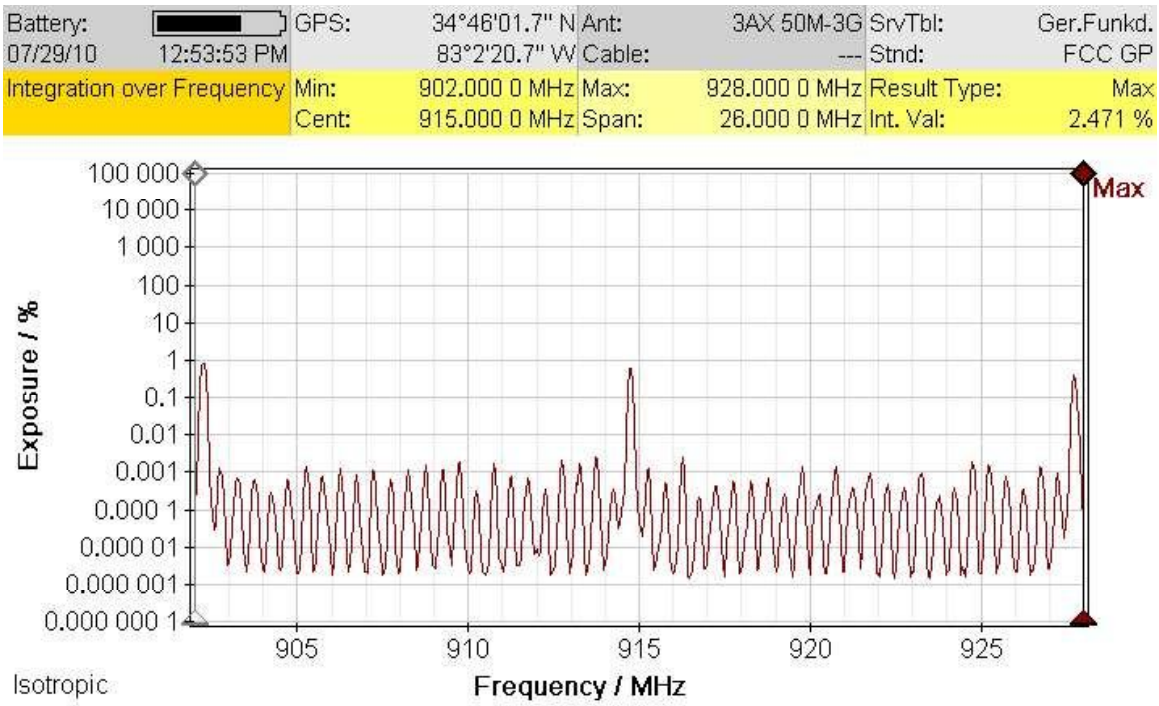

Figure B-3
900 MHz band composite RF field from rack of 10 SmartMeters at 3 feet.

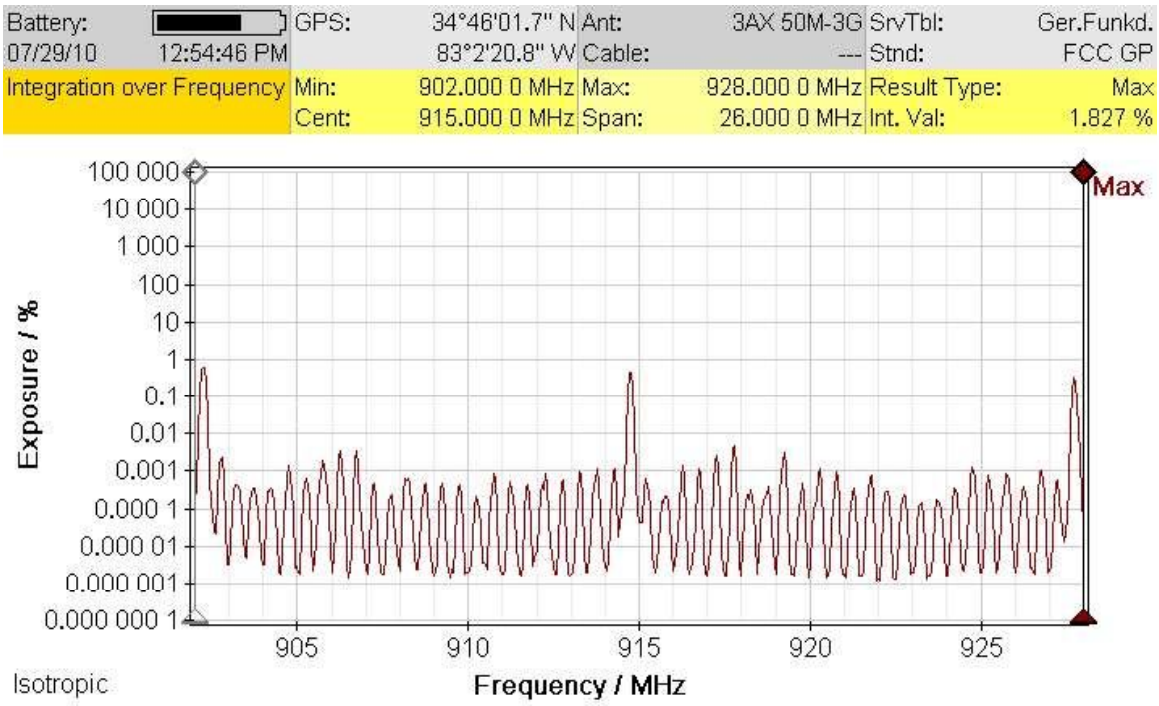

Figure B-4
900 MHz band composite RF field from rack of 10 SmartMeters at 4 feet.

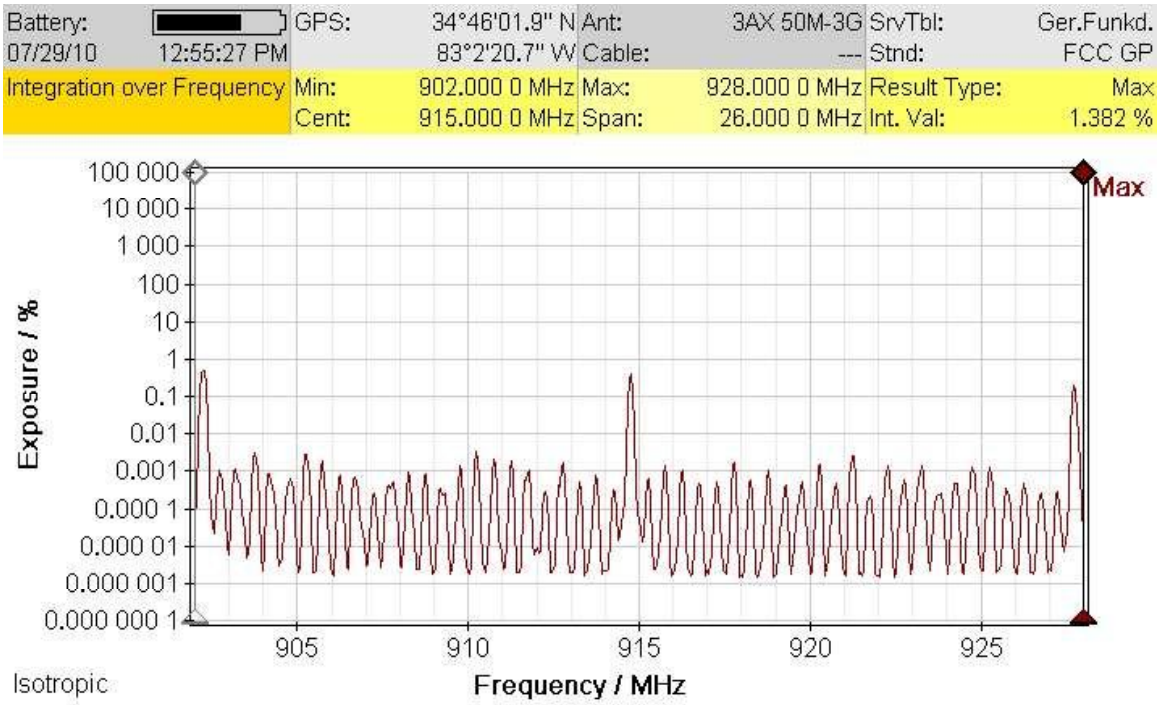

Figure B-5
900 MHz band composite RF field from rack of 10 SmartMeters at 5 feet.

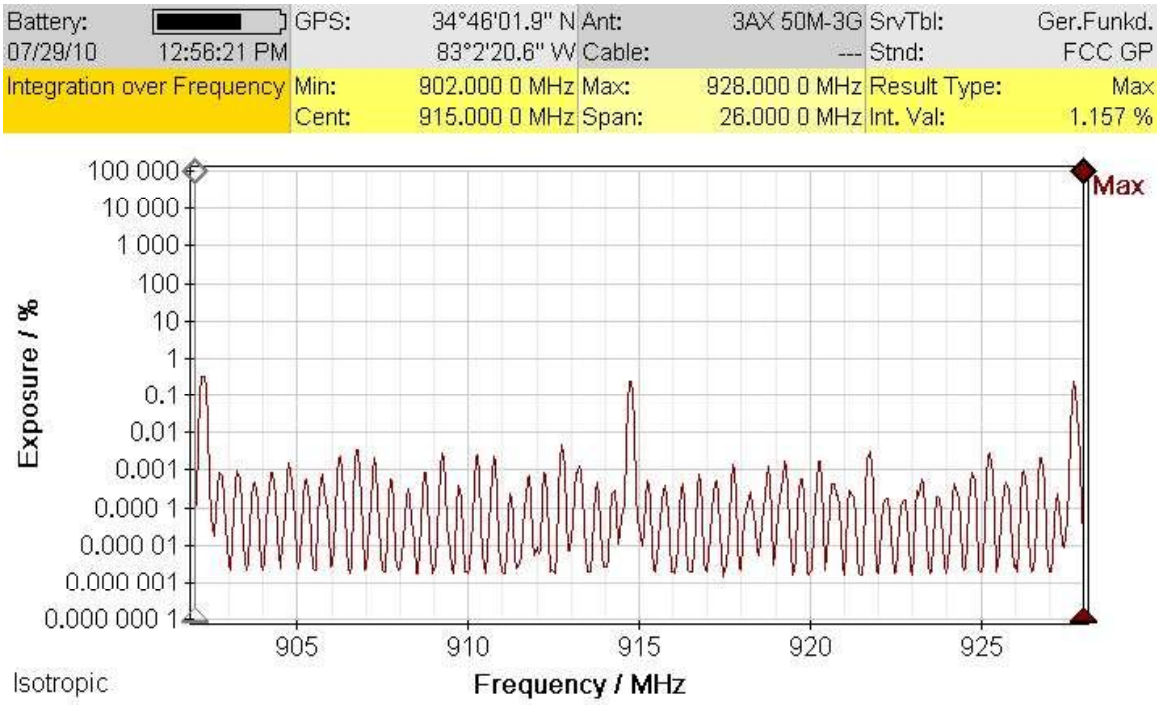

Figure B-6
900 MHz band composite RF field from rack of 10 SmartMeters at 6 feet.

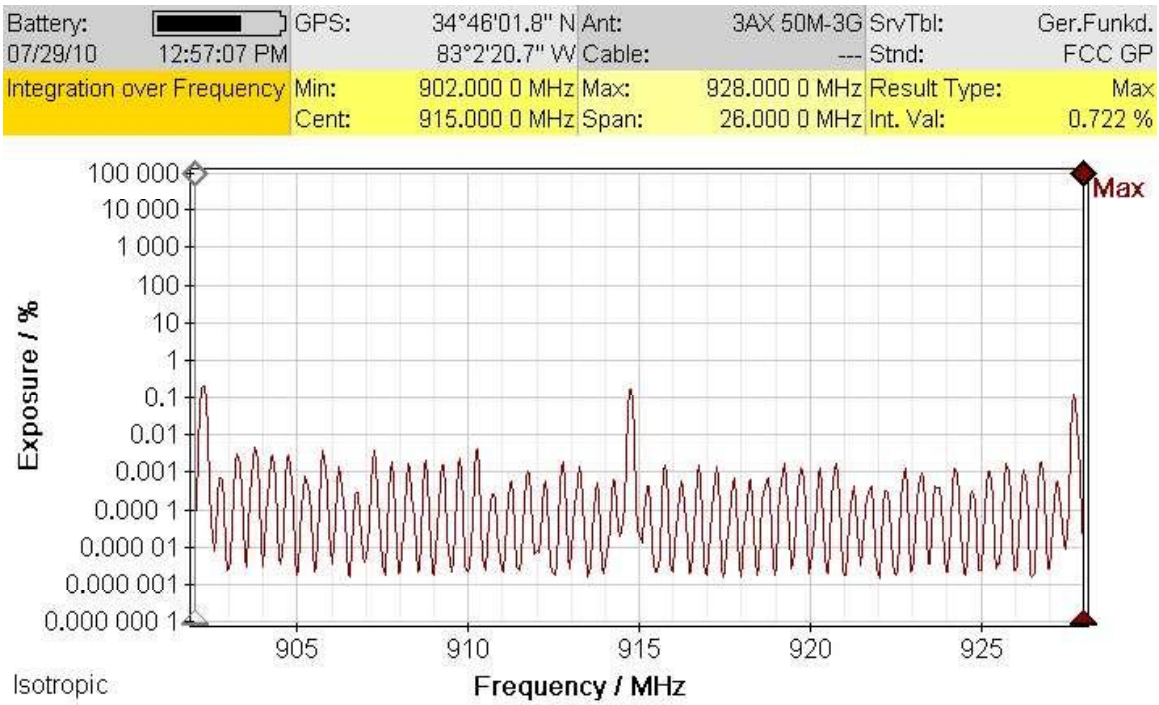

Figure B-7
900 MHz band composite RF field from rack of 10 SmartMeters at 7 feet.

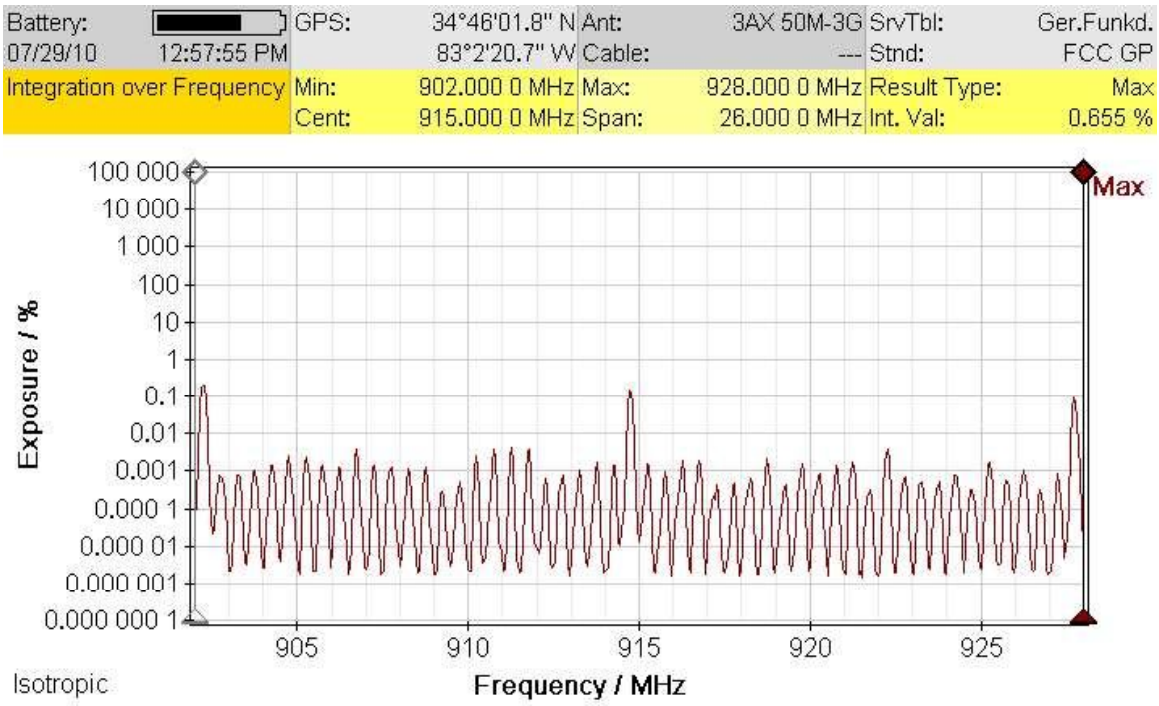

Figure B-8
900 MHz band composite RF field from rack of 10 SmartMeters at 8 feet.

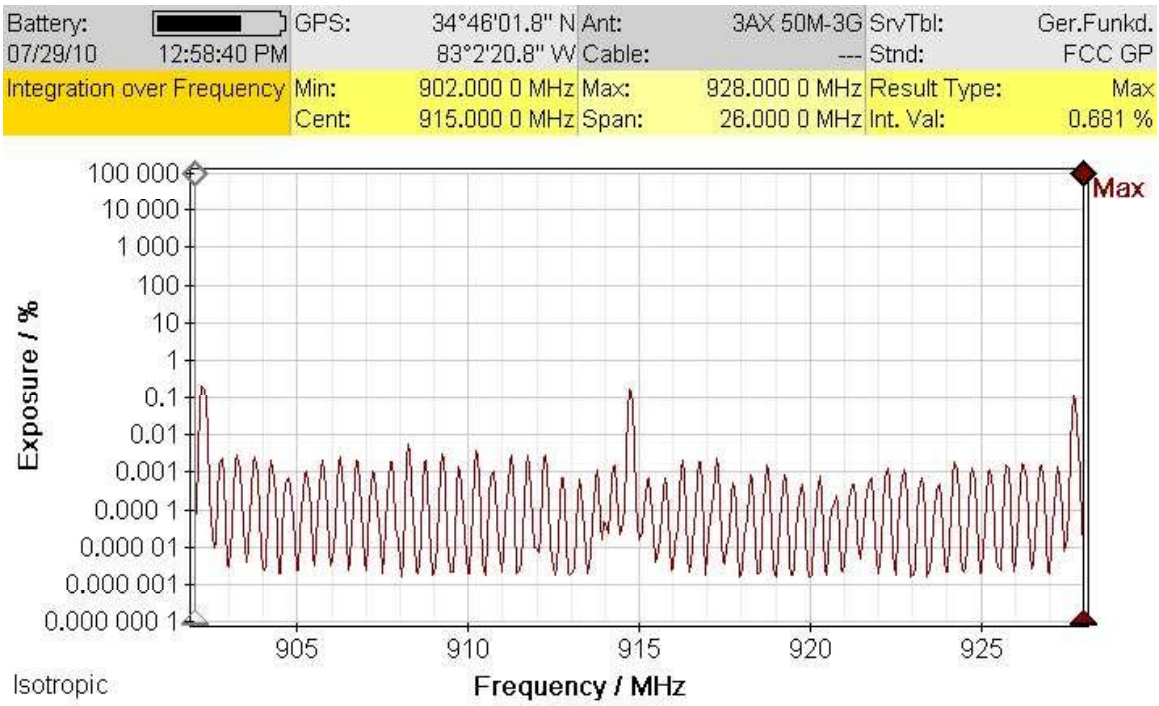

Figure B-9
900 MHz band composite RF field from rack of 10 SmartMeters at 9 feet.

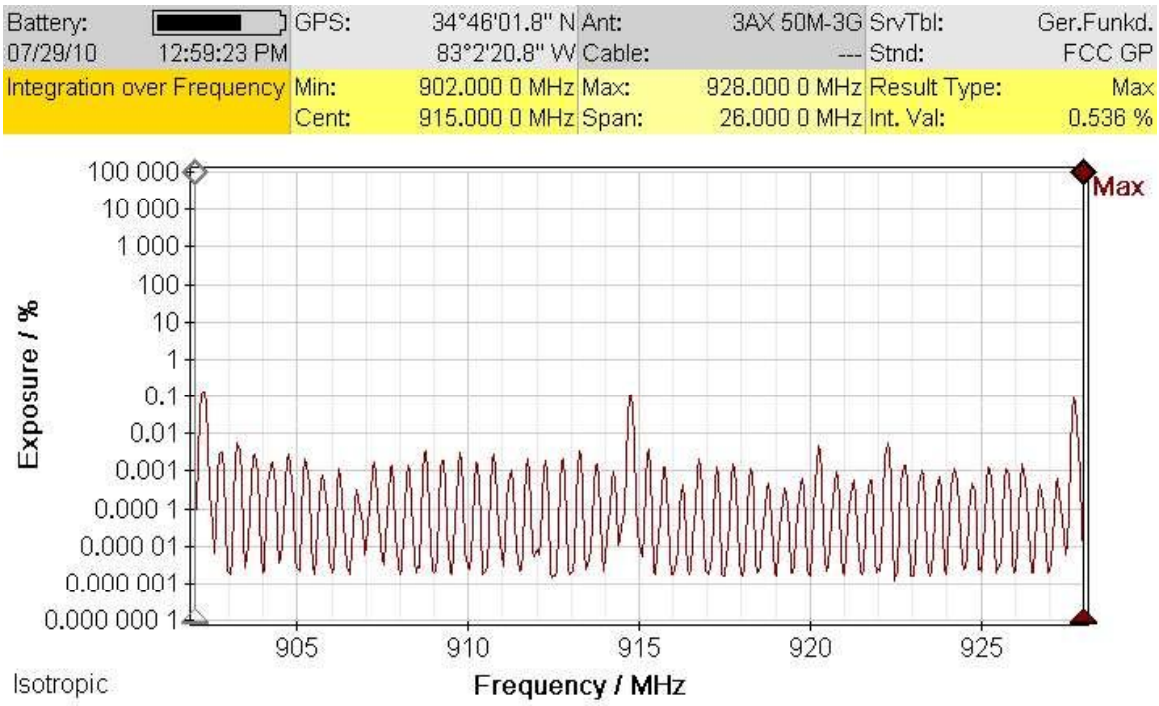

Figure B-10
900 MHz band composite RF field from rack of 10 SmartMeters at 10 feet.

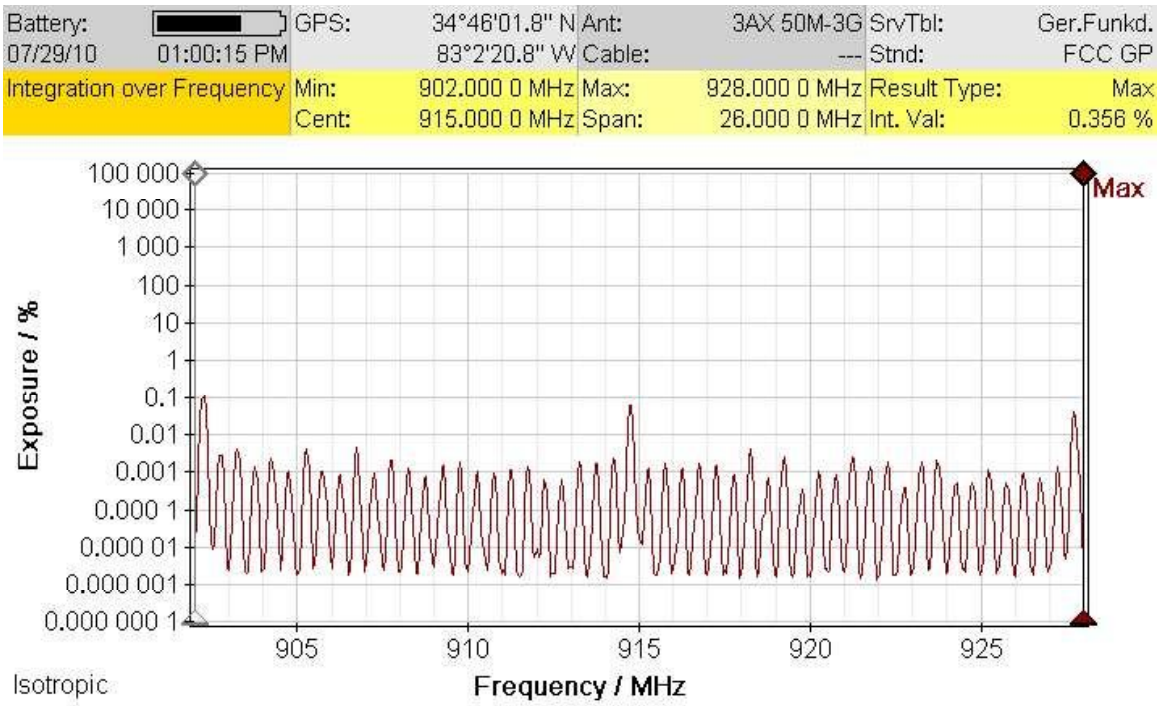

Figure B-11
900 MHz band composite RF field from rack of 10 SmartMeters at 15 feet.

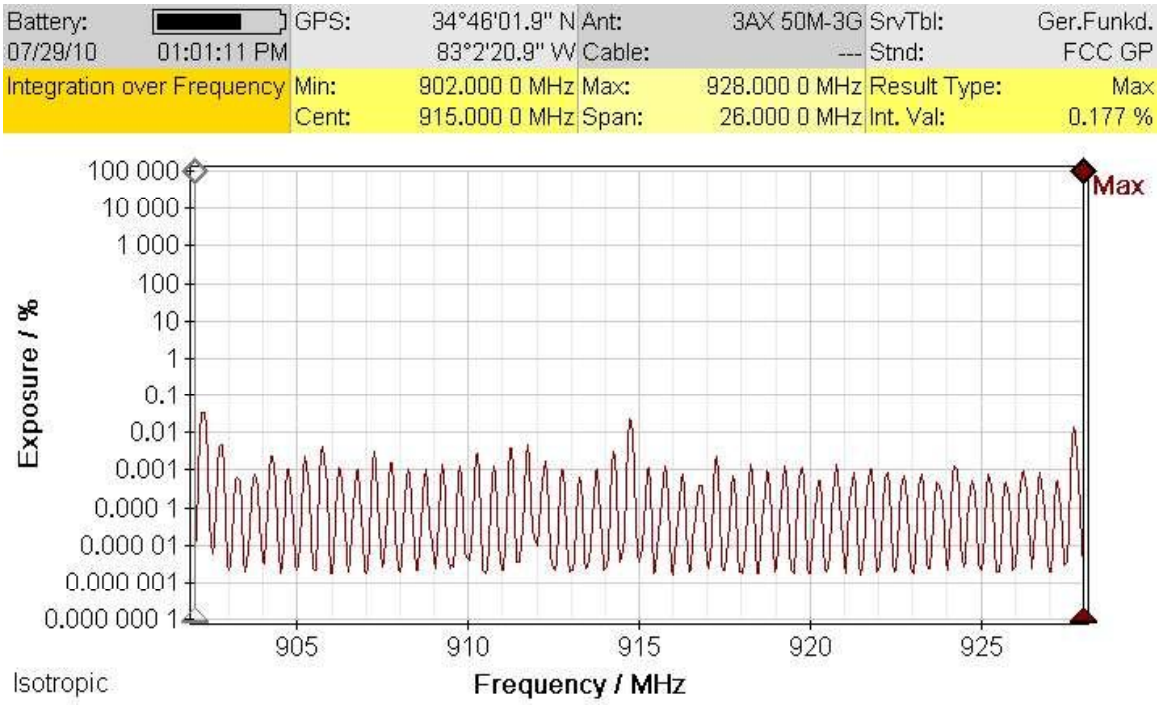

Figure B-12
900 MHz band composite RF field from rack of 10 SmartMeters at 20 feet.

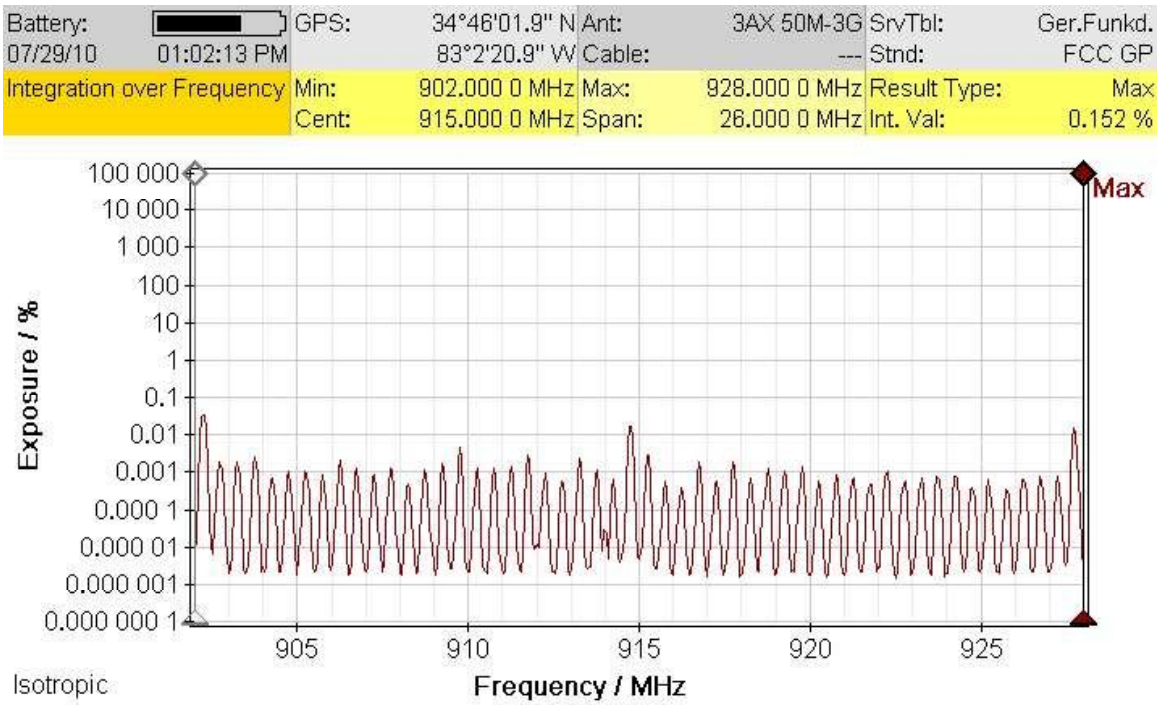

Figure B-13
900 MHz band composite RF field from rack of 10 SmartMeters at 25 feet.

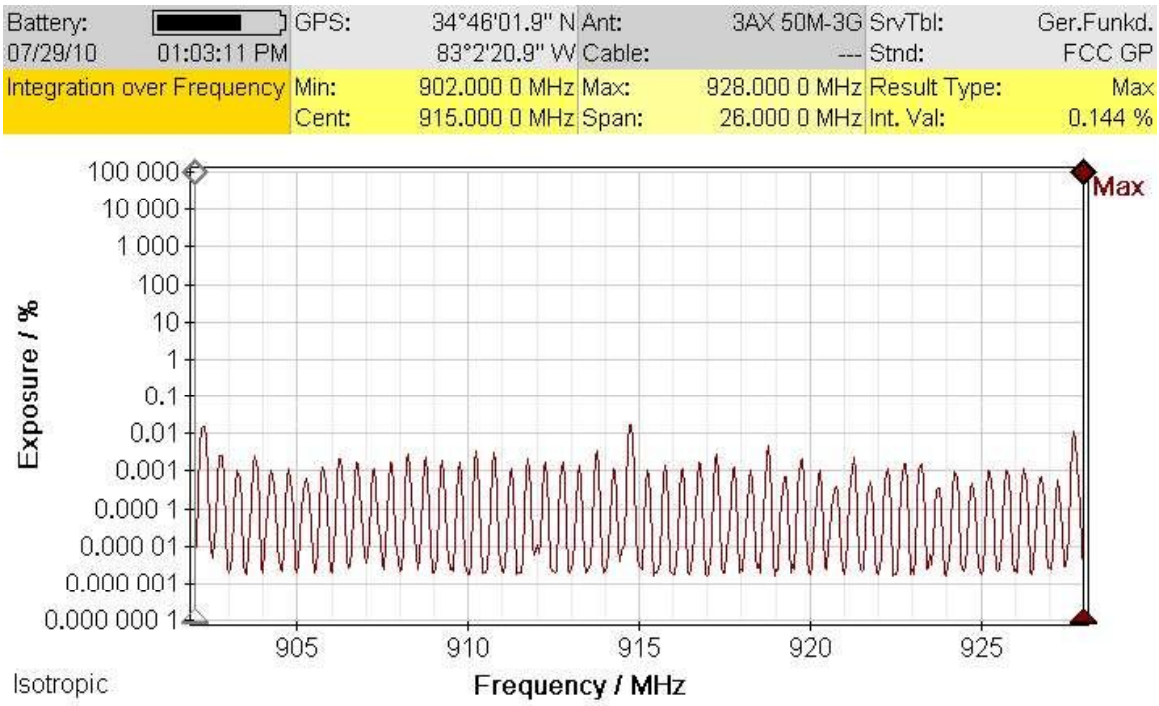

Figure B-14
900 MHz band composite RF field from rack of 10 SmartMeters at 30 feet.

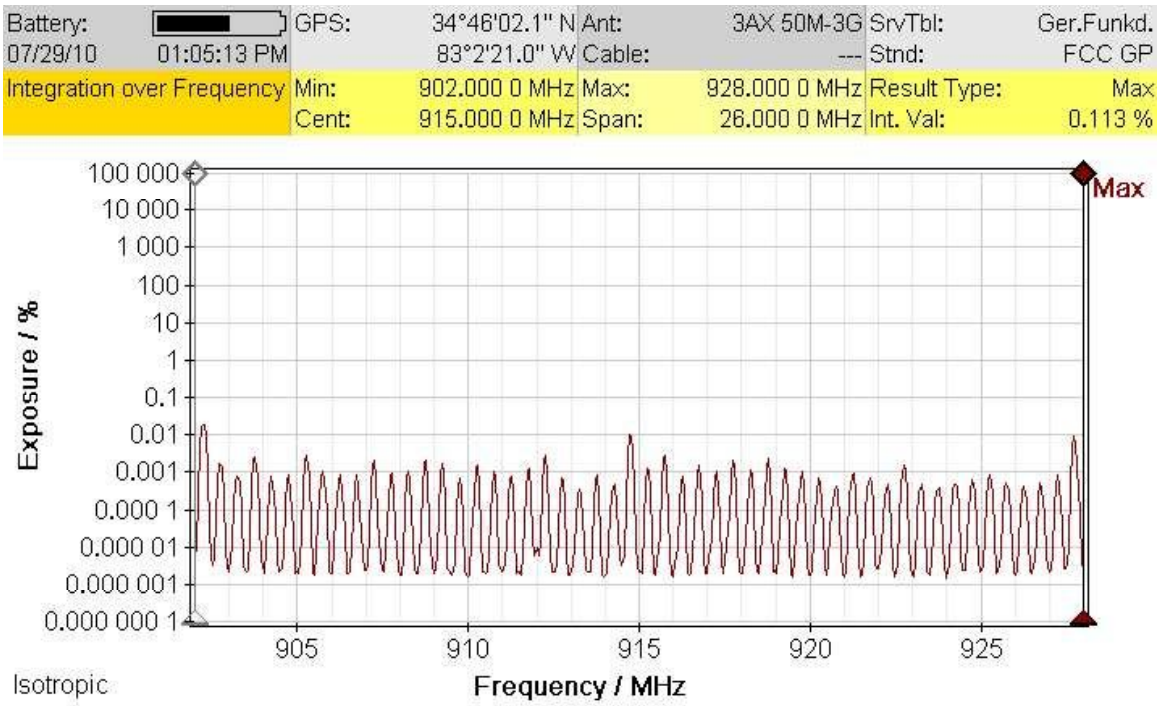

Figure B-15
900 MHz band composite RF field from rack of 10 SmartMeters at 40 feet.

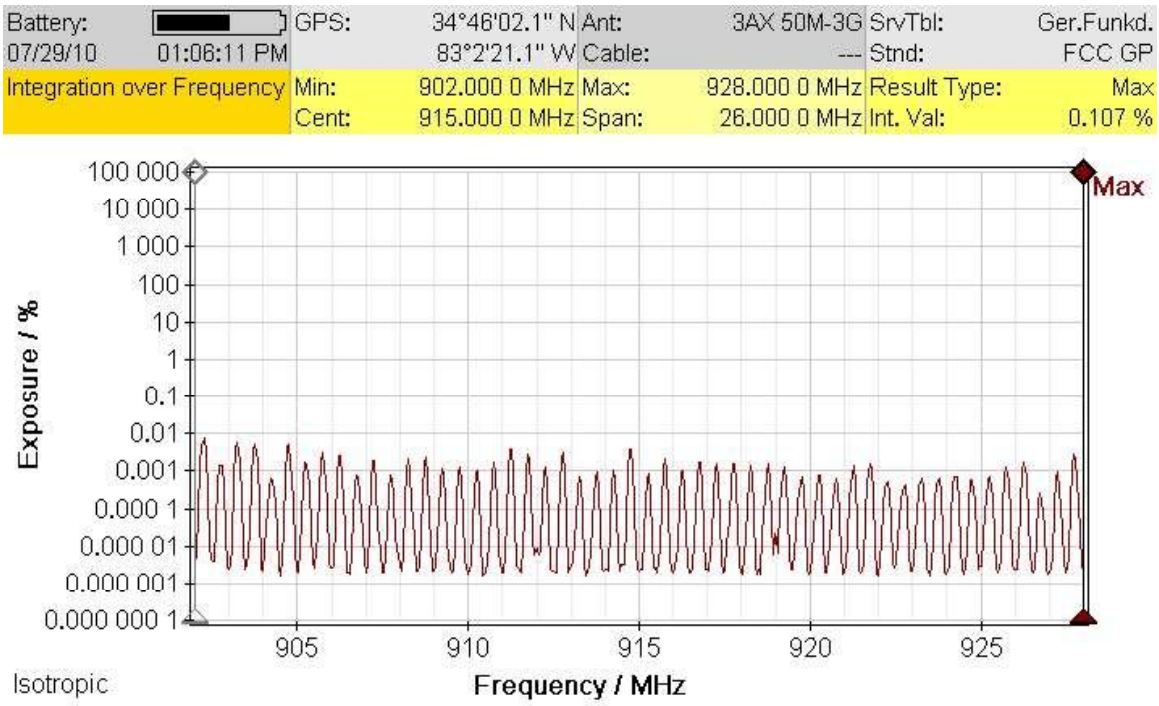

Figure B-16
900 MHz band composite RF field from rack of 10 SmartMeters at 50 feet.

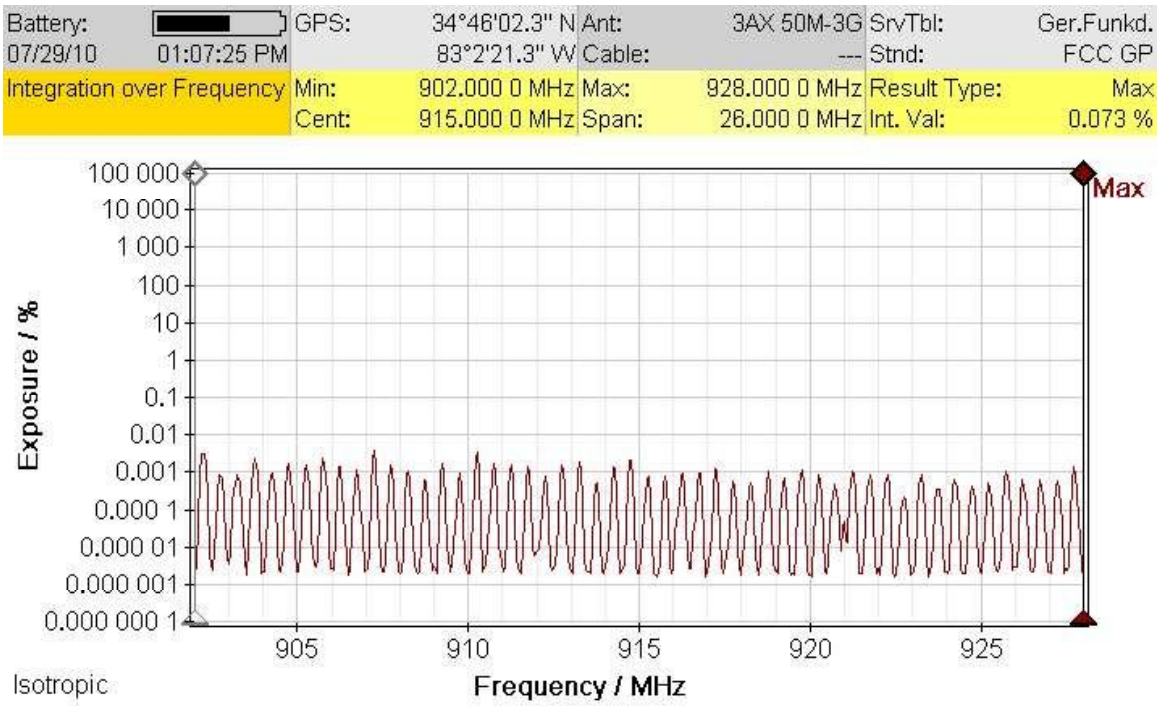

Figure B-17
900 MHz band composite RF field from rack of 10 SmartMeters at 75 feet.

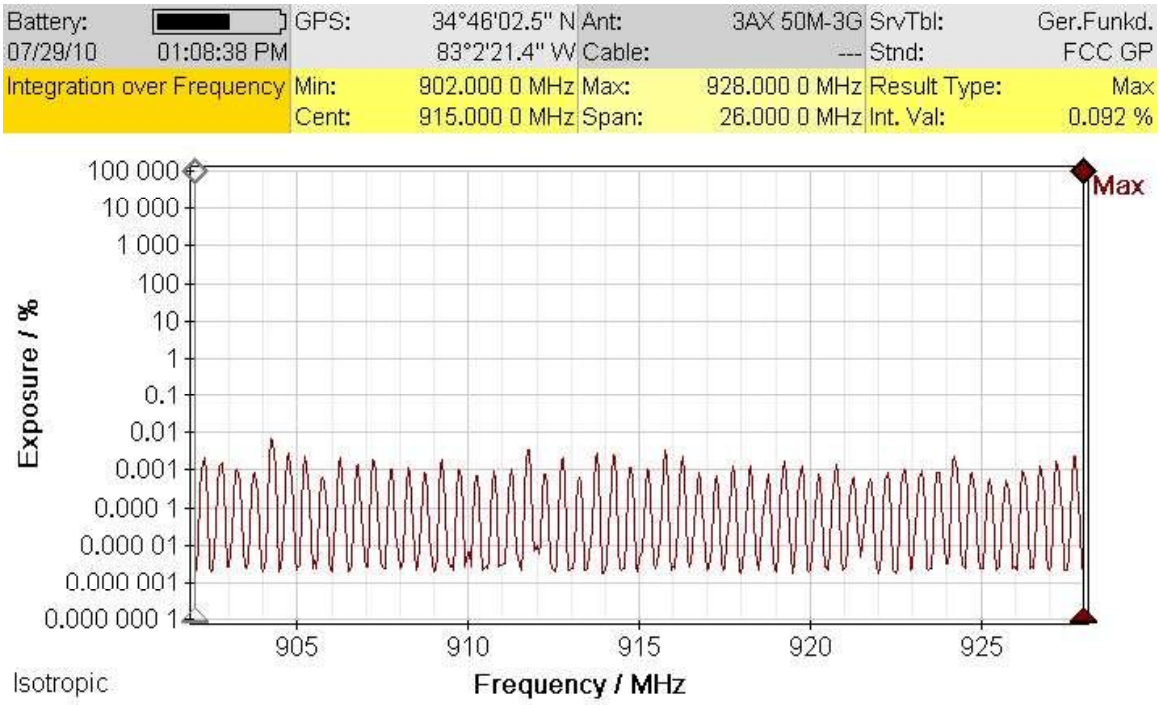

Figure B-18
900 MHz band composite RF field from rack of 10 SmartMeters at 100 feet.

Appendix C: SRM-3006 900 MHz Spectrum Measurements Scans (rear of meters)

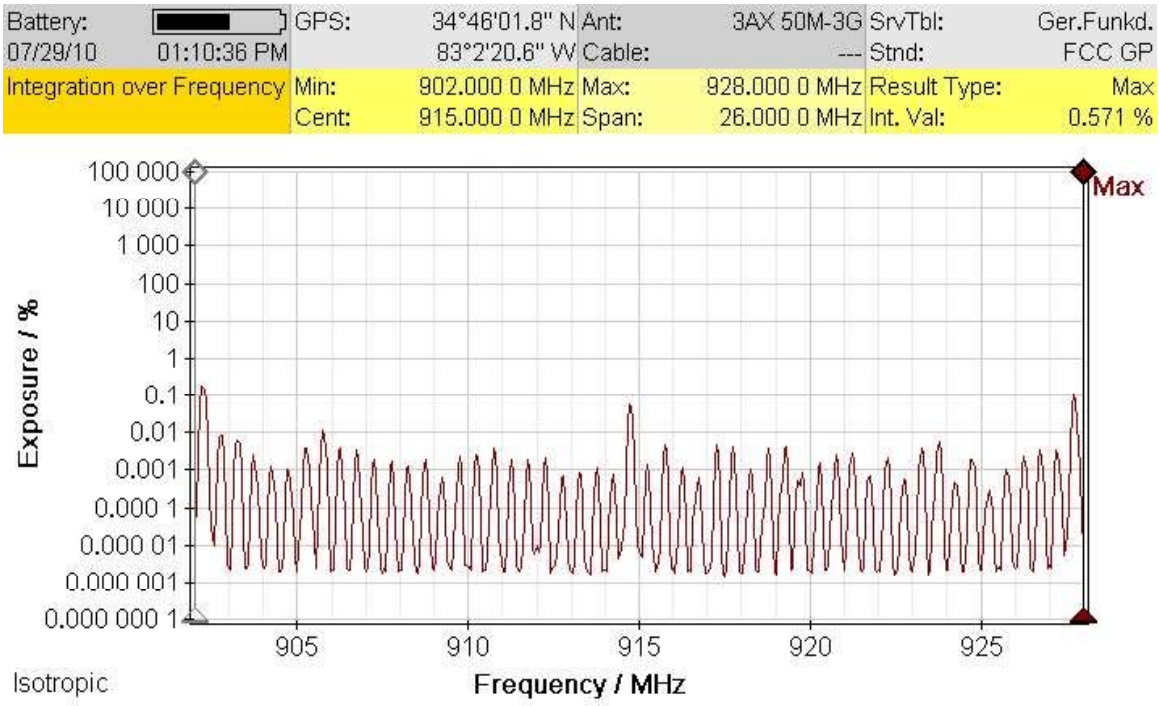

Figure C-1
900 MHz band composite RF field from rack of 10 SmartMeters at 20 cm behind meters.

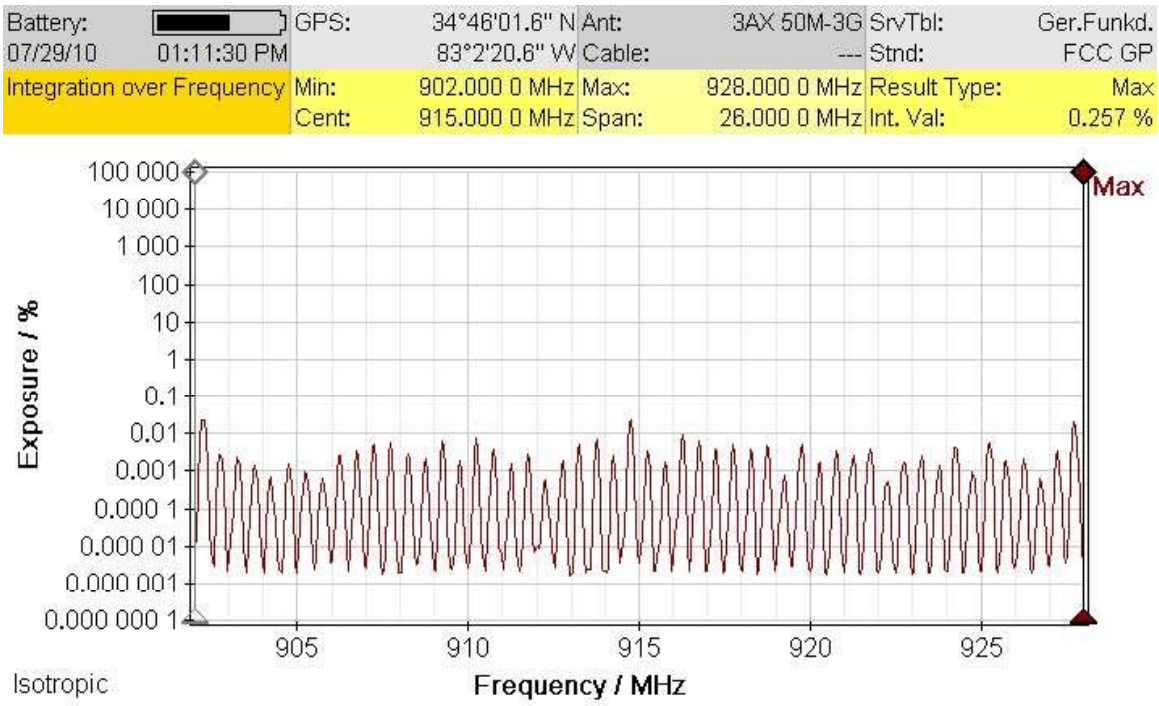

Figure C-2
900 MHz band composite RF field from rack of 10 SmartMeters at 5 feet behind meters.

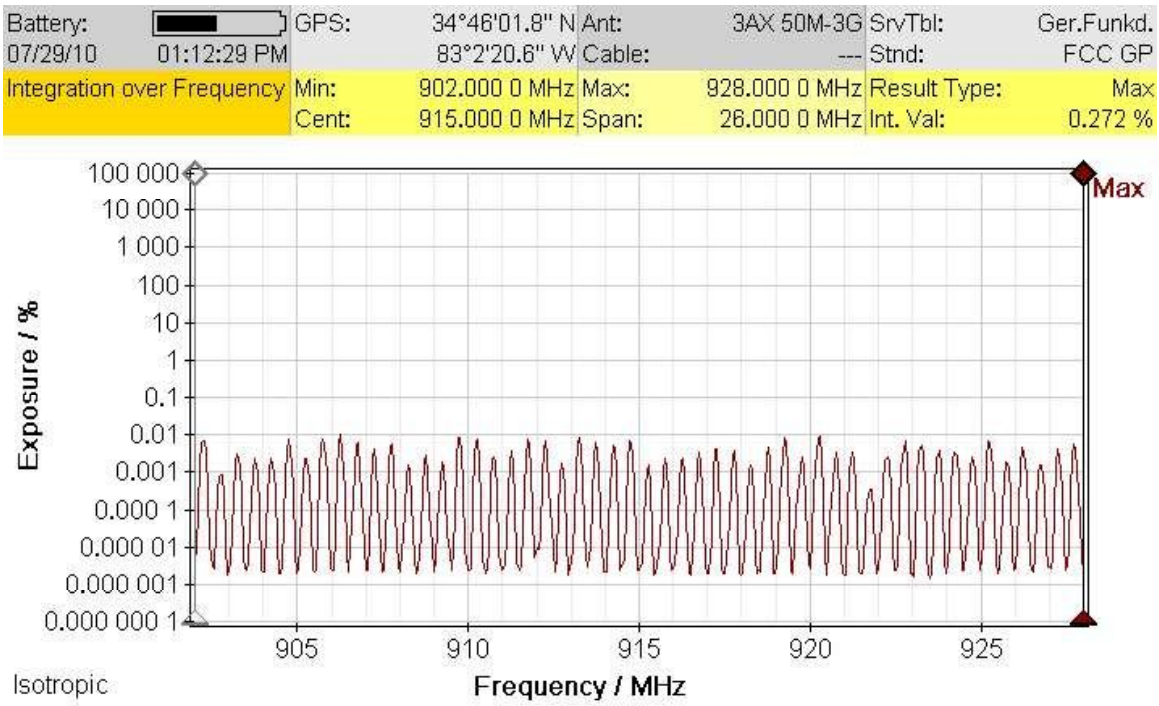

Figure C-3
900 MHz band composite RF field from rack of 10 SmartMeters at 10 feet behind meters.

Appendix D: SRM-3006 2.4 GHz Spectrum Measurement Scans (meter farm)

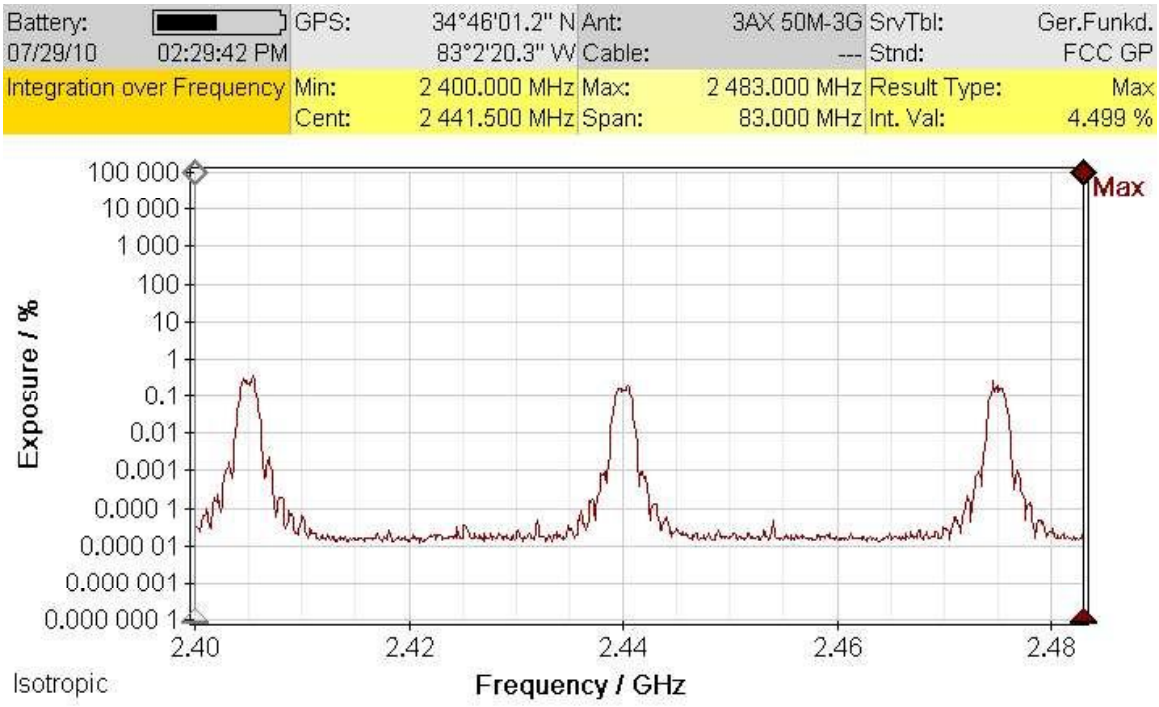

Figure D-1
2.4 GHz band composite RF field from rack of 10 SmartMeters at 1 foot.

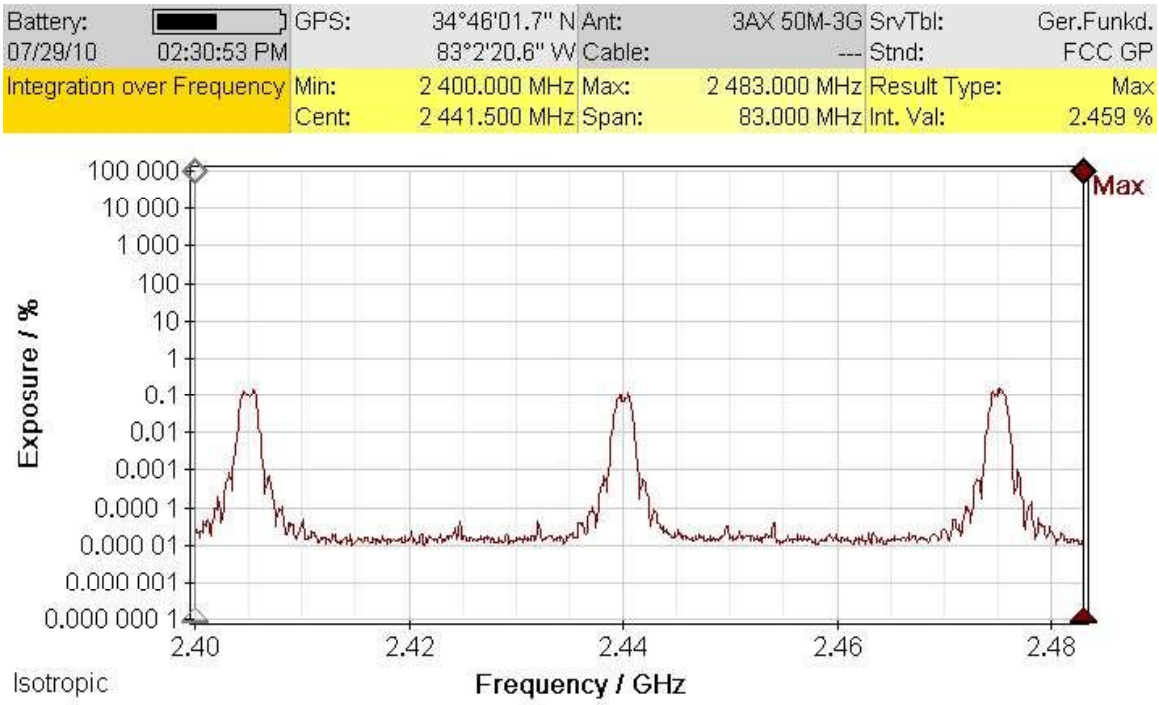

Figure D-2
2.4 GHz band composite RF field from rack of 10 SmartMeters at 2 feet.

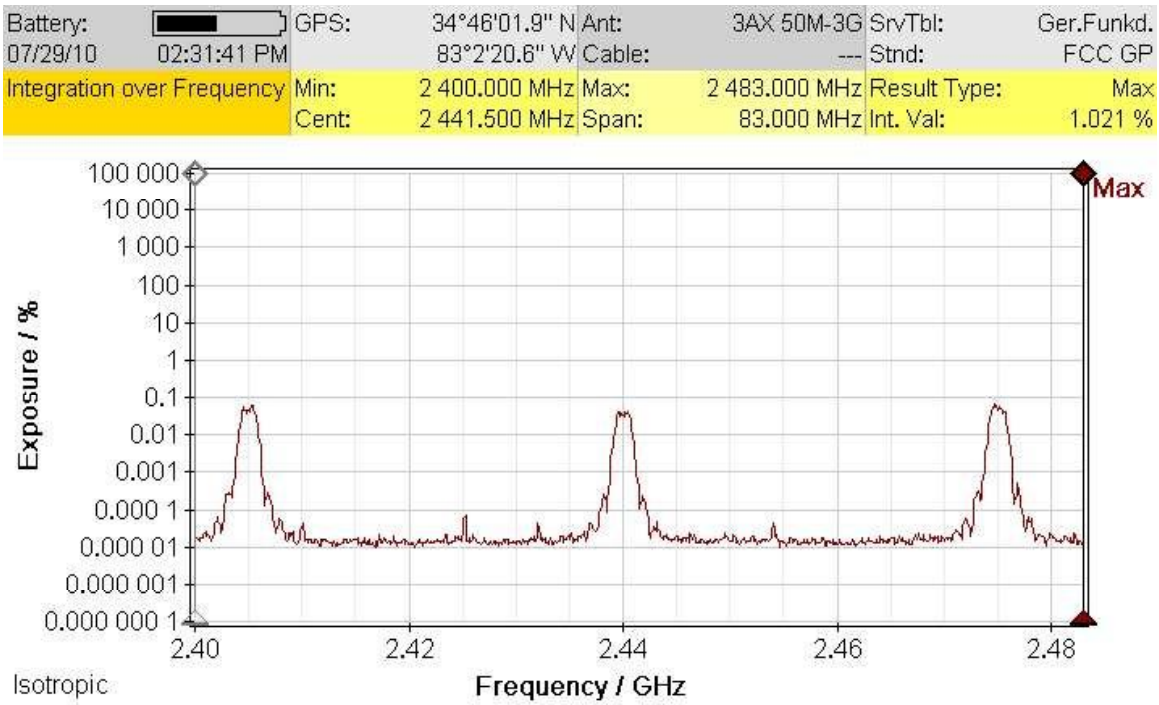

Figure D-3
2.4 GHz band composite RF field from rack of 10 SmartMeters at 3 feet.

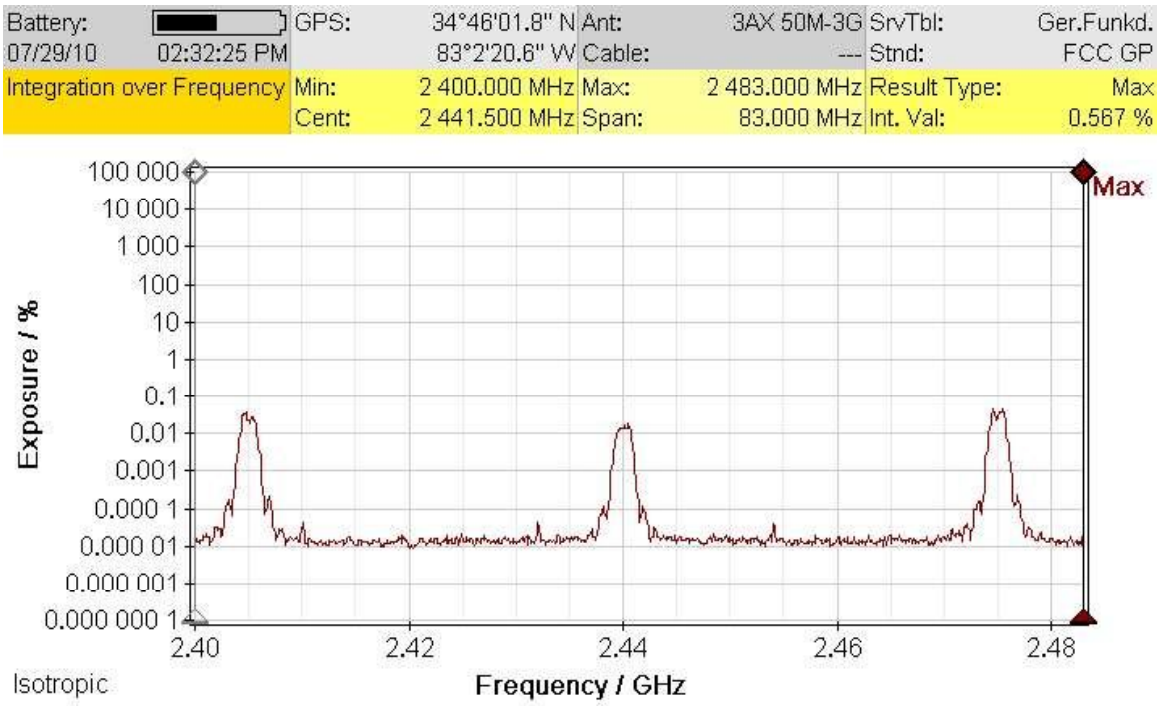

Figure D-4
2.4 GHz band composite RF field from rack of 10 SmartMeters at 4 feet.

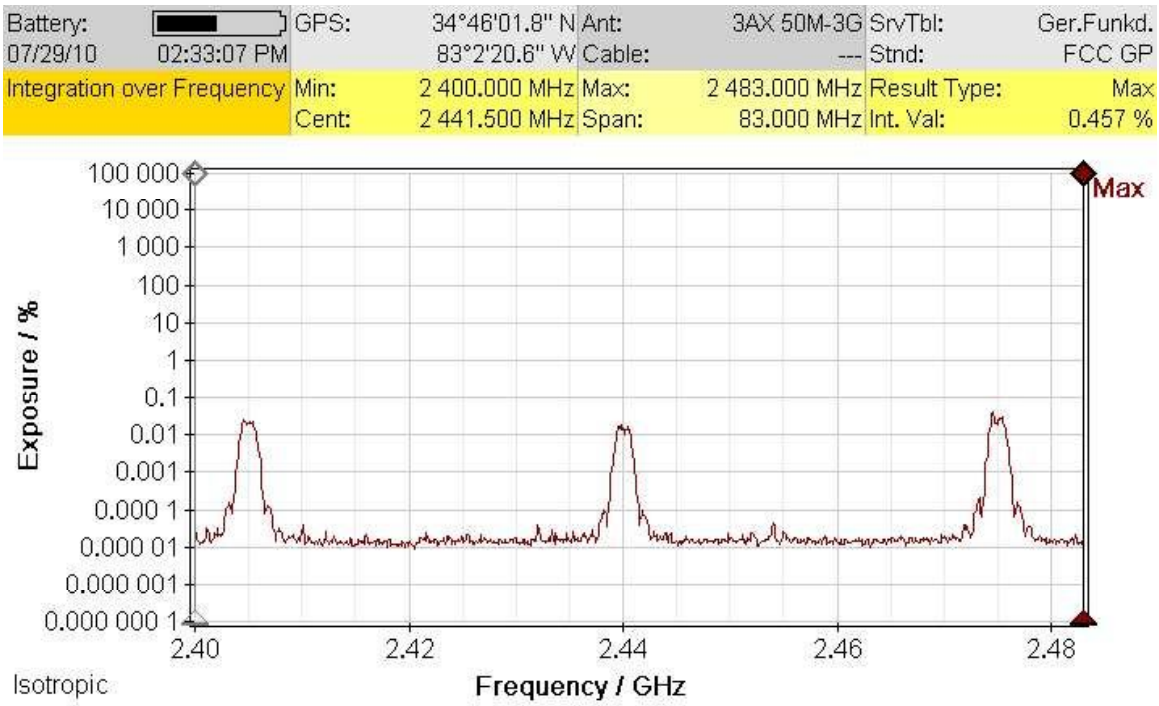

Figure D-5
2.4 GHz band composite RF field from rack of 10 SmartMeters at 5 feet.

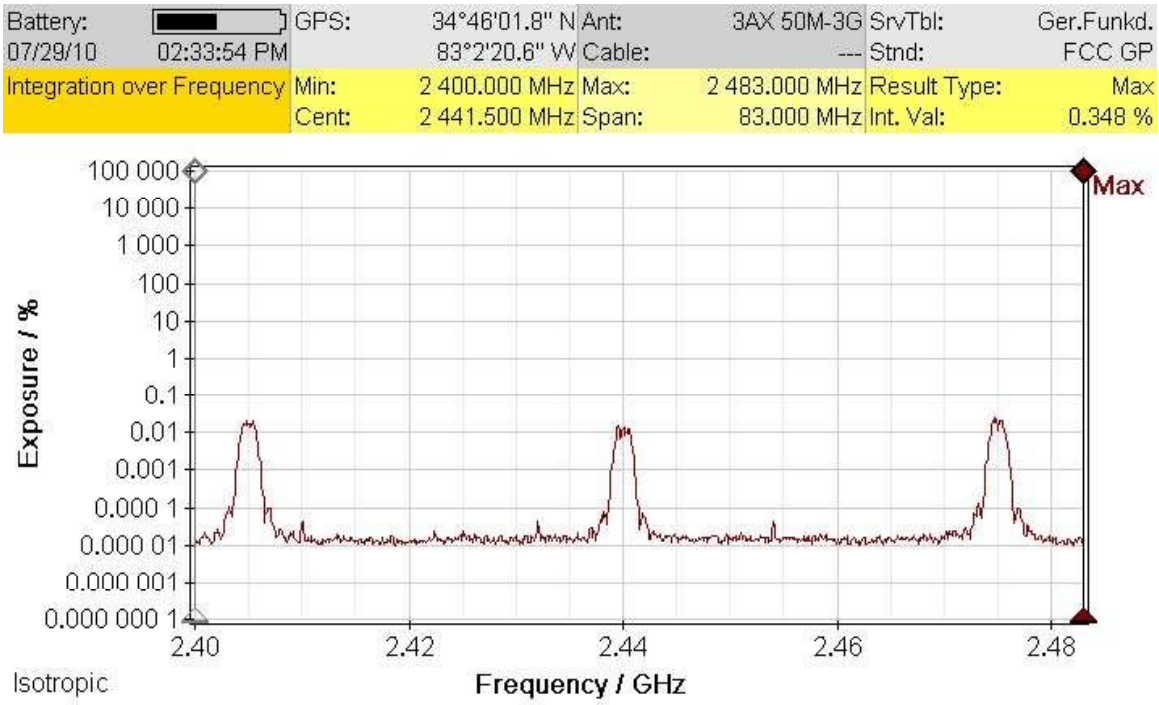

Figure D-6
2.4 GHz band composite RF field from rack of 10 SmartMeters at 6 feet.

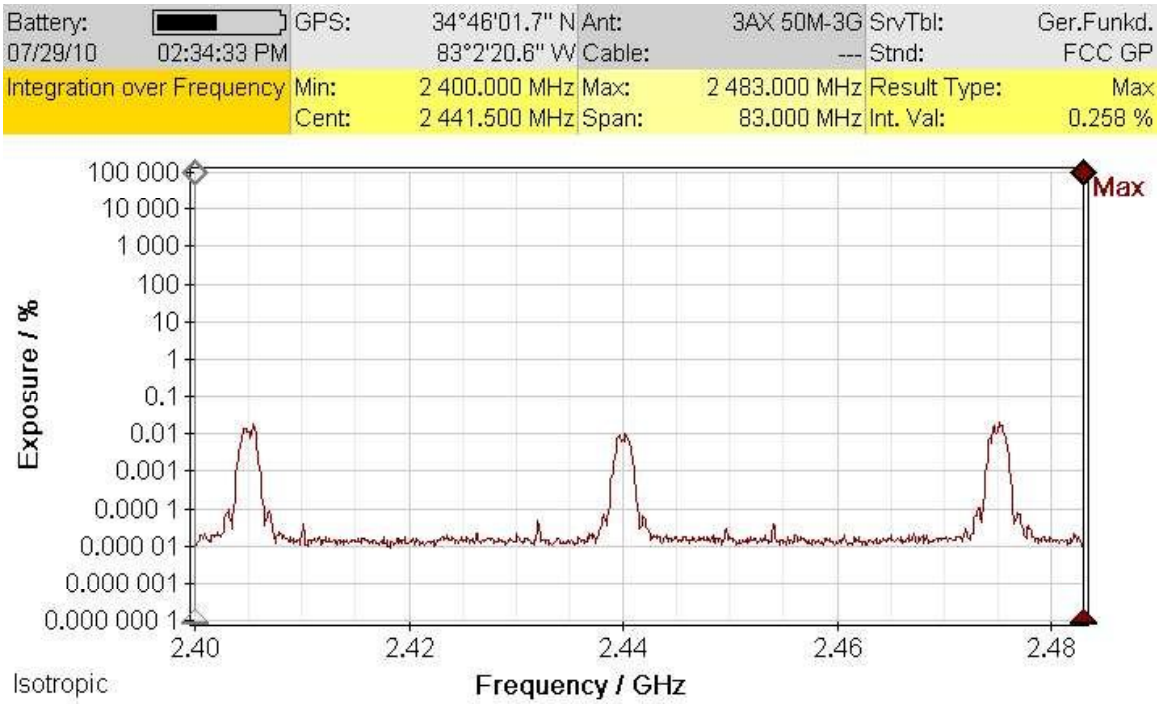

Figure D-7
2.4 GHz band composite RF field from rack of 10 SmartMeters at 7 feet.

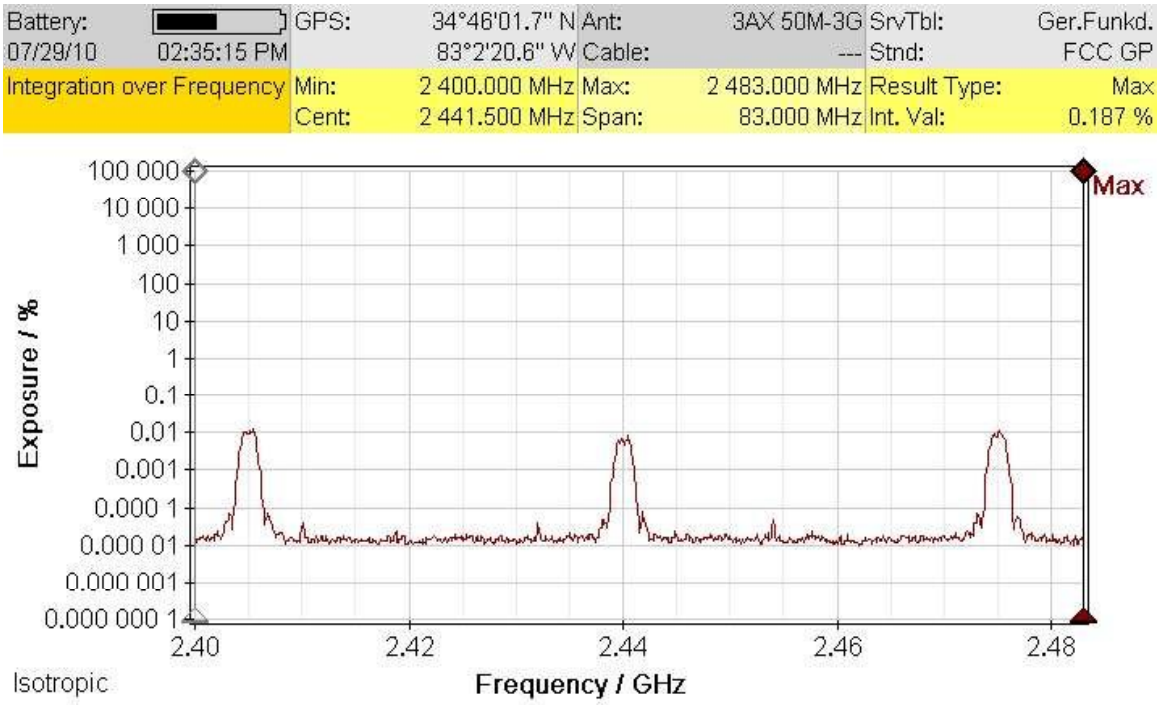

Figure D-8
2.4 GHz band composite RF field from rack of 10 SmartMeters at 8 feet.

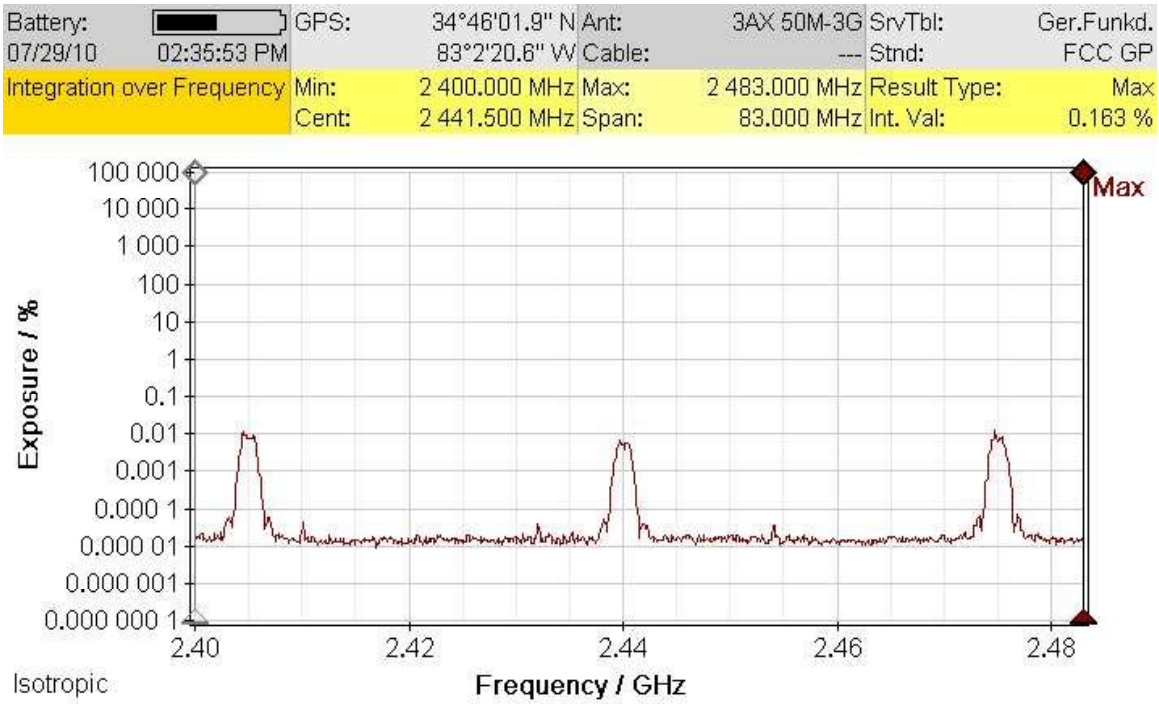

Figure D-9
2.4 GHz band composite RF field from rack of 10 SmartMeters at 9 feet.

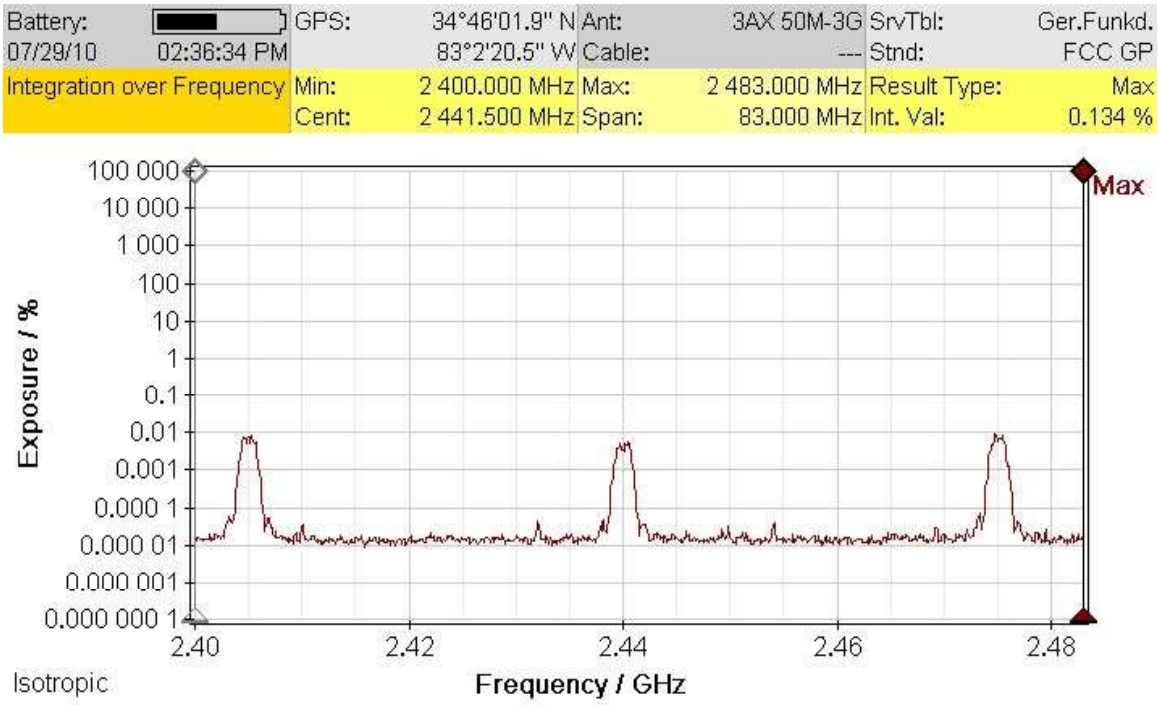

Figure D-10
2.4 GHz band composite RF field from rack of 10 SmartMeters at 10 feet.

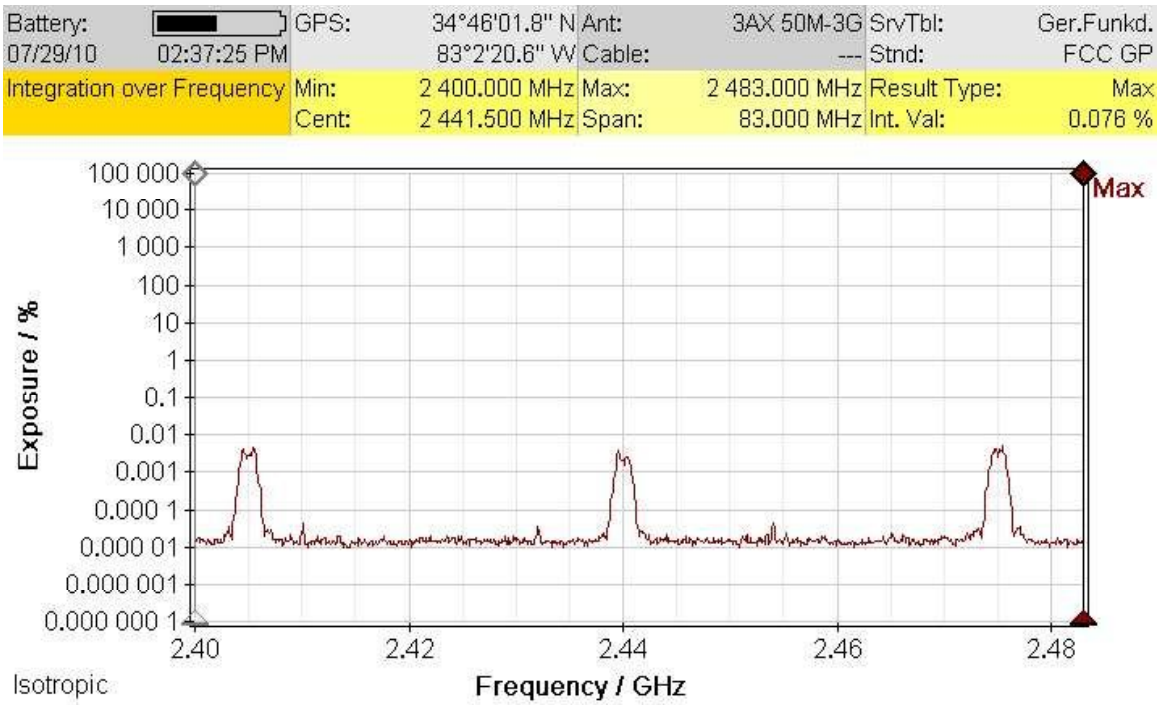

Figure D-11
2.4 GHz band composite RF field from rack of 10 SmartMeters at 15 feet.

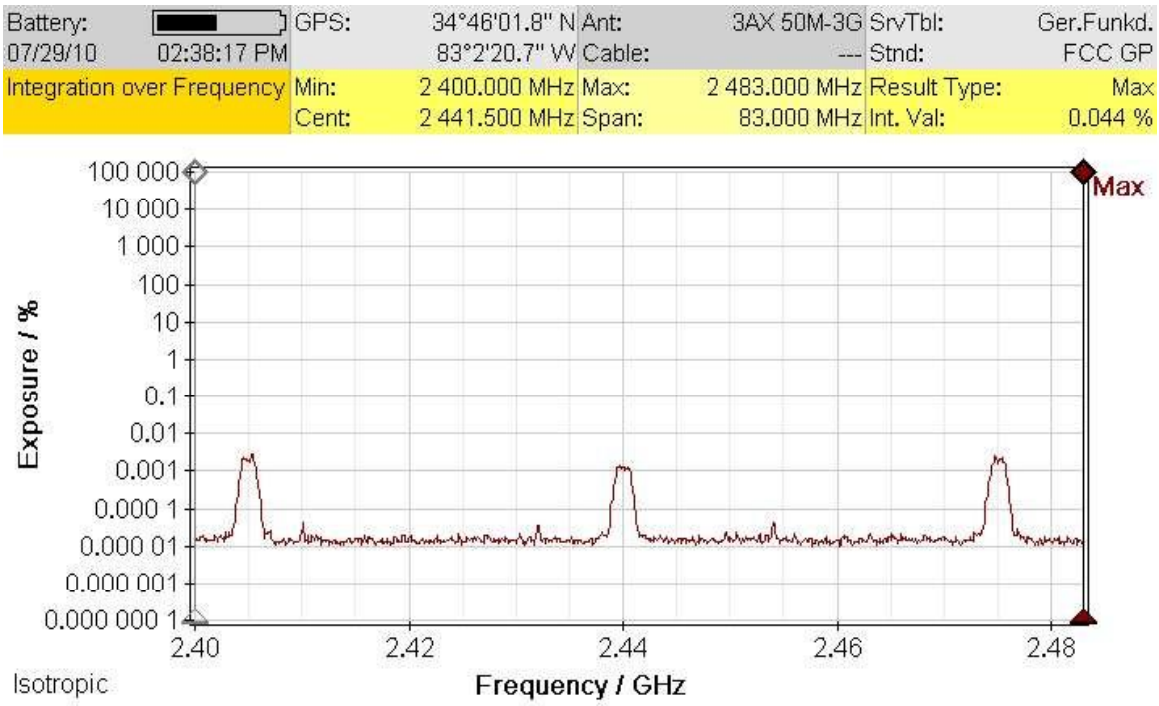

Figure D-12
2.4 GHz band composite RF field from rack of 10 SmartMeters at 20 feet.

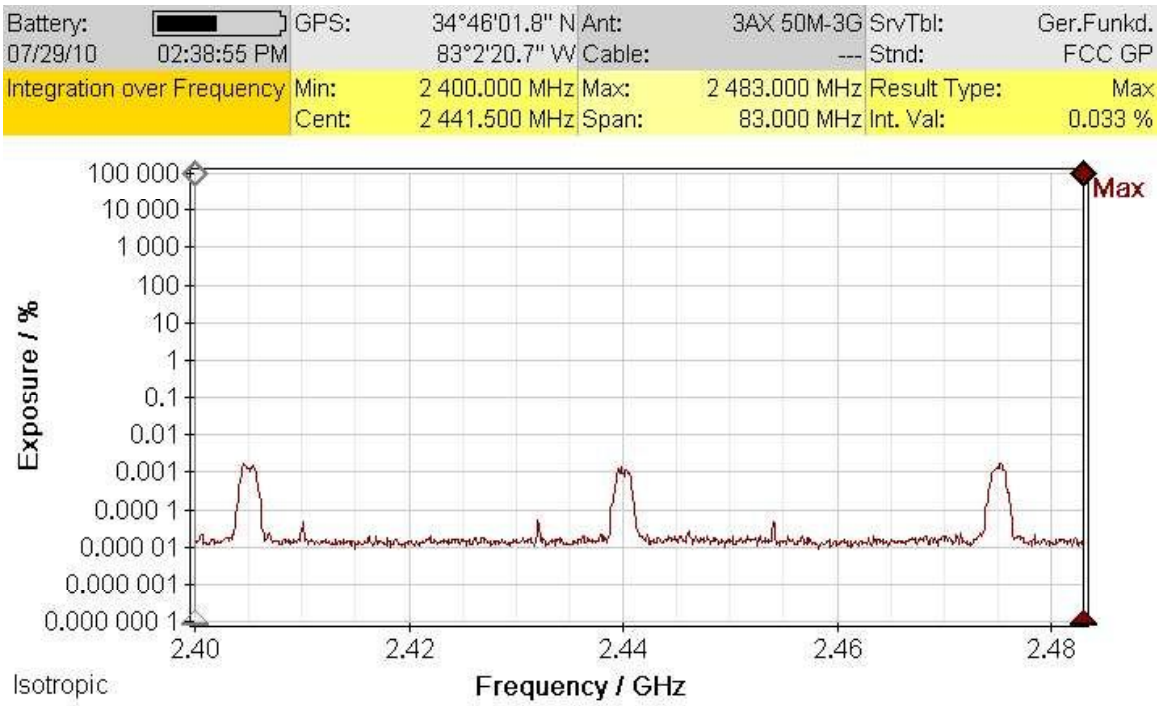

Figure D-13
2.4 GHz band composite RF field from rack of 10 SmartMeters at 25 feet.

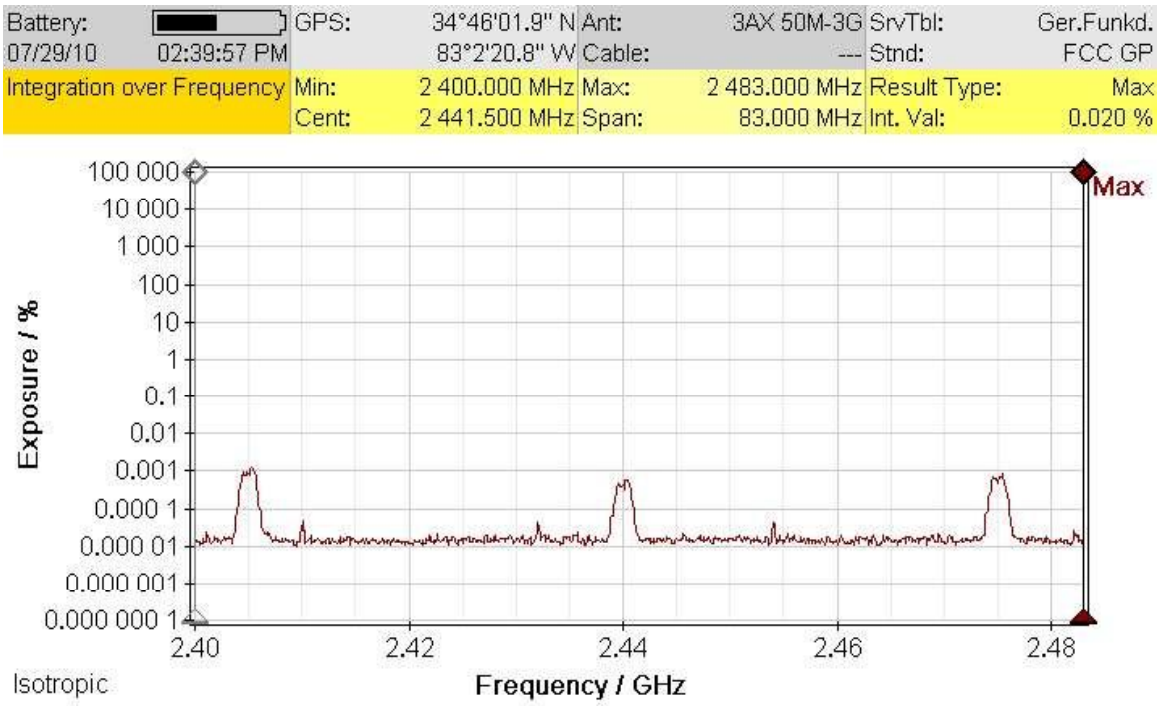

Figure D-14
2.4 GHz band composite RF field from rack of 10 SmartMeters at 30 feet.

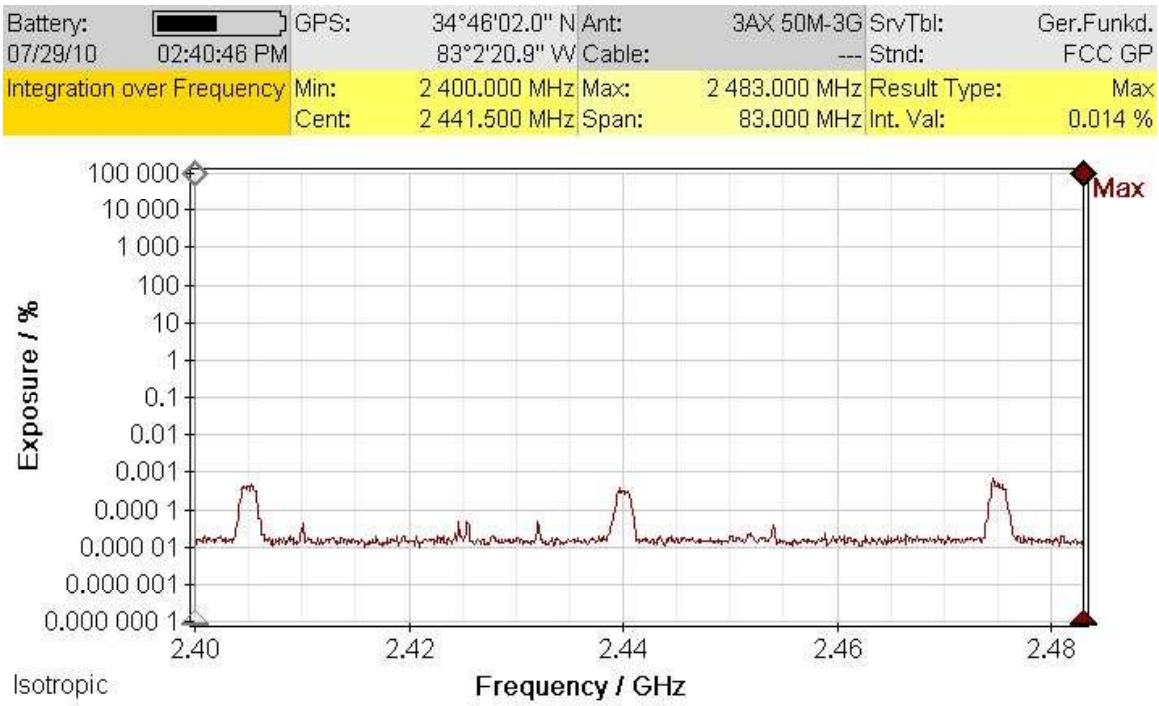

Figure D-15
2.4 GHz band composite RF field from rack of 10 SmartMeters at 40 feet.

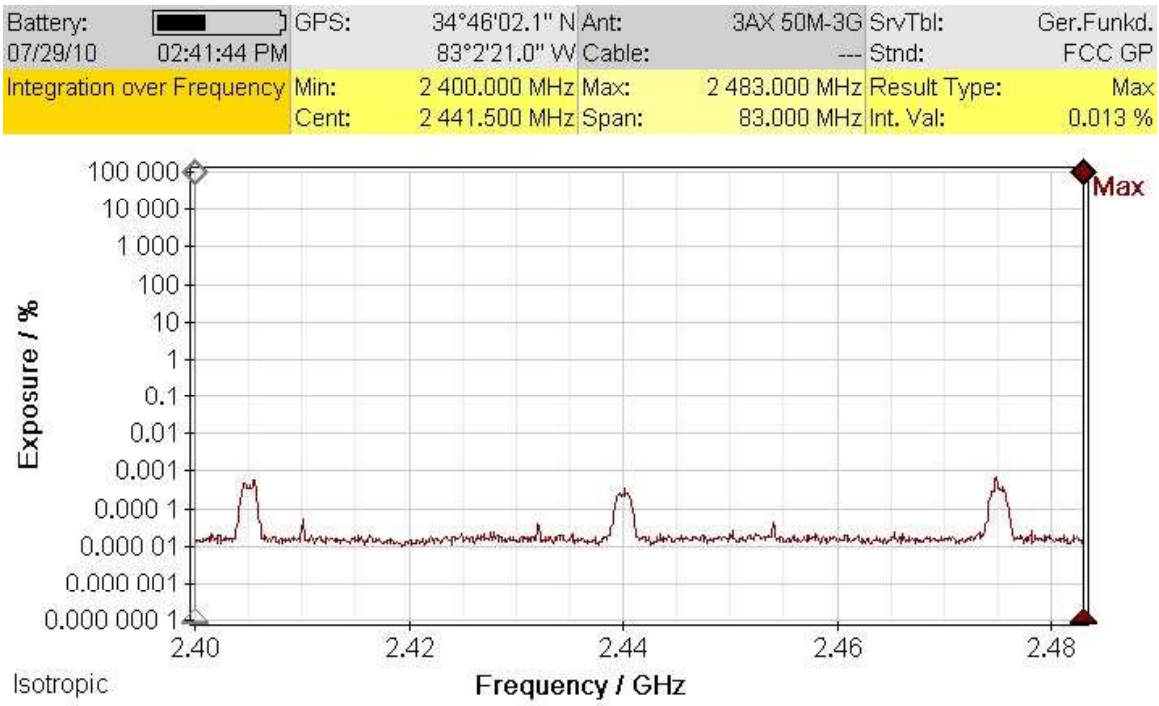

Figure D-16
2.4 GHz band composite RF field from rack of 10 SmartMeters at 50 feet.

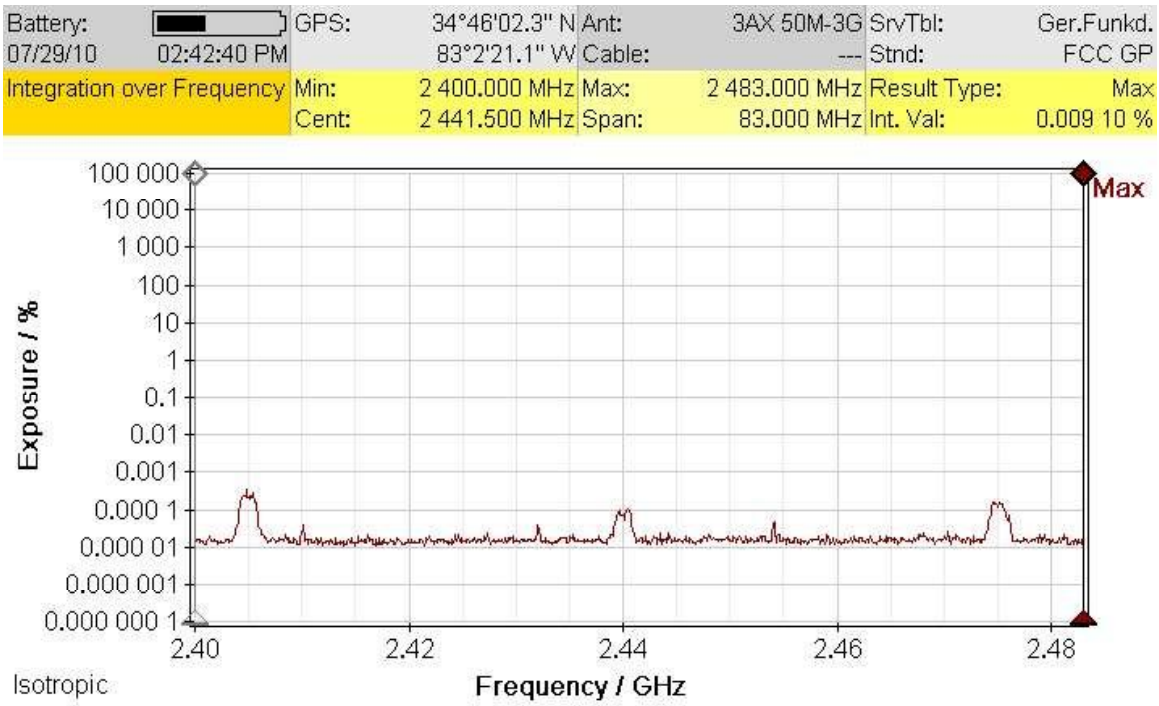

Figure D-17
2.4 GHz band composite RF field from rack of 10 SmartMeters at 75 feet.

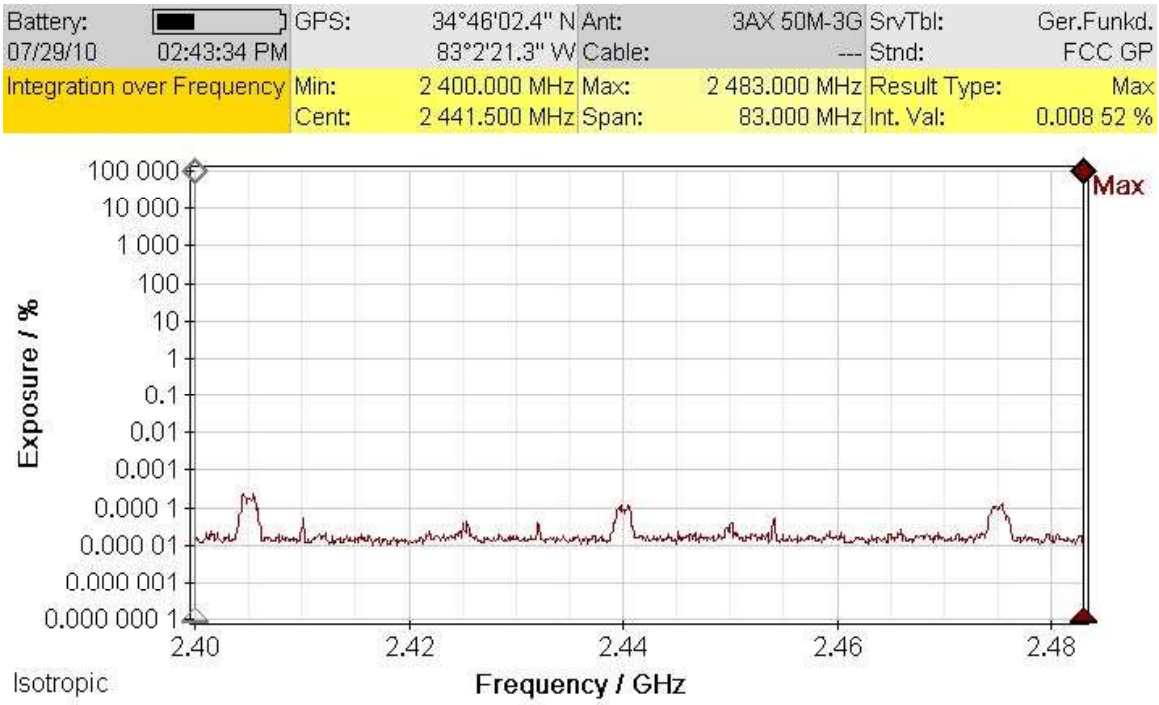

Figure D-18
2.4 GHz band composite RF field from rack of 10 SmartMeters at 100 feet.

Appendix E: SRM-3006 2.4 GHz Spectrum Measurement Scans (rear of meters)

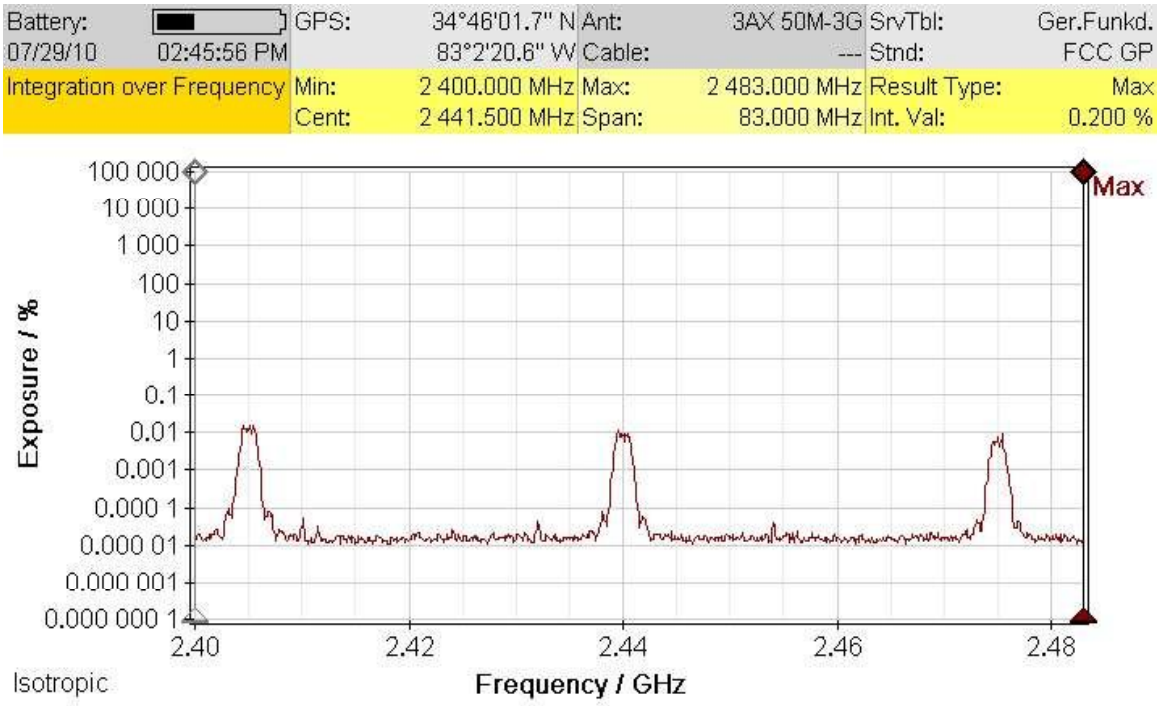

Figure E-1
2.4 GHz band composite RF field at 20 cm behind rack of 10 SmartMeters.

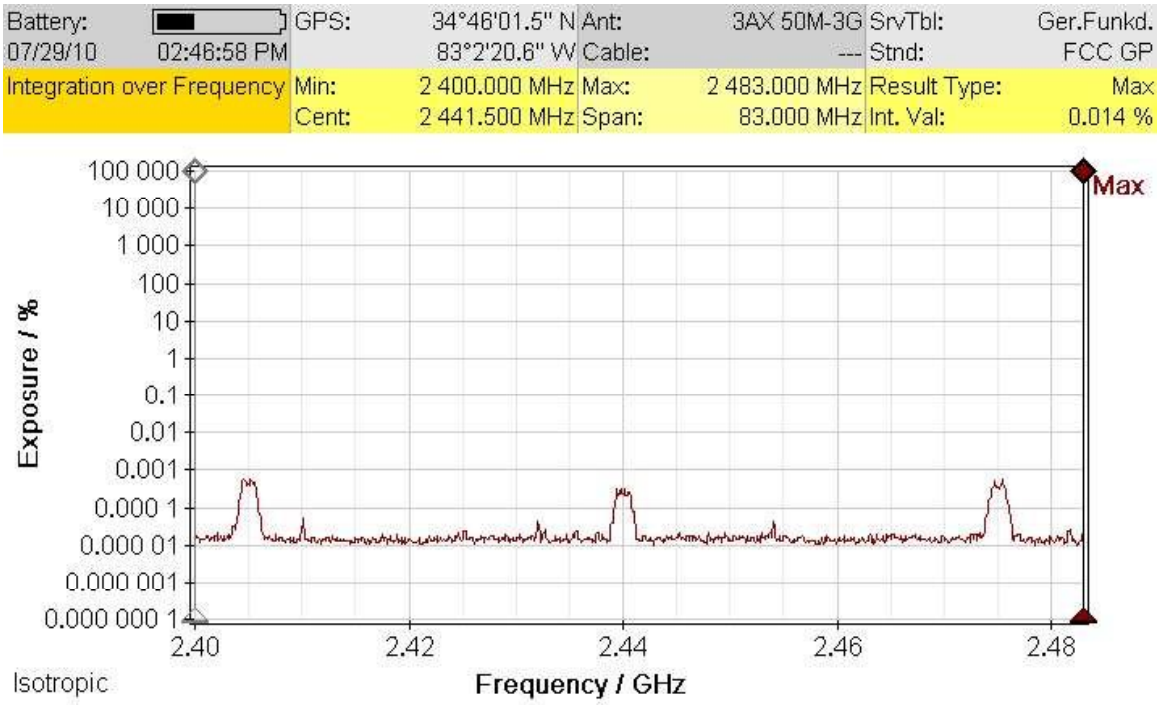

Figure E-2
2.4 GHz band composite RF field at 5 feet behind rack of 10 SmartMeters.

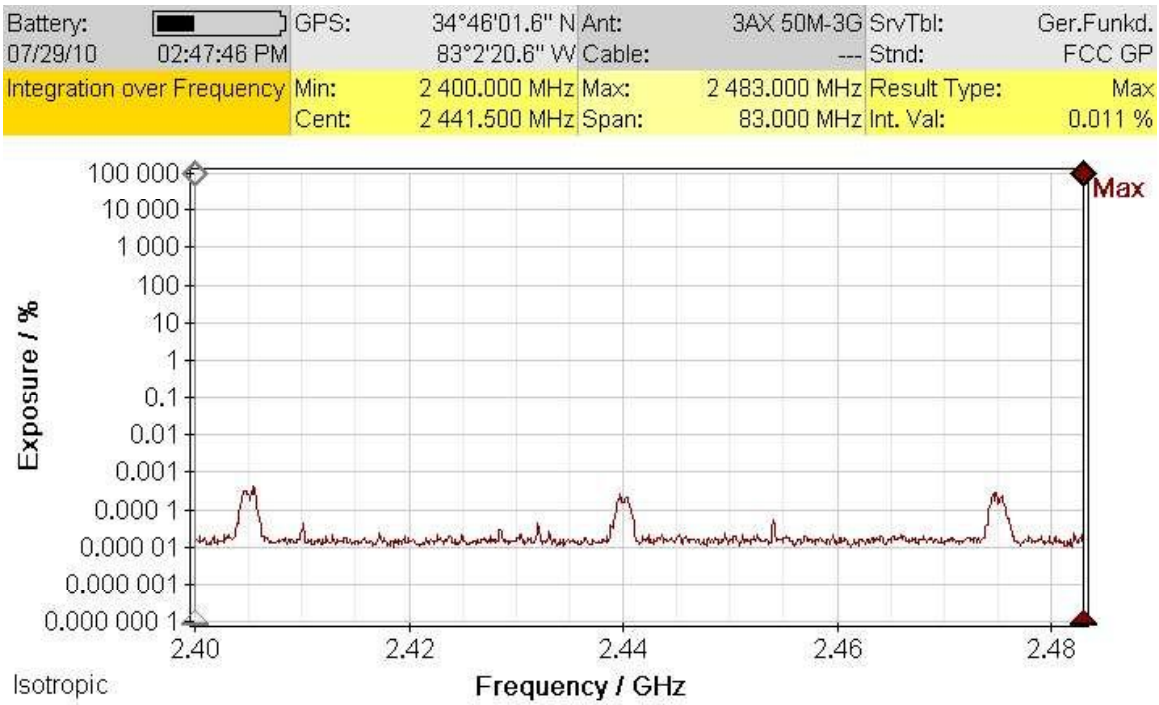

Figure E-3
2.4 GHz band composite RF field at 10 feet behind rack of 10 SmartMeters.

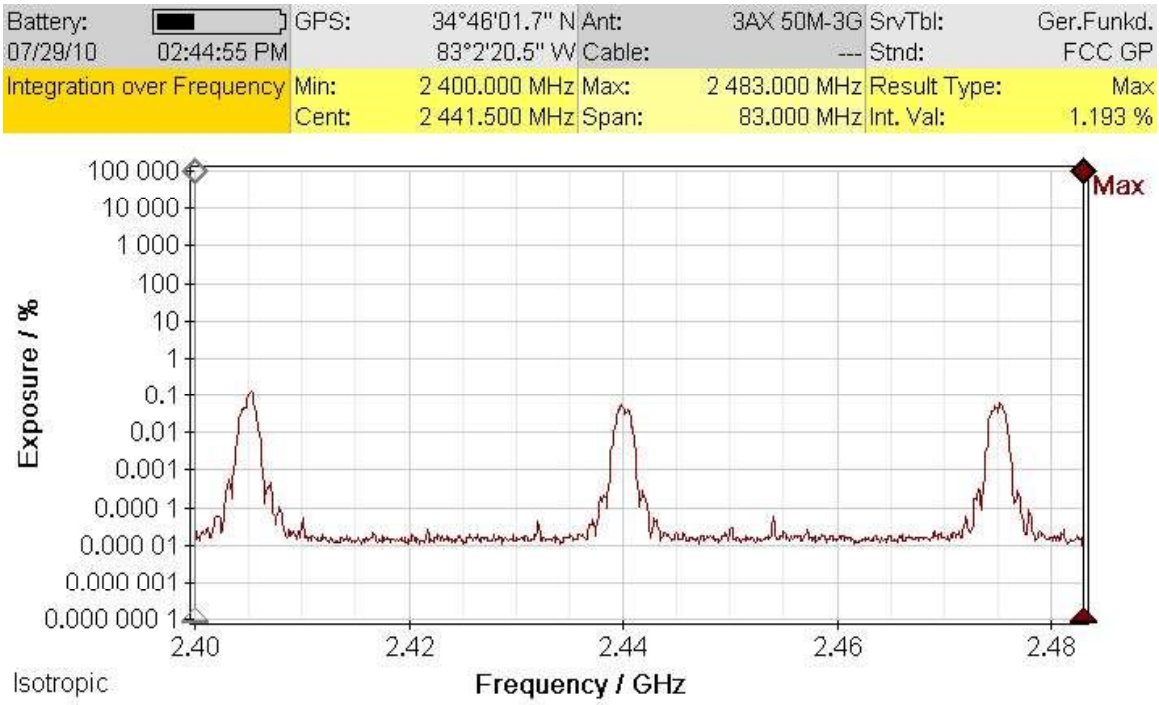

Figure E-4
2.4 GHz band composite RF field obtained with lateral walk at 3 feet in front of rack of 10 SmartMeters from well beyond each side of rack.

Appendix F: Photos of Simulated Stucco Wall During Construction

Figure F-1
Simulated wall section prior to installation of insulation, sheetrock and stucco lath (netting).

Figure F-2
Simulated wall section showing initial installation of stucco scratch coat of stucco during construction with underlying 1.5 inch stucco netting.

Appendix G: Modeling of RF fields of a 915 MHz Dipole for Spatial Averaging

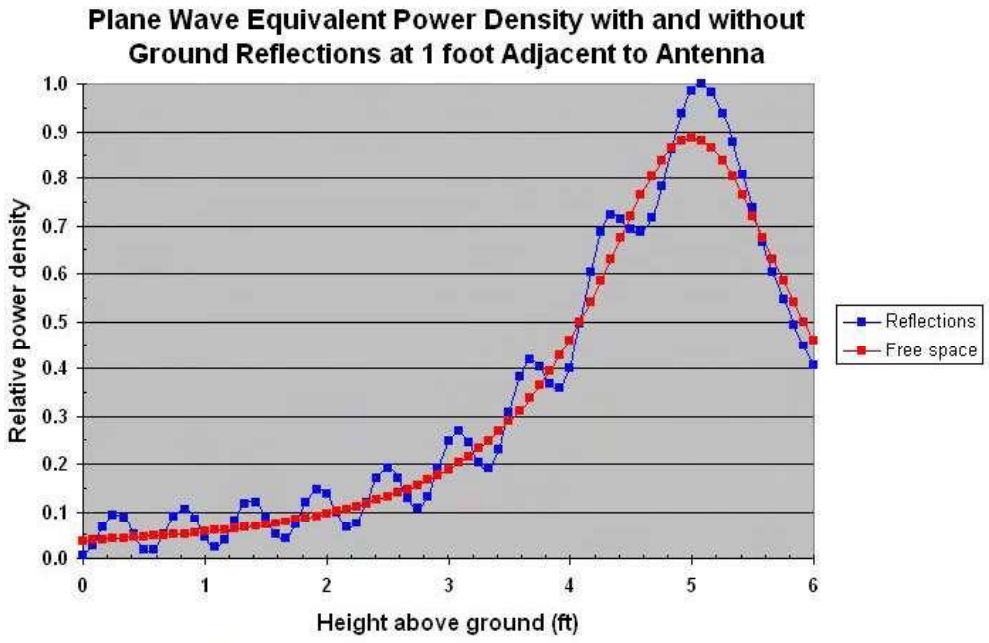

Figure G-1
Plane wave equivalent power density with and without ground reflections along a six-foot vertical line at 1 foot adjacent to a 900 MHz horizontally polarized dipole located at 5 feet above ground. Ratio of spatial average with reflections to spatial average without reflections is 1.032.

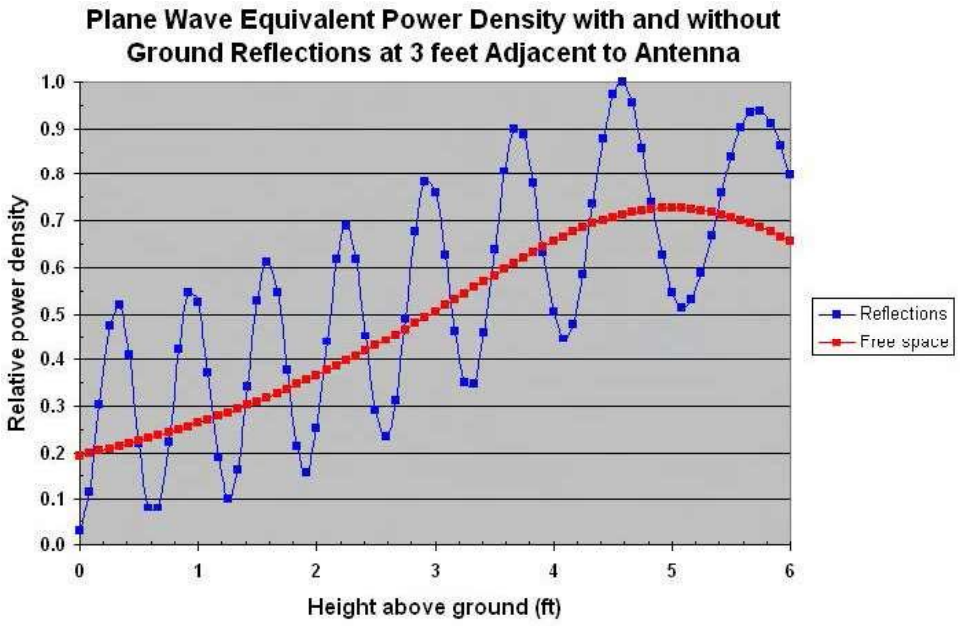

Figure G-2
Plane wave equivalent power density with and without ground reflections along a six-foot vertical line at 3 feet adjacent to a 900 MHz horizontally polarized dipole located at 5 feet above ground. Ratio of spatial average with reflections to spatial average without reflections is 1.103.

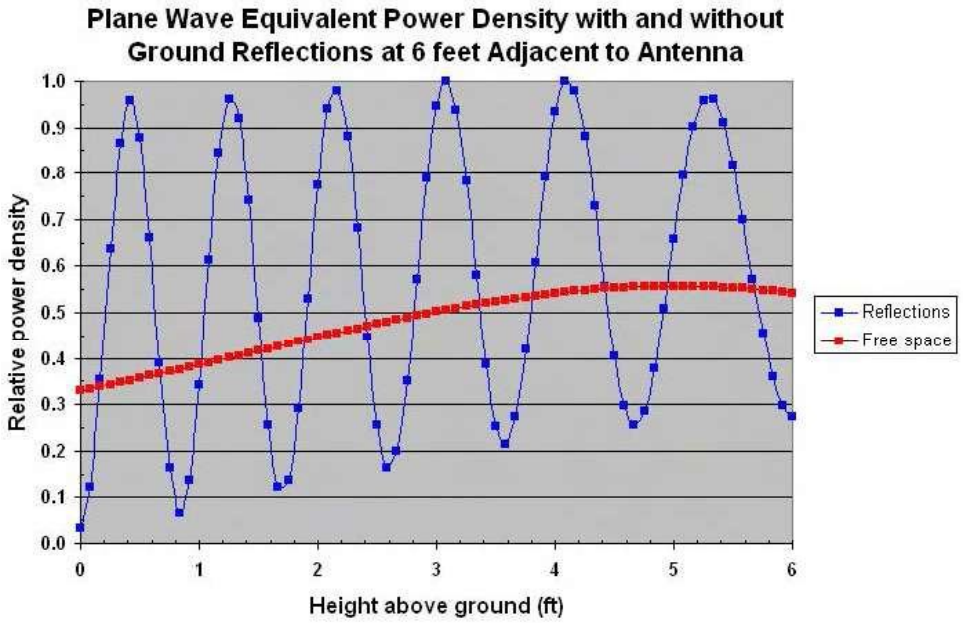

Figure G-3
Plane wave equivalent power density with and without ground reflections along a six-foot vertical line at 6 feet adjacent to a 900 MHz horizontally polarized dipole located at 5 feet above ground. Ratio of spatial average with reflections to spatial average without reflections is 1.190.

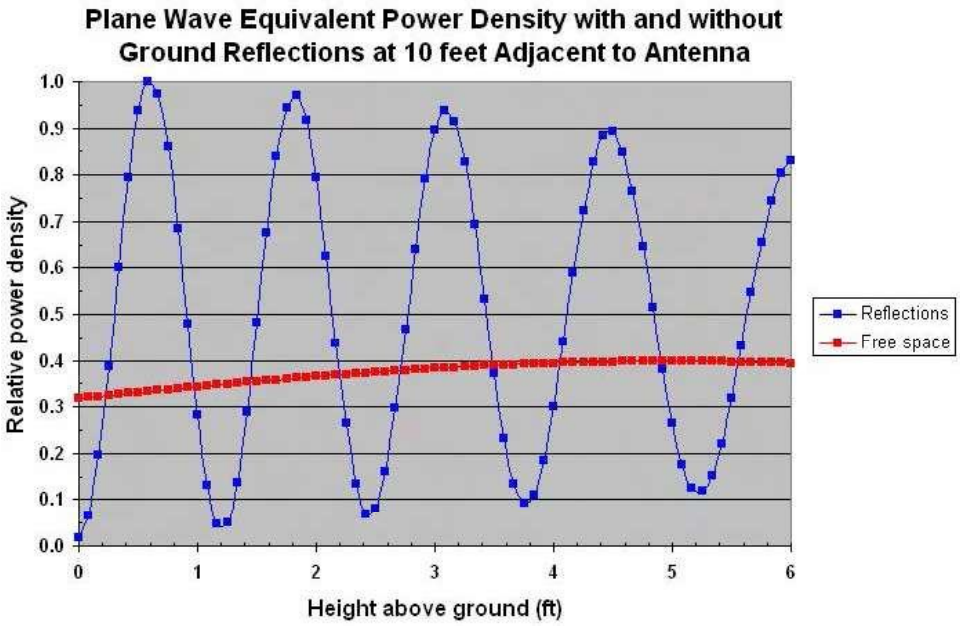

Figure G-4
Plane wave equivalent power density with and without ground reflections along a six-foot vertical line at 10 feet adjacent to a 900 MHz horizontally polarized dipole located at 5 feet above ground. Ratio of spatial average with reflections to spatial average without reflections is 1.344.

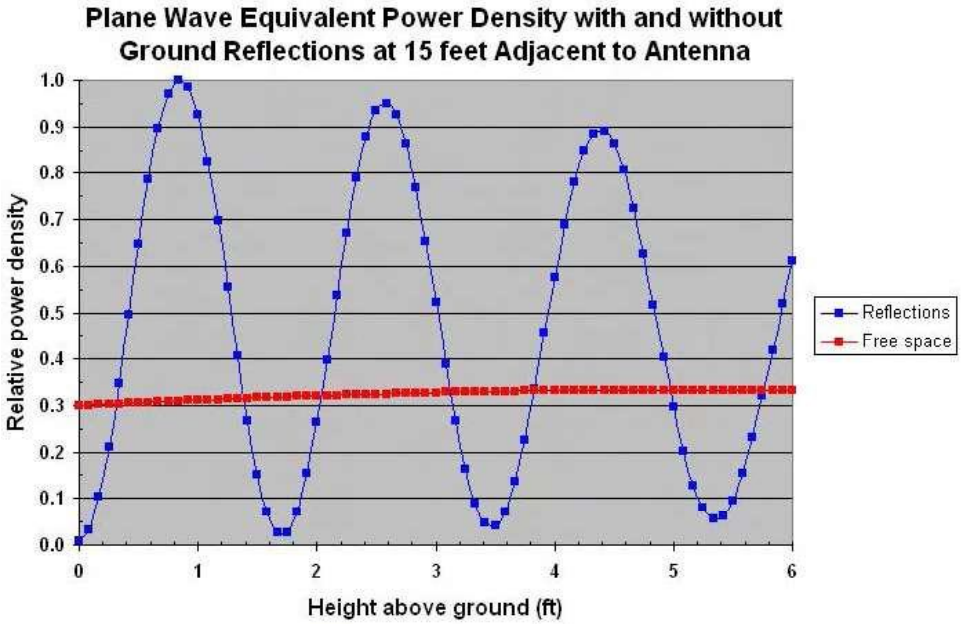

Figure G-5
Plane wave equivalent power density with and without ground reflections along a six-foot vertical line at 15 feet adjacent to a 900 MHz horizontally polarized dipole located at 5 feet above ground. Ratio of spatial average with reflections to spatial average without reflections is 1.432.

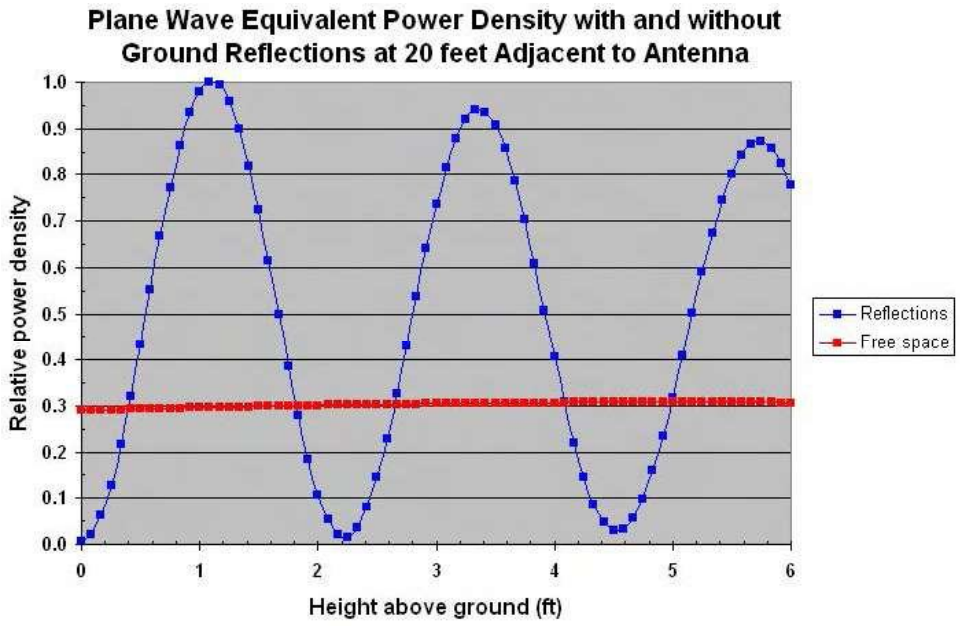

Figure G-6
Plane wave equivalent power density with and without ground reflections along a six-foot vertical line at 20 feet adjacent to a 900 MHz horizontally polarized dipole located at 5 feet above ground. Ratio of spatial average with reflections to spatial average without reflections is 1.650.

Appendix H: Glossary of Terms

AirCard- An AirCard is a device that, typically, is inserted into a laptop computer, that provides access to the Internet via a wireless wide area network (WWAN) normally operated by cellular telephone companies. This is to be distinguished from Wi-Fi wireless capability built into most modern laptop computers that allow communication with a so-called hot spot as in cyber cafes.

AMI- Advanced metering infrastructure.

AMR- Automatic meter reading.

ANSI- American National Standards Institute, issued first standard for protection against intense microwave exposure in 1966.

anechoic- A term meaning without echos or reflections. Anechoic chambers are often used for antenna pattern measurements to minimize any disturbance of the measurement data due to reflections from the local environment.

antenna- A device designed to efficiently convert conducted electrical energy into radiating electromagnetic waves in free space (or vice versa).

antenna pattern- Typically a graphical plot illustrating the directional nature of radiated fields produced by an antenna. The pattern also shows the directional nature of the antenna when used for receiving signals.

attenuation- The phenomenon by which the amplitude of an RF signal is reduced as it moves from one point in a system to another. It is often given in decibels.

averaging Time (T_{avg})- The appropriate time period over which exposure is averaged for purposes of determining compliance with the maximum permissible exposure (MPE). For exposure durations less than the averaging time, the maximum permissible exposure, MPE', in any time interval, is found from:

$$MPE' = MPE\left(\frac{T_{avg}}{T_{exp}}\right)$$

where T_{exp} is the exposure duration in that interval expressed in the same units as T_{avg}. T_{exp} is limited by restriction on peak power density.

azimuth pattern- Commonly a term referring to an antenna pattern showing the distribution of radiated field from the antenna in the azimuth plane (horizontal plane).

bandwidth- A measure of the frequency range occupied by an electromagnetic signal. It is equal to the difference between the upper frequency and the lower frequency, usually expressed in Hertz.

beacon signal- A very short duration signal emitted by Smart Meters (7.5 milliseconds in the case of the Itron meters) to indicate their availability to connect to other meters within a mesh network. Beacon signals occur periodically at different time intervals depending on the state of connectivity with the mesh network and any requirements to transmit data. This interval can vary from approximately once every 3.5 seconds to once an hour but can be absent during times when the Smart Meter must transmit energy consumption data to the network (see text for more detail).

calibration correction factor- A numerical factor obtained through a calibration process that is used to multiply RF field meter readings by to obtain corrected readings to achieve the maximum accuracy possible.

carrier current- A term used to include the use of electric power lines for communication of voice or data signals by imposing a radiofrequency signal on the 60-Hz voltage waveform. The data signals are "received" at some distant point by a receiver connected to the power line, not by use of an antenna to detect a radiated RF fields.

CDMA- Code division multiple access. A method by which several signals can be sent simultaneously over the same channel by encoding each signal with a unique code that allows each signal to be extracted from the total. The other signals within the communications channel are considered noise.

cell relay- A form of Smart Meter that provides the normal function of an end point meter but also allows for data connectivity with the electric utility company via a wireless wide area network that functions in the cellular telephone or personal communications service (PCS) bands.

continuous exposure- Exposure for durations exceeding the corresponding averaging time (usually 6 minutes for occupational exposure and 30 minutes for the general public). Exposure for less than the averaging time is called short-term exposure.

controlled environment- Controlled environments are locations where there is exposure which may be incurred by persons who are aware of the potential for exposure associated with employment, by other cognizant persons or as the incidental result of transient passage through areas where analysis shows the exposure levels are below some standard level for this environment but above the level for uncontrolled environments.

controlled exposure- a term applied by the FCC to occupational human exposures to radio frequency fields when persons are exposed as a consequence of their employment and in which those persons who are exposed have been made fully aware of the potential for exposure and can exercise control over their exposure.

dBi- decibel referenced to an isotropic antenna- a theoretical antenna which transmits (or receives) electromagnetic energy uniformly in all directions (i.e. there is no preferential direction).

dBm- A logarithmic expression for radiofrequency power where 0 dBm is defined as equal to 1 milliwatt (mW). Hence, +10 dBm is 10 mW, +20 dBm is 100 mW, etc., and -10 dBm is 0.1 mW.

decibel (dB)- a dimensionless quantity used to logarithmically compare some value to a reference level. For power levels (watts or watts/m^2), it would be ten times the logarithm (to the base ten) of the given power level divided by a reference power level. For quantities like volts or volts per meter, a decibel is twenty times the logarithm (to the base ten) of the ratio of a level to a reference level.

direct sequence- As used in direct sequence spread spectrum radio transmission, a modulation technique wherein the resulting transmitted bandwidth of a signal is spread over a much wider band and resembles white noise.

duty cycle- a measured of the percentage or fraction of time that an RF device is in operation. A duty cycle of 1.0, or 100%, corresponds to continuous operation. Also called duty factor. A duty cycle of 0.01 or 1% corresponds to a transmitter operating on average only 1% of the time.

effective isotropic radiated power (EIRP)- the apparent transmitted power from an isotropic antenna (i.e. a theoretical antenna that transmits uniformly in all possible directions as an expanding sphere).

effective radiated power (ERP)- the apparent transmitted power from an antenna, taking into account the effect of the antenna to concentrate the power in a given direction rather than emitting it in all directions, expressed in watts (W), and typically referenced to a half-wave dipole type of antenna.

electric field strength- a field vector (E) describing the force that electrical charges have on other electrical charges, often related to voltage differences, measured in volts per meter (V/m).

electromagnetic field- a composition of both an electric field and a magnetic field that are related in a fixed way that can convey electromagnetic energy. Antennas produce electromagnetic fields when they are used to transmit signals.

electromagnetic spectrum- the range of frequencies associated with electromagnetic fields. The spectrum ranges from extremely low frequencies beginning at zero hertz to the highest frequencies corresponding to cosmic radiation from space.

elevation pattern- Commonly a term referring to an antenna pattern showing the distribution of radiated field from the antenna in the elevation plane (vertical plane).

end point meter- A term used to designate a Smart Meter that is installed on a home or business to record and transmit electric energy consumption but that does not provide access point features such as those provided by a cell relay.

EPA- Environmental Protection Agency.

EVDO- Evolution-Data Optimized. A third generation telecommunications standard for wireless transmission of

data via radio signals. EVDO presents an advantage over other technologies since it uses the same transmission frequencies as existing CDMA networks, providing a cost advantage since additional, new spectrum is not required to implement this method of communication. The major EVDO deployments in the U.S. are by Verizon and Sprint.

exposure- exposure occurs whenever a person is subjected to electric, magnetic or electromagnetic fields or to contact currents other than those originating from physiological processes in the body and other natural phenomena.

far field- the far field is a term used to denote the region far from an antenna compared to the wavelength corresponding to the frequency of operation. It is a distance from an antenna beyond which the transmitted power densities decrease inversely with the square of the distance.

Federal Communications Commission (FCC)- the Federal Communications Commission (FCC) is an independent agency of the US Federal Government and is directly responsible to Congress. The FCC was established by the Communications Act of 1934 and is charged with regulating interstate and international communications by radio, television, wire, satellite, and cable. The FCC also allocates bands of frequencies for non-government communications services (the NTIA allocates government frequencies). The guidelines for human exposure to radio frequency electromagnetic fields as set by the FCC are contained in the Office of Engineering and Technology (OET) Bulletin 65, Edition 97-01 (August 1997). Additional information is contained in OET Bulletin 65 Supplement A (radio and television broadcast stations), Supplement B (amateur radio stations), and Supplement C (mobile and portable devices).

free space impedance- an expression of the apparent degree to which free space impedes the flow of electromagnetic energy express in ohms and equal to the ratio of the strength of the electric and magnetic fields (the impedance of free space is equal to 377 ohms).

frequency conformal- A term used to describe broadband RF field probes that have an inherent frequency shaped response that is tailored to a specific frequency dependent RF exposure standard. The output of such a probe is normally expressed in percentage of the exposure standard.

gain, antenna- a measure of the ability of an antenna to concentrate the power delivered to it from a transmitter into a directional beam of energy. A search light exhibits a large gain since it can concentrate light energy into a very narrow beam while not radiating very much light in other directions. It is common for cellular antennas to exhibit gains of 10 dB or more in the elevation plane, i.e., concentrate the power delivered to the antenna from the transmitter by a factor of 10 times in the direction of the main beam giving rise to an effective radiated power greater than the actual transmitter output power. In other directions, for example, behind the antenna, the antenna will greatly decrease the emitted signals. Gain is often referenced to an isotropic antenna (given as dBi).

gigahertz (GHz)- one billion hertz.

ground reflection factor- A factor commonly used in calculations of RF field power densities that expresses the power reflection coefficient of the ground over which the RF field is being computed. The purpose of the factor is to account for the fact that ground reflected RF fields can add constructively in an enhanced (stronger) resultant RF field.

HAN- Home Area Network. In the context of Smart Meters, a local area network for communication between a personal computer and various electrical appliances, equipment or systems to accomplish optimized electric energy consumption at the home. Small sensors with low power radio transmitters are attached to the various electrical appliances for communication in the HAN.

hertz- the unit for expressing frequency, one Hertz (Hz) equals one cycle per second.

IEEE- Institute of Electrical and Electronics Engineers.

insertion loss- A measure of the reduction in transmitted radio frequency energy afforded by some material or structure. The materials used in home construction can attenuate the RF signals produced by Smart Meters such that RF field strengths inside a home will be less than at the same distance but outside the home in front of the meter.

ISM- Industrial, Scientific, and Medical. There are various ISM frequency bands designated by the FCC for

equipment or appliances designed to generate and use RF energy for industrial, scientific or medical purposes.

isotropic antenna- a theoretical antenna which transmits (or receives) electromagnetic energy uniformly in all directions (i.e. there is no preferential direction). The radiated wavefront is assumed to be an expanding sphere.

isotropic probe- Similar to isotropic antenna but normally related to RF measurement instruments designed to evaluate the magnitude of RF fields from a safety perspective. The isotopic character of the probe results in a measurement of the resultant RF field produced by all polarization components.

"license free"- a phrase meaning that an RF transmitter is operated at such low power and within an authorized frequency band that no formal license to operate is required by the FCC. There are restrictions placed on these devices, however, such as they shall not produce interference and/or may not create RF fields exceeding particular field strengths.

magnetic field strength- a field vector (H) that is equal to the magnetic flux density divided by the permeability of the medium. Magnetic field strength is expressed in units of amperes per meter (A/m).

max hold spectrum- A feature often present on instruments such as spectrum analyzers in which the instantaneous peak values of measured signals are captured and continuously displayed so that, over time, the absolute maximum signal values can be determined even if they were only present for a short period.

maximum permissible exposure (MPE)- the rms and peak electric and magnetic field strength, their squares, or the plane wave equivalent power densities associated with these fields and the induced and contact currents to which a person may be exposed without harmful effect and with an acceptable safety factor.

megahertz (MHz)- one million hertz.

mesh network- A term describing a network, typically wireless, in which multiple nodes communicate among themselves and data can be relayed via various nodes to some access point. Mesh networks are self healing in that should a particular pathway become nonfunctional for some reason, alternative paths are automatically configured to carry the data. Mesh networks can expand beyond the normal range of any single node (Smart Meter) by relaying of data among the different meters.

microwatts- one-millonth of a watt, a microwatt (μW) or 10^{-6} watts.

microwatt per square centimeter ($\mu W/cm^2$)- a measure of the power density flowing through an area of space, one millionth of a watt passing through a square centimeter.

microwave- an electromagnetic wave at super high frequencies, typically above 300 MHz, the wavelength of which is very short (micro).

milliwatt per square centimeter (mW/cm^2)- a measure of the power density flowing through an area of space, one thousandth of a watt passing through a square centimeter. One milliwatt per square centimeter is equal to 1,000 microwatts per square centimeter.

mode- A statistical term referring to the most frequently observed value among many. It is distinguished from the mean or median of a distribution.

modem- In the context of Smart Meters, a term commonly used to describe a wireless transceiver capable of receiving and transmitting data over a wireless wide area network. An AirCard is a form of cellular modem. Cellular modems are used in Smart Meter access points to transmit data via the Internet to electric utility companies.

modulation- refers to the variation of either the frequency or amplitude of an electromagnetic field for purposes of conveying information such as voice, data or video programming.

near field- a region very near antennas in which the relationship between the electric and magnetic fields is complex and not fixed as in the far field, and in which the power density does not necessarily decrease inversely with the square of the distance. This region is sometimes defined as closer than about one-sixth of the wavelength. In the near field region the electric and magnetic fields can be determined, independently of each other, from the free-charge distribution and the free-current distribution respectively. The spatial variability of the near field can be large. The near field predominately contains reactive energy that enters space but returns to the antenna (this is

different from energy that is radiated away from the antenna and propagates through space).

nearfield coupling- A phenomenon that can occur when an RF measurement probe is placed within the reactive near field of an RF source such that the probe interacts strongly with the source in a way that typically draws power from the source than would not occur at greater distances. When nearfield coupling occurs, field probe readings are typically erroneously greater than the actual RF field magnitude.

omnidirectional antenna- an antenna that emits a signal of essentially constant strength in all directions, in contrast to a directional antenna.

PCS- Personal communications service. Typically used to designate a band of frequencies in the 1900 MHz range with similar features to cellular telephone base stations but, commonly, with added data transmission performance.

picowatts- picowatts or pW (10^{-12} watts).

planar scan- In the context of this study, a spatial scan over a plane in front of a Smart Meter or a group of Smart Meters at a fixed distance from the Smart Meters.

plane wave- wave with parallel planar (flat) surfaces of constant phase (See also Spherical wave). Note: The cover of this report shows an idealized spherical wave that expands outward- in an appropriate region that this spherical wave can be considered as a plane (flat) wave.

plane wave equivalent power density- the power density associated with an electromagnetic wave propagated in free space in which the front of the wave is flat (plane). Meters used for measuring power density are often calibrated in terms of the plane wave equivalent power density.

polarization- the orientation of the electric field component of an electromagnetic field relative to the earth's surface. Vertical polarization refers to the condition in which the electric field component is vertical, or perpendicular, with respect to the ground, horizontal polarization refers to the condition in which the electric field component is parallel to the ground.

power density- power density (S, sometimes called the Poynting vector) is the power per unit area normal to the direction of propagation, usually expressed in units of watts per square meter (W/m^2) or, for convenience, milliwatts per square centimeter (mw/cm^2) or microwatts per square centimeter ($\mu w/cm^2$). For plane waves, power density, electric field strength, E, and magnetic field strength, H. are related by the impedance of free space, i.e. 120π (377) ohms. In particular, $S = E^2/120\pi = 120\pi H^2$ (Where E and H are expressed in units of V/m and A/m, respectively, S is in units of $W/m^{2)}$. Although many RF survey instruments indicate power density units, the actual quantities measured are E or E^2 or H or H^2.

Poynting vector- a field vector quantity equal to the vector product (cross product) of the electric field and magnetic field of an electromagnetic wave. The Poynting vector (S, also called power density) is equal to E X H, with units of W/m^2.

product performance standard- Typically a numerical value defining a maximum allowed RF emission magnitude at or near the surface of an electronic device. For microwave ovens, the product performance standard specifies a maximum leakage of RF energy from the oven of 5 mW/cm^2 at any point 5 cm from the surface of the oven. A product performance standard is not the same as a whole body exposure standard. Compliance with the microwave oven leakage standard of 5 mW/cm^2 is not inconsistent with the whole body exposure limit of 1 mW/cm^2 since emission intensity decreases rapidly with distance from the oven and whole body exposure values will generally be substantially less than the whole body limit at such a distance that the whole body is exposed.

radiating field- the components of the total electromagnetic field produced by an antenna that contains all of the energy propagated away from the antenna. In the radiation field, both the electric and magnetic fields are codependent with an intensity that varies inversely with distance from the source.

radiation pattern- a description of the spatial distribution of RF energy emitted from an antenna. Two radiation patterns are required to completely describe the transmitting performance of an antenna, one for the azimuth plane and another for the elevation plane.

radio- a term used loosely to describe a radio transmitter or transceiver.

radio frequency (RF)- although the RF spectrum is formally defined in terms of frequency as extending from 0 to 3000 GHz, the frequency range of interest is 3 kHz to 300 GHz.

radio spectrum- the portion of the electromagnetic spectrum with wavelengths above the infrared region in which coherent waves can be generated and modulated to convey information- generally about 3 kHz to 300 GHz.

reflection- an electromagnetic wave (the "reflected" wave) caused by a change in the electrical properties of the environment in which an "incident" wave is propagating. This wave usually travels in a different direction than the incident wave. Generally, the larger and more abrupt the change in the electrical properties of the environment, the larger the reflected wave

resolution bandwidth- A specification for spectrum analyzers that denotes the ability of the analyzer to identify two signals on different frequencies.

resultant field- The combined result of all polarization components of an electromagnetic field found by determining the sum of three orthogonal components of power density or the root sum squared of three orthogonal components of electric or magnetic field strength.

RF - radiofrequency.

root-mean-square (RMS)- the effective value of, or the value associated with joule heating, of a periodic electromagnetic wave. The RMS value of a wave is obtained by taking the square root of the mean of the squared value of the wave.

router, wireless- A device commonly used in homes and offices for wireless distribution of Internet connectivity, most commonly operating in the 2.4 GHz license free band.

safety factor- additional safety is incorporated into MPE limits by the use of a safety factor (SF). A safe level exposure is divided by the safety factor to yield the allowable exposure limits or maximum permissible exposure (MPE). The FCC uses a SF of 5 for occupational and 50 for public exposure limits. This means the MPE for the general public is 50 times less than a level determined to be safe.

shielding effectiveness- A measure of the ability of a material or structure to attenuate RF fields, typically specified in decibels.

slot antenna- An antenna constructed from a metal substrate with a slot cut in the metal. When driven by a transmitter, the slot radiates like a wire dipole. The antenna pattern of the slot antenna is determined by its size and shape and the driving frequency. In the Itron Smart Meters studied in this project, slot antennas are represented by thick lines on printed circuit cards wherein the metal surface has been removed to create slots.

spatial average- For RF exposure limits, a determination of the average value of power density over the projected cross section area of the body. In practice, an average along a vertical line representing the height of a person.

specific absorption rate (SAR)- the time derivative of the incremental energy absorbed by (dissipated in) an incremental mass contained in a volume) of a given density. SAR is expressed in units of watts per kilogram (or milliwatts per gram, mW/g). Guidelines for human exposure to radio frequency fields are based on SAR thresholds where adverse biological effects may occur. When the human body is exposed to a radio frequency field, the SAR experienced is proportional to the squared value of the electric field strength induced in the body.

spectrum analyzer- An electronic instrument, similar to a receiver, that sweeps across a part of the RF spectrum and displays detected signals as peaks on a visual display screen. Spectrum analyzers normally continuously sweep repetitively over a given frequency band at a relatively high rate thereby allowing for the observation of intermittent signals.

spherical wave- a wave with concentric spherical surfaces of constant phase. Far from its source a spherical wave expands to approximate a flat surface or plane wave over discrete areas. Note: the cover of this report shows an idealized spherical wave generated by a rod antenna.

spread spectrum- Refers to a method by which an RF signal that is generated in a particular bandwidth is deliberately spread in the frequency domain resulting in a signal with a wider bandwidth. Such a technique is used to enhance secure communications, to reduce interference and to prevent detection.

time-averaged exposure- In the context of RF exposure limits, an average of the exposure value over a specified

time period. Commonly, for occupational exposures, the averaging time is six-minutes and for members of the general public 30-minutes. All scientifically based RF exposure limits are in terms of time-averaged values.

transceiver- A radio device that has both transmitting and receiving capability. Strictly, the radio devices in Smart Meters are transceivers since they can both transmit data and receive data. Commonly, in the context of evaluating RF fields, the term transmitter or radio is used to refer to the transmitting feature of the transceiver.

uncontrolled environment- uncontrolled environments are locations where there is the exposure of individuals who have no knowledge or control of their exposure. The exposures may occur in living quarters or workplaces where there are no expectations that the exposure levels may exceed some standard level limits for this environment

uncontrolled exposure- a term applied by the FCC to human exposures to radio frequency fields when the general public is exposed or in which persons who are exposed as a consequence of their employment may not be made fully aware of the potential for exposure or cannot exercise control over their exposure. Members of the general public always fall under this category when exposure is not employment-related.

USB- Universal serial bus commonly found on personal computers.

WWAN- wireless wide area network. WWANs are provided by several cellular telephone companies for wireless connectivity directly to the Internet for data transmission. WWANs are different from so-called wireless "hot spots" such as found in cyber cafes and operate in either the 850 MHz cellular or 1900 MHz PCS bands.

yagi antenna- A multi-element antenna composed of a number of dipole elements attached to a boom such that the combination of elements (a driven element and a reflector and possible director elements) and their spacing result in elevated values of gain. Design originally credited in 1926 to Shintaro Uda and Hidetsugu Yagi in Japan and sometimes called a Yagi-Uda antenna.

Zigbee- A specification for a data communications protocol used by small, low power digital radios commonly implemented in low-rate wireless personal area networks or HANs.

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IEEE Standard for Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz

C95.1™

Sponsored by the
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19 April 2006

IEEE Std C95.1™-2005
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Sponsor

IEEE International Committee on Electromagnetic Safety (SCC39)

Approved 3 October 2005

IEEE-SA Standards Board

Abstract: Recommendations to protect against harmful effects in human beings exposed to electromagnetic fields in the frequency range from 3 kHz to 300 GHz are provided in this standard. These recommendations are intended to apply in controlled environments and for general population exposure. These recommendations are not intended to apply to the exposure of patients by or under the direction of physicians and medical professionals.

Keywords: basic restriction (BR), maximum permissible exposure (MPE), radio frequency (RF), RF exposure, RF safety, specific absorption rate (SAR)

The Institute of Electrical and Electronics Engineers, Inc.
3 Park Avenue, New York, NY 10016-5997, USA

Print: ISBN 0-7381-4834-2 SH95389
PDF: ISBN 0-7381-4835-0 SS95389

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Introduction

This introduction is not part of IEEE Std C95.1-2005, IEEE Standard for Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz.

In 1960, the American Standards Association approved the initiation of the Radiation Hazards Standards project under the co-sponsorship of the Department of the Navy and the Institute of Electrical and Electronics Engineers, Inc. Prior to 1988, C95 standards were developed by Accredited Standards Committee C95, and submitted to the American National Standards Institute (ANSI) for approval and issuance as ANSI C95 standards. Between 1988 and 1990, the committee was converted to Standards Coordinating Committee 28 (SCC 28) under the sponsorship of the IEEE Standards Board. In 2001, the IEEE Standards Association Standards Board approved the name "International Committee on Electromagnetic Safety (ICES)" for SCC 28 to better reflect the scope of the committee and its international membership. In accordance with policies of the IEEE, C95 standards are issued and developed as IEEE standards, as well as submitted to ANSI for recognition.

In 2005, SCC 28 and SCC 34 became Technical Committees 95 and 34, respectively, under a new committee, SCC 39, which is now called ICES.

The present scope of IEEE ICES is as follows:

"Development of standards for the safe use of electromagnetic energy in the range of 0 Hz to 300 GHz relative to the potential hazards of exposure of man, volatile materials, and explosive devices to such energy. It is not intended to include infrared, visible, ultraviolet, or ionizing radiation. The committee will coordinate with other committees whose scopes are contiguous with ICES."

Subcommittee 4 of ICES Technical Committee 95 (TC95) is responsible for this standard. There are five TC95 subcommittees, each of whose area of responsibility is described below in correspondence with its designated subcommittee number:

1) Techniques, Procedures, and Instrumentation;
2) Terminology, Units of Measurements and Hazard Communication;
3) Safety Levels with Respect to Human Exposure, 0-3 kHz;
4) Safety Levels with Respect to Human Exposure, 3 kHz-300 GHz;
5) Safety Levels with Respect to Electro-Explosive Devices.

Three standards, three recommended practices and one guide have been issued. Current versions are:

IEEE Std 1460™-1996 (R2002), IEEE Guide for the Measurement of Quasi-Static Magnetic and Electric Fields.

IEEE Std C95.1™-2005, IEEE Standard for Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz.

NOTE—The recommendations in this standard protect against scientifically established adverse health effects in human beings resulting from exposure to radio frequency electromagnetic fields in the frequency range of 3 kHz to 300 GHz. Other effects that have been reported in the literature but have not been confirmed or could not be related to human health have been considered and are discussed in Annex B and Annex C of this standard.

IEEE Std C95.2™-1999 (R2005), IEEE Standard for Radio-Frequency Energy and Current Flow Symbols.

IEEE Std C95.3™-2002, Recommended Practice for Measurements and Computations of Radio Frequency Electromagnetic Fields with Respect to Human Exposure to Such Fields, 100 kHz-300 GHz.

IEEE Std C95.4™-2002, IEEE Recommended Practice for Determining Safe Distances from Radio Frequency Transmitting Antennas When Using Electric Blasting Caps During Explosive Operations.

IEEE Std C95.6™-2002, IEEE Standard for Safety Levels With Respect to Human Exposure to Electromagnetic Fields, 0-3 kHz.

IEEE Std C95.7™-2005, IEEE Recommended Practice for Radio Frequency Safety Programs, 3 kHz to 300 GHz.

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Errata

Errata, if any, for this and all other standards can be accessed at the following URL: http://standards.ieee.org/reading/ieee/updates/errata/index.html. Users are encouraged to check this URL for errata periodically.

Interpretations

Current interpretations can be accessed at the following URL: http://standards.ieee.org/reading/ieee/interp/index.html.

Participants

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When the IEEE-SA Standards Board approved this standard on 3 October 2005, it had the following membership:

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Satish K. Aggarwal, *NRC Representative*
Richard DeBlasio, *DOE Representative*
Alan H. Cookson, *NIST Representative*

Mike Fisher
IEEE Standards Project Editor

Contents

IEEE Standard for Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz

1. Overview

1.1 Scope

Recommendations are made to protect against established adverse health effects in human beings associated with exposure to electric, magnetic and electromagnetic fields in the frequency range of 3 kHz to 300 GHz. The recommendations are expressed in terms of basic restrictions (BRs) and maximum permissible exposure (MPE) values. The BRs are limits on internal fields, specific absorption rate (SAR), and current density; the MPEs, which are derived from the BRs, are limits on external fields and induced and contact current. The recommendations, which protect against effects associated with electrostimulation and tissue and whole-body heating, are intended to apply to all human exposures except for exposure of patients by, or under the direction of, physicians and medical professionals. These recommendations are not intended for the purpose of preventing interference with medical and other devices that may exhibit susceptibility to radio frequency (RF) fields. The recommendations at 300 GHz are compatible with existing recommendations for safe exposure in the infrared frequency range, which begins at 300 GHz, cf., ANSI Z136.1-2000 [B7][1], ICNIRP guidelines [B63], and IEC 60825-1 [B65]. IEEE Std C95.6-2002[2] is the applicable standard for use at frequencies below 3 kHz.

1.2 Purpose

The purpose of this standard is to provide exposure limits to protect against established adverse effects to human health induced by exposure to RF electric, magnetic and electromagnetic fields over the frequency range of 3 kHz to 300 GHz.

[1]The numbers in brackets correspond to those of the bibliography in Annex G.
[2]Information on references can be found in Clause 2.

1.3 Introduction

This standard is a revision of IEEE Std C95.1, 1999 Edition [B70] and IEEE Std C95.1b-2004 [B71]. The recommendations to protect against established adverse health effects from RF exposures have been made on the basis of a comprehensive review of the scientific data. In revising the standard, findings of studies published between 1950 and December 2003[3] were considered, including those studies that involve low level exposures where increases in temperature could not be measured or were not expected. New insights gained from improved experimental and numerical methods and a better understanding of the effects of acute and chronic RF electromagnetic field exposures of animals and humans are included. A lack of credible scientific and medical reports showing adverse health effects for RF exposures at or below similar exposure limits in past standards supports the protective nature of the exposure limits. This standard includes guidance on the necessity of an RF safety program.

This standard presents two separate sets of rules to limit human exposure to electric fields, magnetic fields, and electromagnetic fields, and to induced and contact currents, in order to protect against established adverse health effects identified in the reviewed studies that are associated with exposure to such fields and currents. Specifically, in the frequency range of 3 kHz to 5 MHz, the rules minimize adverse effects associated with electrostimulation; in the frequency range of 100 kHz to 300 GHz, the rules protect against adverse health effects associated with heating. In the transition region of 0.1 to 5 MHz, each of the two sets of rules must be applied. In this transition region the rules based on heating will be more restrictive for long-term exposures to continuous wave (CW) fields, while the rules based on the effects of electrostimulation will be more restrictive for short-term exposure, e.g., short isolated pulses of low duty factor. The rules and the exposure limits incorporate safety factors that account for uncertainties and that provide a margin of safety for all. (See Annex C.6 for the derivation and detailed aspects of the safety factors.) The safety factors are conservative so that exposures that exceed the BR or MPE are not necessarily harmful. The safety factors incorporated in the MPEs are generally greater than the safety factors in the BRs. Thus, it is possible to exceed an MPE while still complying with the BRs.

Two tiers of exposure limits have been established. The upper tier, which is protective for all with an acceptable margin of safety, applies to exposure of persons in controlled environments. While the weight of scientific evidence supports the conclusion that there is no measurable risk associated with RF exposures below the upper tier of this standard, it is scientifically impossible to prove absolute safety (the null hypothesis) of any physical agent. Thus the lower tier, with an additional safety factor, recognizes public concerns and also supports the process of harmonization with other standards, e.g., the NCRP recommendations [B95] and the ICNIRP guidelines [B62]. The lower tier also defines the action level above which implementation of an RF safety program is recommended. The BRs and MPEs of the lower tier may also be used for the general public to address concerns of continuous, long-term exposure of all individuals.

These exposure limits are intended to apply to all people, with the exception of patients undergoing a procedure for medical diagnosis or treatment. This exemption is provided under the expectation that the medical staff is appropriately trained in minimizing the risk of RF hazards concomitant with the provision of a recognized benefit from the exposure. Likewise, this standard does not apply to exposure of informed volunteers in medical or scientific research studies, subject to approval by Institutional Review Boards for the Use of Human Subjects, nor is it intended to prevent interference with medical and other devices that may exhibit susceptibility to RF energy[4].

[3]Although the literature cutoff date was December 2003, several papers published in 2004 and 2005 were included.

[4]While the issue of RF emissions from wireless transmitters causing electromagnetic interference (EMI) with medical devices is outside the scope of the current standard, there are several relevant standards that the reader is directed to that recommend immunity levels for external medical devices, e.g., IEC 60601-1 [B66] and IEC 60601-1-2 [B67], as well as implantable medical devices e.g., ANSI/AAMI PC69-2000 [B5]. ISO TC215 Technical Report 21073 [B75] offers guidance for the use and operation of mobile wireless transmitters within healthcare facilities.

1.3.1 Safety factor and margin of safety

Below 100 kHz, the effect being minimized is aversive or painful electrostimulation. Because the predominant interaction mechanisms are different above and below 100 kHz, the nature of and the rationale for the safety factors differ. At low frequencies, electrostimulation has a characteristic response time that is much less than one second and exposures are assessed in terms of instantaneous fields or currents. The estimated safety factor in terms of currents or fields is between 3 and 10 (10–20 dB) in the worst case even though for many situations and people the safety factor is considerably greater. An upper tier, which is applicable to exposures in controlled environments, incorporates a smaller safety factor approaching a minimum of unity, even though in most cases the safety factor is considerably greater. A margin of safety near unity, equivalent to no margin of safety, is justified for the upper tier MPEs below 100 kHz for the following reasons: a) the maximum electrostimulation that might occur at the upper tier has no lasting adverse effect, b) the requirement of an RF safety program, and c) the general awareness of workers in occupational situations. A greater margin of safety is provided in the lower tier for frequencies below 100 kHz.

Above 100 kHz there can be a sensation of heating, which is not considered adverse. The limits in this standard may not prevent such thermal sensations; they are designed to protect against adverse health effects resulting from tissue heating, the only established adverse effect of exposure to RF energy at frequencies above 100 kHz. Above 100 kHz, exposures are assessed with reference to an averaging time that varies with frequency and at some frequencies depends on the tier designation (action level and controlled environment). The frequency 100 kHz nominally represents a “thermal crossover” below which electrostimulation effects dominate, and above which thermal effects dominate for continuous wave exposure. However, for pulsed waveforms, especially those of a low duty factor, the thermal crossover can extend to much higher frequencies (in the megahertz region). This standard contains criteria to protect against adverse electrostimulation effects for pulsed waveforms having fundamental frequencies above 100 kHz.

For short duration exposures (less than the averaging time) the BRs and MPEs are related to energy, i.e., specific absorption (SA) or energy density. It is possible, however, to continue to use the BRs and MPEs expressed in power terms, specific absorption rate (SAR), or power density or equivalent fields, recognizing their time dependence. In this case the adverse effect to be protected against is tissue damage that can result from excessive heating. For exposure durations considerably greater than the averaging time, the effect to be protected against is that shown to be the most sensitive to RF exposure, behavioral disruption observed in animals and extrapolated to humans.

The safety factor for whole-body exposure durations greater than the averaging time has been estimated to be in the range of 10 to 50 in power (10 to 17 dB) for the upper tier BRs or MPEs. The corresponding BRs and MPEs of the lower tier incorporate an additional safety factor of 5 relative to the upper tier, i.e., an additional 7 dB. The safety factors for special exposure measures, such as peak (short pulse) limits and contact and induced currents in the limbs, are often related to the safety factors incorporated in the BRs or MPEs for fields. This factor is generally of the order of at least 10 dB.

1.3.2 RF risk assessment and RF safety programs

Throughout the RF spectrum to which this standard is applicable, the MPEs apply to *exposure of individuals*. Areas wherein intense RF fields exist (that exceed the MPEs) would be an exposure issue only when individuals have access to those areas and may become exposed. Hence, compliance with this standard is to be determined by assessing whether persons may be exposed to RF fields exceeding the MPEs and not necessarily by whether RF fields simply exceed the MPEs. This standard recommends that when and where there may be access to RF fields, currents, and/or voltages that exceed the lower tier MPEs (action levels), exposures are to be controlled through the implementation of an RF safety program, as described in IEEE Std C95.7-2005. Application of an RF safety program results in various control measures that can be taken to reduce the probability of a person's exposure exceeding the BRs and MPEs of the upper tier.

2. References

The following referenced documents are indispensable for the application of this document. For dated references, only the edition cited applies. For undated references, the latest edition of the referenced document (including any amendments or corrigenda) applies.

IEEE Std C95.3™-2002, IEEE Recommended Practice for Measurements and Computations of Radio Frequency Electromagnetic Fields with Respect to Human Exposure to Such Fields, 100 kHz–300 GHz.[5, 6]

IEEE Std C95.6™-2002, IEEE Standard for Safety Levels With Respect to Human Exposure to Electromagnetic Fields, 0 to 3 kHz.

IEEE Std C95.7™-2005, IEEE Recommended Practice for Radio Frequency Safety Programs, 3 kHz to 300 GHz.

3. Definitions, acronyms, abbreviations, and letter symbols

3.1 Definitions

For the purposes of this standard, the following terms and definitions apply. *The Authoritative Dictionary of IEEE Standards Terms* [B72], should be referenced for terms not defined in this clause. For the convenience of the reader, terms used in this standard that are defined in The Authoritative Dictionary of IEEE Standards Terms are contained in a glossary (see Annex E).

3.1.1 action level: The values of the electric and magnetic field strength, the incident power density, contact and induced current, and contact voltages above which steps should be initiated to protect against exposures that exceed the upper tier, specifically, implementation of an RF safety program.

3.1.2 adverse health effect: A biological effect characterized by a harmful change in health. *See also:* **established adverse health effect**.

NOTE 1—Adverse effects do not include biological effects without a harmful health effect, changes in subjective feelings of well-being that are a result of anxiety about RF effects or impacts of RF infrastructure that are not physically related to RF emissions, or indirect effects caused by electromagnetic interference with electronic devices.

NOTE 2—Sensations (perceptions by human sense organs) per se are not considered adverse effects. Thus a sensation of warmth at millimeter and other wavelengths and the microwave auditory effect under the underlying special conditions are not recognized as effects to be protected against by this standard. Painful or aversive electrostimulation resulting from exposure at frequencies below 0.1 MHz is treated as an adverse effect.

3.1.3 adverse effects exposure level: The condition or set of conditions under which exposure to an electric, magnetic, or electromagnetic field can produce a harmful change in health. Conditions can be a property of the source (such as field strength, polarization, power density, frequency, modulation, pulse duration and repetition frequency), a dosimetric quantity (such as current, current density, specific absorption, or specific absorption rate), and an exposure characteristic (such as exposure duration and recurrence interval). This standard is based on the lowest known exposure levels for all established adverse effects. The maximum permissible exposure (MPE) values in this standard are derived from these exposure levels incorporating appropriate margins of safety.

[5]IEEE publications are available from the Institute of Electrical and Electronics Engineers, 445 Hoes Lane, P.O. Box 1331, Piscataway, NJ 08855-1331, USA (http://standards.ieee.org/).

[6]The IEEE standards or products referred to in this clause are trademarks of the Institute of Electrical and Electronics Engineers, Inc.

3.1.4 average (temporal) power ($\overline{P}$): The time-averaged rate of energy transfer

$$\overline{P} = \frac{1}{t_2 - t_1}\int_{t_1}^{t_2} P(t)dt$$

where

$P(t)$ is the instantaneous power.

The SI unit of average power is the watt (W).

NOTE—The time duration (t_2–t_1) could be source related (e.g., the source repetition period, duty cycle) or use related (e.g., the averaging time specified in exposure guidelines).

3.1.5 average (temporal) power density: The instantaneous power density integrated (averaged) over a specific time duration. The time duration could be source related, e.g., the source repetition period, or use related, e.g., the averaging time specified in exposure guidelines. The SI unit of average power density is the watt per square meter (W/m^2).

3.1.6 averaging distance: The distance over which the *in situ* electric field is averaged when determining compliance with basic restrictions.

3.1.7 averaging time (T_{avg}): The appropriate time period over which exposure is averaged for purposes of determining compliance with a maximum permissible exposure (MPE) or reference level.

NOTE—Averaging time T_{avg} has an unambiguous meaning only at frequencies above 0.1 MHz, i.e., for the MPEs relating to heating effects. For an exposure time greater than T_{avg}, the MPE is an entity expressed in power units, e.g., SAR or power density, or in terms of field units, while for an exposure time smaller than T_{avg}, the MPE can be expressed as a power or field function of time or an equivalent integral quantity, e.g., specific absorption or energy density either on a volume or area basis.

3.1.8 averaging volume: The volume over which the peak spatial-average specific absorption rate is averaged when determining compliance with the basic restrictions.

3.1.9 basic restrictions: Exposure restrictions that are based on established adverse health effects that incorporate appropriate safety factors and are expressed in terms of the *in situ* electric field (3 kHz to 5 MHz), specific absorption rate (100 kHz to 3 GHz), or incident power density (3 GHz to 300 GHz).

3.1.10 biological effect: A biological effect is an established effect caused by, or in response to, exposure to a biological, chemical or physical agent, including electromagnetic energy. Biological effects are alterations of the structure, metabolism, or functions of a whole organism, its organs, tissues, and cells. Biological effects can occur without harming health and can be beneficial. Biological effects also can include sensation phenomena and adaptive responses.

3.1.11 chronic exposure: A sequence of many repeated or continuous exposures over a long period of time, e.g., months to years, depending on the biological system being considered and its lifespan.

3.1.12 confirmed adverse health effect: *See:* **established adverse health effect.**

3.1.13 confirmed effect: *See:* **established effect**.

3.1.14 contact current: Current induced in a biological medium via a contacting electrode or other source of current.

3.1.15 contact voltage: Voltage between a biological medium and an electrode or current source in the absence of direct contact with the body.

3.1.16 controlled environment: An area where the occupancy and activity of those within is subject to control and accountability as established by an RF safety program for the purpose of protection from RF exposure hazards. *See also:* **general public**.

NOTE—Implementation of an effective RF safety program such as IEEE Std C95.7-2005 is to ensure that persons are not exposed in excess of the "Controlled Environment" MPEs.

3.1.17 continuous exposure: For purposes of this standard, exposure for durations exceeding the corresponding averaging time.

NOTE—In this context, exposure for less than the averaging time is considered a *short-term exposure*. For cellular studies in the laboratory, continuous exposure refers to exposures for most of the cell-cycle of a proliferating cell system (or longer), while for non-proliferating cells *in vitro* or in tissues, "continuous exposure" is arbitrary. For cellular studies, *short term* refers to, at most, exposure over a small portion of the cell cycle time. With respect to non-proliferating cells *in vitro* or in tissues, the definition is arbitrary.

3.1.18 derived limits: *See:* **maximum permissible exposure**.

3.1.19 environmental limit: A limit on the electric and magnetic fields permitted in the general environment, whether or not people are present.

3.1.20 equivalent plane-wave power density (plane-wave equivalent power density) (S): A commonly used term associated with any electromagnetic wave, equal in magnitude to the power density of a plane wave having the same electric (E) or magnetic (H) field strength. Specifically, the normalized value of the square of the electric or the magnetic field strength at a point in the near field of a radiating source. The SI unit of equivalent plane-wave power density is the watt per square meter (W/m^2) and is computed as follows:

$$S = \frac{|E|^2}{\eta} = \eta|H|^2$$

where

E and H are the root mean square (rms) values of the electric and magnetic field strengths, respectively, and

η is the wave impedance ($\cong$ 377 ohms in free space).

Synonym: **equivalent plane-wave power flux density**.

3.1.21 established adverse health effect: A biological effect characterized by a harmful change in health that is supported by consistent findings of that effect in studies published in the peer-reviewed scientific literature, with evidence of the effect being demonstrated by independent laboratories, and where there is consensus in the scientific community that the effect occurs for the specified exposure conditions. The development of this standard is based on the following established adverse health effects: 1) aversive or painful electrostimulation due to excessive RF internal electric fields, 2) RF shocks or burns due to contact with excessively high RF voltages, 3) heating pain or tissue burns due to excessive localized RF exposure, and 4) behavioral disruption, heat exhaustion or heat stroke due to excessive whole body RF exposures. *See:* **adverse health effect**.

3.1.22 established effect: An effect is considered *established* when consistent findings of that effect have been published in the peer-reviewed scientific literature, with evidence of the effect being demonstrated by independent laboratories, and where there is consensus in the scientific community that the effect occurs for the specified exposure conditions.

3.1.23 established mechanism: For purposes of this standard, a mechanism with the following characteristics: (1) It can be used to predict a biological effect in cells, animals, or humans; (2) An explicit model can be proposed using equations or parametric relationships; (3) It has been verified in humans, or animal data supporting the mechanism can be confidently extrapolated to humans; (4) It is supported by strong evidence; and (5) It is widely accepted among experts in the scientific community.

3.1.24 exposure: For purposes of this standard, exposure of a person to electric, magnetic, or electromagnetic fields or to induced and contact currents other than those originating from physiological processes in the body and other natural phenomena.

3.1.25 extremities: For purposes of this standard, the parts of the arms and legs distal from the elbows and knees, respectively.

3.1.26 general public: Individuals of all ages and varying health status, some of whom may be subject to requirements of the controlled environment. *See:* **controlled environment.**

NOTE 1—Generally, unless specifically provided for as part of an RF safety program, the general public includes, but is not limited to, children, pregnant women, individuals with impaired thermoregulatory systems, individuals equipped with electronic medical devices, and persons using medications that may result in poor thermoregulatory system performance.

NOTE 2—Unless specifically provided as part of an RF safety program, individual members of the public may not be aware of their exposure.

3.1.27 grasping contact: An electrical connection with a large energized conductor made by firmly holding the conductor in the hand. In this standard, a contact area of 15 cm^2 is assumed for such contact.

3.1.28 hazard: An intrinsic property or condition that has the potential to cause an adverse health effect.

3.1.29 hazard threshold: The point above which some parameter related to exposure (e.g., SAR, *in situ* electric field strength) is associated with the existence of some hazardous effect.

3.1.30 *in vitro*: Refers to studies and/or effects that occur in an artificial environment outside a living organism.

3.1.31 *in vivo*: Refers to studies and/or effects that occur within the body of living organisms.

3.1.32 *in situ*: For purposes of this standard, *in situ* means within a biological tissue in its normal anatomical position.

3.1.33 localized exposure: For frequencies exceeding 100 kHz, an exposure of a portion of the body wherein the incident plane-wave equivalent power density, or the squares of the field strength exceed 20 times the spatially averaged value over the projected (cross-sectional) area of the body. *See:* **RF hot spot**.

3.1.34 long term exposure: Exposure for a duration much longer than the corresponding averaging time. *See:* **chronic exposure**.

3.1.35 lower tier: A set of limits that provide an additional margin of safety, i.e., a margin of safety greater than that for the upper tier. *See:* **action level**.

NOTE—The lower tier, which is recommended as an action level above which an RF safety program should be implemented, recognizes public concerns, uncertainties in exposure assessment, and supports the process of harmonization with other standards.

3.1.36 low-level effects: Biological effects ascribed to exposure to low-level fields, i.e., at or below the corresponding basic restrictions in the frequency range 3 kHz to 300 GHz.

3.1.37 low-level fields: Electromagnetic fields in the frequency range 3 kHz to 300 GHz that produce induced (*in situ*) electric fields, SAR, or power density at or below the corresponding basic restrictions.

3.1.38 margin of safety: The ratio of the minimum hazard threshold (HT) level to the maximum exposure level, with accounting for all uncertainties in HT and the exposure level, in a specific exposure situation.

NOTE—This is equivalent to the minimum possible safety factor when uncertainties are accounted for, while the nominal safety factor is interpreted as that derived from median measures of HT and basic restriction or MPE. The margin of safety can approach equality with the safety factor if the uncertainties are small or if there is a very large separation between HT and the MPE. The margin of safety is a value judgment based on informed opinion.

3.1.39 maximum permissible exposure (MPE): The highest rms or peak electric or magnetic field strengths, their squares, or the plane-wave equivalent power densities associated with these fields, or the induced and contact currents to which a person may be exposed without incurring an established adverse health effect and with an acceptable margin of safety. The MPEs are derived or estimated from the basic restrictions (induced electric field, SAR, or power density). If an exposure is proven to be below the basic restrictions, the MPE can be exceeded. MPEs are sometimes called *reference levels*, *derived limits*, or *investigation levels*.

3.1.40 mixed frequency fields: The superposition of two or more electromagnetic fields of differing frequency.

3.1.41 near field exposure: Exposure that occurs in the near field region of a source. *See:* **near field region**.

3.1.42 near-field region: A region, generally in proximity to an antenna or other radiating structure, in which the electric and magnetic fields do not have a substantially plane-wave character, and vary considerably from point to point. The near-field region is further subdivided into the reactive near-field region, which is closest to the radiating structure and contains most or nearly all of the stored energy, and the radiating near-field region where the radiation field predominates over the reactive field, but lacks substantial plane-wave character and is complicated in structure.

NOTE—For most antennas, the outer boundary of the reactive near-field region is commonly taken to exist at a distance of $\lambda/2\pi$ from the antenna surface, where λ is the wavelength. The radiating near field region extends out to the far field region.

3.1.43 non-uniform field: A field that is not constant in amplitude, direction, and relative phase over the dimensions of the body or body part under consideration.

3.1.44 non-thermal effects: *See:* **low-level effects**, which is the preferred term.

3.1.45 non-thermal fields: *See:* **low-level fields**, which is the preferred term.

3.1.46 partial-body exposure: *See:* **localized exposure** and **RF hot spot**.

3.1.47 peak electric field: The instantaneous value of the electric field strength at the time of its maximum value.

3.1.48 peak power density: The maximum instantaneous power density occurring during the interval when power is transmitted. The SI unit of peak power density is the watt per square meter (W/m^2).

3.1.49 phase duration (t_p): The time between zero crossings of a waveform. For a sine wave of frequency f, $t_p = 1/(2f)$; for an exponential waveform, t_p is interpreted as the duration measured from the waveform peak to a point at which it decays to 36.8% (e^{-1}) of its peak value.

3.1.50 pinna (*pl.* pinnae): The largely cartilaginous projecting portion of the outer ear consisting of the helix, lobule, and anti-helix. The pinna is also called the *auricle*.

3.1.51 plane-wave equivalent power density: *See:* **equivalent plane-wave power density**.

3.1.52 probe length: The maximum physical dimension of the sensing element, e.g., dipole or loop of an electric or magnetic field probe, respectively, or the dimension of the largest sensing element in a multiple array.

3.1.53 projected area: For purposes of this standard, the geometric area equivalent to the vertical cross section of the human body in the configuration of interest, e.g., sitting or standing.

3.1.54 pulse-modulated field: An electromagnetic field characterized by a form of amplitude modulation in which a continuous wave is abruptly shifted in amplitude from zero to a level at or near the maximum and returning to zero; often characterized by a series of such shifts in a repeated pattern.

3.1.55 radio frequency (RF): A frequency that is useful for radio transmission. For purposes of this standard, the frequency range of interest is 3 kHz to 300 GHz.

3.1.56 reference level: The exposure field and contact current values derived or estimated from the basic restrictions, i.e., induced electric field, SAR or power density. For frequencies above 3 GHz the basic restriction and the reference levels are the same. *See:* **maximum permissible exposure**.

3.1.57 re-radiated field: An electromagnetic field resulting from currents induced in a secondary, predominantly conducting, object by electromagnetic waves incident on that object from one or more primary radiating structures or antennas. Re-radiated fields are sometimes called “reflected” or more correctly “scattered fields.” The scattering object is sometimes called a “re-radiator” or “secondary radiator.”

3.1.58 RF “hot spot”: A highly localized area of relatively more intense RF energy that manifests itself in two principal ways:

1) Near a conductive object that is the immediate source of intense electric or magnetic fields and is surrounded by ambient fields of lower intensity (often referred to as re-radiation), and

2) From reflections and/or narrow beams produced by high-gain radiating antennas or other highly directional sources. In both cases, there are very rapid changes in field strength over distances that are small with respect to the objects and wavelength. RF hot spots are normally associated with very non-uniform exposure of the body (localized exposure). This is not to be confused with an actual thermal hot spot within the absorbing body.

3.1.59 RF safety program (RFSP): An organized system of policies, procedures, practices and plans designed to protect against hazards associated with RF fields, contact voltage, and contact and induced currents. RFSPs shall be documented in writing.

NOTE 1—Implementation of an effective RF safety program is to ensure that persons are not exposed in excess of the MPEs of the upper tier.

NOTE 2—A program typically includes RF awareness training, implementation of protective measures such as signage and the use of personal protective equipment (PPE), incident response, periodic evaluation of program effectiveness, and assigned responsibilities for implementing the program (see IEEE Std C95.7-2005).

3.1.60 rheobase: The minimum threshold intensity in a strength-duration or strength-frequency relation for electrostimulation (applicable to a stimulus duration that is long in comparison with the strength-duration time constant). In a strength-duration curve, the rheobase forms a minimum asymptote to thresholds for pulsed stimulus durations greater than a strength-duration time constant, τ_e. In a strength-frequency curve,

the rheobase forms a minimum asymptote to thresholds for sinusoidal stimuli with frequencies less than a strength-frequency constant, f_e.

3.1.61 risk: The likelihood or probability that a person will be harmed by a particular hazard.

3.1.62 safety factor (F_s): A multiplier (≤ 1) or a divisor (≥ 1) used to derive maximum permissible exposure (MPE) values, which provides for the protection of individuals, and uncertainties concerning threshold effects due to pathological conditions or drug treatment, uncertainties in reaction thresholds, and uncertainties in induction models.

NOTE 1—F_s is usually taken as the ratio of the value of the threshold for an adverse effect, i.e., hazard threshold (HT), to the basic restriction (BR) or maximum permissible exposure value (MPE). Thus, $F_s = HT_{BR}/BR$ or HT_{MPE}/MPE, where HT_{BR} is expressed in the same units as BR and HT_{MPE} is expressed in the same units as MPE. This is usually considered the nominal safety factor derived from the median values of HT, BR, and the MPE.

NOTE 2—Upon consideration of statistical variation and uncertainties in the data on HT, extrapolation to humans, the models for calculation and measurement of BR or MPE and biological variability in humans, a safety factor may be increased to ensure a "margin of safety for all."

NOTE 3—Allowance is also made to account for the fact that measurement error and other uncertainties attend any evaluation of the compliance of actual exposure to the BR or MPE.

NOTE 4—Since the entities in the ratio F_s could be current, voltage, field strength, power, or energy, when comparing safety factors, their expression in decibels (dB) ensures a meaningful and fair comparison. The actual (true) safety factor in any specific situation could be larger or smaller than the nominal safety factor.

3.1.63 scattered radiation: *See:* **re-radiated field**.

3.1.64 short-term exposure: Exposure for a duration less than the corresponding averaging time.

3.1.65 spark discharge: The transfer of current through an air gap requiring a voltage high enough to ionize the air, as opposed to direct contact with a source.

3.1.66 spatial average: For frequencies up to 0.1 MHz, the root-mean-square of the field over an area equivalent to a specified cross section of the adult human body, as applied to the measurement of electric or magnetic fields in the assessment of whole-body exposure.

NOTE—The spatial average is measured by scanning (with a suitable measurement probe) a planar area equivalent to the area occupied by a standing adult human (projected area). In most instances, a simple vertical, linear scan of the fields over a 2 meter height (approximately 6 feet), through the center of the projected area, will be sufficient for determining compliance with the MPEs. For frequencies from 0.1 MHz to 3 GHz, the plane wave equivalent power densities or squares of the electric or magnetic field strengths are to be averaged along a line representing the height of an individual.

3.1.67 specific absorption (SA): The quotient of the incremental energy (dW) absorbed by (dissipated in) an incremental mass (dm) contained in a volume (dV) of a given density (ρ).

$$SA = \frac{\mathrm{d}W}{\mathrm{d}m} = \frac{\mathrm{d}W}{\rho \mathrm{d}V}$$

The SI unit of specific absorption is the joule per kilogram (J/kg).

3.1.68 specific absorption rate (SAR): The time derivative of the incremental energy (dW) absorbed by (dissipated in) an incremental mass (dm) contained in a volume element (dV) of given density (ρ).

$$SAR = \frac{\mathrm{d}}{\mathrm{d}t}\left(\frac{\mathrm{d}W}{\mathrm{d}m}\right) = \frac{\mathrm{d}}{\mathrm{d}t}\left(\frac{\mathrm{d}W}{\rho\,\mathrm{d}V}\right)$$

The SI unit of SAR is the watt per kilogram (W/kg).

NOTE 1—SAR can be related to the electric field at a point by

$$SAR = \frac{\sigma|E|^2}{\rho}$$

where

σ is conductivity of the tissue (S/m)
ρ is mass density of the tissue (kg/m^3)
E is rms electric field strength in tissue (V/m)

NOTE 2—SAR can be related to the increase in temperature at a point by

$$SAR = \left.\frac{c\Delta T}{\Delta t}\right|_{t=0}$$

where

ΔT is the change in temperature (ºC)
Δt is the duration of exposure (s)
c is specific heat capacity (J/kg ºC)

This assumes that measurements are made under "ideal" non-thermodynamic circumstances, i.e., no heat loss by thermal diffusion, radiation, or thermoregulation (blood flow, sweating, etc.).

3.1.69 specific absorption rate—peak spatial-average: The maximum local SAR averaged over a specified volume or mass, e.g., any ten-grams of tissue in the shape of a cube. The SI unit of peak spatial-average SAR is the watt per kilogram (W/kg).

3.1.70 specific heat capacity: The amount of heat necessary to raise the temperature of a unit mass of a substance 1 ºC. The SI unit of specific heat capacity is the joule per kg per kelvin (J/kg·K) or joule per kilogram degree Celsius (J/kg·ºC).

3.1.71 strength-duration curve: The functional relationship between the threshold of excitation and the duration of an excitatory stimulus. In this standard, the strength-duration curve is approximated by two straight lines which are asymptotes to analytic or experimental representations of the curve displayed on a log/log scale: for pulsed stimulus durations greater than a critical time parameter, τ_e, the threshold asymptote is a horizontal straight line (see *rheobase*); for durations less than or equal to τ_e, the threshold asymptote rises in inverse proportion to the pulse duration. This approximation necessarily imposes a margin of conservatism in the representation of thresholds in the vicinity of τ_e.

3.1.72 strength-duration time constant (τ_e): The temporal point in the asymptotic representation of a strength-duration curve that describes the transition between the rheobase and the rising threshold segment of the curve. *See also:* **strength-duration curve**.

3.1.73 strength-frequency constant (f_e): The frequency in the asymptotic representation of a strength-frequency curve that describes the transition between the rheobase and the rising threshold segment of the curve. *See also:* **strength-frequency curve**.

3.1.74 strength-frequency curve: The functional relationship between the threshold of excitation and the frequency of an excitatory stimulus. In this standard, the strength-frequency curve is approximated by two straight lines which are asymptotes to analytic or experimental representations of the curve displayed on a log/log scale: for frequencies less than a critical time parameter, f_e, the threshold asymptote is a horizontal straight line (see rheobase); for durations greater than or equal to f_e, the threshold asymptote rises in proportion to the frequency. This approximation necessarily imposes a margin of conservatism in the representation of thresholds in the vicinity of f_e.

3.1.75 thermal effects: Changes in an organism associated with heating of the whole body or an affected region that are sufficient to increase temperature by a physiologically significant amount; thermoregulatory mechanisms of heat loss (sweating, blood flow) may delay, reduce, or prevent a measurable increase in temperature. Established adverse changes are associated with whole-body heating at levels that usually increase temperature by approximately 1 ºC or more.

3.1.76 thermal level (RF fields): RF fields that are sufficiently strong to significantly increase the temperature of exposed bodies, tissues, and experimental samples.

NOTE—If strong enough, biological mechanisms of heat loss (sweating, blood flow) can reduce or effectively eliminate a temperature change or, alternatively, laboratory techniques (tissue cooling via water bath) can prevent a temperature rise in a biological sample.

3.1.77 touch contact: A contact of small area made between the human body and an energized conductor. In this standard, a contact area of 1 cm^2 is the assumed touch contact area.

3.1.78 uniform field: A field that is constant in amplitude, direction, and relative phase over the dimensions of the body or body part under consideration. In the case of electric fields, the definition applies to an external field undisturbed by the presence of the body.

3.1.79 upper tier: A set of RF exposure limits that are scientifically based and that provide a margin of safety for all, including those in a controlled environment.

3.1.80 weight of scientific evidence: For purposes of this standard, the outcome of assessing the published information about the biological and health effects from exposure to RF energy. This process includes evaluation of the quality of test methods, the size and power of the study designs, the consistency of results across studies, and the biological plausibility of dose-response relationships and statistical associations.

3.1.81 whole-body-exposure: The case in which the entire body is exposed to the incident fields.

3.2 Abbreviations

BR	basic restriction
CW	continuous wave
dB	Decibel
FDTD	finite-difference time-domain
GSM	Global System for Mobile Communications
HF	high frequency (3–30 MHz)
HT	hazard threshold
ICES	International Committee on Electromagnetic Safety
ICNIRP	International Commission on Non-Ionizing Radiation Protection
LSWG	Literature Surveillance Working Group

MF	medium frequency (0.3–3 MHz)
MPE	maximum permissible exposure
NOAEL	no observable adverse effects level
PW	pulsed wave
RF	radio frequency
RAWG	Risk Assessment Working Group
rms	root mean square
SA	specific absorption
SAR	specific absorption rate
SASB	Standards Association Standards Board
SI	Système International d'Unités (international system of units)
UHF	ultra high frequency (300 MHz–3 GHz)
VHF	very high frequency (30–300 MHz)

3.3 Letter symbols for quantities

B	magnetic flux density
c	specific heat capacity
ε_r	relative permittivity
E	electric field strength
E_i	maximum allowed in situ electric field strength
E_o	rheobase in situ electric field strength
f	frequency
f_e	strength-frequency constant
F_s	safety factor
H	magnetic field strength
I	current
J	current density
MPE	maximum permissible exposure value
η	wave impedance
P	power
$P(t)$	instantaneous power
$\overline{P}$	average power (temporal)
q	charge
ρ	mass density
SA	specific absorption
SAR	specific absorption rate
S	power density
T_{avg}	averaging time
t_p	phase duration
W	energy
σ	conductivity
τ_e	strength-duration time constant
λ	wavelength
μ	permeability

3.4 Unit symbols

A	ampere
ºC	degree Celsius
GHz	gigahertz (10^9 Hz)
h	hour
J	joule
K	kelvin
kHz	kilohertz (10^3 Hz)
MHz	megahertz (10^6 Hz)
min	minute
s	second
V	volt
W	watt

4. Recommendations

4.1 Basic restrictions (BRs) and maximum permissible exposures (MPEs) for frequencies between 3 kHz and 5 MHz

This standard provides recommendations to minimize aversive or painful electrostimulation in the frequency range of 3 kHz to 5 MHz and to protect against adverse heating in the frequency range of 100 kHz to 300 GHz. In the transition region of 100 kHz to 5 MHz, protection against both electrostimulation and thermal effects is provided through two separate sets of limits. Below 100 kHz only the electrostimulation limits apply, above 5 MHz only the thermal limits apply, and both sets of limits apply in the transition region. In the transition region, where both sets of limits apply, the limits based on electrostimulation will generally be more limiting for low duty cycle exposures, while the thermal-based limits will be more limiting for continuous wave fields.

4.1.1 BRs: *in situ* electric field

For human exposure to electromagnetic energy at frequencies from 3 kHz to 5 MHz, the basic restrictions refer to limits on the *in situ* electric fields that minimize adverse effects associated with electrostimulation. Such restrictions are derived with consideration of adverse electrical thresholds, their distribution among the population, and safety factors, as documented in IEEE Std C95.6-2002.

Table 1 lists basic restrictions for particular areas of the body in terms of the electric field within the biological tissue (*in situ*). The listed parameters apply to frequencies above and below 3 kHz to show continuity with standards adopted below 3 kHz, i.e., IEEE Std C95.6-2002. Two parameters are listed in the table: the rheobase *in situ* field, E_0, and a strength-frequency parameter, f_e. Limits are determined from Table 1 as:

$$E_i = E_0 \text{ for } f \leq f_e \tag{1}$$

$$E_i = E_0 \, (f/f_e) \text{ for } f \geq f_e$$

where

E_i is the maximum allowed induced in situ electric field.

The basic restrictions on the in situ electric field apply to an arithmetic average determined over a straight-line segment of 5 mm length oriented in any direction within the tissue identified in Table 1. The averaging

time for an rms measurement is 0.2 s. Basic restrictions expressed in Equation (1) apply to frequencies in the range of 0 to 5 MHz.

Table 1—BRs applying to various regions of the body

		Action level[a]	Persons in controlled environments
Exposed tissue	f_e (Hz)	E_0 (rms) (V/m)	E_0 (rms) (V/m)
Brain	20	5.89×10^{-3}	1.77×10^{-2}
Heart	167	0.943	0.943
Extremities	3350	2.10	2.10
Other tissues	3350	0.701	2.10
[a]Within this frequency range the term "action level" is equivalent to the term "general public" in IEEE Std C95.6-2002.			

NOTE—Entries in Table 1 and elsewhere in this standard are sometimes given to three significant digits. This degree of precision is provided so that the reader can follow the various derivations and relationships presented in this standard, and does not imply that the numerical quantities are known to that precision.

4.1.2 MPE for the magnetic field

4.1.2.1 Exposure of head and torso to sinusoidal magnetic fields

Table 2 lists the MPEs for the magnetic field (flux density, *B*, and magnetic field strength, *H*) for exposure of the head and torso. The averaging time for an rms measurement is 0.2 second.

Table 2—MPE for exposure of head and torso: *f* = 3 kHz to 5 MHz

Frequency range (kHz)	Action level[a]		Persons in controlled environments	
	B_{rms} (mT)	H_{rms} (A/m)	B_{rms} (mT)	H_{rms} (A/m)
3.0–3.35	0.687/*f*	547/*f*	2.06/*f*	1640/*f*
3.35–5000	0.205	163	0.615	490
NOTE—*f* is expressed in kHz.				
[a]Within this frequency range the term "action level" is equivalent to the term "general public" in IEEE Std C95.6-2002.				

NOTE—The MPEs in Table 2 minimize adverse effects associated with electrostimulation; Tables 8 and 9 apply to effects associated with tissue heating. All three tables must be considered and the corresponding values for the appropriate tier satisfied at all applicable frequencies.

Compliance with Table 2 ensures compliance with the basic restrictions of Table 1. However, lack of compliance with Table 2 does not necessarily indicate lack of compliance with the basic restrictions, but rather

that it may be necessary to evaluate whether the basic restrictions have been met. This would typically be done using analytical methods. If the basic restrictions in Table 1 are not exceeded, then the MPEs in Table 2 can be exceeded. Consequently, it is sufficient to demonstrate compliance with either Table 1 or Table 2.

4.1.2.2 Non-uniform exposure to sinusoidal magnetic fields

When the magnetic field is not constant in magnitude, direction, or relative phase over the head, torso, or limbs, the maximum field over the head, torso, or limbs shall be limited to the values in Table 2. Alternatively, demonstration of compliance with the basic restrictions is permitted.

4.1.2.3 Exposure of the limbs

The MPEs for the limbs (entire arms and legs) are listed in Table 3. The averaging time for an rms measurement is 0.2 s. Compliance with Table 3 ensures compliance with the basic restrictions of Table 1. However, lack of compliance with Table 3 does not necessarily indicate lack of compliance with the basic restrictions, but rather that it may be necessary to evaluate whether the basic restrictions are met.

Table 3—MPE for the limbs: *f* = 3 kHz to 5 MHz

Frequency range (kHz)	Action level[a]		Persons in controlled environments	
	B_{rms} (mT)	H_{rms} (A/m)	B_{rms} (mT)	H_{rms} (A/m)
3.0–3.35	3.79/*f*	3016/*f*	3.79/*f*	3016/*f*
3.35–5000	1.13	900	1.13	900
NOTE—*f* is expressed in kHz.				
[a]Within this frequency range the term "action level" is equivalent to the term "general public" in IEEE Std C95.6-2002.				

4.1.2.4 Pulsed or non-sinusoidal magnetic fields

When the magnetic flux density waveform is non-sinusoidal, such as with pulsed or mixed frequency waveforms, the MPE shall conform to the rms values of Table 2 and Table 3. In addition, the maximum exposure shall conform to either 4.1.2.4.1 or 4.1.2.4.2. Since both criteria are conservative, adherence to either is sufficient to demonstrate compliance with the MPEs or BRs.

4.1.2.4.1 Restriction based on peak (temporal) field

Demonstration of compliance with either 4.1.2.4.1.1 or 4.1.2.4.1.2 is sufficient to demonstrate compliance with restrictions based on the temporal peak field. Subclause 4.1.2.4.1.1 applies to the in situ induced electric field; 4.1.2.4.1.2 applies to the external field.

4.1.2.4.1.1 Peak *in situ* electric field

The temporal peak of the *in situ* electric field shall be restricted to a value obtained by multiplying the rms values of Table 1 by $\sqrt{2}$. To interpret this table for non-sinusoidal waveforms, frequency, *f*, is defined as $f = 1/(2\ t_p)$, where t_p is the phase duration of a peak excursion of the *in situ* electric field. Phase duration is defined as the time between zero crossings of a waveform. For an exponential waveform, t_p is interpreted as the duration measured from the waveform peak to a point at which it decays to e^{-1} (~36.8%) of its peak

value. Peak limits apply to instantaneous values measured through a bandwidth from zero to the highest frequency content of the waveform under consideration.

4.1.2.4.1.2 Peak external field

The temporal peak of the external magnetic field, B, shall be limited according to the following procedure, where B is a time-varying flux density waveform whose compliance is under evaluation. For conversion of magnetic field intensity, H, to magnetic flux density, B, note that $B = 4\pi \times 10^{-7} H$.

a) Determine the time derivative of the external field, $\frac{dB}{dt} = \dot{B}$

b) Identify the peak and phase duration of any excursion of $\dot{B}$. Phase duration shall be determined as in 4.1.2.4.1.1.

c) Determine the allowable peak limit on B from Table 2 or Table 3 as $\dot{B}_p = \sqrt{2} MPE_{\mathrm{B}}(2\pi f)$,

where

$\dot{B}_p$ is the maximum permissible value of $\dot{B}$,

MPE_{B} is the peak flux density consistent with Table 2 or Table 3,

$f = 1/(2t_{\mathrm{p}})$, and

t_{p} is the phase duration of a peak excursion of $\dot{B}$.

4.1.2.4.2 Restriction based on Fourier components

The requirements of this subclause can be satisfied as an alternative to 4.1.2.4.1.

For an exposure waveform consisting of multiple frequencies, a test for compliance of the exposure waveform shall satisfy the following criterion:

$$\sum_{0}^{5\ \mathrm{MHz}} \frac{A_i}{MPE_i} \le 1 \qquad (2)$$

where A_i is the magnitude of the i^{th} Fourier component of the exposure waveform, and MPE_i is the maximum permissible exposure (Table 2 or Table 3) or the basic *in situ* field restriction (Table 1) for a single sinusoidal waveform at a frequency f_i. The summation is carried out from the lowest frequency of the exposure waveform to a maximum frequency of 5 MHz. Note that A_i and MPE_i must measure the same quantity, as well as be in the same units. For instance, if A_i is the magnitude of a flux density, then MPE_i must also be a measure of flux density. Alternatively, both A_i and MPE_i could be measures of the time derivative of the field, the induced in situ electric field, or induced current density.

It may be necessary to evaluate Equation (2) at frequencies outside the limits of this standard. For purposes of such evaluations, the MPE_i values applying to frequencies less than 3 kHz shall be determined as follows:

a) *Basic restrictions* (Table 1). The BRs from 0 Hz to 3 kHz shall be determined as given in Table 1.

b) *Magnetic field MPEs* (Table 2 and Table 3). The MPE for B or H below 3 kHz shall be determined as given in IEEE Std C95.6-2002.

c) *Electric field MPEs* (Table 4). The MPE for the external electric field below 3 kHz shall be determined as given in IEEE Std C95.6-2002. The MPE applicable to 3 kHz shall be assumed up to a maximum frequency of 5 MHz.

d) *Induced and contact current MPEs* (Table 5). Values of induced and contact current below 3 kHz shall be determined as given in IEEE Std C95.6-2002.

4.1.3 MPE for the external electric field

4.1.3.1 Uniform whole-body exposure to sinusoidal electric fields

Table 4 lists MPEs in terms of the undisturbed (absent a person) external electric field, E. It is assumed that the undisturbed field is constant in magnitude, direction, and relative phase over a spatial extent that would encompass the human body. The averaging time for an rms measurement shall be 0.2 s. For a controlled environment in which an exposed individual is not within reach of a grounded object, it may be acceptable to exceed the MPEs in Table 4. This standard does not specify limits for situations involving contact with ungrounded objects, e.g., a person above ground on an elevated, insulated platform. (See Annex C.)

Table 4—Electric field MPE–whole body exposure: *f* = 3 kHz to 100 kHz

	Action level[a]	**Persons in controlled environments**
Frequency range (kHz)	E (rms) (V/m)	E (rms) (V/m)
3 kHz to 100 kHz	614	1842
[a]Within this frequency range the term "action level" is equivalent to the term "general public" in IEEE Std C95.6-2002.		

4.1.3.2 Non-uniform or localized exposure to sinusoidal electric fields

When the external electric field is not constant in magnitude, direction, and relative phase over the dimensions of the human body, the spatially averaged external field (i.e., the fields are averaged as opposed to averaging the squares of fields as at higher frequencies) shall be restricted to the MPE in Table 4. For a controlled environment in which an exposed individual is not within reach of a grounded conducting object, it may be acceptable to exceed the MPE in Table 4. This standard does not specify limits for such cases. In no case shall the BRs of Table 1 or the contact current limits of Table 5 be exceeded.

4.1.3.3 Pulsed or non-sinusoidal electric fields

When the waveform of the external electric field is non-sinusoidal, such as with pulsed or mixed frequency waveforms, the rms value of the spatially averaged external field shall conform to the MPEs of Table 4, and also to either of the criteria stated in 4.1.2.4.1 and 4.1.2.4.2. For this application, the external magnetic field is replaced by the undisturbed electric field; A_i is understood to represent the magnitude of the i^{th} Fourier component of the external electric field waveform, and MPE_i is the maximum permissible electric field magnitude at frequency f_i.

4.1.4 Contact and induced current limits

4.1.4.1 Sinusoidal current

Contact and induced current shall be limited as specified in Table 5, subject to the following conditions:

a) Table 5 limits for freestanding individuals without contact with conducting objects shall not exceed the induced current values listed in the rows labeled "Both feet" and "Each foot."

b) Contact limits in Table 5 assume a freestanding individual who is insulated from ground while touching a conductive path to ground. The criteria do not necessarily protect against aversive sensa-

tions from spark discharges just prior to, and just after the moment of direct contact with the ground path.

c) The averaging time for rms current measurements shall be 0.2 s. The limits for peak exposure refer to instantaneous values measured through a bandwidth from zero to the maximum frequency determined by Fourier decomposition of the waveform of interest.

d) In controlled environments, limits for grasping contacts apply where personnel are trained to make grasping contact and to avoid touch contacts with conductive objects that present the possibility of painful contact current. A grasp contact area is assumed to be 15 cm^2. The use of protective gloves, the prohibition of metallic objects, or the training of personnel may be sufficient to assure compliance with the contact current limit in controlled environments. For the general public, it is assumed that access, methods of contact, and protective measures are unconstrained.

e) A touch contact is assumed to have a contact area of 1 cm^2.

f) For long exposure duration (t >>1 s), the values of induced and contact currents in Table 7 for protecting against heating effects in the RF range, are more restrictive than the corresponding values of currents in Table 5 for frequencies greater than 100 kHz. Hence, for long exposure duration, compliance with this standard at frequencies greater than 100 kHz will be associated with meeting the limits of Table 7.

g) The limits in Table 5 protect against adverse electrostimulation effects; the MPEs in Table 8 and Table 9 apply to effects associated with tissue heating.
NOTE—All three tables must be considered and the corresponding values for the appropriate exposure group satisfied at all applicable frequencies.

Table 5—RMS induced and contact current limits for continuous sinusoidal waveforms, *f* = 3 kHz to 100 kHz

Condition	Action level[a] (mA)	Persons in controlled environments (mA)
Both feet	0.90*f*	2.00 *f*
Each foot	0.45*f*	1.00 *f*
Contact, grasp[b]	—	1.00 *f*
Contact, touch	0.167*f*	0.50 *f*
NOTE 1—f is expressed in kHz.		
NOTE 2—Limits apply to current flowing between the body and a grounded object that may be contacted by the person.		
NOTE 3—The averaging time for determination of compliance is 0.2 s.		
[a]Within this frequency range the term "action level" is equivalent to the term "general public" in IEEE Std C95.6-2002.		
[b]The grasping contact limit pertains to controlled environments where personnel are trained to make grasping contact and to avoid touch contacts with conductive objects that present the possibility of painful contact.		

4.1.4.2 Non-sinusoidal (pulsed or mixed frequency) current

When the current waveform is non-sinusoidal, such as with pulsed or mixed frequency waveforms, exposures shall conform to the rms MPEs of Table 5, and also to either of the criteria stated in 4.1.2.4.1 and 4.1.2.4.2. For this application, the external field is replaced by the applied current, A_i is understood to represent the magnitude of the i^{th} Fourier component of the current waveform, and MPE_i is the maximum permissible current magnitude at frequency f_i.

4.2 BRs and MPEs for frequencies between 100 kHz and 3 GHz

4.2.1 BRs for whole-body exposure for frequencies between 100 kHz and 3 GHz

The whole-body-average BRs shown in Table 6 are based on established adverse health effects associated with heating of the body during whole-body exposure (see Annex C.2 for explanation). Consistent with the approach used in the prior standard to derive exposure limits, a traditional safety factor of ten (10) has been applied to the established SAR threshold for such effects, yielding an SAR of 0.4 W/kg averaged over the whole body. In the absence of an RF safety program, the BRs of the lower tier (action level) may also be used for the general public. Applied to members of the general public, the lower tier provides more assurance that continuous, long-term exposure of all individuals in the population, will be without risk of adverse effects.

Table 6—BRs for frequencies between 100 kHz and 3 GHz

		Action level[a] SAR[b] (W/kg)	Persons in controlled environments SAR[c] (W/kg)
Whole-body exposure	Whole-Body Average (WBA)	0.08	0.4
Localized exposure	Localized (peak spatial-average)	2[c]	10[c]
Localized exposure	Extremities[d] and pinnae	4[c]	20[c]
[a]BR for the general public when an RF safety program is unavailable.			
[b]SAR is averaged over the appropriate averaging times as shown in Table 8 and Table 9.			
[c]Averaged over any 10 g of tissue (defined as a tissue volume in the shape of a cube).*			
[d]The extremities are the arms and legs distal from the elbows and knees, respectively.			

*The volume of the cube is approximately 10 cm^3.

4.2.2 BRs for localized exposure for frequencies between 100 kHz and 3 GHz

In the transition region of 100 kHz to 5 MHz, two sets of BRs apply: c.f. Table 1 and Table 6. The localized exposure BRs shown in Table 6 are established to protect against excessive temperature rise in any part of the body that might result from localized or non-uniform exposure. When averaging SAR over a 10-g volume of tissue in the extremities and the pinnae, only SAR values for that tissue may be considered. If any cubic volume contains tissue from both the body and the extremities or pinna, each must be considered separately. Specifically, when determining the average SAR in a 10 g cube of tissue in the body, any lack of tissue contained in the cube from the extremities or pinna should be treated as air, i.e., mass = 0 and SAR = 0. In addition, the orientations of the cubes used for SAR averaging should align with the coordinate axes

used in the experimental measurement or numerical computational procedures. Detailed methodology for measurement and calculation using the appropriate averaging volume can be found in IEEE Std C95.3-2002.

4.2.3 Contact and induced current limits, 100 kHz to 110 MHz

In the transition region of 100 kHz to 5 MHz, two sets of contact and induced current limits apply. The limits in Table 5 protect against effects associated with electrostimulation and the limits in Table 7 protect against effects associated with tissue heating. Contact and induced current shall both be limited as specified in Table 7, subject to the conditions enumerated in 4.1.4.1, except for a greater averaging time. Figure 1 (upper tier) and Figure 2 (lower tier) provide E-field values below which induced current does not have to be measured. The electric field strength values plotted in Figures 1 and 2 are derived from estimated induced body currents from exposure to uniform electric fields (typically far field exposures) aligned with the axis of the body of a 1.75 m tall individual standing in good conductive contact with ground (Gandhi et al. [R346][7], Tofani et al. [R575]). These assumed exposure conditions will often not be applicable to realistic exposures with the result that substantially higher electric field strengths will be required to produce the induced body or touch current limits specified in this standard. For example, normal footwear can significantly reduce induced body current. In addition, the currents specified in this standard in the 100 kHz to 100 MHz frequency range are to be time-averaged over either 6 minutes or 30 minutes. Moreover, the values for induced current are based on the assumption that all current will flow through one foot to ground, resulting in a conservative indication of field strength below which induced current measurements are not required.

Table 7—RMS induced and contact current limits for continuous sinusoidal waveforms, *f* = 100 kHz to 110 MHz

Condition	Action level[a] (mA)	Persons in controlled environments (mA)
Both feet	90	200
Each foot	45	100
Contact, grasp[b]	—	100
Contact, touch	16.7	50
NOTE 1—Limits apply to current flowing between the body and a grounded object that may be contacted by the person.		
NOTE 2—The averaging time for determination of compliance is 6 minutes.		
[a]MPE for the general public in absence of an RF safety program.		
[b]The grasping contact limit pertains to controlled environments where personnel are trained to make grasping contact and to avoid touch contacts with conductive objects that present the possibility of painful contact.		

[7]The number in brackets preceded by "R," e.g., [R342] corresponds to citations in the International EMF Project (IEEE/WHO) database and are listed in Annex F.

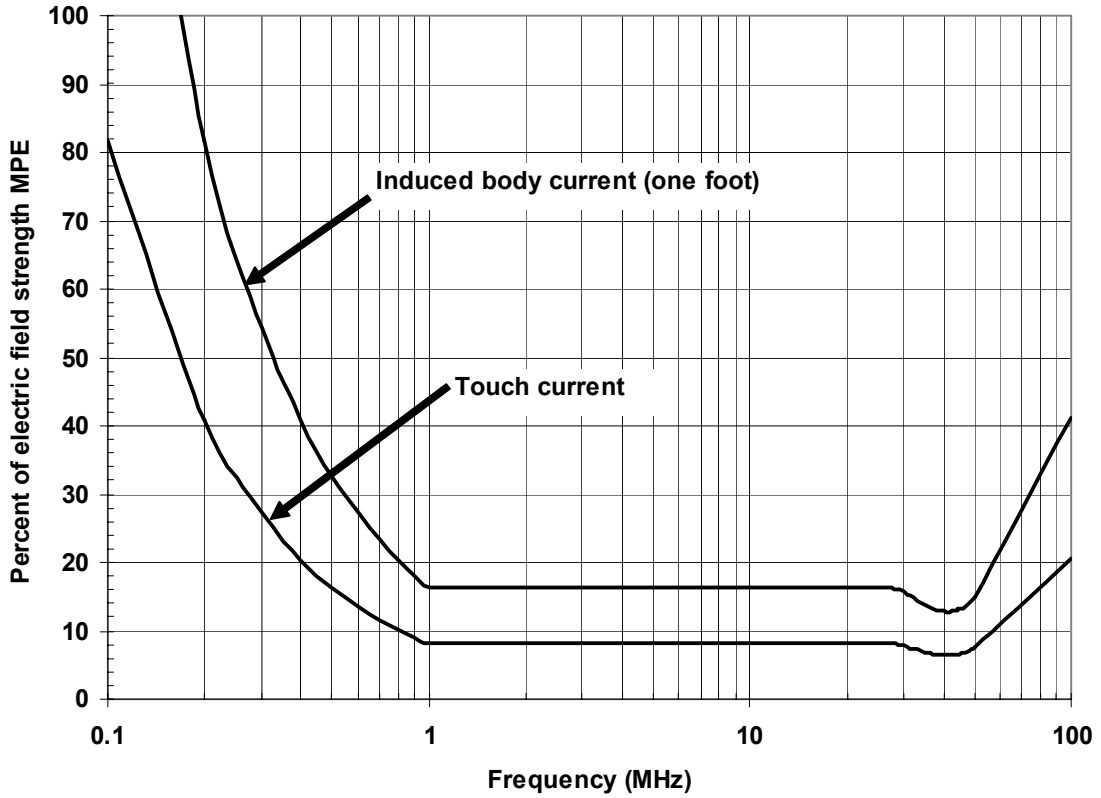

Figure 1—Percent of electric field strength MPE below which the induced current through one foot, or the touch current, will meet the MPEs of Table 7 for the upper tier (exposures in controlled environments); based on a body height of 1.75 m.

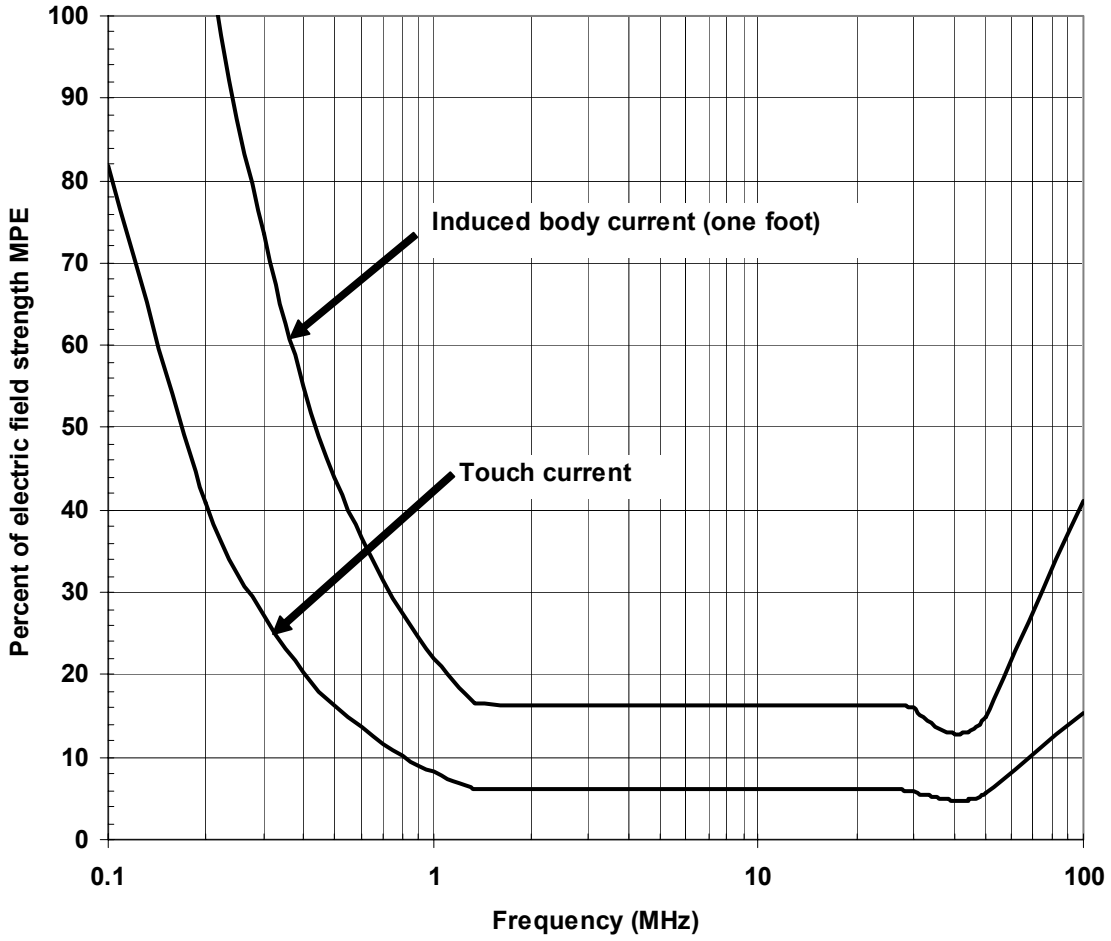

Figure 2—Percent of electric field strength MPE below which the induced current through one foot, or the touch current, will meet the MPEs of Table 7 for the lower tier; based on a body height of 1.75 m.

4.3 BRs for frequencies between 3 GHz and 300 GHz

BRs to protect against adverse effects associated with heating are established for incident power density for frequencies between 3 GHz and 300 GHz. Such restrictions are derived with consideration of adverse effects thresholds (based on the literature review and evaluation), their distribution among the population, and safety factors. The BRs for frequencies between 3 GHz and 300 GHz are the same as the corresponding MPEs shown in Table 8 and Table 9, and are considered appropriate for all human exposure.

4.4 MPEs for frequencies between 100 kHz and 300 GHz

Because of the difficulty in determining whether an exposure complies with the BRs, derived limits (MPEs) to protect against adverse effects associated with heating are provided below for convenience in exposure assessment. For human exposure to electromagnetic energy at radio frequencies from 100 kHz to 300 GHz, the MPEs, in terms of rms electric (E) and magnetic (H) field strengths and the equivalent plane-wave free-space power densities (S) are presented as a function of frequency in Table 8 and Table 9. For multiple field exposure situations, e.g., different frequency field sources, compliance is determined by summing the per-

centages of the applicable MPEs that each frequency field represents and ensuring that this sum does not exceed 100 percent. When fields at multiple frequencies are present, the use of Figure 1 or Figure 2 to assess whether induced or contact currents need to be measured is not practical. If exposure levels are determined via theoretical analysis, consideration of possible reflections of fields must be included.

Compliance with Table 8 and Table 9 ensures compliance with the BRs on whole-body average SAR. However, lack of compliance with Table 8 and Table 9 does not necessarily imply lack of compliance with the BRs, but rather that it may be necessary to perform additional evaluations to determine whether the BRs have been met. If the BRs given above are not exceeded, the MPEs in Table 8 and Table 9 can be exceeded. Consequently, it is sufficient to demonstrate compliance with either the whole-body BRs or Table 8 and Table 9.

Table 8—MPE for the upper tier (people in controlled environments) (see Figure 3 for graphical representation)

Frequency range (MHz)	RMS electric field strength (E)[a] (V/m)	RMS magnetic field strength (H)[a] (A/m)	RMS power density (S) E-field, H-field (W/m^2)	Averaging time $\|E\|^2$, $\|H\|^2$ or S (min)
0.1–1.0	1842	$16.3/f_M$	$(9000, 100\,000/f_M^2)$[b]	6
1.0–30	$1842/f_M$	$16.3/f_M$	$(9000/f_M^2, 100\,000/f_M^2)$	6
30–100	61.4	$16.3/f_M$	$(10, 100\,000/f_M^2)$	6
100–300	61.4	0.163	10	6
300–3000	–	–	$f_M/30$	6
3000–30 000	–	–	100	$19.63/f_G^{1.079}$
30 000–300 000	–	–	100	$2.524/f_G^{0.476}$

NOTE—f_M is the frequency in MHz, f_G is the frequency in GHz.

[a]For exposures that are uniform over the dimensions of the body, such as certain far-field plane-wave exposures, the exposure field strengths and power densities are compared with the MPEs in the Table. For non-uniform exposures, the mean values of the exposure fields, as obtained by spatially averaging the squares of the field strengths or averaging the power densities over an area equivalent to the vertical cross section of the human body (projected area), or a smaller area depending on the frequency (see NOTES to Table 8 and Table 9 below), are compared with the MPEs in the Table.

[b]These plane-wave equivalent power density values are commonly used as a convenient comparison with MPEs at higher frequencies and are displayed on some instruments in use.

Table 9—Action level (MPE for the general public when an RF safety program is unavailable) (see Figure 4 for graphical representation)

Frequency range (MHz)	RMS electric field strength (E)[a] (V/m)	RMS magnetic field strength (H)[a] (A/m)	RMS power density (S) E-field, H-field (W/m^2)	Averaging time[b] $\|E\|^2$, $\|H\|^2$ or S (min)	
0.1–1.34	614	$16.3/f_M$	$(1000, 100\,000/f_M^2)$[c]	6	6
1.34–3	$823.8/f_M$	$16.3/f_M$	$(1800/f_M^2, 100\,000/f_M^2)$	$f_M^2/0.3$	6
3–30	$823.8/f_M$	$16.3/f_M$	$(1800/f_M^2, 100\,000/f_M^2)$	30	6
30–100	27.5	$158.3/f_M^{1.668}$	$(2, 9\,400\,000/f_M^{3.336})$	30	$0.0636\,f_M^{1.337}$
100–400	27.5	0.0729	2	30	30
400–2000	–	–	$f_M/200$	30	
2000–5000	–	–	10	30	
5000–30 000	–	–	10	$150/f_G$	
30 000–100 000	–	–	10	$25.24/f_G^{0.476}$	
100 000–300 000	–	–	$(90f_G-7000)/200$	$5048/[(9f_G-700)f_G^{0.476}]$	

NOTE—f_M is the frequency in MHz, f_G is the frequency in GHz.

[a]For exposures that are uniform over the dimensions of the body, such as certain far-field plane-wave exposures, the exposure field strengths and power densities are compared with the MPEs in the Table. For non-uniform exposures, the mean values of the exposure fields, as obtained by spatially averaging the squares of the field strengths or averaging the power densities over an area equivalent to the vertical cross section of the human body (projected area) or a smaller area depending on the frequency (see NOTES to Table 8 and Table 9 below), are compared with the MPEs in the Table.

[b]The left column is the averaging time for $|E|^2$, the right column is the averaging time for $|H|^2$. For frequencies greater than 400 MHz, the averaging time is for power density S

[c]These plane-wave equivalent power density values are commonly used as a convenient comparison with MPEs at higher frequencies and are displayed on some instruments in use.

NOTES TO TABLE 8 AND TABLE 9

a) The MPEs refer to exposure values obtained by spatially averaging the electric and magnetic field strengths, the squares of the electric and magnetic field strengths, or the plane wave equivalent power densities along a line corresponding to the axis of the human body as follows:

Frequencies between 100 kHz and 3 GHz: The MPE for fields between 100 kHz and 3 GHz are derived on the basis of limiting the whole body averaged (WBA) SAR, which is proportional to the spatial average of the incident plane wave equivalent power density (or squares of electric and magnetic field strengths), averaged over the projected area of the body. Therefore, the MPE corresponds to the spatially averaged plane wave equivalent power density or the spatially averaged values of the squares of electric and magnetic field strengths. In practice, a measurement over the length of the body is sufficient for assessing exposures for comparison with the MPE.

Frequencies greater than 3 GHz: For frequencies greater than 3 GHz, the MPE is expressed in terms of the incident power density. To provide a transition in the frequency range 3 GHz to 6 GHz, compliance with this standard may be demonstrated by evaluation of either incident power density or local SAR. From 3 GHz to 30 GHz, the power density is spatially averaged over any contiguous area corresponding to 100 λ^2, where λ is the free space wavelength of the RF field in centimeters. For frequencies exceeding 30 GHz, the power density is spatially averaged over any contiguous area of 0.01 m^2 (100 cm^2), not to exceed a maximum power density of 1000 W/m^2 in any one square centimeter as determined by a calculation or a conventional field measurement.

b) For near-field exposures at frequencies below 300 MHz, the applicable MPE is in terms of rms electric and magnetic field strength, as given in Table 8 and Table 9, columns 2 and 3. For convenience, the MPE may be expressed as equivalent plane-wave power density, given in Table 8 and Table 9, column 4. For frequencies below 30 MHz, both the rms electric and magnetic field strength must be determined; for frequencies between 30 and 300 MHz, either field component will be sufficient provided that the point in question is in the far-field of the source. For frequencies above 300 MHz, either field component may be used, when expressed as equivalent plane wave power density, for determining compliance with the MPEs in Table 8 and Table 9.

c) For mixed or broadband fields at a number of frequencies for which there are different values of the MPE, the fraction of the MPE [in terms of E^2, H^2, or power density (S)] incurred within each frequency interval should be determined and the sum of all such fractions should not exceed unity. See Annex D for an example of how this is accomplished.

d) In a similar manner, for mixed or broadband induced currents at a number of frequencies for which there are different values of the basic restriction, the fraction of the induced current limits (in terms of I^2) incurred within each frequency interval shall be determined, and the sum of all such fractions should not exceed unity.

e) For exposures to pulsed RF fields, in the range of 100 kHz to 300 GHz, the peak (temporal) value of the MPE for the instantaneous peak E field is 100 kV/m.

f) For exposures to pulsed RF fields in the range of 100 kHz to 300 GHz, the peak pulse power densities are limited by the use of time averaging and the limit on peak E field, with one exception: the total incident energy density during any one-tenth second period within the averaging time shall not exceed one-fifth of the total energy density permitted during the entire averaging time for a continuous field (1/5 of 144 J/kg), i.e.,

$$\sum_{0}^{0.1s} (S_{\text{pk}} \times \tau) \leq \frac{MPE_{\text{avg}} \times T_{\text{avg}}}{5} \leq 28.8 \text{J/kg}$$

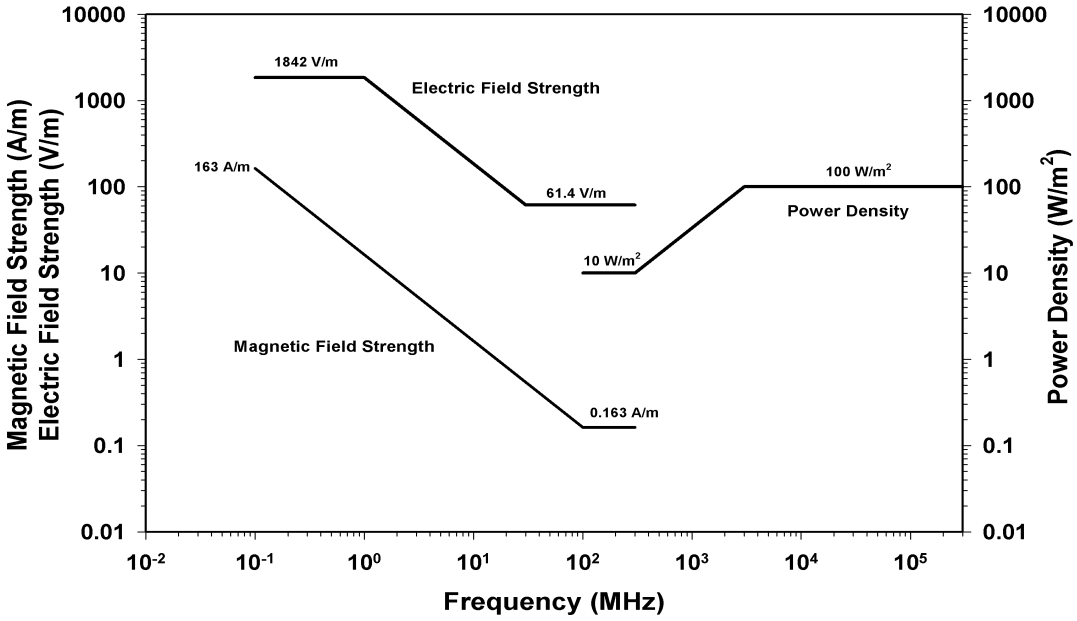

Figure 3—Graphic representation of the MPEs in Table 8 (exposures in controlled environments)

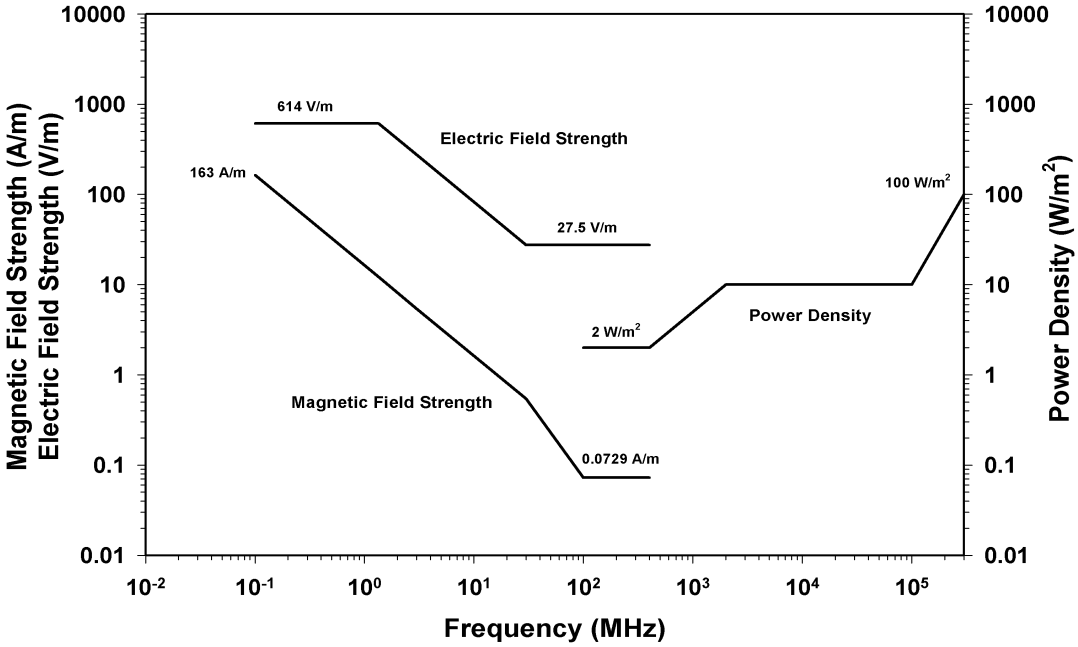

Figure 4—Graphic representation of the MPEs in Table 9 (lower tier—action level)

4.5 Suggested limit for contact voltage to protect against RF burns

In addition to the limits recommended for contact and induced currents, this standard also specifies an MPE for the open circuit voltage that exists on objects exposed to electric and magnetic fields in the frequency range of 0.1 to 100 MHz with which an individual may come into contact. The open circuit voltage is the factor most likely to lead to an electrical arc between the object and a person approaching contact, and hence a localized RF burn of the skin. The maximum suggested open circuit voltage is 140 volts (rms), as measured between any two points of contact with the body, unless it can be shown for specific situations via measurements that a higher open circuit voltage can be tolerated without surface arcing and an attendant RF burn. This suggested limit and rationale should be considered tentative until such time as a more thorough scientific and technical basis for the limit is developed (NAVSEA [B93]).

4.6 Relaxation of the power density MPEs for localized exposures

The following relaxation of the power density MPE is allowed for exposure of any part of the body. Compliance with the MPE of Table 8 (upper tier) is determined from spatial averages of power density or the mean squared electric and magnetic field strengths over an area equivalent to the vertical cross section of the human body (projected area) at a distance no closer than 0.2 m from the field source. The spatial peak value of the power density or mean squared field strength shall not exceed 20 times the square of the allowed spatially averaged values (Table 8) at frequencies below 300 MHz, and shall not exceed the equivalent power density of 200 W/m^2 at frequencies between 300 MHz and 3 GHz, 200 $(f/3)^{1/5}$ W/m^2 at frequencies between 3 and 96 GHz (f is in GHz), and 400 W/m^2 at frequencies above 96 GHz.

Compliance with Table 9 (lower tier) is determined from spatial averages of power density or the mean squared electric and magnetic field strengths over an area equivalent to the vertical cross section of the human body (projected area) at a distance no closer than 0.2 m from the field source. The spatial peak value of the power density or mean squared field strength shall not exceed 20 times the square of the allowed spatially averaged values (Table 9) at frequencies below 400 MHz, and shall not exceed the equivalent power density of 40 W/m^2 at frequencies between 400 MHz and 3 GHz, 18.56 $(f)^{0.699}$ W/m^2 at frequencies between 3 and 30 GHz (f is in GHz), and 200 W/m^2 at frequencies above 30 GHz.

4.7 Assessing compliance with this standard

Compliance with this standard would ideally include a determination that the basic restrictions are not exceeded. For the upper tier in the RF range, this means that the whole-body average and local SARs do not exceed 0.4 W/kg and 10 W/kg, respectively. In practice, however, assessing compliance with this standard will generally consist of determining whether potential exposure of an individual in terms of the various exposure parameters of electric and magnetic fields, plane wave equivalent power densities, contact and induced currents, and contact voltages, exceeds any of the corresponding MPEs. This is less complicated than an assessment of SAR. SAR evaluation may be necessary for some exposure conditions, however, especially for evaluating exposure when the body is extremely close to an RF field source (within the reactive near-field region) and for highly localized exposures. Evaluating whether exposure conditions exceed the upper or lower tier will normally not involve a direct assessment of SARs, but only that the exposure parameters, e.g., Table 8 and Table 9, are not exceeded.

Assessment of exposure to RF fields may be accomplished via either measurements or analysis, using appropriate instrumentation and measurement techniques or numerical/analytical methods. For measurements, reference should be made to IEEE Std C95.3-2002. The MPEs in terms of the RF field or power densities specified in this standard were derived to ensure compliance with the basic restriction on whole-body average (WBA) SARs, and are intended to be applied to field exposure over the entire body in terms of a spatial average. Two issues must be kept in mind when assessing compliance. While the MPEs of this standard are intended to protect against exposures that would result in the WBA SAR exceeding the basic

restrictions, assessment of exposure under conditions wherein the RF fields are strongly non-uniform over the body, typical of near-field exposures, may not in some cases ensure that local SARs will comply with the basic restriction on local SAR of this standard. Moreover, application of the MPEs for fields at locations extremely close to the RF field source (typically in the reactive near-field region wherein there can be substantial coupling between the individual and the source) may not ensure compliance with the basic restriction on local SAR. In both of these special cases, it may be necessary to directly evaluate local SAR through measurement or analysis. Further, care should be used in any RF field measurements conducted extremely close to an RF source to ensure conformance with the minimum measurement distance specified in IEEE Std C95.3-2002; this will prevent inaccurate readings due to probe-field coupling. Generally, however, most commercially available isotropic field probes, even when coupling to the source due to proximity, will read high compared with the actual value. Hence, while it is recommended that the minimum measurement distances prescribed in C95.3-2002 be used, measurements at closer distances will usually be conservative. An exception to this rule is when the measurement probe is large relative to the wavelength of the RF fields being measured.

In cases where the measured exposure parameters approach or exceed the MPE, the more complex evaluation of SAR may be used to make a further determination of compliance with the standard. In many cases, such evaluations may reveal that the SAR basic restrictions are not exceeded. A practical guideline for eliminating the need to assess whether the whole-body average SAR exceeds the basic restriction of 0.4 W/kg (or 0.08 W/kg when the lower tier is used as an exposure limit for the public) is to determine if the power of the source(s) exceeds 28 W (upper tier) or 5.6 W (lower tier) for an average man (70 kg). If the cumulative power of the relevant RF field sources is less than these values, the exposure will not exceed the basic restriction on whole-body average SAR. Such a determination, however, does not necessarily imply that the basic restriction on local SAR would not be exceeded.

4.8 RF safety programs

Throughout the RF spectrum applicable to this standard, the MPEs apply to exposure of people, i.e., compliance with this standard is determined by whether or not exposures of people to RF fields, currents and voltages exceed the applicable MPEs. Where there may be access to RF fields, currents, and/or voltages that exceed the lower tier (Action Level) of this standard, an RF safety program such as detailed in IEEE Std C95.7-2005 shall be implemented to ensure that exposures do not exceed the MPEs or BRs for persons in a controlled environment. Application of an RF safety program results in various mitigative measures that can be taken to reduce the probability of exceeding the MPE for the upper tier. This program typically includes RF awareness training, implementation of protective measures such as signage and the use of personal protective equipment (PPE), incident response, periodic evaluation of program effectiveness, and assigned responsibilities for implementing the program (IEEE Std C95.7-2005).

Annex A

(informative)

Approach to revision of IEEE Std C95.1, 1999 Edition

A.1 ICES revision process

The revision process established by the IEEE International Committee on Electromagnetic Safety (ICES) is a continuing rigorous and open scientific process that is transparent at all levels and includes the opportunity for input from all stakeholders.

A.1.1 Continuity of the IEEE standards revision process

IEEE Std C95.1, 1999 Edition [B70] was first approved by the IEEE Standards Board in 1991 and published as IEEE Std C95.1-1991. The 1991 standard was then reaffirmed in 1997, and then an updated version, containing minor revisions and clarifications, and incorporating IEEE Std C95.1-1991 with IEEE Std C95.1a-1998, was approved and published in 1999, and an amendment that addresses the peak spatial average SAR in the pinnae (IEEE Std C95.1b™-2004 [B71]) was approved in 2004. This standard is a complete revision of IEEE Std C95.1-1991; the revision process implemented by ICES Subcommittee 4 is described below.

A.1.2 Open nature of the IEEE ICES standards development process

IEEE ICES and its Subcommittees are composed of volunteers representing all stakeholders. A balance of representatives from government, industry, academia, and the general public is maintained in accordance with the membership requirements of all standards committees sponsored by the IEEE Standards Association Standards Board (SASB). Subcommittee membership is open to all and consists of volunteers in engineering, physics, statistics, epidemiology, life sciences, medicine, and the public. This wide-ranging participation, including thorough discussions and open decision making, is the hallmark of the process that led to this standard.

A.1.3 Complete reassessment of the technical rationale

IEEE Std C95.1-1991 (and the 1999 Edition) was based on research published before 1986. Research has continued since 1986; a reevaluation of the RF biological effects database was therefore performed. A new risk assessment based on the results of this reevaluation was undertaken. Attempts were made to include and to evaluate all of the relevant literature in the database.

A.1.4 Process for interpretations, clarifications, and appeals

The evaluation of an IEEE standard is a process that is continually ongoing, i.e., IEEE standards are “living” documents. Requests for interpretation and clarification submitted to IEEE ICES by the Secretary of the IEEE-SASB are resolved by special working groups of the ICES subcommittees. The rules and procedures for responding to such requests are included in the ICES Policies and Procedures and are approved by the IEEE-SASB. Valid and applicable comments, received since the last revision, are incorporated in the current revision of the standard by consensus. Appeals of an approved standard are resolved in accordance with the IEEE-SASB Policies and Procedures.

A.1.5 The literature surveillance effort

A Literature Surveillance Working Group (LSWG) was established to compile a citation list of all relevant published literature. At the literature cutoff date of 31 December 2003, approximately 2200 papers had been identified. These were augmented by a few papers and documents appearing in 2004 and 2005. The committee agreed that only peer-reviewed papers and technical reports of original research would constitute the primary database on which any risk analysis would be based. Abstracts and presentations at scientific meetings or technical conferences were expressly excluded from the database. A list of all of the citations is provided in Annex E.

A.1.6 Literature evaluation process

Working groups (WGs) were established to review and evaluate the literature database. These WGs evaluated engineering, epidemiology, *in vivo*, and *in vitro* aspects of individual citations. Additionally, a WG on mechanisms assessed the role of mechanisms of interaction in standard setting and was available to evaluate the technical significance of particular interaction mechanisms. The Engineering WG was tasked with reviewing all papers. The *in vivo* and *in vitro* studies were evaluated in terms of the adequacy of engineering design. The engineering evaluation included assessment of the exposure systems, field characteristics and measurements, dosimetry, specific absorption rates, induced currents and fields, and temperature/humidity measurements. The sufficiency of the information provided in each publication, to allow a full understanding of how the experiment was performed, was paramount.

The Epidemiology WG was originally tasked with the evaluation of each paper for study design and population segments, quality of the methods and implementation, merit of data acquisition and analysis for specific endpoints, and presence or absence of positive statistical associations. Individual papers included possible effects both on specific segments of the general population and on subpopulations occupationally exposed to electromagnetic fields. Because the Chair of the Epidemiology WG changed hands several times; few papers were reviewed because of the lack of available volunteer epidemiologists and a review paper was used instead (see below).

The *In Vivo* and *In Vitro* WGs were tasked similarly to examine the technological methodologies employed in each published paper. Both groups considered the biological entities studied in each paper and their special characteristics. The RF exposure conditions, specific organ systems and/or biological endpoints examined, the engineering and statistical methodologies employed, and provided assessments of the relevance of each study for setting human exposure standards, were evaluated. The *in vitro* papers typically emphasized possible effects at the cellular level, including those on cell viability and proliferation, genotoxicity, cell transformation, molecular synthesis, and cell function. The *in vivo* papers typically examined possible effects of exposure on the whole organism or on specific organ systems, including effects on the embryo/fetus, reproductive ability, immunological system, functional alterations of the metabolic or thermoregulatory system, various histological endpoints, and behavioral changes. As for the engineering evaluations, the adequacy of the information provided in each *in vitro* and *in vivo* publication, which would allow a reasonably knowledgeable research scientist to understand how the study was performed and to independently reproduce it, was essential. The *In Vivo* WG reviewed more than 90% of the *in vivo* papers, but the Epidemiology and *In Vitro* WGs reviewed only a small portion of the papers in their respective areas. This left gaps that, where critical, were filled by the hazard identification process and several review papers, as described in A.1.7, below. See also A.1.9.

The chair of each WG was responsible for providing copies of each paper to two independent reviewers, together with specially designed and approved review forms. These forms were in a computer format that required numerical scoring by individual reviewers for entry into a computerized database. When a review was completed, the reviewer gave the paper an overall technical merit rating on a 5-point scale. The rating scale was: Very High = 5; Moderately High = 4; Acceptable = 3; Low = 2; and Very Low = 1. For ratings of 1 or 2, a request was made for justification in writing by the reviewer. This was not requested for ratings of

3 and above, which were considered acceptable. Strong discordance between the two reviews of a given paper required a third independent review. Periodically, the chair of each WG submitted a summary of the reviews completed to the Chair of the Risk Assessment WG (RAWG). All of the reviews were performed by volunteers, who were randomly selected from within each working group. The identification of each reviewer in a specific paper will remain confidential; the list of reviewers who were active at any time in the review process will be publicly available.

A.1.7 Hazard identification and review papers

As the literature review process proceeded, it became clear that such a very large database would require many years of intense effort to accomplish the goal of identifying any potential hazard to human health or safety resulting from exposure to RF electromagnetic fields (3 kHz to 300 GHz). A special Revision WG was created to prepare a framework for the new standard and to discuss both the extent of the normative content and the informative annexes. As more reviews were being completed, certain individuals with considerable expertise in specific areas volunteered or were asked to prepare review papers to summarize the findings in specific topic areas. These included, for example, cancer induction or promotion, teratologic effects, ocular effects, epidemiology, thermoregulation, and animal behavior [cf. B.1 and B.5]. In each topic area, one of the goals was to search for definable hazards. Summaries and conclusions from each review paper appear in Annex B.

A.1.8 Role of the Risk Assessment Working Group

A literature cutoff date was established (December 31, 2003) as the literature evaluation process continued moving forward. The evaluations of the published papers continued to be submitted by the four WG Chairs to the Chair of the RAWG. The texts and conclusions of the various review papers were made available to the RAWG, whose charge was to evaluate the implied risk for human beings of exposure to RF electromagnetic fields. This activity was supported by a dialogue between RAWG members and a Revision Working Group (ongoing for several years) concerning the format, basis, and details of the new standard.

A.1.9 Current status of the literature evaluation and review process

As the current version of the revised C95.1 standard neared completion and was balloted by SC-4, it became clear that the literature evaluation process would not be completed on time. While the engineering WG evaluated nearly all of the papers in the database and the *In Vivo* WG evaluated more than 90% of their assigned papers, few epidemiology and *in vitro* papers were evaluated by members of their respective WGs. A lack of qualified reviewers was the principal reason for the latter. On the other hand, review summaries of the biological papers (~1300) in the principal database are presented in Annex B. These summaries are further enhanced by the 12 review papers published in Supplement 6, 2003 of *Bioelectromagnetics* [B15], including reviews of the epidemiology and in vitro literature (cf. B.1). The conclusions derived from this extensive review process are based on the weight of evidence approach throughout and form the basis of the current revision of the standard. Committee members believe that the literature review process should be a continuous, ongoing effort; if any new adverse effect is established which would require a change in the standard, the standard can be promptly revised by amendments.

A.2 Basic concepts for developing the MPEs

The process followed by the committee for establishing MPEs with respect to human exposure to RF electromagnetic fields, 3 kHz to 300 GHz, was dependent on the weight of the scientific evidence, a procedure used to develop guidance for assessment of risk from chemical and other physical agents known to be hazardous. These methods have been developed over the years and are widely reviewed (NRC [B99], [B100], [B101]). The process began with a detailed evaluation of the relevant literature in the scientific database,

took advantage of all completed evaluations in the computerized database, and proceeded to a determination of potential hazards to human beings exposed to RF energy (cf. A.1.6 to A.1.9 above), from which thresholds of individual responses and dose response functions were determined.

A.2.1 Publication of novel findings, supportive data, and general acceptance by the scientific community

Many novel experimental studies have been published in the peer reviewed scientific literature, and while of interest, cannot be applied to setting standards for allowable human exposure to RF energy. A number of these studies suffer from poor design, inappropriate or no controls, inadequate dosimetry, physical artifacts, defective measurements, or improper statistical analysis. Other studies suffer from erroneous conclusions and lack of scientific detail. Many published studies failed to replicate or support initially reported effects of RF exposure. The results of other published studies, of high-quality design or exceptional importance, although not independently replicated in the published literature, were seriously considered as part of the risk assessment because supporting evidence was available in that literature. While the body of potentially pertinent science is generally discussed and commented upon in scientific meetings or other forums, informal interchanges do not constitute contributions to a valid risk analysis, and were excluded as anecdotal. Painstaking review by experts of the papers in the scientific database was the only dependable means of sorting the meaningful data from the mediocre or unusable data. These reviews, performed as part of the process for establishing this standard, were careful to differentiate between evidence for a biological effect and that for an adverse human health effect. The procedures detailed above provide the basis for the evaluation of RF hazards and the associated risk assessment used in establishing this standard.

A.2.2 Assessing thresholds and dose-response relationships

For exposures to nonionizing electromagnetic energy, observed individual biological effects, whether adverse or beneficial, are characterized by thresholds and are a function of exposure level. For any given biological response, a threshold can be determined below which the specific response does not occur or is undetectable. Above the threshold level, a function that relates dose rate, e.g., SAR, to response magnitude is determined and the lowest level at which a potential hazard occurs is identified. Exposure limits can then be developed to protect against the occurrence of the effects to human beings.

A.2.3 Selection of safety factors and development of MPEs

Once a hazard threshold has been identified and enough supporting information is available, a safety factor can be applied to the threshold to derive an exposure limit that is based on the best available scientific information using the conservative approach common in standard setting. In practice, the better the hazards involved are understood, the better the numerical foundation for the safety factor, but the choice always relies on professional judgment.

The selection of a “safety factor” is generally an arbitrary process, which presupposes that a hazard has been identified and a threshold has been determined. The safety factor is influenced by the uncertainty in our knowledge of the degree of hazard associated with the hazard exposure threshold and is selected to prevent exceeding the threshold value in human exposure with a sufficiently wide margin. The magnitude of a safety factor may range from unity at low frequencies, where electrostimulatory effects may occur, to significantly greater values at frequencies above 100 kHz, where heating effects may occur. In all cases, however, the selection of the appropriate safety factor is based on informed expert opinion after considering the underlying biological and engineering uncertainties applicable to the exposed population for a broad range of exposure conditions. In this standard, for frequencies above 100 kHz, safety factors are equivalent to SAR or power density reduction factors relative to those exposure values representing the exposure thresholds for hazardous effects. These factors are not necessarily numerically equivalent to the degree of reduction in the resulting manifestation of RF energy absorption, such as an increase in localized tissue temperature.

Annex B

(informative)

Identification of levels of RF exposure responsible for adverse effects: summary of the literature

NOTE—References denoted in brackets with the letter "R" before the number (e.g., [R119]) are references from the IEEE/WHO Literature Database and are found in Annex F. References denoted with the letter "B" before the number (e.g., [B115]) are references that are not in the IEEE/WHO database and are found in the Bibliography (Annex G).

B.1 Introduction

The following summary of the literature is based on critical reviews of studies within the IEEE/WHO RF literature database[8] (see Annex F). Although this standard considers the entire frequency range between 3 kHz through 300 GHz, a majority of the studies evaluated employ RF signals relevant to various communications, industrial, and radar technologies. In general, studies are not identified or separated according to specific frequencies or modulation characteristics, as the evidence does not support frequency or modulation-specific effects except for geometrical resonances for animal and human exposures that result in enhanced heat deposition and thermoregulation in mammalian models at certain frequencies.

Detailed review papers were drafted for twelve general subject areas by individual members of IEEE ICES TC-95/SC4 and were published together at the end of 2003 as Supplement 6 of the journal *Bioelectromagnetics* [B15][9]. These include:

1) Historical Review of RF Exposure Standards and ICES (Osepchuk and Petersen [R1088]),
2) Thermoregulatory Responses to RF Energy Absorption (Adair and Black [R1091]),
3) Behavioral and Cognitive Effects of Microwave Exposure (D'Andrea et al. [R1093]),
4) Cancer, Mutagenesis, and Genotoxicity (Heynick et al. [R1095]),
5) Lifespan and Cancer in Laboratory Mammals Exposed to RF Energy (Elder [R1092]),
6) Microwave Effects on the Nervous System (D'Andrea et al. [R1089]),
7) Ocular Effects of RF Energy (Elder [R1099]),
8) Auditory Responses to Pulsed RF Energy (Elder and Chou [R1096]),
9) Epidemiological Studies of RF Exposures and Human Cancer (Elwood [R1097]),
10) RF Effects on Blood Cells, Cardiac, Endocrine, and Immunological Functions (Black and Heynick [R1111]),
11) RF Fields and Teratogenesis (Heynick and Merritt [R1098]), and
12) RF Exposure and Biological Effects: In vitro Studies with in vivo Correlation (Meltz [R1090]).

The review for this standard includes studies conducted under many different exposure conditions, some using levels of RF energy too low to produce significant heating in animal or *in vitro* test systems (herein referred to as "low-level" exposures rather than "non-thermal" exposures), others using levels of RF energy producing clear RF heating ("thermal"), and others employing conditions where RF currents can cause burns or nerve and muscle stimulation ("shocks"). In all categories particular attention was paid to variables that might occur prior to, or concurrent with RF exposure, and possibly result in effects at lower RF field levels. The IEEE/WHO database was used in developing this revision. References from this database

[8]The entire IEEE/WHO database can also be found at Internet site http://www10.who.int/peh-emf/emfstudies/IEEEdatabase.cfm.
[9]The individual papers can be found at Internet site http://grouper.ieee.org/groups/ scc28/sc4/ contents.html.

(shown in Annex F) are denoted [Rxx]; the number following each citation, e.g., [IEEE-xxx], is the IEEE Accession Number. References that are not in the IEEE/WHO database (usually because they are not studies specifically examining bioeffects of RF) are included in the Bibliography, Annex G, and denoted [Bxx].

B.2 Executive Summary

A review of the extensive literature on RF biological effects, consisting of well over 1300 primary peer reviewed publications published as early as 1950, reveals no adverse health effects that are not thermally related (except for electrostimulation discussed in B.2.4). This conclusion is consistent with those reached by other scientific expert groups and government agencies including the:

- Australian Government, Australian Radiation Protection and Nuclear Safety Agency, Committee on Electromagnetic Energy Public Health Issues (ARPANSA [B9], [B10]),
- European Commission Expert Group (McKinlay et al. [B88]),
- European Committee on Toxicology, Eco-toxicology and the Environment (CSTEE [B27], [B28], [B29]),
- France's Commission for Consumer Safety (the French Expert Report - 'Zmirou report' to the French Health General Directorate) (Zmirou et al. [R787]),
- French Environmental Health and Safety Agency (AFSSE [B2]), (Aran et al. [B8])
- Health Council of the Netherlands (HCN [B55], [B56]),
- Hong Kong-Office of the Telecommunications Authority [B59],
- International Commission on Non-Ionizing Radiation Protection (ICNIRP [B62], [B64]),
- Japanese Ministry of Post and Telecommunications [B76],
- New Zealand Ministry of Health and Ministry of Environment [B99],
- Royal Society of Canada Expert Panel (RSC [B109])
- Singapore Health Sciences Authority (SHSA [B122], [B123]),
- Swedish State Radiation Protection Authority (SSI [B126]),
- U.K. Independent Expert Group on Mobile Phones (IEGMP [B73]),
- U.K. National Radiological Protection Board (NRPB [B103]),
- U.S. Food and Drug Administration (FDA [B42]), and the
- World Health Organization (WHO [B135], [B136]).

Further examination of the RF literature reveals no reproducible low level (non-thermal) effect that would occur even under extreme environmental exposures. The scientific consensus is that there are no accepted theoretical mechanisms that would suggest the existence of such effects. This consensus further supports the analysis presented in this section, i.e., that harmful effects are and will be due to excessive absorption of energy, resulting in heating that can result in a detrimentally elevated temperature. The accepted mechanism is RF energy absorbed by the biological system through interaction with polar molecules (dielectric relaxation) or interactions with ions (ohmic loss) is rapidly dispersed to all modes of the system leading to an average energy rise or temperature elevation. Since publication of ANSI C95.1-1982 [B6], significant advances have been made in our knowledge of the biological effects of exposure to RF energy. This increased knowledge strengthens the basis for and confidence in the statement that the MPEs and BRs in this standard are protective against established adverse health effects with a large margin of safety.

B.2.1 Thermal physiology and associated behavioral responses form the basis of the RF standard

Behavioral studies indicate that a threshold of ~4 W/kg causes disruption of complex behavioral performance in several animal species, including non-human primates, and under diverse exposure conditions. The

disruption of behavior is often (but not always) accompanied by an increase in core body temperature of ~1.0 ºC. These accommodating responses to a thermal challenge, while not considered detrimental, can be compared to the response when humans take off or put on a light sweater to adjust to mild temperature changes. It is this level of impact that is significant in establishing the basis for this standard. However, the extrapolation of these behavioral results from animals to humans is considered conservative. This is because comparable increases in core body temperature are not easily produced in humans due to their more efficient thermoregulatory system. Even at exposure levels considerably higher than current standards allow, human body temperature is efficiently regulated by the mobilization of appropriate heat loss mechanisms, such as sweating and skin blood flow.

Exposure to RF energy produces a sensation of warmth. The sensitivity to exposures has been shown to increase monotonically from microwave to millimeter wave frequencies. Thus much less energy is needed at the higher frequencies to produce a thermal sensation because the depth of penetration at the higher frequencies deposits energy closer to the skin where most thermal sensors are located. The threshold of cutaneous thermal pain is 44–45 ºC and, if generated by RF energy, will result in a prompt effort to escape from the field, thereby preserving normothermia.

B.2.2 Non-cancer related studies supportive of the standard

Studies on teratogenic effects of RF exposure, and other conditions that cause heat stress in animal models, have demonstrated that significant increases in the incidence of heat-induced abnormalities are seen at maternal temperature increases of approximately 2–2.5 °C. This mostly occurs following exposures of tens of minutes up to one hour or so. The results of a few studies reporting teratogenic, reproductive and developmental effects at low levels of RF exposure are generally weak in design, and have not been confirmed independently. The weight of evidence from animal studies supports the conclusion that teratogenic, reproductive, or developmental effects do not occur unless the RF exposure is >4 W/kg and causes a significant temperature increase above the normal body temperature. The weight of evidence from studies of human populations exposed to RF fields from video display units, magnetic imaging devices, medical diathermy units, heat sealers and radar does not suggest that teratogenic, reproductive or developmental effects occur at exposures lower than the upper tier MPEs in this standard.

While studies have reported effects on hematological and immunological endpoints in animals and *in vitro* models, the majority was performed at thermal levels of RF exposure and is most likely the result of heating and elevated temperature. In the few studies that have reported effects at low-level (non-thermal) exposure levels (i.e., below the MPEs), the findings are generally inconsistent with each other, as well as with the larger body of evidence reporting no effects at these exposure levels.

The results of many investigations have confirmed that the permeability of the blood brain barrier (BBB) can be affected by a significant increase in temperature caused by absorption of RF energy, but fail to support a repeatable low-level effect. Based on modeling studies, the localized exposure limit for the lower tier will produce an increase in brain temperature of about 0.2 °C (Van Leeuwen et al. [R711]), (Bernardi et al. [R725], [R1109]), (Gandhi et al. [R1105]), (Van de Kamer and Lagendijk [R1114]) (see B.6.3.2). This increase is very small in comparison with the increase in temperature that is associated with reported changes in BBB permeability. Published reports of permeability changes in the BBB at SARs <4 W/kg have not been confirmed and no exposure- or dose-response relationship is evident.

Adverse effects of RF exposure of the eye (e.g., cataracts) are associated only with significant temperature increases due to the absorption of RF energy. There is no evidence of other significant ocular effects (including cancer) that would support a change in the adverse effect level of 4 W/kg.

The phenomenon of RF hearing in humans is a well-established biological effect with no known adverse health consequence. The RF-induced sounds are similar to other common sounds. A quiet environment is needed for the sounds to be heard.

A few studies have reported effects of RF exposure on *in vitro* membrane function and protein leakage through artificial and cellular membranes. However, significant variability and a lack of a consistent correlation with SAR were common in these responses. It is possible that the RF exposure resulted in local temperature increases, which may have contributed to the observed effects. Numerous studies have documented effects of higher (thermal) levels of RF exposure on membrane fluidity and ion transport.

Several reports that have reviewed the calcium efflux effects literature support the conclusion that, notwithstanding unresolved research questions, calcium efflux effects from exposure to low-level amplitude modulated RF fields cannot be used in setting RF exposure standards. WHO EHC 137 [B137] concluded that insufficient information is available to define these reported weak field interactions, and this observation could not be characterized as a potential adverse health effect. An NRPB report [B103] observed that if the phenomenon of calcium efflux were biologically significant, concomitant changes would be expected in the functions of nervous tissues that depend on the movement of calcium ions. No such functional alterations have been demonstrated unambiguously; the report included the statement that there was no reason to believe that 16-Hz modulation has special effects.

Increases and decreases in both evoked and spontaneous population spikes in hippocampal slices exposed *in vitro* to CW RF energy have been reported, but not supported by similar studies. Reports that modulated RF exposure decreased electrical activity in isolated snail neurons seem to contradict reports that RF exposure either increased firing rate or had no effect on isolated neurons. A number of studies have reported that clearly thermal levels of exposure can result in decreased firing amplitude and a prolonged refractory phase in isolated neurons. However, no effects of even very high levels of RF exposure were observed if cooling techniques were used to prevent temperature elevation.

Various other non-cancer endpoints affected by acute thermal RF exposures to animals have included altered digestive function, increased serum triglyceride and beta-lipoprotein levels, increased rate of liver regeneration, increased tissue water content, and conductivity. These un-replicated studies present no consistent evidence of effects due to RF exposure and are in general inconsistent with long term animal study results that indicate no detrimental effects of exposure at SARs up to 4 W/kg.

A review of human provocation studies, including cognitive function and memory, EEG, sleep disturbances, event related potentials, headache and fatigue, hypersensitivity, and effects on blood pressure/heart rate, showed no consistent evidence of an adverse effect of low-level RF exposure on the nervous system. However, because of the variety of different effects reported by some investigators and many contradictory reports, research in this area continues.

B.2.3 Cancer-related studies

The scientific weight of evidence for 35 animal bioassay studies completed to date provide no evidence of physiological, pathological or disease-specific effects of long-term RF exposure, including lifetime exposures, at levels up to 4 W/kg. Those few studies that have reported effects are either not corroborated in similar studies, or the results could not be verified in specific replication attempts. These long term studies clearly indicate a lack of evidence that RF exposure causes or promotes tumor induction or any other life shorting disease. No adverse effect was found on longevity or body mass as a result of chronic RF exposures at SARs in the range of 0.5–4 W/kg. Even though these studies do not give clear thresholds for thermal effects, they are helpful in defining no observable adverse effect levels (NOAEL) in the long-term studies.

A review of numerous supportive studies addressing cancer and basic cellular interactions show no consistent evidence for a reproducible biological effect of low level (non thermal) RF exposure. These studies include examination of DNA breaks, mutation, specific DNA absorption, chromosome aberration induction, micronucleus formation, sister chromatid exchange induction, DNA repair synthesis, inhibition of DNA repair synthesis, phenotypic mutagenesis, transformation, cell cycle elongation, cell toxicity, proliferation, growth rate, cell cycle analysis, gene and protein expression and activity, and oxidative stress. The majority

of studies report no effect. The magnitude of the reported effects are generally very small, often in the range of variability that normally occurs in clinical laboratory tests ordered by physicians, and thus the direct health implication of such reports would still remain unclear even if they were independently verified.

The epidemiological studies to date do not show clear or consistent evidence to indicate a causal role of RF exposures in connection with human cancer or other disease endpoints. Many of the relevant studies, however, are weak in terms of their design, their lack of detailed exposure assessment, and have potential biases in the data. While the available results do not indicate a strong causal association, they cannot establish the absence of a hazard. They do indicate that for commonly encountered RF exposures, any health effects, if they exist, must be small. Even though epidemiological evidence cannot rule out a causal relationship, the overall weight-of-evidence is consistent with the results of the long term animal studies.

B.2.4 Electrostimulation and effects below 100 kHz

At frequencies below 100 kHz, electrostimulation reaction thresholds will typically be lower than thermal reaction thresholds. Above 100 kHz, however, thermal effects typically exhibit lower thresholds of reaction than do electrostimulation effects when the stimulus waveform is of a continuous oscillatory nature. However, with pulsed waveforms of low duty factor, the frequencies at which electrostimulation thresholds are lower than thermal thresholds can extend into the megahertz region. This occurs because the heating capacity of electric current (i.e., its rms value) is proportional to the square root of the duty factor.

B.3 Role of mechanisms in determination of levels for adverse effects

A sound working knowledge of mechanisms of interaction is desirable for unification and simplification of health and safety standards in face of the variety and complexity of biological systems, the multitude of technological applications that constitute the electromagnetic environment, and the resulting potential for compounded complexity upon their interaction. Ideally, a thorough understanding of interaction mechanisms can be used to develop quantitative models for exposure that would allow calculation of dose in a biologically significant manner. The analysis of biological and biophysical mechanisms also permits evaluation of the plausibility of various theories proposed to explain laboratory results and others offered as speculations. Of even more importance, well-established quantitative mechanisms reduce uncertainty for the physical and biological measures used to assess health and safety effects for exposed people. Consequently, there could be reduced uncertainty about the sufficiency of the health and safety guidelines.

Mechanisms of interaction play a critical role in application of results from studies with laboratory animals to human beings. In the case of exposure to fields over the range 3 kHz to 300 GHz, physical mechanisms of interaction greatly influence which tissues and organs are affected and to what degree. Biological mechanisms for thermoregulation, sensory responses to skin temperature and auditory responses to pulsed fields (“microwave hearing”) are critical factors for utilization of results from studies with laboratory animals and human subjects. Likewise, meaningful investigations of speculative mechanisms for biological effects require that the mechanism be specified in a way that allows conduct of experiments at comparable levels. The foregoing remarks also apply to *in vitro* research, where the experimenter needs to establish plausibility for the hypothesis in terms of both dosimetry and biological mechanisms.

Standards development requires differentiation between proven and speculative mechanisms. Proven mechanisms have been established for RF interactions in human beings, with these exhibiting thresholds of reaction that are understood in terms of established biophysical and biological principles. An established mechanism that does not produce adverse effects, even at high doses, is not useful for setting the quantitative limits required in standard setting. On the other hand, speculative mechanisms are those that are not sufficiently well understood to define the threshold of interaction in human beings, and may not have confident support from the experimental literature. However, a speculative mechanism can be useful for designing experiments that will allow for an experimental determination of biological activity. A well established

effect on biological cells might be speculative because its application to intact human beings is not presently understood or demonstrated. Mechanisms established in one species, but of uncertain applicability to humans, provide another example of a speculative mechanism in the context of standard setting. Such speculative mechanisms require monitoring and possible reevaluation in the future.

A number of speculative and established mechanisms of RF interactions with biological systems have been proposed and representative samples are listed below. Most of these are speculative and have no support from a review of the biological literature, i.e., no consistent low level effect. The last three categories (thermal, strong field effects, and electrostimulation) are established effects that are used as the basis of this standard.

a) Resonant Interactions
    1) Vibrational
        i) Molecular resonance in atom-atom interaction models
        ii) Water damping makes all features at potentially interesting frequencies into bulk (thermalized) modes below several hundred gigahertz
        iii) Very soft modes exist without a sharp limit; softest yet demonstrated and calculated is at 150 GHz
    2) Electronic
        i) Chemistry - redox reactions
        ii) Electron tunneling in proteins
        iii) Radical pair mechanism
b) Non-resonant interactions (dynamical, chemical, statistical, etc.)
    1) Electric dipole interaction (E)
        i) Cooperative interactions (including dipole-dipole interactions suggested by Fröhlich [R577])
        ii) Magnetic dipole interaction, e.g., atomic magnetic moments, magnetite (heating and mechanical forces on gating charges)
        iii) Ion transporters, channels that depend on charge and voltage; transporters of molecules (transmitters, hormones)
    2) Conformational change in two classes:
        i) Chemical, where there is thermal activation and chemical rate constants are defined (e.g., Na-K ATPase, polymerases, cyclohexane);
        ii) Molecular absorption of RF energy
    3) Molecular motors for transfer of neurotoxicants, hormones, general exocytosis, etc.
    4) Anomalous energy diffusion via normal modes
    5) Non-equilibrium dynamical effects
    6) Nonlinear molecular energy transfer (solitons)
c) Thermal
    1) Systemic reactions (thermoregulatory system)
    2) Localized heating
    3) Microthermal (putative, shown to be insignificant)
d) Strong field effects having no weak field analogy
    1) High field strength short pulsed fields
    2) Short pulsed RF fields
    3) RF shocks and burns
e) Electrostimulation

The speculative mechanisms among those above have been evaluated by several theoreticians who concluded from fundamental physical principles that such mechanisms would not produce detectable effects in biological systems for the exposure levels and frequencies considered in this standard (Adair [R3], [R805], [R978], Weaver and Astumian [R134], Astumian and Weaver [R559]). Establishment of the mechanisms (e.g., electrostimulation and thermal) that may cause harm is important for standard setting, especially insofar as it provides the technical means to extrapolate data from animals to humans, to determine thresholds using mathematical models, and to extrapolate results obtained at specific radio frequencies to all frequencies within the RF spectrum.

B.4 Improvements in dosimetry

Accurate dosimetry is essential for an understanding of biological effects, since even uniform exposures lead to non-uniform absorption in almost all *in vivo* and *in vitro* exposure situations. Developments in this area have been very successful. Sophisticated models can now be used to reliably estimate *in situ* electric fields and SARs for a wide range of frequencies and other technical and anatomical parameters. Numerical methods of calculation that use the finite-difference time-domain (FDTD) technique to estimate *in situ* fields and SAR have grown in sophistication and usefulness. FDTD results are notable because of the ability to resolve RF fields at the millimeter level using accurate anatomical models based on high-resolution MRI images. The FDTD method joins other methods for dosimetric calculations that have played an essential role in setting the correspondence between exposures to external fields and the *in situ* electric field, SAR, and current density (see C.7.6). The last three are direct measures that can be related to any adverse effects of RF energy on body tissues, organs, and the whole body, although there are no practical means to set standards that require direct measurement of *in situ* fields. Spectral content, temporal and spatial patterns, and polarization are some of the additional factors of the electromagnetic environment that may be important for assessment of a biological effect.

It bears emphasis that SAR is a measure of the rate of energy absorption in a unit mass of tissue and does not in itself define a mechanism of interaction. As discussed elsewhere, the mechanism of interaction that has been selected as a basis for parts of the RF portion of this standard is heating of the body, which may be accompanied by an increased body temperature if heating overcomes the heat loss mechanisms that act to maintain constant body temperature. In principle, SAR might also be used to quantify mechanisms of interaction that do not involve a temperature increase. However, the electric field strength in tissue, which can easily be calculated from SAR (and vice versa) if tissue conductivity and density are known, is the more appropriate measure for cell membrane polarization effects in excitable tissue that are the basis for some parts of this standard. The spatial and temporal distribution of electrical forces that influence excitable membrane effects will be quite different from those that determine a thermal effect. In general, the amplitudes and time variations in SAR, electric field strength and magnetic field strength may each be appropriate for specific proposed alternative mechanisms of interaction.

B.5 Established effects forming the basis of the standard

This subclause extensively reviews the known database of established thermal effects at RF frequencies above 100 kHz and provides a brief overview of electrostimulation, which is the basis for the standard below 100 kHz.

B.5.1 Thermoregulation

B.5.1.1 Review of thermoregulation studies

In humans, efficient thermophysiological responses exist for maintaining an optimal body temperature in response to added thermal energy. The usual range of body temperature in humans extends from 35.5 to 40 °C, and is routinely influenced by circadian variation, vigorous exercise, variations in ambient conditions, sequelae of food intake, menstrual variation in women, emotional factors, and assorted effects of drugs and alcohol. Age can also play an important role due to differences in surface to volume ratio, sweating capacity, and cardiac function and output (Makrides et al. [B85], [R1009], [R1010], and Webster [B134]). At elevated body temperatures, increases in metabolism, heart and respiration rate, and nerve conduction velocity can occur. At temperatures above ~42 °C, central nervous system function can deteriorate and convulsions may occur. At this level protein denaturation may begin and cells may be damaged. Sustained exposure to this level in humans often leads to irreversible neurological and cardiac damage (Mambo et al. [R1011], Britt et al. [B18], and Hales et al. [B51]).

Other consequences of severe and prolonged hyperthermia include confusion, unconsciousness, increased heart rate, lowered blood pressure (Gathiram et al. [R1110]), elevated enzyme activity, and damage to the heart and kidneys. Thermoregulatory responses may cease above 43 °C (heat stroke), after which body temperature may rise rapidly if external cooling is not imposed. Several factors can influence the thermal sensitivity of specific tissues in response to occupational or accidental exposure to high RF fields, including thermal tolerance, pH, nutrition, and pressure effects. Additional factors include the phenomena referred to as “step up” and “step down” heating. The effects of these factors on thermal sensitivity are fairly well characterized, and can be described quantitatively based on Arrhenius analysis (Dewhirst et al. [R1080]). As an example, the intestines contain a large quantity of highly toxic lipopolysaccharide (LPS, an endotoxin) that can be sloughed from the walls of gram-negative bacteria residing in the intestine; hyperthermia to 42–43 °C can lead to significant damage due to increased entry of LPS into the circulation.

The initial response to thermal loads in animals involves a lowering of metabolic rate to reduce heat generation (Adair and Adams [R293]). This response occurs in humans only in very cold environments when heat production is elevated through shivering. During intense warming or vigorous exercise, or whenever the temperature of heated tissue exceeds ~41 °C (Cunningham [R878]), peripheral and deep blood vessels dilate causing peripheral and/or local blood flow to increase as much as 10-fold (Gordon et al. [R53], [R54], Lotz and Saxton [R91], [R92], Adair et al. [R297], Bruce-Wolfe and Adair [R314], Candas et al. [R317], Jauchem and Frei [R589], Adair [R898], and Gordon [R903]). Each liter of blood (at 37 °C) that flows to the skin can return as much as 1 °C cooler and allow the body to lose up to 1.16 W·h (watt hour) of heat (Hardy [R881]). Sweating is activated when the ambient temperature rises above ~30 °C or the internal body temperature rises above ~37 °C (Wenger [R897]), although the rate of sweating may be influenced by many factors including physical fitness, state of hydration, and heat acclimatization. Most young, healthy humans have the capacity to cope with thermal loads that are up to 15 times their resting metabolic rate of ~1.25 W/kg, even in thermally stressful environments. When thermal loads are low and continuous, core temperature will initially rise and then stabilize at an elevated level. If thermoregulatory mechanisms are impaired, the maximal SAR at which thermal equilibrium can be maintained will be lower.

The deposition of thermal energy associated with RF absorption deep within tissues of the body is in contrast to conventional surface heating mechanisms involving radiant heat sources, ambient air temperature, humidity, air velocity, clothing, etc. Exercise, like RF exposure, can deposit thermal energy directly in deep tissues. Equivalent absorbed energy in the two cases (one active, the other passive) yields equivalent thermoregulatory responses (Nielsen and Nielsen [R910]). Studies of multiple work environments and locations within 15 metropolitan areas of the United States have estimated that ~99% of the population was exposed to background RF of less than 10 mW/m^2 (1 $\mu W/cm^2$) (Tell [R523]), (Mantiply et al. [R631]). At the resonant frequency range for humans, this would represent a whole-body SAR of 0.0004 W/kg, or about 0.03% of the normal resting metabolic rate. Even the current whole-body SAR limit of 0.4 W/kg for exposure in a controlled environment represents only 35% of the resting metabolic rate in humans. Heating at this level

would be comparable to donning a light sweater and would be of little or no physiological significance during most daily activities.

The potential effects of RF exposure, and the mechanism of elevated body temperatures in febrile individuals, must be differentiated from that in normothermic individuals. Strenuous exercise often elevates deep body temperature above a normal “set point” level controlled by the medial preoptic/anterior hypothalamic (PO/AH) brainstem area, which generates signals for efficient heat loss through the mechanisms of vasodilation, increased blood flow, and sweating. During fever, however, heat loss mechanisms are curtailed and heat production/storage mechanisms predominate because the set point is elevated (Shimada and Stitt [R914]). Stitt [R893] demonstrated that when a pyrogenic substance was introduced intra-hypothalamically in animals, thermoregulatory mechanisms were mobilized to increase the body's storage of heat to the level of the elevated set point. Adair [R623] extended these studies to show that febrile monkeys could use RF energy to generate a fever in response to a pyrogen injected into the PO/AH, thereby sparing metabolic energy stores or body fluids. These results imply that RF energy could be utilized by humans to generate a fever, instead of the mobilization of thermoregulatory responses of heat production (shivering and vasoconstriction). Similarly, Pound [R888] has proposed that absorbed RF energy can increase the thermal comfort of people in cold environments.

The goal of thermoregulatory research involving RF exposure of animals is the prediction of adverse thresholds for human RF exposure. However, comparative analysis and extrapolation of animal data to humans must be performed cautiously as smaller animals, particularly rodents, require a high metabolic heat production in order to maintain thermal balance. This is due to their larger surface area to volume ratio, and lack of efficient mechanisms for heat dissipation (Gordon [R348], [R349], [R903]). Threshold levels of RF exposure that trigger various thermophysiological responses in many species of animals have been determined experimentally across a range of RF frequencies, intensities, and under various ambient conditions (Gordon et al [R53], [R54]) (Gordon [R55], [R56], [R348], [R349], [R903]) (Gordon and Ali [R57]) (Jauchem et al [R62], [R168], [R169], [R170], [R587], [R661]) (Gordon and Ferguson [R238]) (Frei et al. [R42], [R271], [R272], [R583]) (Lu et al. [R276]) (Walters et al [R284], [R896]) (Adair and Adams [R293]) (Adair et al. [R295], [R297], [R623]) (Candas et al. [R317]) (Guy et al. [R350]) (Ho and Edwards [R352]) (Phillips et al. [R417]) (Morrissey et al. [R584]) (Frei and Jauchem [R585]) (Jauchem and Frei [R588], [R590]). Studies on rats (Spiers and Adair [R126]) (Chou et al. [R138]) and monkeys (Adair et al. [R297]) have suggested that no long term effects on normal metabolism and thermoregulation occur from chronic thermal RF exposures. Moderate RF exposure might be a safe, rapid, and cost effective energy source for body heating and rewarming (Olsen et al. [R108]) (Hesslink et al. [R239]) (Lloyd and Olsen [R883]) (Olsen and David [R886]) (Olsen [R887], [R972]) (Pound [R888]). Thermoregulation in nonhuman primates has been studied in detail (Adair [R1], [R899]) (Adair and Adams [R292]), [R293], [R294]) (Adair et al. [R295], [R296], [R297]) (Bruce-Wolfe and Adair [R314]) (Candas et al. [R317]) and has shown that while thermoregulation is somewhat less efficient in response to RF exposure at resonance (Lotz [R91], [R247]) (Adair et al. [R137]) (de Lorge [R233]) (Krupp [R241]) autonomic heat loss mechanisms are still rapidly mobilized as a result of the efficient stimulation of central thermal sensors (a situation similar to that occurring in humans during exercise (Adair [R874]). Computerized thermoregulatory models, based on physiological data, have predicted human thermoregulatory responses with good accuracy (Adair and Berglund [R2], [R140], [R671]) (Stolwijk [R283]). Exposures of neonates have demonstrated the young rat's ability to maintain a constant body temperature through efficient thermoregulatory mechanisms (Spiers and Adair [R126], [R892]), (Guillet and Michaelson [R971]). In studies of sheep exposed to MRI, involving head and whole body SARs of up to 4 W/kg for 20 - 104 minutes, no apparent adverse consequences or significant core body temperature increases were observed (Barber et al. [R940]). However, when thermoregulatory responses were disabled (internal temperature responses impaired by anesthesia, panting prevented by controlled ventilation through an endotracheal tube, and convective and radiant heat loss prevented by intact fleece), core temperature continued to rise during exposure (Gordon [R143]).

RF exposure can influence the action of various psychoactive drugs, ethanol, corticosteroids, anesthetics, and other agents that normally influence the thermoregulatory balance (Hjeresen et al. [R58], [R59]) (Lai et al. [R69], [R70], [R71], [R72], [R73], [R74], [R75], [R76], [R77], [R78], [R79], [R176], [R244], [R369],

[R369], [R370], [R371]), (Jauchem et al. [R170], [R489]), (Lotz and Michaelson [R391], [R392]), (Smialowicz et al. [R439], [R440]]), (Cleary and Wangemann [R541], [R882]), (Michaelson [R885]), (Spiers et al. [R891]), (Blackwell [R970]), (Putthoff et al. [R973]). Many of these studies have limited generality, because the impacts of SAR, drug dose and ambient temperature have yet to be explored. In some studies, the lack of appropriate controls is a problem. Several papers claim that ethanol administration interferes with heat loss from the body because the animals become hypothermic. However, careful parametric studies (Spiers et al. [R891]) have shown that acute ethanol administration interferes with metabolic heat production, not heat loss.

Several studies have determined threshold levels of RF energy that generate changes in heat production and heat loss responses in human volunteers (Adair et al. [R639], [R660], [R782], [R875]), (Walters et al. [R713]), (Adair [R873]). Whole-body exposures at 100 and 220 MHz and partial-body exposures at 450 and 2450 MHz were studied. Subjects were exposed or sham exposed in controlled thermal environments to RF fields having local peak SARs of up to ~15 W/kg. No significant changes in metabolic heat production or deep body (esophageal) temperature (±0.1 °C) occurred during 45-min exposures (Adair et al. [R639], [R660], [R782], [R792], [R875]); heat loss responses such as increases in local sweating rate and skin blood flow were mobilized. In general, these fields exceeded the 200 mW/m² controlled environment limit for partial-body exposure specified in IEEE Std C95.1, 1999 Edition [B70]. No consistent difference in response to PW and CW exposures at comparable average field strengths has been observed (Lu and de Lorge [R837]). Humans exposed to MRI (64 MHz, peak SARs of 2–4 W/kg) under assorted exposure regimes showed slight elevations in corneal temperature, skin temperature, blood flow, sweating, and heart rate, but no significant rise in core body temperature (Adair and Berglund [R140], [R671]) (Gordon [R143]) (Shellock and Crues [R180], [R181]) (Shellock et al. [R182], [R183], [R816]) (Shellock [R184]) (Schaefer [R889]). Local high-power RF exposures were used in China to heat testicular tissue to 40–42 ºC for short periods of time for human contraceptive applications (Chiang et al. [R24]) (Liu et al. [R89], [R1005]) with no apparent adverse or long-term tissue effects. Science-based simulation models of human physiological responses have predicted that the scenario after 100 watts of power were deposited in the head for 30 minutes, or a whole body MRI scan of a 70 kg patient for an indefinite duration at SAR = 5 W/kg, would not be sufficient to overcome the available heat loss mechanisms or raise core body temperature (Adair [R2], [R873]) (Stolwijk [R283], [R895]) (Adair and Berglund [R671]) (Stolwijk and Hardy [R894]). Even with skin blood flow restrictions of up to 67%, an MRI scan of the trunk at an SAR = 4 W/kg for 40 minutes would still result in a temperature rise equal to or less than 1 ºC. While some accidental RF exposures at high levels in humans and associated adverse effects have been reported, (Hocking et al. [R60]) (Hocking and Westerman [B58]) most have been shown to be benign.

The absorption profile for the higher microwave frequencies (10 GHz and above) is similar to that for infrared radiation (Stevens [R974]) and millimeter waves (Frei et al. [R586]) (Ryan et al. [R649]) with RF energy absorbed principally in the most superficial layers of skin and in close proximity to temperature-sensitive nerve endings. Although lower RF frequencies will be absorbed in complex patterns at additional depths, thresholds for the detection of RF fields at frequencies of 2.45 GHz and above by human observers have been determined in several studies (Seinkowicz et al. [R124]) (Justesen et al. [R362]) (Hendler and Hardy [R548]) (Hendler et al. [R549]) (Hendler [R550]) (Blick et al. [R615]) (Riu et al. [R632]) (Walters et al. [R713]) (Adair et al. [R792], [R875]) (Cook [R876], [R877]) (Eijkman and Vendrik [R879]) Michaelson [R884]) (Schwan et al. [R890]) (Vendrik and Vos [R975]) using brief exposures (≤ 10 seconds) and exposures of restricted areas of the forehead, back, or forearm skin. In general, the shorter the wavelength, the less energy is required to produce a cutaneous thermal sensation. Using the Penne's bio-heat equation as the basis for a theoretical analysis, Riu et al. [R632] suggested that a constant temperature increase of ~0.07 °C at or near the surface of the skin was necessary for thermal sensation. This analysis also indicated that the depth at which the thermal receptors are located is not a relevant parameter, as long as it is within 0.3 mm of the surface. Early studies to identify the pain threshold suggested a correlation with a final skin surface temperature of ~46.1 ± 1.0 °C (Cook [R876], [R877]), although this threshold depended upon the area exposed, exposure time, initial skin temperature, anatomical site, and thermal conductivity.

B.5.1.2 Summary of thermoregulation

Significant core temperature increases (on the order of ~1 ºC or more) can be induced in laboratory rodents and non-human primates as a result of RF exposures at levels of ~4 W/kg, resulting in significant physiological and behavioral effects. Comparable increases in core body temperature are not easily produced in humans by RF exposures due to a more efficient thermoregulatory system. Even at exposure levels considerably higher than current standards allow, human body temperature is efficiently regulated in healthy individuals by the mobilization of appropriate heat loss mechanisms, such as sweating and skin blood flow. Exposure to RF frequencies produces a sensation of warmth for which the threshold power density is less as the frequency increases. The threshold of cutaneous thermal pain is 45–47 ºC, and if generated by RF energy, the pain will result in a prompt effort to escape from the field to preserve normothermia.

B.5.2 Animal behavior, neurochemistry, neuropathology

B.5.2.1 Review of animal behavior studies

Behavioral disruption in animals has served as the basis for human RF exposure guidelines since the early 1980's (ANSI [B6]) (ICNIRP [B62]) (NCRP [B95]) and studies of human thermal sensation of RF exposures (Brown et al. [R230]) (Justesen et al. [R363]) (Hendler and Hardy [R548], [R549]) (Hendler [R550]) (Blick et al. [R615]) (Riu et al. [R632]) (Walters et al. [R713]) (Adair et al. [R792], [R875]) (Cook [R876], [R877]) (Eijkman and Vendrik [R879]) (Michaelson [R884]) (Schwan et al. [R890]) (Justesen [R906]) (Vendrik and Vos [R975]) reinforce the conclusion that behavioral changes observed in RF exposed animals are likely to be thermally motivated. Acute thermal responses in animals can range from perception to aversion, work perturbation, work stoppage, endurance reduction, and even convulsions and death in the extreme (Phillips et al. [R417]) (Frei et al. [R586]) (Guy and Chou [R904]) (Justesen [R905]) (Modak et al. [R909]). RF effects on behavior, however, may reflect an animal's attempts to engage in other thermoregulatory activities (Stern [R915]). Further, hot spots generated in certain parts of the body at non-resonant frequencies and in locations where blood flow is minimal (D'Andrea et al. [R33], [R34], [R328]) (Grandolfo et al. [R216]) (Lin et al. [R866]) (Gandhi [R902]) as well as RF hearing effects that occur with high peak pulses (see B.11) may be involved in the influence of behavior by RF exposure.

Animals are generally more sensitive to thermal effects of RF exposure at frequencies closest to their resonant frequency (~2500 MHz for mice, ~600–700 MHz for rats, ~70 MHz for adult humans), as it takes less incident energy to increase core body temperature. Thermal exposures at or near the resonant frequency have had noticeable effects on animal behavior (Gordon et al. [R53], [R54]) (Gordon [R55], [R56], [R903]) (Gordon and Ali [R57]) (Mitchell et al. [R103], [R104]) (Gordon and Ferguson [R238]) (D'Andrea et al. [R269], [R327]) (de Lorge and Ezell [R331]) (Gordon [R348], [R349]) (Smialowicz [R439], [R440], [R901]). In a series of studies, de Lorge and colleagues disrupted learned behavior in mice, rats, and monkeys with acute RF exposures at various frequencies (de Lorge [R232], [R233]) (D'Andrea and de Lorge [R270]) (de Lorge and Ezell [R331]) (Knepton and de Lorge [R493]) (Knepton et al. [R494]) (Nelson [R508]) (Sanza and de Lorge [R913]). Whole-body specific absorption rates of ≥ ~4 W/kg were generally required to affect behavioral changes across species at 2.45 GHz, although different behavioral thresholds were observed across species at 5.7 GHz and 1.3 GHz. In general, as animal size increases, higher power densities are required to affect behavior changes and colonic temperature increases. Across species, an increase of 1 °C in colonic temperature is generally correlated with disruption of behavior. Other investigators have confirmed correlations in animals between behavioral changes, increased core body temperature, and acute whole body RF exposure levels of ~≥ 4 W/kg with either CW or high peak power pulses (Akyel et al. [R4]) (D'Andrea et al. [R35], [R210], [R231]) (Quock et al. [R114], [R279]) (Brown et al. [R230]) (Schrot et al. [R432]). Most studies at low levels of RF exposure, and even some at thermal levels, report no effects on behavior (Akyel et al. [R4]) (Gage [R338]) (Gage et al. [R339]) (Gage and Guyer [R340]) (Lebovitz [R375], [R376]) (Thomas et al. [R456], [R457], [R461]) (Liddle et al. [R499]) (Sagan and Medici [R503]) (Bornhausen and Scheingraber [R746]) although positive reports of behavioral changes at near-thermal (Schrot et al. [R432]) and apparent non-thermal acute (Frey and Spector [R43]) and chronic (Bruderer and

Bolt [R848]) exposure levels do exist. Studies of acute RF exposure effects on cognitive performance generally report no effects (Sienkowicz et al. [R712]) (Dubreuil and Edeline [R840]) unless exposures reach the thermal range (Thuroczy et al. [R743]) (Mickley et al. [R810]) (Mickley and Cobb [R811]) although studies by Lai et al. reported changes in maze testing of rats at RF exposure levels of 0.6 W/kg (Lai et al. [R244]) (Wang and Lai [R705]). The high peak pulses used in these later studies may have generated RF hearing effects. Recent and well documented efforts by two laboratories to confirm the maze result were unsuccessful (Cassel et al. [R1137]) (Cosquer et al. [R1140]) (Cobb et al. [R1113]).

Some enhancement of active and passive avoidance behavior in mice acutely exposed to RF at thermal levels has been reported (Luttges [R502]) (Beel [R1004]) while continued daily repeated exposures lead to performance deterioration (Beel [R1004]). The ability of acute high peak pulsed RF to influence aversive and escape behavior have produced equivocal results (Justesen [R63]) (Carroll et al. [R318]) (King et al. [R365]) (Levinson et al. [R378]) (Monahan and Ho [R407]) (Monahan and Henton [R408] [R409]) (Justesen [R905], [R906]) (Justesen et al. [R907]). In many studies, animals failed to learn aversive behaviors in response to intense acute RF exposures, even at lethal field strengths, although stimuli such as foot shock are consistent reinforcers. Justesen [R63] has suggested the inability of animals to learn an escape response in the presence of intense RF fields suggests a delay in timely sensory feedback. Some reports suggest differences between CW and PW exposures of the same average power on affecting aversive behavior (Frey [R334]) (Lebovitz [R376]) (Thomas et al.[R461]) although the possibility of a RF auditory effect specific to high peak power PW exposures in these later studies cannot be ruled out (Stern [R915]).

Acute RF exposure can affect changes in thermoregulatory response and behavior as well (Adair [R1], [R899]) (Lotz and Saxton [R91]) (Vitulli et al. [R132], [R133]) (Lotz [R247]) (Lu et al. [R276]) (Adair and Adams [R292], [R293], [R294]) (Adair et al. [R296], [R297], [R623]) (Candas et al. [R317]) (Stern et al. [R448]) (Berglund [R900]) (Gordon [R903]) (Nielsen and Nielsen [R910]) (Shimada and Stitt [R914]). In studies with resonant vs. non-resonant RF, trained monkeys in a cold environment maintained a consistently optimal skin temperature. A slightly greater increase in deep body temperature was preferred by animals when the RF exposure was at the resonant frequency (resulting in deeper body penetration of the RF energy). RF exposure was effective only to a limited degree as a positive reinforcer for operant behavior in animals in response to cold environments (Marr et al. [R96]) (Vitulli et al. [R132], [R133]) (Bruce-Wolfe and Adair [R314]). Studies have also reported on the ability of acute RF exposures to interact with the thermoregulatory action of various drugs (Lai et al. [R69], [R70], [R71], [R72], [R73], [R74], [R75], [R76], [R77], [R78], [R79], [R176], [R244], [R368], [R369], [R370], [R371]) (Lotz and Saxton [R91]) (Lotz [R247]) (Monahan and Ho [R407]) (Monahan and Henton [R408], [R409]) (Thomas [R458]) (Thomas et al. [R459], [R460]).

Reports on the effects of chronic low-level RF exposure have been generally negative (D'Andrea et al. [R31], [R32], [R269], [R327]), (DeWitt [R37]) (Chou et al. [R138]), (Lebovitz [R375], [R376]) although positive effects at near-thermal levels have been reported (Mitchell et al. [R406]). Reports from Eastern Europe and the Soviet Union (summarized in D'Andrea and de Lorge [R270]) have reported effects at lower levels. Prenatal exposure at low levels has been reported by some laboratories to be ineffective in producing behavioral changes in the offspring after birth (Galvin et al. [R45]) (Kaplan et al. [R363]) although other laboratories have reported effects at ~4 W/kg or higher including decreased activity, thermal sensitivity, and decreased term weight in rat pups (Jensh et al. [R356], [R357], [R358], [R359]) (Jensh [R360], [R361], [R646]) (O'Connor [R911]).

B.5.2.2 Summary of animal behavior studies

A threshold of ~4 W/kg for disruption of complex behavioral performance in several animal species, including non-human primates, under diverse exposure conditions, often (but not always) accompanied by an increase in core body temperature of ~1.0 ºC, has been used as a basis for setting human exposure guidelines since 1982. Alteration (but not necessarily stoppage) of a variety of other learned and unlearned behaviors in animals can occur at SARs between 1–4 W/kg, depending upon the frequency and the size of the animal. Essentially all behavioral changes due to RF exposure at these levels are reversible, and no consistent evi-

dence exists for long-term or permanent effects. Thermoregulatory behavior in the presence of RF fields appears to be quite efficient in most species and under most conditions, even at SARs equal to twice the resting metabolic rate, although exceptions may exist at the resonant frequencies. Extrapolation of available animal data to humans is useful on an interim basis for setting standards. Because of better thermoregulatory mechanisms in humans, as well as a superior ability to discriminate and cognitively act upon perception of intense RF fields, the animal data may tend to underestimate the threshold levels for safety for humans.

B.5.2.3 Neurochemistry

Neurochemical changes found at RF exposure levels causing a significant increase in rat body temperature include the following: decreased brain concentrations of serotonin and 5-hydroxyindoleacetic acid (Snyder [R741]); lower concentrations of norepinephrine, serotonin and dopamine (Merritt et al. [R924], [R925]); changes in norepinephrine and acetylcholine (Gandhi and Ross [R47]); and reduced norepinephrine, increased 5-hydroxyindoleacetic acid and no change in serotonin (Inaba et al. [R595]). Reduced brain acetylcholine levels were measured in rats following RF exposure producing brain temperature increases of 2–4 ºC (Modak et al. [R909]) and at 6.5 W/kg, but not 3.5 W/kg, (2450 MHz CW) and 0.3 W/kg (800 MHz) (Testylier et al. [R834]). Mausset et al. [R923] showed that SARs of 4 and 32 W/kg reduced gamma-aminobutyric acid (GABA) levels in the rat cerebellum. Under exposure conditions (2.86 GHz PW, 10 mW/cm^2 for 4 h/day, 5 d/week, for up to 4 or 8 weeks) producing "only moderate signs of heat stress" with no significant increase in body temperature of rats, there was no change in metabolism of the inhibitory neurotransmitter GABA (Zeman et al. [R930]). Browning and Haycock [R17] showed that neither acute nor chronic RF exposure at non-hyperthermia levels had any effect on rat brain synapsin I, an indicator of neurotoxicity.

Lai [R139] summarized a decade of his research on the role of endogenous opioids in biological responses to RF exposure, mostly to pulsed waveforms (2 μs, 500 pulses per second) with whole-body average exposure of 0.6 W/kg, as follows: 1) exposure enhanced morphine-induced catalepsy in the rat (Lai et al. [R368]); 2) exposure attenuated the naloxone-induced wet-dog shake, a morphine withdrawal symptom, in morphine-dependent rats (Lai et al. [R69]); 3) narcotic antagonist blocked a transient increase in body temperature after exposure (Lai et al. [R496]); 4) the effect of acute exposure on amphetamine-induced hyperthermia (Lai et al. [R70]) and ethanol-induced hypothermia (Lai et al. [R370]) can be blocked by narcotic antagonist; 5) RF-induced changes in high-affinity choline uptake (HACU), an index of cholinergic activity, in the brain can be blocked by narcotic antagonists (Lai et al. [R71], [R76]); 6) changes in concentrations of muscarinic cholinergic receptors in the brain after repeated sessions of RF exposure can be blocked by pretreatment with narcotic antagonists before each session of RF exposure (Lai et al. [R78]); and 7) three major subtypes of opioid receptors are involved in the effect of RF exposure on HACU (Lai et al. [R79]). In addition, Lai reported that biological responses were influenced by RF exposure parameters such as duration of exposure, the pattern of energy absorption in the body (Lai et al. [R369]) and waveforms. An example of the latter was the finding that HACU was affected by PW fields and not CW fields. As explained by Lai [R139], the differential effect due to waveform was possibly due to the auditory response to pulsed RF fields (see B.6.5).

In addition to the studies on cholinergic systems mentioned above, Lai published other studies on these systems because of their role in many physiological and behavioral functions (Lai et al. [R74], [R76], [R497], [R620]). RF exposure reduced HACU in the frontal cortex and hippocampus of the rat. The effect on the hippocampus, but not the effect on frontal cortex, could be blocked by a narcotic antagonist, a response similar to acute restraint-induced stress (Lai et al. [R71], [R72]). A learning deficit was found to be correlated to the decrease in cholinergic activity (Lai et al. [R75]). Changes in muscarinic cholinergic receptors were dependent on endogenous opioids in the brain because the effect was blocked by the narcotic antagonist naltrexone (Lai et al. [R78]). All three subtypes of opioid receptors were affected (Lai et al. [R76], [R176]). Based on his results, Lai [R176] proposed a model of neural mechanisms mediating the effects of low-level RF exposure on cholinergic activity in the frontal cortex and hippocampus of the rat. The RF exposure somehow activated corticotropin-releasing factor, which in turn caused a decrease in activity of cholinergic innervations in the frontal cortex and hippocampus (Lai et al. [R77]). The endogenous opioids, via three receptors, are the intermediate step before the hippocampal change occurs. The activation process might be

a stress response. Lai et al. [R79] tested this possibility by studying the concentration of benzodiazepine receptors in the cortex and hippocampus. The increased level in the cortex showed adaptation after repeated exposure, i.e., less stress. Based on his decade of research on opioids and cholinergic systems, Lai [R139] 1) speculated that low-level RF exposure is a "stressor" (Lai et al. [R73]) because of the similarity of RF effects and those of established sources of stress and 2) concluded that there is no convincing evidence that repeated exposure to low-level RF fields could lead to irreversible neurological effects.

The stress response was also addressed by Lu et al. [R394] who evaluated the effects of RF exposure on body temperature and neuroendocrines [thyroxine, thyrotropin (TSH), growth hormone and corticosterone] in rats subjected to 2450 MHz CW exposure at 1–70 mW/cm^2 for 1–8 h. It was noted that body temperature was the most sensitive parameter. Adrenocorticol stimulation was correlated with inhibition of growth hormone and TSH in exposed animals and the authors stated that the pattern of adenohypophyseal response in rats was consonant with a stress response. This is consistent with the observation that none of the endocrine changes occurred without a thermogenic RF exposure.

In other neurochemical studies, Hjeresen et al. [R59] investigated effects of RF exposure on ethanol-induced interactions with neurotransmitter systems and Monahan [R105] reported that 1 and 10 W/kg affected the cholinergic drug scopolamine and physostigmine on shock latency and motor activity of mice. Results from the latter study suggest RF enhancement of cholinergic activity (D'Andrea et al. [R1089]). Ashani et al. [R306]) investigated the hypothermic interaction of pulsed RF exposure on drugs affecting cholinesterase.

Based on results from a series of studies on brain energy metabolism, Sanders et al. [R428], [R429], [R430], Sanders and Joines [R983] hypothesized that RF exposure could inhibit energy production by affecting the mitochondrial electron transport chain. Related work showed that RF exposure affected mitochondrial marker enzymes in mouse brain (Chiang et al. [R480]) and pulsed RF fields induced subtle changes in succinate dehydrogenase levels in the developing mouse brain (Chiang and Yao [R23]).

In an in vitro study, Gandhi and Ross [R48] described changes in the metabolism of inositol phospholipids in rat brain synaptosomes exposed at 10 and 30 W/kg. Millar et al. [R405] found no effect of pulsed 2.45 GHz fields on acetylcholinesterase (AChE) activity in samples maintained at a constant temperature while being exposed at SARs ranging from 4–2460 W/kg. In addition, a wide variety of pulse widths, repetition rates, and duty cycles were also without effect. In neuroblastoma cells exposed in vitro to amplitude modulated RF energy, Dutta et al. [R39] reported different responses including increased and decreased AChE activity and no effect over a range of SARs from 0.001–0.1 W/kg. In young rats exposed at 0.1–0.4 W/kg, decreased brain AChE was found (Kunjilwar and Benhari [R636]).

Mausset et al. [R1138] exposed the rat head for 15 min to a pulsed 900 MHz signal at a brain-averaged SAR of 6 W/kg. In addition to a strong glial reaction in the brain, effects were found on a GABA receptor and dopamine transporters. The effects were claimed to be the first evidence for such changes in the rat brain following an acute, high-power GSM exposure; however, the molecular and cellular changes did not translate into an effect on the exposed rat's general locomotor behavior.

In human subjects exposed to GSM signals for 2 h/day, 5 days/week for 1 month, no significant effects were found on anterior pituitary hormones (serum adrenocorticotropin, thyrotropin, growth hormone, prolactin, luteinizing hormone, and follicle stimulating hormone) (de Seze et al. [R640]) and no effect was measured on melatonin in subjects exposed at the maximum power of commercially available mobile phones (de Seze et al. [R690]). Mann et al. [R709] found no changes in nocturnal hormones (growth hormone, cortisol, luteinizing hormone and melatonin) in human subjects exposed to a pulsed 900 MHz signal (0.2 W/m^2). Radon et al. [R783] also demonstrated a lack of effect of pulsed 900 MHz fields (1 W/m^2, maximum SAR averaged over 10 g in the head estimated at 0.025 W/kg) on melatonin and cortisol in human males exposed to ten 4 hour periods (across night and day) in a double blind study. In rats and hamsters exposed to 900 MHz (CW and PW) at 0.04–0.36 W/kg, Vollrath et al. [R614] also failed to find nocturnal melatonin changes.

Reviews that address neurochemical effects of RF exposure include Michaelson et al. [R926], Lai [R139], Vander Vorst and Duhamel [R1143], Hermann and Hossmann [R717], Hossmann and Hermann [R981] and D'Andrea et al. [R1089].

B.5.2.4 Summary of neurochemistry

Neurochemical effects are found when RF exposures are sufficiently high to induce significant increases in body temperature. The results of studies reporting effects at non-hyperthermic RF levels, e.g., the effects on brain energy metabolism (Sanders et al. [R428], [R429], [R430]), have not been confirmed/replicated by independent investigators. Some effects were reported to occur after pulsed, but not CW, RF exposure (Lai [R139]). It is known that the auditory system is very sensitive to pulsed RF energy (see B.6.5) and Lai [R139] explained that differential effects of PW and CW exposures possibly could be due to the RF auditory response. Although it has been hypothesized that RF exposure acts as a stressor (Lai et al. [R73]) because of the similarity of RF effects and those of established sources of stress, Lai [R139] concluded that there is no convincing evidence that repeated exposure to low-level RF fields could lead to irreversible neurological effects. It is noted that results from the human studies described above show no changes in a variety of neurochemicals following exposure of the head to pulsed 900 and 1800 MHz signals used in telecommunications.

B.5.2.5 Neuropathology

In the early 1970s, there were reports in the Eastern European literature describing changes in nervous system structure in laboratory animals exposed to microwave fields (Gordon et al. [R921]). A study in the Western literature, however, found no histologic changes after acute RF exposure causing brain temperature increases of 4.4-6.5 ºC (Lin et al. [R862]).

The rationale for a series of histologic studies by Albert and his colleagues (Albert and DeSantis, [R916]), (Albert et al. [R299], [R300]) was based in part on the results of the research mentioned above. In Chinese hamsters, Albert and DeSantis [R916] found that high intensity RF fields of 15 W/kg caused cellular alterations in hypothalamic and subthalamic regions of the brain and 7.5 W/kg caused vacuolation of neurons, but not glia, in the hypothalamic region. In other studies, rats and monkeys were exposed to RF fields during their fetal and postnatal life to examine effects of RF exposure on the developing brain (Albert et al. [R299], [R300]), (Albert and Sherif [R6]). In rats, exposure to two frequencies (100 and 2450 MHz) resulted in a decrease in the number of Purkinje cells. At 2450 MHz, rats exposed postnatally (5 days, 7 h/day) at 2 W/kg beginning at one and six days of age and examined immediately after exposure had morphological changes suggestive of effects on cerebellar microneurons and the metabolic status of Purkinje cells (Albert and Sherif [R6]) in addition to fewer Purkinje cells than control animals; however, this latter change was reversible because there was no change in number of Purkinje cells at 40 days after exposure (Albert et al. [R299]). In contrast to this result, there were fewer Purkinje cells in experimental rats than in control animals at 14 months after long-term exposure at 2.8 W/kg that began with *in utero* exposure, i.e., pregnant rats were exposed from gestation day 6 through the end of pregnancy and their offspring were exposed for 97 days for 4 h/day at 100 MHz (Albert et al. [R299]). In a non-human primate study, Albert et al. [R300] examined Purkinje cells in the offspring of pregnant squirrel monkeys exposed at 3.4 W/kg (2450 MHz) for 3 h/day, 5 days/week, until the offspring were 9.5 months of age. Unlike the results from the rat studies, no significant effect on Purkinje cells was found in monkeys. Although there are many experimental differences between the rat and monkey studies (see Albert et al. [R300] and D'Andrea et al. [R1089]), it is noted that 1) the distribution of RF energy absorption in the monkey is more similar to that of human beings because its body shape better resembles human body shape and 2) there was no effect on Purkinje cells in the monkey exposed to 3.4 W/kg, a level that is 8.5 times greater than the limit for controlled environments.

As described in more detail in C.7.13.1, an extensive investigation of mammalian brain development found no histological changes in the developing rat brain (Inouye et al. [R781]). In contrast to the effect reported by Albert et al. [R299], there were no changes in Purkinje cells. In this study, rats were exposed prenatally

and postnatally to brain SARs up to about five times greater than the threshold SAR for established adverse effects.

Most importantly, histopathological analysis of the brain and other CNS tissues was a special focus of life-time RF exposure studies in rats (Zook and Simmens [R778]), some of which included exposure of the animals during gestation (Adey et al. [R677], [R727]) (Anderson et al. [R1120]). These studies are described in detail in C.7.13.2.1 and B.7. No neuropathology was observed in animals exposed to RF energy during critical periods of CNS development in the fetus, as well as throughout young and adult life.

In a study involving only a few animals, Guy and Chou [R904] reported histological changes in the brains of rats exposed to a single high-intensity microwave pulse at 915 MHz (10 kW at 60 and 100 ms). The SARs were sufficiently high to cause the brain temperature to increase by about 8 ºC.

An in vitro study reported morphological changes in mouse neuroblastoma cells exposed to a pulsed RF field (Webber et al. [R524]) while another study found minor changes in cellular structure in snail ganglia exposed at 12.9 W/kg, a level more than three times greater than the adverse effect level found in live animals (Arber et al. [R287]).

B.5.2.6 Summary of neuropathology

A review of the literature investigating neuropathological changes in animals exposed to RF energy, particularly two-year exposure studies, does not provide evidence to change the 4 W/kg adverse effect level. Albert et al. [R299] reported changes in Purkinje cells in rats exposed below 4 W/kg; however, as discussed above, this effect is not supported by results from Inouye et al. [R781] or Albert et al. [R299].

B.5.3 Review of 3 kHz to 100 kHz studies

B.5.3.1 Long-term exposures (3–100 kHz)

There are now many major reviews of the RF literature, including those of the Advisory Group on Non-ionizing Radiation of the UK National Radiological Protection Board [B3], the Health Council of the Netherlands [B55], the Institution of Electrical Engineers [B68], the International Commission on Non-ionizing Radiation Protection [B62], and the US National Research Council [B100].

None of the above reviews established a hazard from long-term RF exposure. This Standard does not propose limits on exposures that are lower than those necessary to protect against adverse short-term effects in the frequency range below 100 kHz, because there is no evidence that these levels would not protect against long term exposures at lower levels. The Subcommittee will continue to evaluate new research and will revise this standard should the resolution of present uncertainties in the research literature identify a need to limit long-term exposures to values lower than the limits of this standard. The Subcommittee will also continue to evaluate new research on short-term effects and modeling.

B.5.3.2 Short-term exposures (3–100 kHz)

In the frequency range from 3 to 100 kHz, this standard was developed with respect to *established* mechanisms of biological effects that could lead to adverse effects in humans from electric and magnetic field exposures. These have been described in IEEE C95.6-2002. These established mechanisms fall within the category of short-term effects known as *electrostimulation*, which refers to the induction of a propagating action potential (a “nerve impulse”) in excitable tissue (nerve and muscle) by an applied electrical stimulus. Such effects are understood in terms of recognized interaction mechanisms. The standard regarding such effects does not apply to exposure encountered during medical procedures, nor does it necessarily protect against interference of medical devices or problems involving metallic implants.

Maximum exposure limits in this frequency range are based on avoidance of short-term reactions of electrostimulation. A review of the literature pertaining to electrostimulation effects, and the rationale for maximum permissible exposure levels, is provided in this standard and the following reactions are discussed: (a) aversive or painful stimulation of sensory or motor neurons, (b) muscle excitation that may lead to injury while performing potentially hazardous activities, and (c) cardiac excitation.

B.6 Non-cancer related studies

B.6.1 Teratogenicity, reproduction, and development

B.6.1.1 Teratogenicity

Studies in animal models of possible teratogenic effects of RF exposure, and other conditions causing heat stress, have demonstrated that significant increases in the incidence of heat-induced abnormalities are seen after maternal temperature increases of approximately 2–2.5 ºC (mostly following exposures of tens of minutes up to one hour or so). Higher temperature increases, of up to ~5 ºC, for shorter durations are teratogenic (Edwards et al. [R1081]). Fetal malformations were observed in offspring of pregnant rodents (mice, rats and Syrian hamsters) exposed to whole body average SARs ≥9 W/kg (Brown-Woodman and Hadley [R19]) (Lary et al. [R81], [R373], [R374]) (Berman et al. [R536]) (Chazan et al. [R540]) (Rugh et al. [R552]). The teratogenic effects of RF exposure were attributed to thermal stress because many of the studies recorded elevations of 2 ºC or more in the maternal core body temperature.

Exposures at lower SARs (3.6–7.3 W/kg) did not cause deformities in rats (Berman et al. [R308]) (Jensh et al. [R356], [R358]) (Jensh [R360], [R646]). Reduced fetal body weight in rats was observed at 7.3 W/kg (Jensh [R360]) and 4.8 W/kg, but not at 2.4 W/kg (Berman et al. [R228]), following long-term exposure of pregnant rats. The studies involved virtually continuous exposure during gestation. These studies and another report (Berman and Carter [R537]) support the observation that exposure levels of 4.8–7.3 W/kg, i.e., levels somewhat less than those causing malformations, result in reduced fetal mass in rats. In comparison to the rat, higher SARs are required for teratogenicity and reduced fetal mass in the mouse, because the smaller animal is able to dissipate heat more efficiently (Berman et al. [R309]) (Inouye et al. [R354]) (Nawrot et al. [R410], [R411]).

Very high SARs for short periods of time (Chernovetz et al. [R156], [R157]) or low SARs for long periods of time (causing no significant thermal stress) have generally not been associated with teratogenic effects (Chiang and Yao [R23]) (Schmidt et al. [R721]) (Larry et al. [R737]) (Cobb et al. [R744]).

Several studies have investigated the interaction between RF exposure and known teratogens such as ionizing radiation (183), 2-methoxyethanol (Nelson et al. [R219], [R277], [R613]) (Nelson and Conover [R599]), salicylic acid (Nelson and Snyder [R674]), and arabinoside (Marcickiewicz et al. [R95]). Some of these studies reported potentiation of teratogenic effects at exposure levels below the threshold for RF exposure alone, although RF exposure levels in these co-teratogen studies caused significant body temperature increases {with the exception of one unconfirmed study (Marcickiewicz et al. [R95])}.

There are a few reports (Brown-Woodman and Hadley [R18]) (Tofani et al. [R129]) (Berman et al. [R305]) that are inconsistent with the weight of evidence indicating that teratogenic effects of RF exposure are thermally based; the results of these studies have not been confirmed or replicated by other laboratories. One study (Heinrichs et al. [R488]) of mice exposed to the MRI conditions used for human clinical imaging reported no overt embryotoxicity (resorptions, stillbirths) or teratogenicity. A slight, significant decrease in the fetal crown-to-rump length was recorded.

No teratogenic effects were found following continuous exposure of pregnant mice during gestation days 0–18 to 20 kHz magnetic fields, such as those associated with video display terminals (VDTs) (Huuskonen et

al. [R730]). In humans, no association between VDT exposure and teratogenesis was found (Kurppa et al. [R243]).

In addition to mammalian models, avian and insect species have been examined for teratogenic effects following RF exposure. In avian eggs, no effects were found on hatching, malformations, embryo weight, or hematologic parameters at SARs (14 W/kg) that maintained the proper incubation temperature of 37 ºC (McRee et al. [R770]) (Hamrick and McRee [R771]), although effects could be precipitated with exposures causing higher temperature elevations (Byman et al. [R315]) (Clarke and Justesen [R324]) (Hills et al. [R531]). Hatchability of chicken eggs was not affected at 2.9 W/kg (Braithwaite et al. [R185]). Those studies reporting terata in avian models in the absence of RF heating (Saito et al. [R149]) (Fisher et al. [R333]) Saito and Suzuki [R650]) (Youbicier-Simo et al. [R653]) have not been confirmed or replicated by other independent laboratories.

Overall, the investigations of teratogenic effects in insects after RF exposures are consistent with the weight of evidence showing that malformations are caused by RF heating (Pickard and Olsen [R418]) (Lindauer et al. [R738]), (Carpenter and Livstone [R763]), (Liu et al. [R764]), (Green et al. [R765]), (Olsen [R766], [R769]), (Schwartz et al. [R804]).

B.6.1.2 Reproduction

Sterility can occur when mammalian testes, which are normally at a temperature of 33–35 ºC, are heated by a variety of methods (e.g., hot water, infrared radiation, ultrasound) to temperatures approaching normal abdominal temperature (37–38 ºC). Likewise, RF energy, due to its ability to heat and raise the temperature of the testes, can adversely affect fertility and sperm morphology (Goud et al. [R193]), (Kowalczuk et al. [R861]). Permanent changes in reproductive efficiency in rats have been associated with RF exposures causing temperatures in the testes greater than 45 ºC (Fahim et al. [B38]). At less extreme RF exposure conditions, temporary sterility has been demonstrated in male rodents with core temperatures of ~41 ºC and intra-testicular temperatures ≥37.5 ºC (Lebovitz and Johnson [R82], [R377]) (Lebovitz et al. [R218]) (Berman et al. [R307]). A lower sperm count and necrosis of testicular tissue was observed in testes heated to 39 ºC or more by either microwave heating or through the use of a water bath (Reed et al. [R424]). An RF exposure at an SAR of 6.3 W/kg, which caused a body temperature increase of about 1.5 ºC, did not affect spermatogenesis in rats (Johnson et al. [R490]).

After reporting that exposure of rats to mobile phone emissions caused a reduction in the diameter of seminiferous tubules (Akdag et al. [R688]) (Dasdag et al. [R733]), the same laboratory performed “a more thorough study” that failed to confirm the effect and also failed to find effects on additional measures of testicular function and structure (Dasdag et al. [R1108]). A study reporting effects of low-level RF exposure on reproductive ability in rodents (Magras and Xenos [R619]) is not useful because of flaws in study design, including inappropriate control groups. The reduction in fertility in exposed rats in the absence of a significant increase in body temperature (Brown-Woodman et al. [R20]) has not been independently confirmed, and remains inconsistent with the weight of evidence indicating that reproductive effects of RF exposures are thermally based.

An *in vitro* study reported reduced fertility of sperm at SARs ≥50 W/kg, i.e., exposures that are much higher than the established adverse effects threshold of 4 W/kg (Cleary et al. [R27]).

A slight but significant reduction in litter size was reported in the second litter born to rats exposed throughout their first pregnancy for 6 h daily at 3.6 W/kg. Control animals curled up, but exposed animals splayed their bodies indicating that the RF exposure caused some heat stress (Jensh et al. [R357]).

In avian studies, the number and fertility of sperm maintained at their normal temperature during RF exposures at 10 and 50 W/kg were not affected (Hall et al. [R775], [R776]). Reports of effects on fecundity in chickens are not useful, because the exposures took place in metal cages (Krueger et al. [R495]), (Giarola

and Krueger [R922]). Reproductive parameters in quail exposed during development are discussed in the following section (see B.7.3).

In Drosophila melonagaster, RF fields produced reproductive effects, but only at very high exposure levels (Pay et al. [B108], [R748]).

The literature on human reproductive studies includes reports of workers using VDTs, MRI devices, RF heat sealers, medical diathermy units and radar. Some reports found no association between exposure to VDTs and pregnancy outcome (Nurminen and Kurppa [R198]) (Larsen [R245]) (Michaelson [R248]) (Schnorr et al. [R253]) (Taskinen et al. [R255]), including miscarriage (Bryant and Love [R196]) (Ericson and Kallen [R235], [R236]), while other studies found an increased risk of infertility (Smith et al. [R628]) and a slightly elevated risk of miscarriage (Goldhaber et al. [R223]) (McDonald et al. [R224]). Studies of female MRI workers concluded that there was no major elevation in risk of adverse reproductive outcomes (Evans et al. [R237]) (Kanal et al. [R240]). Work with RF heat sealers reportedly did not affect male semen quality or hormone levels (Grajewski et al. [R761]). In China, intentional RF exposures of human testes, sufficient to cause scrotum surface temperatures of 40–42 ºC, have been reported to be an effective contraception method (Liu et al. [R89]).

A weak association exhibiting an exposure-response relationship was reported between miscarriages in female physical therapists and occupational exposure during pregnancy from medical diathermy units (915 and 2450 MHz) (Ouellet-Hellstrom and Stewart [R226]), (Stewart and Ouellet-Hellstrom [R696]). However, a commentary on the exposure-response relation showed that there was no association between absorbed RF energy and the reported effect (see Hocking and Joyner [R274] and Ouellet-Hellstrom and Stewart [R668]).

No association was reported between miscarriages and use of shortwave (27.12 MHz) diathermy units. In other studies, the use of shortwave equipment by female physiotherapists was reported to be associated with low birth weight of offspring (Lerman et al. [R784]) and dead or malformed infants (Kallen et al. [R145]). In Danish physiotherapists (Larsen et al. [R197]), use of high-frequency electromagnetic devices was associated with a higher ratio of female births and lower birth weight of males. The authors, however, cautioned that the results were based on sparse data and needed to be interpreted with caution; the results were not confirmed in a study of Swiss physiotherapists (Gubéran et al. [R678]). In Finland, no firm evidence of increased spontaneous abortions or congenital malformations was found in offspring of female physiotherapists (Taskinen et al. [R255]). Daels [R666] administered RF energy during uterine contractions to 2000 females during parturition. No adverse side effects of RF heating were observed; the temperature of the newborn was slightly increased but never exceeded 37.8 ºC.

A possible association between the incidence of Down's syndrome and paternal radar exposure (Sigler et al. [R153]) was not confirmed in an extended study by the investigators (Cohen et al. [R141]). Lower sperm concentration, motility and number of normal sperm have been reported in RF workers (Lancranjan et al. [R372]). Schrader et al. [R681] and Weydant et al. [R682], however, could not confirm their own finding of a decrease in sperm numbers in U.S. soldiers exposed to radar.

B.6.1.3 Development

In an important long-term study, squirrel monkeys were exposed at 2450 MHz at three SARs (0.034, 0.34 and 3.4 W/kg) beginning during the second trimester of pregnancy. Mothers and offspring were exposed for an additional 6 months after parturition and the offspring were exposed for an additional 6 months. In the offspring, no significant changes were found upon examination of a wide array of endpoints. These included growth rate, four of five tests of behavioral development, EEG, biochemistry and hematology (Kaplan et al. [R363]). The effect measured in one of the behavioral studies was observed in the highest exposure group (3.4 W/kg); this group had a high mortality rate, an effect that was not replicated by the same laboratory (Kaplan et al. [R363]). Exposure of rats during gestation to 2450 MHz fields at thermal levels (16.6–22 W/kg) resulted in lower brain weight (Shore et al. [R437]) (Berman et al. [R538]). Long-term, continuous

exposure of rats during gestation at 0.4 W/kg (2450 MHz PW) caused no effect on development, fetal body weight, brain weight, or the DNA, RNA or protein content of the brain (Merritt et al. [R404]). A decrease in Purkinje cells in the cerebellum of rats after 100 and 2450 MHz exposures at ~3 W/kg could not be confirmed in squirrel monkeys exposed at 2450 MHz by the same laboratory (Albert et al. [R299], [R300]). Histologic examination of the brains of rats at 15, 20, 30 and 40 days of age following prenatal and postnatal 2450 MHz exposure from day four of gestation to 40 days of age (except for two days) revealed no effect on brain development, including no change in the relative number of Purkinje cells in the cerebellum. The brain SAR was ≥ 9.5 W/kg in 2–40 day old rats and the whole-body average SAR was 1.76 W/kg (Inouye et al. [R781]). There is no independent confirmation of reduced brain weight in 308 day old mice exposed *in utero* to 20 kHz magnetic fields (pulsed, 15 μT peak to peak) (Dimberg [R564]). Effects on the adrenal gland were observed in neonatal rats exposed to 2450 MHz at 9–10 W/kg (Guillet and Michaelson [R971]).

Rats exposed at 2450 MHz prenatally (days 5–20 of gestation) and perinatally (days 5–20 of gestation plus days 2–20 postnatally) had larger body mass and less swimming endurance at 30, but not 100 days of age. The estimated SAR in the fetal rats was 4 W/kg, and the SAR of rats aged 2–20 days was 5.5–16.5 W/kg (Galvin et al. [R45]). Exposure of rats throughout pregnancy at 3.6–5.2 W/kg (2450 MHz) did not significantly alter postnatal growth or physiological development, and no alterations were observed in five of six adult behavioral paradigms (Jensh et al. [R359]). Exposed females showed a significantly higher activity. Exposures at 7.3 W/kg (6000 MHz) produced effects on eye opening, postnatal growth, and behavior in a water maze as well as open field tests (Jensh [R361]). The SARs associated with behavioral effects are consistent with the conclusion that the threshold for such effects is about 4 W/kg. Prenatal exposure of rats to mobile phone signals had no effect on operant behavior of the rats in adulthood (Bornhausen and Scheingraber [R746]). RF fields at SARs of 0.2, 1.0 and 5 W/kg had no effect on development of rat embryos grown in culture (Klug et al. [R718]).

A series of studies at 2450 MHz investigated the development of the quail embryo exposed *in ovo* Gildersleve et al. [R49], [R50], [R51], [R52]) (Clark et al. [R288]) (Galvin et al. [R341], [R342]) (Hamrick and McRee [R351], [R771]) (McRee et al. [R770], [R780]) (Hamrick et al. [R772]) (Inouye et al [R773]) (McRee and Hamrick [R774]). Continuous exposure of quail embryos during the first eight days of incubation at 4 and 16 W/kg had no affect on the development of the heart (Galvin et al. [R341]). Brief exposures from 0.3–30 W/kg (CW and PW) had no affect on the heart rate of quail embryos that could not be attributed to temperature changes (Hamrick and McRee [R351]). Following hemorrhagic stress (30% blood lost) in young quail that had been exposed *in ovo* continuously to 2.45 GHz for the first 12 days of development at 4 W/kg, changes were found in the response of one enzyme (Gildersleve et al. [R51]) and changes limited to one sex were found in corticosterone levels (Gildersleve et al. [R52]) and leucopoiesis (Clark et al. [R288]). This exposure 1) slightly retarded the development of the external granular, molecular and the Purkinje cell layers in the cerebellum prior to hatching, while at eight weeks of age, no morphological changes in Purkinje cells were noted (Inouye et al. [R773]); 2) produced hematological changes (McRee and Hamrick [R774]), 3) reduced male reproductive capacity (McRee et al. [R780]), but 4) did not affect the immune response in both sexes (Gildersleve et al. [R50]) (Galvin et al. [R342]) (Hamrick et al. [R772]). Also not affected were the following parameters at 224 days of age: mortality after hatching, egg production or weight, fertility, hatchability of eggs produced and reproductive performance of the progeny (Gildersleve et al. [R49]). The RF effects that were reported are considered to be thermal effects, because continuous RF exposure of quail eggs during the first 12 days of development at 4 W/kg increased the egg temperature by 2.5–3 ºC. At an ambient temperature of 37 ºC, the RF exposure caused the temperature of the eggs to rise to 39.5–40 ºC, and only 7% of the eggs hatched. Therefore, to maintain the egg at the normal incubation temperature of 37 ºC, the ambient temperature was reduced to 35.5 ºC during exposure at 4 W/kg. This procedure was used in a number of the studies summarized above and resulted in a higher hatchability in exposed eggs compared with control eggs (McRee and Hamrick [R774]). In related studies with chicken eggs, 2450-MHz exposure during incubation at 2.9 W/kg did not affect hatchability (Braithwaite et al. [R185]) and temperature increases of 0.25–2.3 ºC were measured in embryonic and amniotic fluid in eggs exposed at 1250 MHz to 1.45-10.44 W/kg (Talau et al. [R1132]).

B.6.1.4 Summary of teratogenicity, reproduction, and development

Studies on the teratogenic effects of RF exposure, and other conditions that cause heat stress in animal models, have demonstrated that significant increases in the incidence of heat-induced abnormalities are seen at maternal temperature increases of approximately 2–2.5 ºC, mostly following exposures of tens of minutes up to one hour or so (Edwards et al. [R1081]). Some studies have reported that RF exposure could potentiate the effects of known teratogens, for example chemical teratogens and ionizing radiation, although the RF exposures produced significant maternal temperature increases as discussed above. The results of a few studies reporting teratogenic, reproductive, and developmental effects at low levels of RF exposure are generally weak in design and have not been confirmed independently. The weight of evidence from animal studies supports the conclusion that teratogenic, reproductive, or developmental effects do not occur unless the RF exposure is >4 W/kg, an SAR that causes a significant temperature increase above the normal body temperature. The weight of evidence from studies of human populations exposed to RF fields from video display units, magnetic imaging devices, medical diathermy units, heat sealers and radar does not suggest that teratogenic, reproductive, or developmental effects occur within the BRs and MPEs recommended in IEEE Std C95.1, 1999 Edition [B70] and those recommended in this standard.

B.6.2 Hematology and endocrinology

B.6.2.1 Hormone changes

A handful of reports cite changes in melatonin and various other hormones (Gildersleve et al. [R49], [R50], [R51], [R52]) (Abhold et al. [R291]) (Saddiki-Traki and Lescoat [R515]) (Deschaux and Pelissier [R547]) and neurotransmitters (Mausset et al. [R923]) in laboratory animals after low levels of RF exposure, although most hormone changes observed in animals have been at clearly thermal RF exposure levels (Lu et al. [R93], [R94], [R173], [R393], [R394], [R395]), (Michaelson et al. [R504], [R926]), (Merritt et al. [R924], [R925]). In some cases (Saddiki-Traki and Lescoat [R515]), (Deschaux and Pelissier [R547]) it is difficult to determine whether exposure levels were actually thermal or not, because of the absence of temperature measurement, inadequate temperature measurement, or inadequate reporting/description of dosimetric measurements. Small sample size is frequently a problem. A number of other studies reported no change in hormones following low-level, non-thermal RF exposures (Bonasera et al. [R15]), (Toler et al. [R130]), (Vollrath et al. [R614]), (Heikkanen and Juutilianen [R1051]). In humans, a marginal melatonin increase was associated with a study of occupational mobile phone use (Burch et al. [R1050]), although more controlled human provocation studies performed in multiple independent laboratories have not confirmed any effects on melatonin, growth hormone, luteinizing hormone, cortisol, or other hormones (de Seze et al. [R640], [R690]), (Mann et al. [R709]), (Radon et al. [R783]).

B.6.2.2 Immune function and hematology

A number of studies in animals have reported that at levels insufficient to cause a significant thermal increase, RF exposure does not cause any significant change in differentiation, mitogenic activity, function of immune cells, or other hematological endpoints in animals (Liddle et al. [R85], [R86]) (Djordjevich et al. [R161]) (Chou et al. [R322]) (Gandhi et al. [R345]) (Liddle et al. [R386]) (Guy et al. [R387]) (Smialowicz et al. [R441]) (Chagnaud and Veyret [R658]) (Braithwaite et al. [R1057]). This is also the case in isolated cell lines of hematopoitic origin or primary lymphocytes (Brown and Marshall [R16]), (Roberts et al. [R116]), (Cleary et al. [R325]). Some of these *in vitro* studies have even used extremely high SAR levels in conjunction with temperature control. Reports do exist of low-level RF exposures causing both increases and decreases in spleen immune cell subpopulations (Nakamura et al. [R648]) (Elekes et al. [R734]) (Dasdag [R1054]) and increased (Shao and Chiang [R123]), decreased (Lyle et al. [R396]), or mixed effects (Veyret et al. [R630]) in immunoglobulin titers and cellular immunity function. One study (Liburdy and Wyant [R383]) reported a possible RF induced shape change in Ig proteins exposed to low levels of RF energy in an LGC fractionation column.

A series of studies from a single laboratory in Poland reported that exposure of rabbits and guinea pigs to low RF levels depressed erythrocyte numbers and erythroblast proliferation, while conversely the same exposure was reported to stimulate lymphocyte proliferation. The exposure also was reported to cause mitotic disturbances, and changes in nuclear structure, and generated various other effects in combination with drugs on CNS function (Baranski and Edelwejn [R470]) (Baranski [R471], [R472], [R473]). Little information was provided on the actual conditions of exposure, making interpretation and confirmation of non-thermal conditions impossible.

When thermal levels of RF exposure are used, some studies continue to find no effect on autoimmune response (Anane and Veyret [R1052]) or other hematologic or immunologic endpoints (Galvin et al. [R343], [R344]) (Ortner et al. [R413]) (Ragan et al. [R420]) (Dunscombe et al. [R1059]) in animals and tissue culture. Many more studies at thermal levels of exposure report either increased or decreased immune cell function (Bogolyubov et al. [R14]) (Deschaux et al. [R160]) (Rotkovska et al. [R200]) (Smialowicz [R281]) (Bogolyubov et al. [R290]) (Huang and Mold [R353]) (Liburdy [R379], [R380], [R381]) (McRee et al. [R399]) (Rama Rao et al. [R421], [R422], [R423]) (Rotkovska et al. [R426]) (Smialowicz et al. [R438]) (Smialowicz et al. [R442], [R443], [R444], [R445], [R446], [R447]) (Takashima and Asakura [R455]) (Wiktor-Jedrzejczak et al. [R464]) (Yang et al. [R465]) (Galvin et al. [R546]) (Wiktor-Jedrzejczak et al. [R553], [R554], [R555]) (Nakamura et al. [R648]) (Logani et al. [R694]) (Ortner and Galvin [R1053]) (Dwivedi et al. [R1058]) (Pazderova-Vejlupkova and Frank [R1060]) (Pazderova-Vejlupkova and Josifsko [R1061]) (Logani et al. [R1062]), as well as the induction of stress markers (Cleary et al. [R158]) (Wangemann and Cleary [R463]) (Nakamura et al. [R648]) (Pazderova-Vejlupkova and Frank [R1060]), similar to the effects of non-RF heating to elevated temperatures (Rama Rao et al. [R421], [R422], [R423]). Other studies have shown no effect of RF exposure using GSM (Global System for Mobile Communications) signals on the immune system *in vitro* (Sultan et al. [R449], [R450]).

In a single Italian study of women living near radio-television broadcasting towers (500 kHz–3 GHz) with electric field strengths of 4.3 ± 1.4 V/m on their balconies (Boscolo [R1012]), the authors reported a reduction in immune cell numbers and activity. The study did not report any dose response, and seemed to leave many potential confounding factors uncontrolled. Another study in humans (Tuschl et al. [R1056]) reported no effect on immune cell population or function in humans occupationally exposed to RF during diathermy treatments. The same group did report an increase in natural killer cells, as well as the occurrence of oxidative bursts in monocytes in a more recent study of hospital personnel operating MRI units and industrial workers using induction heaters (Tuschl et al. [R1055]).

B.6.2.3 Summary of hematology and endocrinology

While studies have reported effects on hematological and immunologic endpoints in animals and *in vitro* models, the majority of the studies were performed at thermal levels of RF exposure and the reported effects are most likely the result of heating and elevated temperature. In the few studies that have reported effects at low-level (non-thermal) exposure levels, the findings are generally inconsistent with each other as well as with the larger body of evidence reporting no effects at these exposure levels.

B.6.3 Blood brain barrier (BBB) permeability

B.6.3.1 Review of BBB Studies

Two reports from the former Soviet Union were the first to describe effects of RF exposure on the BBB (Kleyner et al. [R366]) (Polyashuck [R928]). The first article on this subject in the western literature appeared in 1975 when Frey et al. [R335] reported that an SAR of approximately 1 W/kg caused increased BBB permeability in rats. In 1977, Oscar and Hawkins [R415] reported increased BBB permeation at 0.4 W/kg (CW) and 0.1 W/kg (PW). Later, in response to criticism from Preston et al. [R419] that the changes in Oscar and Hawkins [R415] may have been due to blood flow changes, Oscar et al. [R416] measured and found increased local brain blood flow after RF exposure. Because of this finding, Oscar et al. concluded

that their earlier BBB permeability effects may have been smaller than reported. Oscar then co-authored the paper by Gruenau et al. [R167], who used a technique to measure BBB permeability that is insensitive to blood flow change; no effect of RF exposure was found. The effect in their original report was most likely an artifact; this conclusion is supported by the results of other studies. With techniques used by Oscar and Hawkins [R415], other investigators (Preston et al. [R419]), (Preston and Prefontaine [R799]) could find no effect of RF exposure on BBB permeability at whole body SARs estimated to be 0.02–6 W/kg, or at SARs in the head ranging from 0.08–1.8 W/kg. Additional attempts to replicate or confirm the effects in Oscar and Hawkins [R415] and Frey [R335] have been unsuccessful (Ward et al. [R257]) (Ward and Ali [R258]) (Lin and Lin [R388]) (Merritt et al. [R402]). Frey [R485] also reported that RF exposure of rats caused a small increase in the permeability of the blood-vitreous humor barrier but, based on preliminary experiments, there was no reported effect on the blood-placental barrier [R1027].

A series of studies from Albert's laboratory (see [R299], [R533], [R1016], [R1017], [R1018]) reported increased BBB permeability using a different technique (electron microscopy) than those used by Frey [R335] and Oscar and Hawkins [R415]. Effects were reported in rats and hamsters exposed at SARs ranging from 0.9–2.5 W/kg, but later work (Tsurita et al. [R723]) failed to confirm the effects. Ward et al. [R257] used RF exposure conditions similar to those of Albert [R1016] and found no increase in permeation, after correcting the data for thermal effects due to absorbed RF energy.

Sutton et al. [R452] exposed pigs repeatedly for 1 min followed by a 9-min pause for 8 h/d for 90 days by fitting the animal's head with a leather harness holding a standard two-way portable radio; the peak brain SAR was 8.1 W/kg. The BBB remained intact in the exposed animals and, in addition, neurohistological and enzyme-histochemical preparations failed to show any evidence of damage to nervous tissue or chronic reaction to injury in the brain. Other recent animal studies showed no BBB permeability changes after a one hour exposure at 4 W/kg (whole body), or after lifetime exposures at SARs ranging from 0.25, 0.5, 1.0 and 4.0 W/kg (whole body) (Finnie et al. [R841], [R851]).

Sutton and Carroll [R451] found that RF exposure of the rat head, which produced a brain temperature of 40 ºC or more, caused increased BBB permeation. When the body core temperature of the rat was kept at 30 ºC during RF exposure of the head, the exposure time had to be extended to observe effects on the BBB. These results indicate that hyperthermia caused by absorbed RF energy disrupted the BBB, as this disruption could be prevented or decreased by perfusion of the brain with cooled blood. The animal's body temperature was maintained well below normal by the transit of the cooled blood. Merritt et al. [R402] showed that BBB permeation was affected in rats heated to 40 ºC by hot air or RF exposure, and concluded that hyperthermia was the causative factor, not RF energy per se. In a series of four papers, Williams and colleagues [R259], [R260], [R261], [R262] concluded that RF effects on the BBB are mediated by temperature dependent changes, and are not a direct non-thermal effect of the RF energy. Similarly, Fritze et al. [R735] found blood brain barrier permeability changes in rats in a pattern consistent with thermal effects. Other papers have demonstrated that changes in BBB permeability are due to the thermal effects of RF exposure (Neilly and Lin [R106]) (Goldman et al. [R347]) (Lin and Lin [R388], [R389]) (Moriyama et al. [R598]) (Ohmoto et al. [R927]).

Two papers describe effects on the BBB resulting from an RF exposure in combination with exposure to a virus or a drug, domperidone. RF exposures that increased the rectal temperature of mice by 1.5 ºC or more reduced survival following inoculation with Japanese encephalitis virus; the expression of lethality of this virus requires entry into the central nervous system (Lange and Sedmak [R80]). High-level RF exposure (45.5 W/kg) facilitated drug action by increasing BBB permeability in mice (Quock et al. [R114]). These results are consistent with the weight of evidence demonstrating that BBB permeability is affected by RF exposures that cause a significant increase in brain temperature.

It has been suggested that the magnetic field associated with MRI exposure may alter BBB permeability (Prato et al. [R573]). However, no effect on the BBB was found when exposure was to the RF signal only (Garber et al. [R486]).

Other reports are not consistent with the evidence presented above. Schirmacher et al. [R722] reported an increase in permeability in a cell culture model of the BBB, when it was exposed at a low SAR. Neubauer et al. [R107] found that 2 W/kg, but not 1 W/kg, caused a BBB change in rats. Chang et al. [R319] reported that one of six RF exposure levels affected BBB permeability in dogs, although no exposure-response relationship was found. Persson et al. [R753] reported that exposures at 915-MHz (CW and PW) affected the BBB. Although CW exposures were reported to increase the number of rats exhibiting increased BBB permeability by about 3-fold, the change did not follow an SAR-response relationship over four ranges spanning 0.02–8.3 W/kg. The results with modulated RF fields also were not SAR dependent. The lowest SAR range (0.0004–0.008 W/kg) showed the highest increases at all modulations (4, 8.3, 16, 50 and 217 Hz) and, at the highest SAR range (1.7–8.3 W/kg), no modulation frequency was effective. The data for 217 Hz showed that this modulation frequency was not effective at the highest SAR range or at the next to lowest range, but 217 Hz was effective at the other two ranges, including the lowest SAR range. The 1997 paper by Persson et al. [R753] stated that their earlier reports (Salford et al. [R251], [R651]), (Persson et al. [R740]) were preliminary results and the 1997 paper appears to include data from all previous studies in their laboratory. Persson et al. stated also that their "…method for detection of albumin is extremely sensitive and reveals even minute amounts of albumin leaking through the BBB, so small that they may be harmless to the brain." A more recent report [R980] from this group describes effects on neurons and the BBB in rats exposed to SARs ≤ 0.2 W/kg.

In drafting this standard, reports of the effects of RF exposures on the blood brain barrier that could (or could not) result in other changes that were cumulative with time were discussed. Assuming that changes in the blood brain barrier do occur at or below 4 W/kg, it would have to be demonstrated that an intermittent chronic (a few hours per day) or continuous chronic (almost 24 hours per day) exposures had resulted in measurable morphologic, histopathologic, functional, or behavioral change. Any of these could be reflected by alterations of the performance of the animal or individual exposed, or the function of a wide-range of organs in the body, since the different tissues in the brain play an important role in many body functions. Even if evidence was substantiated of a blood brain barrier effect, it would be important to know that adverse morphologic, histopathologic, functional, or behavioral changes resulted from the exposures. Based on a weight-of-evidence analysis of the available literature, there is no substantiated *in vivo* literature demonstrating such adverse effects for any RF exposure at SARs ≤ 4 W/kg.

B.6.3.2 Summary of blood brain barrier (BBB) permeability

In contrast to the lack of confirmation of effects of low-level RF exposure on the BBB, when no heating is measured or expected to occur, the results of many investigators have confirmed that the permeability of the BBB can be affected by a significant increase in temperature caused by absorption of RF energy. In most reports, thermal effects have been demonstrated by uptake of radiotracers, dyes and large proteins such as albumin. Two studies have shown increased uptake of virus particles and drugs. Based on modeling studies, a localized exposure of the head at 2 W/kg will produce an increase in localized brain temperature of about 0.2 ºC. This increase in temperature is very small in comparison with the increase that is associated with the changes in BBB permeability described above. The published reports of permeability changes in the BBB at SARs <4 W/kg are not useful in the development of exposure guidelines, because the effects have not been confirmed and no dose-response relationship is evident.

B.6.4 Eye pathology

B.6.4.1 Review of eye pathology studies

Whole-body (far-field) RF exposure studies show that cataracts form in rabbit eyes only if intense fields at or near lethal levels are applied (Williams et al. [R817]) (Hirsch et al. [[R1000]]). Cataracts can also be produced by localized (near-field), high-intensity exposures of the eyes of the dog (Baillie [R527]) (Baillie et al. [R528]) (Daily et al. [R943]) and rabbit (Cogan et al. [R326]) (Carpenter and Van Ummersen [R942]) (Hagan and Carpenter [R944]). Continuous and pulsed RF exposures at the same average power were shown to be equally effective in producing cataracts in rabbits (Birenbaum et al. [R807]), a result that is consistent with a thermal mechanism. In general, cataractogenic near-field exposure levels were so thermally stressful that localized exposure of the eye (and head) caused the whole body (rectal) temperature of rabbits to increase by 1.2–2.7 ºC (Foster et al. [R40]) (Carpenter et al. [R988], [R998]); whole-body exposure at these levels produced extreme body temperatures resulting in death (Appleton et al. [R264]) (Appleton [R467]).

In localized (near-field) studies at 2.45 GHz, threshold exposure conditions for cataracts of ≥ 150 W/kg for ≥30 min have been determined; these conditions are associated with temperatures ≥ 41 ºC in or near the lens of the rabbit eye (Kramar et al. [R654], [R947]), (Guy et al. [R698]), (Carpenter et al. [R941]), (Carpenter [R1003]). At the same frequency, cataracts were not observed in the monkey eye exposed to similar high intensity fields (Kramar et al. [R1001]). This difference reflects the different patterns of RF energy absorption in rabbit and monkey heads due to their different facial structure. Since the monkey head is similar in structure to the human head, the results of the non-human primate study indicate that the frequency dependence of cataractogenesis in rabbits and human beings would be different. While it is reasonable to assume that an RF exposure that would induce temperatures ≥ 41 ºC in or near the lens in the human eye would produce cataracts by the same mechanism (heating) that caused cataracts in the rabbit lens, such an exposure would greatly exceed the currently allowable limits for human exposure and would be expected to cause unacceptable thermal effects in other parts of the eye and face. For example, human eye modeling studies at 1500 MHz (Taflove and Brodwin [R951]) and 2450 MHz (Neelakantaswamy and Ramakrishnan [R507]) suggest that power densities greater than 1000 W/m^2 could cause SARs and temperatures in or near the human lens that are known to produce cataracts in rabbit eyes; such exposures would increase the temperature of the cornea by about 6 °C (Taflove and Brodwin [R951]). This temperature increase is twice that of the corneal surface of the rabbit eye, which, when exposed at 26.5 W/kg, caused corneal edema and other ocular effects (Saito et al. [R695]). Two relevant modeling studies of the human eye showed that for 50 W/m^2, the MPE at 1.5 GHz for controlled environments (e.g., FCC [B41]), a temperature change in the lens of less than 0.3 ºC at frequencies from 0.6–6 GHz (Hirata et al. [R946], [R999]) would be expected.

RF exposures that produced lens opacities in rabbits almost always caused inflammation of the iris (Birenbaum et al. [R806]). Other ocular effects, including corneal lesions, retinal effects and changes in vascular permeability, were reported in non-human primates by Kues' laboratory following both CW and PW exposures (Kues and Monahan [R66]), (Kues et al. [R67], [R242]). However, the inconsistencies in Kues' results, the failure by Kamimura et al. [R580] to independently confirm corneal lesions after CW exposure, the failure by Lu et al. [R739] to independently confirm retinal effects after PW exposure, and the absence of functional changes in vision (Lu et al. [R739]), (McAfee et al. [R950]) are reasons why the ocular effects reported by Kues and colleagues are not useful in defining the adverse effect level for RF exposure. Kues et al. [R684] did not observe corneal damage, changes in vascular permeability of the iris, or lens opacities in the rabbit or monkey eye exposed to 60 GHz fields at 100 W/m^2. Histological examination of the cornea of rabbit eyes exposed at a high intensity (2250 W/m^2) to both CW and PW fields showed no effects, but neither SAR nor temperature data were given (Williams and Finch [R813]). A high intensity pulsed RF exposure causing a temperature rise to 40 ºC near the retina of rabbits resulted in degenerative retinal changes but no cataracts; no effect on blood-brain barrier permeability or retinal vascular permeability (Paulsson et al. [R511]) was observed. In contrast to these findings, Frey [R485] reported increased permeability of the blood-vitreous humor barrier in rats exposed to pulsed fields at low average power. An appropriate control group was not used for one exposure group, however, and no information on SAR or temperature in the eye was provided.

A comparative study (Hagan and Carpenter [R944]) of relative effects at 2.45 and 10 GHz found that the cataractogenic potential was greater at the lower frequency, a result consistent with peak energy absorption at the higher frequency occurring in tissues near the surface of the eye and not in or near the lens. At the higher frequencies of 35 and 70 GHz, that did not cause opacities in the lens (Rosenthal et al. [R551]), effects were observed in other tissues of the rabbit eye, e.g., inflammation of the cornea. The frequency-dependent distribution of RF energy observed in the rabbit eye demonstrates that higher frequencies have greater potential for effects on the structures near the outer surface of the eye, and lower potential for effects within the eye, such as lens opacities.

Changes in DNA synthesis and mitosis (Van Ummersen and Cogan [R952]) and ascorbic acid levels (Kinoshita et al. [R1008]) in lenses of rabbits receiving a cataractogenic exposure are attributed to thermal effects. Also, the effects on glutathione level and peptidase activity in the lens of rabbits exposed to RF fields, which caused a 2–3 ºC rise in the interior of the eye, are attributed to thermal effects (Bernat [R266]). An *in vitro* experiment with rabbit lenses found no difference in ascorbic acid concentrations in RF-exposed and control samples subjected to identical time-temperature conditions and in samples exposed to CW and PW fields at the same average power (Weiter et al. [R812]). Stewart-DeHaan and colleagues [R30], [R174], [R519] reported effects in the rat lens exposed *in vitro* to RF energy, but the usefulness of these studies, and a related modeling study (Wyeth [R526]), have not been established. The effects have not been independently confirmed. Threshold values for similar effects, if they occur in live animals, are not known. Based on changes at the cellular level, two recent papers from China (Ye et al. [R1069]), (Juan et al. [R1072]), speculated that an acute, low-level microwave exposure would cause cataracts in rabbits. The papers do not provide sufficient experimental details, including SAR values, to allow replication of the results.

Long-term exposure of rats (Utteridge et al. [R846]), rabbits (Guy et al. [R350]), and monkeys (McAfee et al. [R397], [R950]) did not cause cataracts or other ocular effects. In these studies, rats were exposed at 0.25–4.0 W/kg, rabbits received 17 W/kg in the head and monkeys received 20 and 40 W/kg in the face. These and other study results (Chou et al. [R321], [R322]) support the conclusion that clinically significant ocular effects, including cataracts, are unlikely to occur in human populations exposed for long periods of time to low-level RF fields. Case reports of cataracts involving a few workers (Issel and Emmerlich [R355]) are not supported by studies of larger populations. Five human studies, some without statistical evaluation and most with little or no RF exposure data, failed to demonstrate clinically significant ocular effects (Hollows and Douglas [R144]) (Aurell and Tengroth [R203]) (Cleary and Pasternack [R206]) (Majewska [R949]) (Odland [R1002]). A number of other studies reported no ocular effects in human populations (Siekierzynski et al. [R152]) (Appleton and McCrossan [R201]) (Appleton et al. [R202]) (Cleary et al. [R205]) (Shacklett et al. [R225]) (Hathaway et al. [R945]). One of these studies (Hathaway et al. [R945]) did not confirm the retinal effects reported in an earlier study (Aurell and Tengroth [R203]). The data in Appleton and McCrossan [R201] was analyzed by Frey [R222], who came to the conclusion that there was a statistically significant increase in lens abnormalities in the RF exposed group. A further independent evaluation found that Frey's analysis was improper and led to an erroneous conclusion (Wike et al. [R208]). This independent statistical analysis [R208] confirmed the results of studies of U.S. military personnel, which showed no association between RF exposure and ocular effects (Appleton and McCrossan [R201]) (Appleton et al. [R202]).

An ocular effect (abnormal cone function) was reported in a man exposed twice for 15 min to 6000 MHz while inspecting a satellite antenna (Lim et al. [R948]). The exposures were sufficiently intense to cause facial erythema (eyelid burns), bilateral foreign body sensation and blurred vision, but no cataracts were reported. These observations support the conclusion that the high exposure levels required to produce cataracts in the human eye would cause undesirable effects on other parts of the eye and face.

Four studies addressed eye cancer in human populations exposed to RF energy. Two of these studies reported an association between RF exposure and uveal melanoma, a cancer of the pigmented vascular tissue in the eye including the iris (Stang et al. [R749]) (Holly et al. [R838]). The authors of one of these papers, however, concluded that several methodologic limitations prevented their results from providing clear evidence for the hypothesized association (Stang et al. [R749]). In an attempt to confirm these observa-

tions, Johansen et al. [R808] contrasted the incidence rate of this rare cancer with the number of mobile phone subscribers in Denmark. No increasing trend in the incidence rate of ocular malignant melanoma was found, while the number of mobile phone subscribers is increasing exponentially. In earlier work, Johansen et al. [R767] found no association between mobile phone use and eye and brain cancer, leukemia and more than 20 other cancers in a cohort study of 420,000 users of mobile phones. The three most recent studies of eye cancer (Stang et al. [R749]) (Johansen et al. [R767], [R808]) and mobile phone use therefore failed to provide clear supporting evidence for the results described in the earliest study (Holly et al. [R838]).

B.6.4.2 Summary of eye pathology

In summary, adverse effects of RF exposure of the eye, i.e., cataracts, are associated with significant temperature increases due to the absorption of RF energy. The maximal permissible RF exposures in this standard are therefore protective against the significant temperature increases that can result in adverse effects on the eye, such as cataracts. There is no evidence of other significant ocular effects, including cancer, which would support a change in the adverse effect threshold of 4 W/kg.

B.6.5 Auditory pathology and RF hearing

B.6.5.1 Review of RF hearing studies

Exposure of the human head to high peak pulsed RF power can result in the perception of sound. This phenomenon, which is known as “RF hearing,” or “microwave hearing” is a well-established biological effect (Frey [R824], [R1065]) (Frey and Messenger [R828]) (Airborne Instrument Labs [R953]), which of itself has no known adverse health consequence. RF-induced sound has been characterized as a click, buzz, hiss, knock or chirp, and is best detected in extremely quiet environments, often with subjects inserting earplugs to reduce background noise (Cain and Rissmann [R204]) (Guy et al. [R487]) (Frey [R824], [R825], [R826]) (Ingalls [R957], (Khizhnyak et al. [R958]) (Tyazhelov et al. [R963]) (Constant [R1067]). RF hearing requires the ability of the exposed person to detect high-frequency acoustic waves in the range of ~5–8 kHz as well as bone-conduction hearing responding to lower acoustic frequencies (Cain and Rissmann [R204]), (Frey [R824], [R1065]) (Airborne Instrument Labs [R953]) (Rissmann and Cain [R1066]). The fundamental frequencies able to produce RF sound in the human head, based on animal data and modeling are similar, e.g., 7–10 kHz (Chou et al. [R955]), 8-15 kHz (Lin [R863], [R864], [R865]) and 7–9 kHz (Watanabe et al. [R965]). Effective radiofrequencies reported in the literature range from 2.4 to 10,000 MHz (Cain and Rissmann [R204]) (Frey [R825], [R826]) (Ingalls [R957]) (Roschmann [R1075]). Since there are no reports of human perception of RF energy at frequencies higher than 10,000 MHz, the physiological significance of calculated RF hearing thresholds at 30–300 GHz is unknown (Gandhi and Riazi [R46]).

The pathway by which acoustic waves are detected by the ear and interpreted by the brain as sound involves mechanical distortion of cochlear hair cells, due to thermoelastic expansion, resulting in cochlear microphonics, i.e., electrical potentials that mimic the sonic waveforms of acoustic stimuli. Subsequent to the detection of sound by the cochlea, electric potentials associated with the detection of sound may be recorded by electrodes in neurons at various locations along the auditory pathway. Chou et al. [R481] reported recording of cochlear microphonics from RF-exposed animals after two other attempts were unsuccessful (Chou et al. [R487]) (Frey [R827]). This discovery, that RF sound is perceived by the auditory system, provided evidence against the proposal that RF pulses directly stimulate the central nervous system (Frey [R825]). Other research demonstrated that the RF-induced auditory sensations were similar to acoustic sound detection once the cochlea was stimulated; that is, RF stimuli and acoustic stimuli gave similar electrophysiological responses along the auditory pathway (Chou et al. [R481]) (Lebovitz and Seaman [R498], [R697]) (Taylor and Ashleman [R742]) (Frey [R827]) (Lin et al. [R869]). The middle ear, however, is not required, as RF-induced auditory responses were found in animals in which the middle ear had been ablated (Chou and Galambos [R482]), (Chou et al. [R487]), (Wilson et al. [R525]), (Taylor and Ashleman [R742]). Several studies have reported thresholds for the RF-induced auditory sensation in laboratory animals (Seaman and Lebovitz [R122]), (Cain and Rissmann [R204]), (Guy et al. [R487]), (Lebovitz and Seaman [R498]).

The RF hearing phenomenon depends on the energy in a single pulse and not on the average power density. Guy et al. [R487] found that the threshold for RF-induced hearing of pulsed 2450-MHz signals was related to an energy density of 0.4 J/m^2 (40 μJ/cm^2) per pulse, or energy absorption per pulse of 16 μJ/g. The rapid thermoelastic expansion that produces audible sounds results from only a 5×10^{-6} °C temperature rise in tissue due to the absorption of the energy in the RF pulse (Foster and Finch [R484]) (Gournay [R956]) (Sommer and von Gierke [R962]) (White [R966]). The literature on microwave auditory effects indicates that the energy in a pulse delivered within the first 30 μs to 70 μs would be most efficient at producing acoustic pressure waves, while the efficiency for pulses longer than about 50 μs depends primarily on peak SAR level, this being in the range of about 10,000 W/kg peak (ARPANSA [B11]). The experimental weight-of-evidence, and the results of modeling studies, support the thermoelastic expansion theory (Lin et al. [R87]) (Chou and Guy [R320]) (Chou et al. [R323], [R954]) (Foster and Finch [R484]) (Guy et al. [R487]) (Lebovitz and Seaman [R498], [R697]) (Olsen and Lin [R509], [R510]) (Frey and Messenger [R828]) (Lin [R864], [R871]) (Joines and Wilson [R1073]) (Roschmann [R1075]). This evidence does not support an alternate proposal by Frey [R825], [R827] that pulses of RF energy directly stimulate the central nervous system. The failure (Frey and Coren [R829]) to measure thermoelastically induced mechanical vibrations in the head predicted by the thermoelastic expansion theory was shown to be due to lack of sensitivity of the holographic technique (Chou et al. [R960]). No published report supports the suggestion by Tyazhelov et al. [R963] that the theory does not explain all characteristics of RF hearing.

One of the studies that confirmed the finding that RF hearing does not involve the middle ear reported similar changes in the auditory system of rats exposed to continuous wave and pulsed fields (Wilson et al. [R525]). The results with a continuous wave field have not been independently confirmed. There are no other reports of continuous wave signals causing auditory responses in animals, and there are no reports of continuous wave signals causing RF-induced sound in humans.

Although the RF field was not pulsed and no RF-induced sound would occur, one group has investigated functional effects in the auditory system of RF exposed rats by measuring cochlear emission as an indicator of pathological changes in outer hair cells. No changes in otoacoustic emissions were found at average SARs in the head of 0.2 and 1 W/kg (Marino et al. [R831]).

Additional information on RF hearing is available in reviews and fact sheets listed in the following references (ARPANSA [B11]) (Chou et al. [R594]) (Lin [R390], [R867], [R868], [R870], [R872], [R1006]) (Postow and Swicord [R961]) (Elder and Chou [R1096]) (Stewart [R1133]) (Elder and Cahill [R1134]).

B.6.5.2 Summary of auditory pathology and RF hearing

The phenomenon of RF hearing in humans is a well-established biological effect with no known adverse health consequence. The RF-induced sounds are similar to other common sounds. They can be characterized as the perception of sounds of low intensity because, in general, a quiet environment is needed for the sounds to be heard. The RF fields in experimental magnetic resonance studies of the human head can cause RF-induced sound pressures about 10,000 times the threshold for RF hearing. There is no evidence, however, for detrimental health effects from RF induced sounds caused by magnetic resonance systems (Roschmann [R1075]). A comparison with ultrasound pressures during routine medical diagnosis, including exposure of the fetus, suggests that RF-induced pressures more than five orders of magnitude greater than the pressure at the hearing threshold would be unlikely to cause adverse health effects (Watanabe et al. [R965]). Based on this comparison, the exposure limit in the IEEE C95.1, 1999 Edition [B70] and this standard for a single RF pulse of 576 J/kg (spatial peak), although 36,000 times greater than the threshold for RF hearing in humans, is below potentially adverse effects levels (Elder and Chou [R1096]).

B.6.6 Membrane biochemistry

A few studies have reported effects of RF exposure on *in vitro* membrane function (Phillipova et al. [R112]), [R250]), (Alekseev and Ziskin [R286]) and protein leakage through artificial and cellular membranes

(Savopol et al. [R478]). One *in vivo* study reported that 2.45 GHz RF exposure at 1.4 W/kg to mice and cell lines resulted in changes in intestinal, brain, and cell surface membrane morphology, as well as changes in cell surface charge distribution, in a manner dependent upon the AM modulation (Somosy et al. [R220], [R282]). However, significant variability and a lack of a consistent correlation with SAR were common in these responses. It is possible that the RF exposure resulted in local temperature increases, which may have contributed to the observed effects. Many studies have documented the effects of higher (thermal) levels of RF exposure on membrane fluidity and ion transport (Liu and Cleary [R88]) (Phelan et al. [R111]) (Phillipova et al. [R112], [R250]) (Sandweiss [R119]) (Orlando et al. [R249]) (Bergqvist et al. [R265]) (Neshev and Kirilova [R278]) (Allis and Sinha [R301], [R302]) (Friend et al. [R336]) (Kim et al. [R364]) (Liburdy and Penn [R382] (Liburdy and Magin [R384]) (Liburdy and Vanek[R385]) (Olcerst et al. [R412]) (Shynrov et al. [R436]) (Arber and Lin [R469]) (Barsoum and Pickard [R474], [R642]) (Pickard and Barsoum [R512]) (Portella et al. [R513]) (Saalman et al. [R516]) (Sandblom and Thenander [R517]) (Webber et al. [R524]) (Baranski et al. [R529]) (Brunkard and Pickard [R539]) (Galvin et al. [R544]) (Bliss et al. [R560]) (Fesenko and Gluvstein [R565], [R566]) (Weaver [R571]) (Eibert et al. [R673]) (Benz and Zimmerman [R931]) (Weaver et al. [R939]) (Tyazhelov et al. [R964]).

B.6.7 Calcium studies and neuron conduction

B.6.7.1 Calcium studies

A paper published in 1975 described changes in calcium ions associated with chick brain samples exposed *in vitro* to amplitude-modulated (AM) RF fields (Bawin et al. [R476]). This was called the "calcium efflux effect," a change in the quantity of calcium ions released from brain tissue into a bathing solution shortly after exposure; it does not refer to calcium ion movement across the cell membrane. The 1975 paper sparked considerable interest because brain tissue was used, the effective AM frequencies are found in the electroencephalogram (EEG) of awake animals, the exposure level was too low for RF heating, and the changes in calcium were modulation dependent. Statistically significant effects were reported for modulation at 6, 9, 11, 16, and 20 Hz, with the maximal response at 16 Hz, and no effects for an unmodulated field or for modulation at 0.5, 3, 25, and 35 Hz. Initial interest also was high because the *in vitro* calcium studies were conducted to follow up on animal studies showing an effect on operant conditioning of cat behavior by RF fields that were amplitude modulated at 3, 6, 9, and 16 Hz (Bawin et al. [R476]). The effect also appeared to be power dependent (Blackman et al. [R311]) (Sheppard et al. [R435]), leading to the description that the calcium efflux response occurred only within "windows" in both frequency and power. Numerous calcium ion studies were conducted over many years in attempts to explore the biological significance of exposure to low-level modulated fields and to develop physical models to account for the reported dependence on modulation frequency and power.

The first publication (Bawin et al. [R476]) on calcium efflux describes a result that is often overlooked in interpreting the physiological significance of this effect, i.e., the calcium efflux was shown to be independent of metabolism because the effect was the same in normal and cyanide-poisoned (i.e., dead) brain samples. For this and other reasons, the U.S. Environmental Protection Agency concluded that the physiological significance of the effect on calcium efflux was not established (Elder and Cahill [R1134]), and a later report states that "…no obvious indications of human health hazard currently can be concluded from in vitro RF radiation research results" (EPA [B37]). These EPA reports addressed chick brain studies that were published in the period from 1975–1991 (Albert et al. [R5]) (Blackman et al. [R11], [R12], [R13], [R229], [R310], [R311], [R312], [R313], [R768]) (Shelton and Merritt [R434]) (Sheppard et al. [R435]) (Bawin et al. [R476]), [R477]) (Joines and Blackman [R491]) (Bawin and Adey [R535]).

The chick brain studies stimulated a variety of experiments with other neurological tissue samples exposed to similar and different (i.e., pulsed) RF fields. The following responses have been reported with regard to a 16-Hz (AM) RF field exposure: 1) With cats exposed *in vivo*, irregular increases in calcium efflux from the brain were observed (Adey et al. [R298]); 2) Increased calcium efflux and increased ornithine decarboxylase activity were found in the brains of rats exposed *in vivo* (Paulraj et al. [R1046]); 3) With electron micros-

copy, examination of the brains of mice exposed *in vivo* showed a modified Ca^{++}-ATPase activity and a redistribution of calcium at the synapse, i.e., the exposure induced the appearance of calcium precipitates in the synaptic cleft and on the outside of the neuronal plasma membrane while the calcium content of synaptic vesicles decreased (Kittel et al. [R626]); 4) Studies with neuroblastoma cells from human and rodent cell lines reported effects on calcium efflux at specific AM frequencies and SAR levels similar to those found to be effective in chick brain experiments (Dutta et al. [R38], [R332]); 5) Increased calcium efflux was reported in rat brain synaptosomes exposed *in vitro* (Lin-Liu and Adey [R171]).

In contrast to the AM studies, RF fields pulsed at repetition rates numerically equal to the frequency of sinusoidal modulations (e.g., 8, 16 and 32 Hz) that were used in the chick-brain experiments had no effect on calcium efflux from rat brain tissue exposed *in vitro* (Merritt et al. [R403]) (Shelton and Merritt [R434]) or from the brains of rats exposed *in vivo* (Merritt et al. [R403]).

Calcium efflux has also been examined after RF exposure in pancreatic, skeletal muscle and heart tissue samples. An increase in calcium efflux from slices of rat pancreas exposed *in vitro* was not associated with leucine release, indicating that the 16-Hz AM RF exposure did not affect intracellular calcium (Albert et al. [B4]). The first study with chick brains also reported that electromagnetic fields similar to those causing the effect in brain samples did not affect calcium efflux from chick skeletal muscle (Bawin et al. [R476]). Such fields had no influence on the contractile response and kinetics of calcium efflux from isolated atrial strips of the frog heart (Schwartz and Mealing [R212]). The authors stated that these negative results apparently contradicted previously reported findings from the same laboratory showing that 16-Hz AM RF fields increased calcium efflux from intact frog hearts (Schwartz et al. [R121]). Exposure of frog hearts *in vitro* to 16-Hz modulated CW and pulsed fields had no effect on the beating rate (Yee et al. [R135]) and pulsed RF fields, modulated at 16 Hz, had no effect on the beating rate of rat hearts in the absence of RF heating (Yee et al. [R136]). An increase was observed in the inter-beat interval of chick cardiac cells exposed *in vitro* to unmodulated (CW) RF fields at SARs ≥ 1.2 W/kg, while fields with a modulating square-wave frequency of 16 Hz had no effect (Seaman and DeHaan [R150]). To examine whether reported calcium efflux changes could cause changes in the excitability of cell membranes, myocytes of guinea pig and rat hearts were exposed to RF fields (180, 900 and 1800 MHz) that were pulsed according to the GSM-standard for mobile phones. Measurements were made of membrane potential, action potential, L-type Ca^{++} current and potassium current. None of these electrophysiological parameters were changed by RF exposure (Linz et al. [R685]).

Four studies explored the influence of RF fields on intracellular free calcium concentrations $[Ca^{++}]_i$ in cells exposed *in vitro*. Two studies found no effect and two reported changes that were possibly due to an artifact associated with the $[Ca^{++}]_i$ assay. No relevant effects were found on $[Ca^{++}]_i$ in guinea pig heart cells exposed to three different RF signals that were pulse modulated at frequencies reported to cause calcium efflux in chick brain and other samples (Wolke et al. [R576]). For exposures at 2 W/kg, there was no clear indication that mobile phone signals changed $[Ca^{++}]_i$ or calcium signaling in human lymphocytes exposed at 915 MHz (GSM and CW) (Cranfield et al. [R932]). An effect on $[Ca^{++}]_i$ in mouse neuroblastoma cells exposed to a 5 kHz signal (16 Hz AM) was attributed to an artifact of the UV-A[10] irradiation used with the fluorescent assay for $[Ca^{++}]_i$ (Ihrig et al. [R1122], [R1124]).

The SAR threshold for changes in Na^+, K^+ and Ca^{++} concentrations in blood and salivary glands in rats exposed to pulse modulated RF fields was more than 1.5 times the established adverse effect level of 4 W/kg (Furmaniak [R337]). Also, pulsed 27.1 MHz exposure of rats did not alter the 300% rise of calcium (tissue dry weight) in infarcted brain tissue (Rappaport and Young [R514]).

In the absence of evidence for physiological or health effects attributable to calcium efflux effects, and the inconsistent results of both *in vitro* and animal studies, the available information has not proven useful in the development of exposure standards. For these reasons, the papers on calcium efflux are not reviewed critically or described in detail here, although several of the key papers were cited above. The IEEE database

[10]UV-A is defined as wavelengths in the UV portion of the electromagnetic spectrum between 315 and 400 nm.

includes additional related papers (McLeod et al. [R97]) (Prasad et al. [R113]) (Fisher et al. [R165]) (Geletyuk et al. [R273]) (Athey [R304]) (Bawin et al. [R475]) (Bawin and Adey [R534], [R535]) (Kaczmarek and Adey [R683]) (Greengard et al. [R745]). Detailed reviews of this literature are also available (NRPB [R788]), (UNEP/WHO/IRPA [B129]).

B.6.7.1.1 Calcium studies: summary

Several reports that have reviewed the calcium efflux effects literature support the conclusion that, notwithstanding unresolved research questions, calcium effects from exposure to low-level amplitude modulated RF fields cannot be used in setting RF exposure standards. In its review, a UNEP/WHO/IRPA [B129] report concluded that the original observation was not sufficiently well defined, and could not be characterized as a potential adverse health effect. An NRPB report [B104] observed that if the phenomenon of calcium efflux were biologically significant, concomitant changes would be expected in the functions of nervous tissues that depend on the movement of calcium ions. No such functional alterations have been demonstrated unambiguously; the report included the statement that there was no strong reason to believe that 16-Hz modulation has special effects. A more recent NRPB review [B105] did not mention effects of amplitude modulated RF fields on calcium efflux.

B.6.7.2 Neuron conduction

Exposure of hippocampal slices *in vitro* to 700 MHz (CW) RF was reported to both increase and decrease evoked and spontaneous population spikes, depending upon the exposure level (Tattersall et al. [R797]). Amplitude modulation (AM) at 16 Hz in this system had no effect on the results. In contrast, Pakhomov et al. [R1070] did not observe effects on population spikes using a similar hippocampal slice system exposed at 9.3 GHz (CW). Reports that modulated RF exposures decreased electrical activity in isolated snail neurons (Arber et al. [R287]) (Arber and Lin [R303], [R468], [R469], [R917]) (Lin and Arber [R500]) seem to contradict reports that RF exposure either increased firing rate (Beasond and Semm [R976]) (Shchurov et al. [R1047]) or had no effect (Wang et al. [R602]) on isolated neurons. Further, a number of studies have reported that clearly thermal levels of exposure can result in decreased firing amplitude and a prolonged refractory phase in isolated neurons (McRee and Wachtel [R98], [R398], [R400]) (Wachtel et al. [R462]) (Seaman [R518]). No effects of even very high levels of RF exposure were observed if cooling techniques were used to prevent temperature elevation (Chou and Guy [R643]).

B.6.8 Other animal studies

Various other non-cancer endpoints affected by acute thermal exposures to animals have included altered digestive function (Santini [R431]), increased serum triglyceride and beta-lipoprotein levels (Deficis et al. [R159]), increased rate of liver regeneration (Ottani et al. [R1024], [R1025]), increased tissue water content and conductivity (Mikolajczyk et al. [R505]). These un-replicated studies present no consistent evidence of effects due to RF exposure and are in general inconsistent with long term animal study results that indicate no detrimental effects of exposure up to 4 W/kg (see B.7.1).

B.6.9 Human provocation studies

B.6.9.1 Cognitive function and memory

Studies have reported that mobile phone RF exposures result in either improved (Preece et al. [R664]) (Kellnyi et al. [R707]) (Jech et al. [R795]) (Koivisto et al. [R796]) (Lee et al. [R844]) or hindered (Lass et al. [R985]) cognitive function and memory in humans. These studies report changes of very small magnitude, and at least one laboratory has not been able to replicate their earlier results (Koivisto et al. [R708]), (Haarala et al. [R959]). Studies of Latvian children living in proximity to a radar station reported a decrease in acoustical and visual reaction, neuromuscular function, memory, and attention (Lacal [R1032]), although serious flaws in the study design may have introduced artifacts. Other studies report that mobile phone RF

exposure has no effect on memory performance or cognitive function (Freude et al. [R655]) (Preece et al. [R664]) (Freude et al. [R715]) (Krause et al. [R719]) (Kelly et al. [R1036]). Two recent studies have found no effect on RF exposure from mobile phones on cognitive function in children (Preece et al. [R1141]), (Haarala et al. [R1142]).

B.6.9.2 EEG, sleep disturbances, and event related potentials

Studies in humans exposed to mobile phone signals have reported augmentation of sleep and increases in various EEG spectral bands (Reiser et al. [R600]) (von Klitzing [R601]) (Lebedeva et al. [R1042], [R1043]), mainly around the 10 Hz alpha frequency EEG band (Borbély et al. [R703]) (Huber et al. [R736]) (Lebedeva et al. [R1042], [R1043]). Other reports after similar RF exposures have either observed decreases in these same alpha frequency EEG bands (Croft et al. [R994], [R1087]), or no effects (Hietanen et al. [R856]) (Kim [R1033]) (de Seze [R1034]). In studies looking at event related potentials (ERPs), some effects have been observed (Freude et al. [R655], [R715]).

Initial studies by Mann and Röschke [R597] and Mann et al. [R710] using mobile phone signals for RF exposure did indicate effects on shortening sleep onset time, as well as reduction of REM stage and altering EEG recordings during sleep (i.e., getting to sleep faster). In follow up sleep studies, these parameters were not statistically affected even at levels 100-250 times higher than used in previous studies (Wagner et al. [R638], [R1035]), (Röschke and Mann [R609]), although the authors suggest that different characteristics associated with RF exposure may have caused the seemingly discrepant findings. In awake EEG studies, no statistically significant effects on EEG were reported [R609]. There was also no associated change in growth hormone, luteinizing hormone, or melatonin in exposed subjects, although there was a transient increase in cortisol levels (Mann et al. [R709]). In a summary review, the authors conclude from their findings that while there may be some slight biological effects, their data do not suggest any adverse consequences associated specifically with mobile phone exposure (Mann and Röschke [R1121]).

Disruption of sleep has been reported in subjects exposed to RF energy either occupationally (Bielski [R267]) or living in the vicinity of RF broadcasting towers (Santini et al. [R859], [R989]), (Altpeter et al. [R977]). Some studies of event-related brain potentials in humans have reported mixed effects (Freude et al. [R655], [R715]) (Eulitz et al. [R675]) (Kellenyi et al. [R707]) while other studies reported no effects (Hladký et al. [R758]) (Urban et al. [R794]), (Kim [R1033]). A study in narcoleptics found that ERPs were affected only when the visual stimuli appeared on the same side of a computer screen as the phone (Jech et al. [R795]). Initial findings (Krause et al. [R719], [R802]) of event-related potential changes during working memory tasks were not repeatable in a double blind replication study by the same investigators (Krause et al. [R1063]). It remains a challenge to separate the effect of direct RF fields and the effect due to induced RF current brought into the head by the conductive leads.

B.6.9.3 Headache and fatigue

Seven studies of correlations between headache and RF exposure derived data from subjects through questionnaires. Headache incidence and proximity to RF broadcast towers or use of mobile phones yielded a positive correlation (Hocking [R693]) (Oftedal et al. [R755]) (Sandstrom et al. [R777]) (Chia et al. [R849], [R919]) (Santini et al. [R859], [R989]). However, problems with bias were not clearly addressed in these studies. A lack of relevant exposure assessment disallowed any meaningful dose-response to substantiate the reported effects. In other studies, subjects occupationally exposed to radar at incident field strengths of ≤50 W/m^2 exhibited no headaches, fatigue, or irritability attributable to the microwave exposures (Djordjevich et al. [R162], [R190]). In addition, two controlled laboratory provocation studies examining the effects of RF exposure on headaches have reported no effect (Koivisto et al. [R779]), (Paredi et al. [R1044]).

B.6.9.4 Hypersensitivity

Hypersensitivity has been reported in individuals in association with exposure to computer VDU display units (Stenberg et al. [R574]) (Sandstrom et al. [R819]), as well as with RF from occupational (Bini et al.

[R195]) and other external sources (Flodin et al. [R920]) (Choy et al. [R986]). An early Polish study of RF exposed industrial workers reported an increase in certain subjective endpoints including irritability, perspiration, dizziness, and other bioeffects (Bielski [R267]), but significant limitations in the study design make the results difficult to interpret. Hypersensitivity and subjective symptoms have recently become an issue with RF exposure from mobile phones and cell site antennas. Although well performed laboratory studies with controlled provocation in normal (Koivisto et al. [R779]) and self-claimed hypersensitive subjects (Hietanen et al. [R835]) have reported no association between the self-reported hypersensitivity and RF exposure from mobile phones, Hocking [R693], [R842] reported links between various subjective symptoms and mobile phone exposure. A study from Japan (Kimata [R1048]) reported that subjects with a history of eczema and dermatitis (AEDS) showed increased allergic reaction in a skin wheal assay following exposure for 60 minutes to mobile phone emissions, although the suggestion in a follow-up study that the larger effect may be associated with the annoyance of the ringing phone and its disruption on normal activities (Kimata [R1086]). A preliminary study in Spain (Navarro et al. [R1116]) in the vicinity of an 1800 MHz (GSM) base station reported a correlation between RF exposure and various subjective endpoints associated with "microwave sickness" or "RF syndrome."

B.6.9.5 Effects on blood pressure/heart rate

While a provocation study by Braune et al. [R656] initially reported an increase in blood pressure (BP) and heart rate (HR) following exposure to a GSM mobile phone operating at 900 MHz, these effects were subsequently found not to be repeatable by the same group [R847]. The blood pressure and heart rate increases were not confirmed by independent studies in other laboratories (Tahvanainen et al. [R1049]). A single study (Lu et al. [R663]) in rats exposed to low level ultra-wideband (UWB) exposure indicated the opposite, i.e., a hypotensive effect on blood pressure; this study has not been independently confirmed. Studies exposing the backs of normal human volunteers to RF energy greatly exceeding the levels used in the Braune et al. [R656] and Lu et al. [R276] studies did not cause any change in heart rate (Adair et al. [R782]). While a number of animal studies have reported effects of RF exposure on BP and HR, these have all been at clearly thermal exposure levels (Frei et al. [R42], [R271], [R272]) (Jauchem et al. [R62], [R168], [R169], [R170]) (Phillips et al. [R417]) (Frei and Jauchem [R582]) (Jauchem [R882]).

B.6.9.6 Summary of human provocation studies

No consistent evidence exists to indicate an adverse effect of low-level RF exposure on the nervous system. However, because of the variety of different effects reported by some investigators and the many contradictory reports, research in this area continues.

B.7 Cancer-related studies

B.7.1 Animal cancer bioassays

Animal studies have served as a critical and often primary source of information in toxic and carcinogenic assessments of chemical and physical agents by programs such as the National Toxicology Program (NTP) in the U.S. and the International Agency for Research on Cancer (IARC). Long-term animal bioassays, performed over two years in two species (usually rats and mice), and in both males and females, offer reasonable surrogates for human lifetime exposure when epidemiological data is insufficient, impractical, or otherwise unavailable. Chemical, or ionizing radiation initiated animal models have also been used in studies designed to investigate the possible promoting effects of chemical or physical agents. In addition, genetically altered animals, e.g., *Pim*-1 transgenic mice have been used in RF investigations, although the response of this particular transgenic strain has not been validated against known human carcinogens and non-carcinogens.

B.7.1.1 Long-term animal bioassays

A number of long-term animal bioassays have been performed exposing rats and mice to different RF signals for various daily periods. The vast majority of studies performed at both low-level and thermal levels have indicated no pathological or cancer effects (Toler et al. [R621]) (Frei et al. [R637]) (Spalding et al. [R652]) (Adey et al. [R677], [R727]) (Frei et al. [R692]) (Zook and Simmens [R778]) (Jauchem et al. [R934]) (La Regina and Roti Roti [R1019]). A 1962 study (Prausnitz and Susskind [R558]) reported exposed animals survived longer but reported cancer of white cells; a review of this work (Roberts and Michaelson [R194]) criticized the study for major experimental deficiencies. Thermal level exposures, but not low-level exposures over the lifetime of mice were reported to shorten mean lifespan (Liddle et al. [R246]). As one arm of a large study by Utteridge et al. [R846], mice were exposed (120 per group) to RF at SARs of 0.25, 0.5, 1.0, or 4.0 W/kg (whole body average) for 1 hour/day, 5 days/wk, for 104 weeks. This study, with improved exposure methods, was to verify whether the positive effect reported by Repacholi et al. [R606] is repeatable (see below). No pathological or cancer effects were observed by Utteridge et al. [R846]. The only report of a tumor increase due to long-term RF exposure at low levels was by Chou et al. [R138]. A slight increase in overall tumor incidence was reported in rats exposed for 2 years to 2450 MHz at low SAR levels (0.15-0.4 W/kg). A possible increase in pheochromocytoma (based upon only 7 tumors in exposed vs. 1 in sham exposed animals) was observed. No primary brain tumors were observed. The authors did not interpret these observations as biologically significant due to the lack of a clear and consistent increase in individual tumor types and the absence of an adverse effect on survival. Their conclusion is supported by the studies reporting the absence of induction of tumors after chronic lifetime studies listed above. There was also no effect in the Chou et al. study [R138] on a large number of other physiological and behavioral parameters (a totaling of 155 individual endpoints was examined), including open field behavior, immune function, hematology, serum chemistry, thyroxine, protein parameters, metabolism, and growth. In another study, a single adult squirrel monkey was exposed over a long period of time and at necropsy was found to have a malignant neuroectodermal tumor of the right cerebral cortex (Johnson et al. [R680]). It is impossible, however, to make any conclusion from a finding in a single animal without even a control.

B.7.1.2 Investigation of tumor promotion by RF using animal bioassays

Like long-term animal studies involving investigations of RF exposure alone, studies of promotion of tumor development and growth using initiated animals have been largely negative. Many different initiated (genetically damaged or altered) animal models have been used for RF studies, including ENU-initiated rat brain tumors (Adey et al. [R677], [R727]) (Zook and Simmens [R778]), benz(a)pyrene initiated rat sarcomas (Chagnaud et al. [R689]), DMBA initiated rat mammary tumors (Bartsch et al. [R839]), DMBA initiated +/- TPA co-promoted skin pappillomas in SENCAR mice (Mason et al. [R818], [R1021]), DEN induced GSTp(+) rat hepatomas (Imaida et al. [R699]), dimethylhydrazine induced mouse colon tumors (Wu et al. [R263]), and ionizing radiation induced mouse lymphomas (Heikkinen et al. [R1022]). These have consistently demonstrated an absence of tumor promotion by RF fields. In a recent study (Anane et al. [R1107]) using DMBA initiated rat mammary tumors, rather inconsistent results were reported. The authors concluded that this study did not provide evidence of a promotion effect of RF exposure.

Positive promoting effects of RF exposure on breast tumors in C3H/HeJ mice and benz(a)pyrene initiated skin tumors in normal Balb/C mice were reported in the early work of Szmigielski et al. [R254] and Szudzinski et al. [R192]. These data conflict with all other studies performed in DMBA and similar chemically initiated animal models, and no independent confirmation of the Szmigielski et al. work has yet been reported.

In addition to chemicals and ionizing radiation, genetically initiated animal models in the form of transgenic mice have been employed in RF carcinogenicity testing. A study by Repacholi et al. [R606] using transgenic *Pim*-1 mice did report an association between long-term RF exposure and mortality from a certain subtype of lymphoma (follicular), but did not report a statistically significant increase in lymphoblastoid lymphomas. The *Pim*-1 transgenic model was specifically reported to use appearance of the latter type of lymphoma to reveal carcinogens in a shorter time frame than used for the detection of the follicular

lymphomas. A subsequent study, performed at multiple exposure levels with a more uniform and better characterized exposure field, was not able to confirm the initial *Pim*-1 findings (Utteridge et al. [R846]). Hybrid transgenic mice designed to overexpress ODC and wild type mice were initiated for skin tumors with UV radiation. They were then exposed to 900 MHz (GSM and DAMPS) RF for 5 hours/day, 5 days/week for 1 year at an SAR of 0.5 W/kg (whole body average) (Heikkinen et al. [R1101]). RF exposure did not result in a statistically significant effect on the development of skin tumors in either the transgenic or non-transgenic mice. No effects on body weight, survival, urinary 6-hydroxymelatonin sulphate (6-OHMS) levels, polyamine levels, or ODC activity were found. Another transgenic mouse model (pKZ-1) was used in the investigation of intra chromosomal recombination inversions following exposure at 4 W/kg. The authors stated that the reduction in inversions below the spontaneous frequency that they observed had no biological significance (Sykes et al. [R1020]).

B.7.1.3 Tumor cell line injection bioassays

Studies of tumor progression, performed by injecting established tumor cell lines into the original mouse strains and determining growth rate, survival, and metastatic progression, have reported increased survival of the host, as well as inconsistent evidence of either augmentation or suppression of immune function in response to thermal levels of RF exposure (Santini et al. [R120]) (Preskorn et al. [R191]) (Rozkowski et al. [R1023]). In studies using 2–3 and 6–8 W/kg exposure levels, the Szmigielski laboratory reported increased metastatic growth of L1 lung sarcoma cells injected into Balb/C mice (Szmigielski et al. [R522]). Similarly designed studies in other laboratories using different tumor cell lines reported no such effects (Salford et al. [R252]), (Higashikubo et al. [R702]).

B.7.1.4 Acute animal studies

Several short term studies have been conducted that relate to cancer. Although these studies can only be considered as pilot or range finding investigations, the results are supportive of the longer term and more definitive studies indicating an absence of an RF-induced effect. Two studies (Imaida et al. [R699], [R700]) looked at liver tumor formation in rats exposed to 929 MHz (PDC) signals for 90 min/day, 5 days/week, for 6 weeks at SAR values of 1.7–2.0 W/kg maximal in the liver (0.6–0.8 W/kg whole body average), and found no effect on foci formation. Moderate increases were reported in serum ACTH, corticosterone (stress), and melatonin levels. New Zealand rabbits were exposed to 2450 MHz (CW) microwaves 7 h/day, 5 days/week for 13 wks at an SAR of either 0.7 W/kg in the back (0.5 W/kg in the head) or 7 W/kg in the back (5.5 W/kg in the head) using a horn antenna (Chou et al. [R322]). No effects were observed on body mass, cataract formation, blood chemistry, blood protein, lymphocyte activation, or tissue pathology (indicating no evidence of cancer cells).

B.7.1.5 Summary of animal cancer related studies

The scientific weight of evidence from the 35 animal bioassay studies discussed above provides evidence of no physiological, pathological or disease-specific effects of long-term RF exposure, including lifetime exposures, at levels up to 4 W/kg (Utteridge et al. [R846]). Those few studies that have reported effects after low level exposures are either not corroborated in similar studies, or the results could not be verified in followup studies. These long term studies clearly indicate a lack of evidence that RF exposure causes or promotes tumor induction. Furthermore, no adverse effect was found on longevity or body mass as a result of chronic RF exposures, at SARs in the range of 0.5–4 W/kg. Although these studies do not give clear thresholds for effects, they are helpful in defining no observable adverse effect levels (NOAEL) in the long-term studies.

B.7.2 Other animal and *in vitro* studies addressing cancer

In assessing the health hazard of any agent, including RF energy, human or epidemiological studies are given supreme weight. The results of animal studies become considerably important when human data is weak or absent. *In vitro* laboratory systems for assessing the biological effects of exposure play a supportive role only. The results of *in vitro* studies should never be used by themselves to provide a definitive answer as to whether or not a given agent under a given set of experimental parameters has no physiological effect, or is beneficial or harmful to animals, or by extrapolation, to humans.

B.7.2.1 DNA single strand breaks (SSBs) and/or DNA double strand breaks (DSBs)

Studies by Lai and Singh [R275], [R617] have reported DNA breaks in the brain cells of rats exposed at 2450 MHz. These studies described differences in the ability of 2-h pulsed wave exposures and 2-h continuous wave exposures to cause such breaks at the end of the exposures, and at a later time after exposure. Independent replications, albeit with modifications of the initial procedure (Malyapa et al. [R641]) failed to confirm the finding. An extensive study of this subject comparing different methods of comet assay analysis and including an attempt at exact replication of the original studies failed to demonstrate any increase in DNA damage due to RF exposure (Lagroye et al. [R1117]). A major *in vitro* investigation performed at mobile phone frequencies and modulations with even higher SARs (Tice et al. [R815]) resulted in the absence of induction of DNA SSB. Careful examination of the actual data in another *in vitro* paper (Phillips et al. [R687]), and the inherent inconsistency and small changes reported, lead to concern over the conclusion reached. The overwhelming number of studies using mammalian cell lines and freshly isolated human cells (e.g., peripheral lymphocytes) indicates an absence of DNA strand breaks (Malyapa et al. [R634], [R635]) (Vijayalaxmi et al. [R724], [R752]) (Maes et al. [R754]) (Li et al. [R789]) (Alekseev and Ziskin [R790]) (Tice et al. [R815]) (McNamee et al. [R935], [R936]).

B.7.2.2 Specific DNA absorption

If the DNA is to be damaged, it would have to be due to some type of direct energy absorption by the DNA resulting in chemical damage, or some type of induction of a reactive chemical species resulting from (and during or after) the RF exposure. There have been papers published theorizing that DNA can absorb RF energy (Van Zandt et al. [B133]), and papers have also been published reporting experimental evidence for specific absorption of RF at specific frequencies (Sagripanti et al. [R118]) (Edwards et al. [R163], [R164]) (Swicord and Davis [R521]) (Davis et al. [R562]). Careful subsequent studies by two laboratories independently failed to confirm these observations (Foster et al. [R41]) (Gabriel et al. [R44], [R612]). The initial results appear to have been the result of a measurement artifact.

B.7.2.3 Chromosome aberration induction

In vitro investigations of the possible induction of chromosomal damage have a relatively long history in the field of RF bioeffects. Early studies presented the case for chromosome aberrations and "erosion" (Heller [B57]), and subsequent studies advocated RF effects on chromosome aberrations in several mammalian systems (Chen et al. [R155]), (Yao [R556]). These studies had technical and analysis problems; Chen et al. [R155], for instance, first said that there was not a statistically significant increase, and then proceeded to discuss the increase in selected types of chromosome aberrations. An examination of the induction of chromosome aberrations by Lloyd et al. [R90], [R172] reported no increase due to RF exposures. A very careful and extensive examination was undertaken by Kerbacher et al. [R178]. Chinese hamster ovary cells were exposed to pulsed wave 2450 MHz fields for 2 hours at a very high SAR (33.8 W/kg), which was high enough to cause an increase in the culture medium temperature to approximately 40 ºC. A total of 14 different indicators of chromosome aberrations were scored or calculated, including total aberration events per 100 cells and percentage aberrant cells. In all cases, there were no differences between the RF exposed cells and the 37 ºC incubated cells or temperature control shams. The authors went one step further and explored the hypothesis that the high SAR RF exposure could cause an increase in the extent of chromosome aberrations induced by two known chemical clastogens, mitomycin C and Adriamycin. The result again was the

absence of a statistically significant difference of any of the indices compared to the sham exposed temperature (water bath heated) and chemically treated "control" cells. Many experiments were independently repeated, and there were multiple replicate independent exposure flasks for each exposure condition in each experiment. Subsequent to this study, a number of additional studies looking for the induction of chromosome aberrations from RF exposure have been published using different cell types and different exposure conditions (including wireless frequencies and modulations). The overwhelming evidence is that the induction of chromosome aberrations by exposure to RF fields does not occur (Vijayalaxmi et al. [R731], [R752], [R968]), (Maes et al. [R754], [R793], [R967]). There are reports of RF exposures causing chromosome aberrations *in vitro* (Garaj-Vrhovac et al. [R187], [R188], [R189]); these studies typically have inherent technical flaws, or experimental ambiguities based on the exposure system employed.

B.7.2.4 Micronucleus formation

The examination of exposed cells for micronuclei is a relatively new approach for detecting damage at the chromosomal level, especially since the assay is less costly, less tedious, more rapid, and allows for automated scoring. It has been made clear by a number of authors that there are (at least) two mechanisms of formation of micronuclei (MN). One is an apparent disruption of the mitotic apparatus, resulting in enclosure of a whole chromosome with its centromere present (not an indicator of direct chromosome damage by a clastogen). The second mechanism is the encapsulation in a membrane of a small piece of a chromosome. The occurrence of the latter is taken to indicate clastogenic activity of an agent to which the cells are exposed. It is not clear that cells with damage in the form of MN would continue to survive reproductively. Again, one would expect some evidence of cell death or inhibition of cell proliferation if MN were caused by RF exposure. There are reports of the induction of MN by exposure of mammalian cells in vitro to specific frequencies and modulations (d'Ambrosio et al. [R800], Tice et al. [R815]). At the same time, there is a much more abundant literature describing the absence of the induction of MN (Vijayalaxmi et al. [R752], [R968], [R969]) (McNamee et al. [R935], [R936]) (Bisht et al. [R1026]) sometimes in the same cell type and after exposure conditions similar to that used in studies reporting effects. It should also be noted that if MN are present in cells, some evidence of DNA strand breaks in cells exposed similarly would be hypothesized. This has not been demonstrated in the same studies by Tice et al. [R815] in which MN induction was observed. While it can be suggested that the assay for MN is more sensitive than that for DNA strand breaks, the presence of chromosome aberrations of any type means that there are extensive DNA double strand breaks in at least the cell that has the MN present; it is therefore not clear why there is no evidence of SSBs in some reasonable number of other cells under the same exposure conditions. In any event, this result is being explored further as of the time of the drafting of this standard.

An *in vivo* investigation of MN induction has also been performed. While the increase in MN was initially reported (Vijayalaxmi et al. [R622]) not to be statistically significant in a chronic animal RF tumor induction experiment, the initial publication was corrected (Vijayalaxmi et al. [R732]) to report a statistically significant increase of 1 micronucleus in 2000 cells examined. The authors did not consider this increase biologically meaningful, and no statistically significant increase in MN was found in those animals which were exposed chronically to RF and which did have tumors (although the RF was shown not to be responsible for the tumors present).

B.7.2.5 Sister chromatid exchange (SCE) induction

The assay for sister chromatid induction may or may not truly reflect a genotoxic endpoint; the SCEs could be the result of a problem in the mitotic machinery of the cell. An extensive investigation of SCE induction as a result of RF exposure was undertaken by Ciaravino et al. [R26], [R177] at 2450 MHz, pulsed wave, with a 0.1 duty cycle and a reported SAR of 33.8 W/kg. There was no evidence (even with a temperature increase in the medium due to the RF exposure) of any increase in the frequency of SCEs. Expanding the hypothesis to look for an interaction between the RF exposure and simultaneous treatment with chemical mutagens known to induce SCEs (mitomycin C or Adriamycin), no statistically significant increase was found for the 2 h chemical and RF exposure compared to the chemical exposure alone. In a series of studies by Maes et al., the authors' results were inconsistent. After initially reporting that an RF exposure caused an

increase in the SCEs induced by a subsequent mitomycin C treatment (Maes et al. [R581]), the subsequent study was inconsistent (Maes et al. [R754]), with the last two studies reporting that the effect was not present (Maes et al. [R793], [R967]).

B.7.2.6 DNA repair synthesis

There is only one published study (Meltz et al. [R99]) examining the possible induction of DNA repair synthesis resulting from RF exposures. Cells were exposed at three frequencies at 10, 50 and/or 100 W/m^2, (SAR 0.39–4.5 W/kg depending on frequency) for 1–3 h. The results of the series of experiments, using a normal human fibroblast cell line, at frequencies of 350, 850 and 1200 MHz, and where the cells were exposed either while being maintained at 37 °C or 39 °C during the exposure and repair labeling period, was the demonstration of an absence of any increase in repair labeling due to the RF exposure.

B.7.2.7 Inhibition of DNA repair synthesis

In the same study examining the possible induction of DNA repair synthesis in pre-existing DNA as a result of RF exposures of normal human fibroblast cells, experiments were also performed to determine if RF exposures could interfere with the DNA repair synthesis process after the DNA of the cells was damaged by an acute UV-C[11] exposure (Meltz et al. [R99]). The result of this investigation was that the RF exposures had no effect on the repair rate of the UVC damaged DNA; the evidence is that RF exposure does not interfere with this important type of DNA repair, which occurs after DNA base damage.

B.7.2.8 Phenotypic mutagenesis

Most of the above evidences are related to assessment of direct and immediate damage of the DNA and the genetic apparatus of the cell, or to post-exposure damage of the cell through some unknown mechanism. In both *in vitro* and *in vivo* systems, such damage, if it persisted, would likely lead to cell death or to a decrease or loss of cell function (functional death). If the DNA damage was repaired, to the extent that the cell with any residual DNA alterations survived, the result could be a mutated cell. This may or may not result in a detectable phenotypic alteration in one or more of such mutated cells (and their daughter cells).

There are only a limited number of published studies of phenotypic mutations after *in vitro* or *in vivo* exposures to RF fields. The most comprehensive is the work of Meltz et al. [R100], [R179] with multiple RF exposures, multiple independent treatment flasks for each exposure condition, and multiple replicated experiments. The cells were exposed to pulsed waved 2450 MHz fields for 2 h, at an average SAR that resulted in a temperature increase in the culture medium during the exposures. There was no evidence for the induction of phenotypic mutations as a result of the RF exposures. The RF exposures were repeated while simultaneously treating the cells with either mitomycin C or proflavin, known chemical mutagens. There were no differences in all cases between the RF exposed cells and the comparable sham/temperature controls. A study examining the possibility of RF induced mutations has also been performed *in vivo* (Takahashi et al. [R860]). The exposures of the "Big Blue Mice" to RF energy were for 4 weeks. No statistical evidence was found for RF induced mutagenesis.

B.7.2.9 Transformation

The mammalian cell transformation assay involves exposing specific cell lines that are capable of being transformed *in vitro* to agents that are hypothesized to be carcinogens. A positive in the assay is not definitive, because even before the agent can be proposed to be a human carcinogen, the transformed cells must be demonstrated to be "anchorage independent," i.e., able to form colonies in agar, and then to be able to form tumors in animals. The first reports (Balcer-Kubiczek and Harrison [R8], [R9], [R10]), taken together, indicated that the RF exposure employed, by itself, was unable to transform the cells. The final conclusion of the authors was that if the cells were treated with a tumor promoter, the RF could act as an initiator. This is not

[11]UV-C is defined as wavelengths in the UV portion of the electromagnetic spectrum between 190 and 280 nm.

consistent with the weight of evidence indicating that the RF is not mutagenic. An independent attempt to transform mammalian cells (Roti Roti et al. [R756]) by RF exposures at mobile phone frequencies failed to demonstrate transformation.

B.7.2.10 DNA damage, cell cycle elongation, cell toxicity, and decreased cell proliferation

When the DNA of a normal cell is damaged, a number of significant events will occur. Since the cell is normal, it will have a functioning p53 gene, and therefore the stress response to the agent causing the mutation will include the activation of check point genes at the G1/S border and potentially in G2 before the G2/M border. When these genes function, the progression of some of the treated cells out of G1 and into S phase/stage, and out of G2 into M stage will temporarily cease. Whether or not the cell will ultimately live or die, there will be evidence of mitotic delay (measured by a decreased mitotic index), a prolongation of the individual cell's cycle time, and an increase in the cell population doubling time. All of these events (and any cell death) would lead to a decrease in measured cell proliferation (and not an increase) for some period of time after the treatment. In addition to the association of DNA damage with decreased proliferation, there is an association of gross (light microscope visible) chromosome aberrations and cell death. In fact, if there is evidence that a treatment were to result in chromosome aberrations, and/or micronuclei formation, and/or DNA single strand breaks, and/or any other type of DNA damage, there should be evidence for some type of cell death. This could be apoptosis, reproductive death or giant cell formation in proliferating cells, or apoptosis, necrosis or functional death in differentiated cells. It is therefore important to be aware that the overwhelming weight of evidence from chronic lifetime animal exposures to RF fields at different exposure levels, different frequencies, and different modulations (cf. B.7.1), is the consistent absence of any stated evidence for tissue necrosis in any organ examined using standard histopathological techniques (Chou et al. [R138]) (Repacholi et al. [R606]) (Toler et al. [R621]) (Frei et al. [R637], [R692]) (Adey et al. [R677], [R727]) (Utteridge et al. [R846]) (Takahashi et al. [R860]).

B.7.2.11 Proliferation, growth rate, and cell cycle analysis

The majority of studies have reported no effect of low levels of RF exposure on growth and proliferation in various cell lines *in vitro*, either using mobile phone signals (Hoque and Gandhi [R61]) (Stagg et al. [R610]) (Higashikubo et al. [R1028]) or other RF signals (Krause et al. [R217]) (Wiktor-Jedrzejczak et al. [R464], [R555]) (Czerski et al. [R543]) (Vijayalaxmi et al. [R731]) (Fuhr et al. [R852]) (Hamrick and Fox [R1029]). A few *in vitro* studies have reported effects at low levels, although these are inconsistent in their findings and include both increases (Stodolnik-Baranska [R520]), (Daniells et al. [R714]), (Donnellan et al. [R750]), (de Pomerai et al. [R809], [R1104]) and decreases (Garaj-Vrhovacet al. [R187]), (French et al. [R657]), (Velizarov et al. [R670]), (Kwee and Raskmark [R720], (Szabo et al. [R786]) in growth and proliferation, as well as non-linear and frequency dependent changes in the cell cycle time of yeast (Grundler [R645]).

In one set of *in vitro* studies, mobile phone-type RF exposure at extremely low levels was reported to cause an increase in apoptotic gene expression and a 20% apoptosis, followed by a proliferation increase in the subset of surviving cells (Marinelli et al. [R991], [R992], [R993]). The authors speculated that exposure gave cancer cells a "greater survival chance, a phenomenon linked to tumor aggressiveness," and further promoted a commercial protective device that they claimed could block such effects in mobile phone users. Studies by Cleary et al. [R28], [R29], [R561], [R603], [R604] using very high exposure levels (25 W/kg or more) in a reported thermally controlled system showed increases in proliferation and cell cycle progression in human glioblastoma and CHO cell lines. It has been shown, for example, that very small (≤ 1 °C) incremental changes in culture temperature from non-RF heating can cause significant effects on proliferation in yeast (Pakhomov et al. [R814]).

In studies using bacterial cells, increased growth was reported from RF exposures that resulted in small localized temperature increases (Grospietsch et al. [R567]), while larger temperature effects resulted in decreased growth (Morozov et al. [R572]). Experiments designed to investigate the possibility of using microwaves for spore inactivation found no non-thermal microwave response (Welt et al. [R285]); the authors reported that the effects observed were indistinguishable from conventional heating. A study in

nematodes (de Pomerai et al. [R809]) reported that extremely low levels of RF exposure resulted in an ~10% size increase (hypothesized as a faster rate of progression through the life cycle), and a 30%–40% increase in the proportion of egg-bearing adults (as opposed to a decreased growth and lack of egg-bearing maturation in worms heated to 28 °C using non-RF conventional heating). The results did not directly correlate in a dose dependent manner with SAR modeling of the exposure system,

B.7.2.12 Gene and protein expression and activity

A number of studies have examined the effects of RF exposure on expression of different cell response genes that are known to change in response to treatment with chemicals and other insults/stresses. These studies are largely negative (Parker et al. [R109]) (Bush et al. [R154]) (Morrissey et al. [R584]) (Ivashuk et al. [R607]) (Goswami et al. [R662]) (Stagg et al. [R820]) (Li et al. [R821]), but some do report changes in expression after low-level (Romano-Spica et al. [R704]) (Weisbrot et al. [R1077]) or higher level (~thermal) RF exposures (Fritze et al. [R676]) (Natarajan et al. [R836]). In some cases (Makrides et al. [R1009]) the absence of dosimetry makes the study impossible to evaluate or replicate. When taken together, the positive studies do not demonstrate a consistent effect, with both increases and decreases being reported. There is no successful independent confirmation of any of the positive results. Likewise, studies of ODC and a handful of other enzymes and protein kinases report various increases and decreases (Byus et al. [R21], [R316]) (Chiang and Yao [R23]) (Dutta et al. [R39], [R234], [R483]) (Fisher et al. [R165], [R166]) (Krause et al. [R217]) (Somosy et al. [R220], [R282]) (Litovitz et al. [R501], [R618]) (Baranski [R530]) (Kubinyi et al. [R591]) (Penafiel et al. [R608]) (Porcelli et al. [R627]) (La Cara et al. [R659]) (Seaman et al. [R801]) (Verma and Dutta [R803]) (Pacini et al. [R990]) (Kim et al. [R997]) (Paulraj et al. [R1046]) (Szabo et al. [R1078]) (Markkanen et al. [R1118]); these are also not consistent in their effects. In addition, many studies looking at endpoints similar to those above report either no effect (Allis and Sinha-Robinson [R7]), (Millar et al. [R405]), (Takashima [R453]), (Allis and Fromme [R466]), (Galvin et al. [R545]), (Yeargers et al. [R557]), (Makheja et al. [R857]), or an effect only at thermal levels of exposure (Gandhi and Ross [R47]), (Saffer and Profenno [R117]). While three studies reported that RF exposure might accelerate denaturization and/or polymerization of proteins (Bohr and Bohr [R706]) (Lubec [R1031]) such effects were not repeatable in other laboratories (Ortner et al. [R415]) (Petruchelli and Fisher [R1030]). A study in nematodes reported that very low levels of RF exposure resulted in elevations in heat shock protein expression (hsp 27) (Daniells et al. [R714]) (de Pomerai et al. [R728], [R759], [R1104]), although recently the authors have given presentations reporting that their original findings could not be replicated. A second laboratory reported hsp 27 induction and phosphorylation changes in cell lines following RF exposure, but the increased expression required much higher SAR levels than in the nematode study, and may have been due to local thermal conditions (Leszczynski et al. [R845]). Another study performed at extremely low levels of exposure reported decreases in hsp 70 (di Carlo et al. [R850]). In contrast, multiple studies exposing different mouse and human cell lines at very high SAR levels under thermally controlled conditions have reported no induction in hsp gene or heat shock factor (HSF-1) expression levels (Parker et al. [R109]) (Cleary et al. [R629]). When increased hsp gene expression has been observed, it is often at much higher levels of exposure that produce thermal conditions (Fritze et al. [R676]) (Tian et al. [R938]).

Exposure of rat brain synaptosomes at high SAR levels was reported to result in an increase in phosphorylation (Gandhi and Ross [R48]). A related study performed in live rats using similar exposure conditions reported no effect on synapsin I levels or synaptosomal phosphorylation until animals experienced hyperthermic conditions (Browning and Haycock [R17]). A series of studies reported effects on immune parameters and protein synthesis, but only at thermal levels (Wiktor-Jedrzejczak et al. [R464], [R553], [R554], [R554], [R555]). One study reported sporadically distributed increases and decreases in ADP ribosylation among various tissues of rats in a manner that was not linked to any obvious dose response curve following exposure to very low RF exposure levels (Singh et al. [R280]); this study has not been independently replicated. In studies using millimeter waves, exposure of flies at low levels was reported to result in a change in chromosome puffing and down regulation of a secretory protein (Kremer et al. [R367]). In other *in vitro* studies of respiratory enzymes and phage growth, no effects were observed until thermal levels were reached (Melnick et al. [R401]) (Lukashevsky et al. [R785]). One group exposed 23 day old rats to 147 MHz (CW and modulated) and its two sub harmonics and reported changes in Na^{+}-K^{+}-ATPase activity in brain

tissue (Behari et al. [R686]). The SAR was reported to be 6–9 W/kg. There was no report of measurement of core temperature or of adequate ventilation in the TEM cell exposure system; these effects, therefore, were most likely thermally induced.

B.7.2.13 Oxidative stress

Although oxidative stress has been proposed as a potential mechanism for RF effects (Lai and Singh [R275], [R596], [R617]), this has not been adequately demonstrated in experimental studies. One series of studies did report that exposure of rat olfactory tissue decreased camphor binding in a manner that seemed to be related to oxidative stress (due to its being blocked by DTT), but the effect did not show any obvious linear correlation with SAR (Philippova et al. [R112]), [R250]). An *in vitro* study using millimeter wave exposures at extremely high levels, which resulted in significant temperature elevations, reported no effect on peroxidation of liposomes (Logani and Ziskin [R593]). A study using lower levels of RF energy co-exposure reported protection against temperature-induced oxidative hemolysis in human red blood cells (RBCs) (Kiel and Erwin [R492]). A series of studies using extremely high exposure levels of RF energy demonstrated that significant temperature elevations could effect membrane fluidity, permeability, and protein shedding in a manner that may be related to oxidative stress (Liburdy and Vanek [R83], [R385]) (Liburdy et al. [R84]) (Liburdy and Penn [R382]) (Liburdy and Magin [R384]). A study performed in sheep RBCs reported that RF energy at high SAR levels had no effect on NADH oxidase or glucose oxidase activity under temperature controlled conditions, and further that hyperthermia-induced auto-oxidation could be partially reversed by co-exposure with RF energy (Kiel and Erwin [R64]), (Kiel et al. [R65]). The effect of 2450 MHz at an SAR of 103 W/kg was studied on glucose oxidase conversion of partially purified human oxyhemoglobin to methemoglobin. As controls, base (pH 10), heat (50 ºC), and hydrogen peroxide (5.6 mM) were all effective in promoting the oxidation conversion. RF exposure inhibited thermally induced autooxidation by 28.6%, but did not affect oxidation by hydrogen peroxide (Kiel et al. [R65]).

B.7.2.14 Elevated temperature and carcinogenesis

Boukamp et al. [R1136] showed that long-term exposure of already immortalized and p53 mutated human HaCaT keratinocyte cells to a significantly elevated temperature (40 ºC or 104 ºF) for up to 11 passages resulted in no significant tumor formation when injected into nude mice, although after 13 or more passages at the elevated temperature the cells did finally accumulate enough genetic damage to form tumors upon injection into mice. The results of this study indicate that for heat to act as a cofactor in the carcinogenic process (using a model of already immortalized and p53 mutant human skin cells), the temperature of the cells must be maintained at 40 ºC for at least 13 passages (the equivalent of ~13 weeks and hundreds of replication cycles). While the range of skin temperature in humans can fluctuate below and above the normal range of 32–34 ºC, it is unrealistic to imagine an area of skin on a living human being maintained at 40 ºC continuously for more than 11 weeks. Rather than interpreting these data as suggesting that localized heating of skin regions of the body by RF exposure could have possible tumorigenic consequences, a more reasonable interpretation of these data would be the following. Skin cells exposed to RF energy, or any other heat source sufficient to maintain temperature levels at 40 ºC for up to 11 passages, conditions loosely equivalent to a human being maintaining a localized skin temperature of 104 ºF for 11 weeks, were not tumorigenic. This latter interpretation addresses the safety of hyperthermic exposure even under unrealistic exposure conditions for a human being. In addition, there is no independent confirmation of the results and no evidence that their results can be extrapolated to living animals including human beings.

Other studies on the carcinogenic effects of hyperthermia are discussed in a recent review by Dewhirst et al. [R1079] who concluded that “The bulk of the data presented indicate that hyperthermia alone is not carcinogenic.”

B.7.2.15 Summary of cancer related studies

Overall, there is no consistent evidence from various animal and *in vitro* studies for a reproducible biological effect of low level (non-thermal) RF exposure. The majority of studies report no effect on a wide variety

of biological endpoints. The magnitude of the reported effects is generally very small, often in the range of biological/physiological variability with no known health implications. In contrast, there are a large number of studies described in this section that support the basis for this standard.

B.7.3 Cancer related epidemiology studies

Epidemiology is "*the study of the distribution and determinants of disease in human populations*" (quoted from MacMahon and Pugh [B84]). Such studies provide the most relevant information for determining possible associations between exposure to a chemical or physical agent and adverse human health effects. A detailed description and review of the principles of epidemiological study and the use of the Bradford-Hill criteria (Hill [R1045]) for the assessment of cause and effect in epidemiology, as well as a detailed review of relevant studies, is included in the review paper by Elwood [R1097] and other relevant detailed reviews by Moulder et al. [R667], and Berqvist et al. [R1015].

B.7.3.1 Review of epidemiology studies

Epidemiological studies of RF exposure and cancer fall into the following five groups:

1) Studies of disease clusters;
2) Studies of general populations exposed to RF sources [TV, radio, communication transmissions];
3) Studies of occupational groups;
4) Case control studies;
5) Studies of mobile phone users.

Cluster studies, such as the one performed in Sutton Coldfield in the U.K. in response to a cluster of leukemia and lymphoma in adults living close to an RF broadcasting transmitter (Dolk et al. [R624]), are inherently difficult to interpret because of the impossibility of assessing all of the effects that chance variation might have contributed to the cluster. In the initial Sutton Coldfield study, the authors correctly concluded that no causal association could be drawn between the presence of the cluster and RF exposure from broadcasting towers (Dolk et al. [R625]) (Cooper et al. [R760]). Inconsistent effects have been reported between residential proximity to other RF broadcast towers and adverse health endpoints (Bielski [R267]) (Maskarinec et al. [R579]) (Selvin and Merrill [R823]) (Michelozzi et al. [R858]) (Altpeter et al. [R977]) (Hallberg and Johansson [R995], [R996]) (Boscolo [R1012]), although many of these studies have significant flaws in their study design (making them difficult to interpret). An increased incidence and mortality rate of childhood leukemia was reported in Australia with residential proximity to a specific RF broadcasting tower (Hocking et al. [R633]), although subsequent reanalysis of the data showed the results may have been influenced by other confounding variables within the study location (McKenzie et al. [R669]).

While scattered reports of adverse health effects associated with occupational exposure to RF do exist (Demers et al. [R36]) (Kurt and Milham [R68]) (Pearce [R110]) (Speers et al. [R125]) (Thomas et al. [R128]) (Pearce et al. [R199], [R211]) (Hayes et al. [R207]) (Cantor et al. [R268]) (Davis and Mostofi [R563]) (Tynes et al. [R570], [R605]) (Grayson [R592]) (Richter et al. [R747]) (Holly et al. [R838]) these studies are largely inconsistent with each other in terms of the adverse health endpoints affected, and often show no clear dose response with RF exposure. Many have serious flaws in their study design, contain limited or insufficient RF exposure assessment, and are generally inconsistent with the absence of findings of an association from other occupational studies (Tornqvist et al. [R131]) (Coleman [R142]) (Lilienfeld et al. [R146]) (Robinette and Silverman [R147], [R148]) (Siekierzynski et al. [R151], [R152]) (Wright et al. [R213]) (Coleman et al. [R214]) (Muhm [R506]) (Czerski et al. [R542]) (Hill [R568]) (Lagorio et al. [R616]) (Kaplan et al. [R647]) (Morgan et al. [R701]) (Gallagher et al. [R822]) (Groves et al. [R853]) (Wiklund [R1013]) (Armstrong et al. [R1014]). While micronuclei formation in workers occupationally exposed from broadcast antennas has been reported (Garaj-Vrhovac [R757]) (Lalic et al. [R791]), these findings

were not verified in a larger study of more than 40 Australian linemen exposed under similar conditions (Garson et al. [R186]). No clear association could be established between occupational exposures of parents to a number of agents, including RF, and effects (neuroblastoma) in their offspring (Spitz and Johnson [R289]) (De Roos et al. [R798]). One study reported a slight excess in brain tumors associated with combined exposure to RF and other exposures associated with electrical or electronic jobs, but not with RF alone (Thomas et al. [R128]). A study of a Polish military cohort reported a substantial excess of total cancer and several cancer sub-types with jobs associated with RF exposure (Szmigielski [R578]), (Szmigielski and Kubacki [R982]), although questions have been raised about severe bias in the exposure assessment of this study (Elwood [R665]) (Bergqvist [R1015]) (Stewart [R1133]). Studies by Milham of U.S. amateur radio operators reported an excess in one of nine types of leukemia assessed (see [R101], [R102], [R209], [R215], and [R569]), but not for total tumors, total leukemia, or brain tumors, and potential confounding factors might have included exposure to soldering fumes, degreasing agents and over-representation of a particular social class.

Because of the current popularity of mobile phone technology, mobile phone-use studies represent a majority of recent reports dealing with RF exposure. Many of these have elements of strong study design, although consistent shortcomings include a) the difficulty of obtaining and/or reconstructing accurate and detailed individual exposures associated with mobile phone use over many years, and b) the relatively short period of time the technology has been in widespread use vs. the relatively long latency periods associated with many disease endpoints (e.g., various forms of cancer). Large cohort studies of tumor incidence (Johansen et al. [R767]) and mortality (Dreyer et al. [R691]) have shown no association with mobile phone use. A report by Stang that drew upon data gathered on multiple disease endpoints from a larger cohort reported an association between mobile phone use and melanoma of the eye [R749]. A similar analysis drawn from a large set of cohort data by Johansen [R808] reported no such association. Both analyses of ocular melanoma were based upon small numbers of patients classified into exposure categories, making the collective findings somewhat inconclusive. Of four case control studies of brain tumor incidence and cell phone use, two have been negative (Muscat et al. [R751], [R937]) (Inskip et al. [R762]). A series of studies by Hardell et al. first reported no association between mobile phone use and brain tumors (see [R679], [R716], and [R729]). A larger study population was then employed and an association was found between mobile phone use and benign acoustic neuroma, especially on the same side of the head (ipsilateral) as the mobile phone use (Hardell et al. [R854], [R855], [R933]). After reanalysis of the same data set, malignant astrocytoma was then found to correlate with analogue as well as with GSM mobile phone and cordless phone use (Hardell et al. [R1007]). In the latest analysis of the same study group and data set, an association was reported between analogue mobile phone use and vestibular schwannoma (VS) (Hardell et al. [R1064]). Hardell correlated these findings on VS with the subjective studies by Oftedal [R755] and Sandstrom [R777] that reported increased complaints of tinnitus (a precondition of VS) in Norway. Hardell also provided 3 additional cases of mobile phone users complaining of tinnitus that “contacted” him independently, although none of these individuals had any detectable tumor. In addition to brain tumors, an earlier study by Hardell et al. reported a case study of angiosarcoma of the scalp associated with the use of cordless telephones [R716]. Recent studies by Auvinen [R830] and Kahn [R1112] did not confirm the findings of Hardell et al. with respect to non-malignant acoustic neuroma or tumor laterality. Auvinen et al. [R830] did report a slight association between malignant gliomas (but not other brain tumors or salivary gland tumors) and analogue cell phone use, with a weak increasing trend with duration of subscription. These authors cautioned, however, that the results were preliminary. A mixed meta-analysis of all four case control studies shows no association between brain tumors and either total mobile phone use [combined odds ratio (OR) 1.02, 95% confidence interval (CI) 0.85–1.23] or maximum mobile phone use [combined OR 1.08, 95% CI 0.75–1.57] (see Table 5.6 of Elwood [R1097]). Another preliminary study reported chromosomal aberrations in a small number of mobile phone users (Gadhai et al. [R1115]).

B.7.3.2 Summary of epidemiology studies

The epidemiological evidence to date does not show clear or consistent evidence to indicate a causal role of RF exposures in connection with human cancer or other disease endpoints. Many of the relevant studies, however, are weak in terms of their design, their lack of detailed exposure assessment, and have potential

biases in the data. While the available results do not indicate a strong causal association, they cannot establish the absence of a hazard. They do indicate that for commonly encountered RF exposures, any health effects, if they exist, must be small. Even though epidemiological evidence cannot rule out a causal relationship, the overall weight-of-evidence is consistent with the results of the long term animal studies showing no evidence of physiological, pathological or disease-specific effects.

Annex C

(informative)

Rationale

NOTE—References denoted in brackets with the letter "R" before the number (e.g., [R119]) are references from the IEEE/WHO Literature Database and are found in Annex F. References denoted with the letter "B" before the number (e.g., Reilly [B112]) are references that are not in the IEEE/WHO database and are found in the Bibliography (see Annex G).

C.1 Introduction

A careful literature evaluation process by the ICES working groups and the literature review presented in Annex B have not changed the scientific basis for the adverse effect level between 100 kHz and 3 GHz. The threshold for whole-body average (WBA) SAR of 4 W/kg for established adverse effects remains the same as in the IEEE Std C95.1, 1999 Edition [B70]. Adoption was based on the decision that the threshold for disruption of ongoing behavior in laboratory animals including nonhuman primates may be a potentially adverse effect in human beings. The peak spatial average SAR values have been changed from 1.6 W/kg and 8 W/kg for exposure of the public and exposures in controlled environments to 2 W/kg and 10 W/kg, respectively. This change was based on the scientific considerations explained in C.2.2.2 and was also influenced by the desire to harmonize the basic restrictions with ICNIRP where scientifically justified.

The limits in this standard protect against established adverse health effects in human beings. For whole body exposure, the basis for this standard is derived from the science reviewed in Annex B (see especially B.2.1) and is consistent with the ICNIRP guidelines. For localized exposure, this standard uses recent scientific information [B138] to protect against adverse effects in the tissues most sensitive to thermal effects. Recent modeling studies show that at 10 W/kg per 10 g it may be possible to exceed a 1 °C rise in tissue, which had been suggested earlier as the upper temperature increase that has no detrimental health effects (UNEP/WHO/IRPA [B129]) (ICNIRP [B62]) (WHO EHC 137 [B137]). More recent WHO information indicates that a 1 °C rise in temperature, even in the most sensitive tissues and organs, is not adverse (WHO [B138]).

The upper boundary of the frequency range over which WBA SAR is deemed to be the basic restriction has been reduced from 6 GHz to 3 GHz. The rationale for this change is based on RF penetration depth calculations explained in C.2.2.1. The tissue averaging mass for the peak spatial-average SAR has been changed from 1g to 10 g. This change, which is explained in detail in C.2.2.1, C.2.2.2.1 and C.7.5, is based on the biologically based rationale of ICNIRP related to exposure of the eyes and extensive theoretical biophysical research quantifying RF energy penetration in biological tissue. The results of this research show that RF energy is incapable of causing significant local temperature increases in small tissue volumes within the body.

The rationale to set exposure limits for stimulatory effects at lower frequencies and temperature-related effects at higher frequencies has been explained thoroughly in this standard compared with the previous version. Improved numerical and measurement methods in RF dosimetry have increased knowledge about the SAR-temperature relationship following RF energy deposition in human tissue, which is essential when assessing potential biological and health effects of RF exposures. In addition, in order to explain the rationale for adverse effect levels in the frequency range of 3 kHz to 300 GHz (see C.3), a number of special considerations have been reviewed and explained in detail in C.7 (for example, to cover extreme exposure situations of specific human subpopulations).

In summary, this standard incorporates a reasonably large margin of safety and an RF safety program is required to provide part of the margin of safety for those exposed above the relevant action level (lower tier). This standard may also be considered especially conservative, since the safety factors are applied against perception phenomena (electrostimulation and behavioral disruption), which are far less serious effects than any permanent pathology or even reversible tissue damage that could occur at much higher exposure levels than those for perception phenomena.

This revision of IEEE Std C95.1 maintains many of the characteristics of the previous standard but also contains a number of differences from earlier editions that address new dosimetry findings and that simplify the use and application of the standard. Some of these similarities and differences are described below.

C.1.1 Similarities and differences between this standard and IEEE Std C95.1, 1999 Edition

C.1.1.1 Similarities

a) All relevant reported biological effects at either low ("non-thermal") or high ("thermal") levels were evaluated. Research on the effects of chronic exposure and speculations on the biological significance of low-level interactions have not changed the scientific basis of the adverse effect level.

b) WBA and peak spatial-average SAR remain the basic restrictions of exposure over much of the RF spectrum. The WBA SAR values remain the same as in IEEE Std C95.1, 1999 Edition, i.e., 0.4 and 0.08 W/kg.

c) The MPE for exposures in controlled environments remain the same as in IEEE Std C95.1, 1999 Edition.

d) The averaging time remains six minutes for frequencies below 3 GHz for effects associated with tissue heating. For electrostimulation effects, the averaging time is 0.2 s for an rms measurement. Peak electrostimulation limits apply to instantaneous values within the applicable bandwidth.

C.1.1.2 Differences:

a) IEEE Std C95.1, 1999 Edition contains two tiers; an upper tier for "exposures in controlled environments" and a lower tier for "exposures in uncontrolled environments." In this standard, two tiers have also been set. As in the 1999 Edition of this standard, an upper tier has been set for exposure of persons in controlled environments. While the weight of scientific evidence supports the conclusion that no measurable risk is associated with RF exposures less than the upper tier of this standard, it is impossible to scientifically prove absolute safety (the null hypothesis). Thus a lower tier has been set with an extra margin of safety that applies to all other individuals. The lower tier, called an "action level," recognizes public concerns, takes into account uncertainties in laboratory data and in exposure assessment, and supports the process of harmonization with other standards, e.g., the NCRP recommendations [B95] and the ICNIRP [B62] guidelines. For practical purposes, the lower tier may be used for the general public or as an action level, above which an RF safety program shall be implemented to protect against exposures that exceed the upper tier. (See Clause 3 for definitions of "lower tier," "upper tier," and "action level.")

b) The upper frequency boundary over which WBA SAR is deemed to be the basic restriction has been reduced from 6 GHz to 3 GHz.

c) The MPEs for the lower tier are different from those in IEEE Std C95.1, 1999 Edition and are in general more restrictive between 300 MHz and 300 GHz.

d) The peak spatial-average SAR values have been changed from 1.6 W/kg and 8 W/kg for lower and upper tiers to 2 W/kg and 10 W/kg, respectively (see C.2.2.2).

e) The averaging mass for determining the peak spatial-average SAR has been changed from 1 g of tissue in the shape of a cube to 10 g of tissue in the shape of a cube (see C.2.2.1, C.2.2.2.1 and C.7.5).

f) Although implicit in previous versions of IEEE Std C95.1, the present standard explicitly relies on "basic restrictions" (see Clause 3 for definition).

g) The standard now requires the development and implementation of an RF safety program in controlled environments.

h) The averaging time for both the upper and lower tiers has been changed for frequencies above 3 GHz.

i) The upper frequency at which maximum induced and contact currents are specified is now 110 MHz compared with 100 MHz in the previous standard.

j) The frequency at which the upward ramp begins for the relaxation of the power density limits for localized exposure (see 4.6) has been changed from 6 GHz to 3 GHz.

k) In recognition of the differing impact of exposure to particular frequencies, the standard provides sections devoted to three frequency bands: 3 kHz to 5 MHz, 100 kHz to 3 GHz and 3 GHz to 300 GHz. The limits in the first band minimize adverse effects associated with electrostimulation. This overlaps the second band where the limits also protect against effects associated with heating. The limits in the third band protect against effects associated with heating. Differences within each of those bands are provided below.

   1) **3 kHz to 5 MHz:** The standard defines basic restrictions (BR) in terms of the *in situ* (within biological tissue) electric fields for different regions of the body. Magnetic field MPEs are specified for the arms and legs and for the head and torso, but compliance with the standard can be demonstrated for uniform sinusoidal magnetic fields by showing that either the *in situ* electric field BR or the magnetic field MPE is satisfied. If the magnetic field is not constant over the head and torso, it is sufficient to demonstrate that the basic restrictions are satisfied, or that the spatial peak of the magnetic field MPE is not exceeded. Based on current knowledge of adverse effects on humans within this frequency range, the whole body electric field MPE for the controlled environment has been increased. Similarly, the magnetic field MPEs, with separate requirements for body portions as noted above, have been increased for both the general public and controlled environments and have been made frequency dependent. Averaging time for an RMS measurement is 0.2 second. Formulas have been included for determining maximum permitted peak electric fields for both *in situ* and environmental considerations.

   2) **100 kHz to 3 GHz:** In this frequency range where SAR is the controlling criterion, the revised standard confirms the presumed threshold WBA SAR of 4 W/kg for potentially adverse effects. Localized exposure restriction criteria (peak spatial-average SAR) have been changed for both the upper and lower tiers. Peak spatial average SAR for any body tissue including the hands, wrists, forearms, feet, ankles, lower legs and pinnae, is required to be determined over 10 g of tissue in the shape of a cube. Peak spatial average SAR for the 10-g sample is to be no greater than 10 W/kg for the upper tier and 2 W/kg for the lower tier except for the hands, wrists, forearms, feet, ankles, lower legs and pinnae, where the permitted peak spatial average remains as specified previously in 10 g of tissue, i.e., 20 W/kg for the upper tier and 4 W/kg for the lower tier. The contact current limits for the frequency range of 100 kHz to 110 MHz have been subdivided into touch and grasping conditions, with the grasping condition confined to the controlled environment. The permissible touch contact current has been reduced for both the controlled environment and the general public. The lower part of this frequency range, i.e., 100 kHz to 5 MHz, is a transition region where the limits protecting against electrostimulation *and* the limits protecting against effects associated with heating must be met.

   3) **3 GHz to 300 GHz:** In this frequency range, the interactions become quasi-optical, and the MPEs are expressed in terms of incident power density and exposure duration. The principal change in the standard has been in the MPE frequency dependence above 300 MHz for the lower tier (general public). At 300 GHz, the MPE is equal to the MPE in the laser standards, which begin at 300 GHz (ANSI [B7]) (IEC [B65]).

C.1.2 Risk profile for adverse effects

For some time, the Risk Assessment Working Group (RAWG) has been particularly concerned about the lack of rigor in defining the safety factors used to derive the MPEs in the standard. Selected RF hazard levels based on work stoppage in animals bear little resemblance to recorded RF accidents in both public and occupational environments. In decreasing order of definitive harm to humans, consideration was given to RF accidents including shocks and burns, localized RF heating, surface heating, and whole-body heating. The implications of microwave hearing as a hazard were also given considerable attention. Literature dealing with indices of cell toxicity, mutagenesis, transformation, tumor initiation and promotion, and teratogenic effects after low level (non-thermal) exposures received extensive attention, discussion, and evaluation.

The risk profile shown below is presented to help provide a framework for interpreting the relevance and applicability of this standard. For example, one might argue that the emphasis on WBA SAR in this standard seems misplaced when considering the predominant risks associated with the RF exposures listed below. However, it is important to recall the historical context. The convenience and utility of dosimetric methods for assessing WBA SAR in animals and human models was extremely important for understanding results underpinning the research on behavioral effects, upon which exposure limits of this standard were originally derived. From the practical perspective of managing RF safety issues within industrial environments, the various considerations relevant to RF safety may be ranked as follows:

1) **RF shocks and burns:** These probably constitute the most harmful RF exposure hazard. A substantial proportion of shock and burn accidents are caused by contact with live, high-powered RF conductors. Shocks and burns from passively energized conductors (reradiating structures) are generally only seen in high power RF environments at MF, HF and VHF frequencies. Examples include radio broadcast sites, and locations where long conductors, such as the hoisting cable of a tall crane, are in the vicinity (e.g., within 1000 m) of AM radio broadcast antennas.

2) **Localized RF heating effects:** These are undeniably realistic hazards, but they occur much less commonly than RF shocks and burns.[12]

3) **Surface heating effects:** These are potentially hazardous, though hardly ever experienced in practice. Possibilities for significant exposures could include open waveguides for high powered GHz sources and the potential use of microwave-based non-lethal weapons for crowd control. The much lower exposure thresholds and exposure durations for sensory effects provide a very effective guidance for protecting against physical harm.

4) **Whole body heating effects:** Although RF absorption sufficient to cause whole-body heating is the most discussed interaction between RF fields and humans in this standard, it likely presents an even lower potential risk of adverse effects than any of the items mentioned above. In practice, significant whole-body heating very rarely occurs. Discomfort due to absorbed RF energy requires sustained application of high, (e.g., kW) RF power that is, generally, not associated with most exposure situations. Deliberate exposure of subject volunteers in the laboratory setting, with institutional approval required, may be an exception. From a risk perspective, recommendations against very mild RF whole-body heating effects ranks lower in terms of scientifically based priorities when compared with far more substantial thermal loads that are routinely imposed by the environment (e.g., air temperature, humidity, infrared radiation, air flow, insulation, etc.). When whole-body heating does occur, it is usually associated with workers climbing on energized broadcast antenna towers, working close to high power active broadcast antennas, or working close to unshielded RF “heaters” and “sealers.”

5) **Microwave hearing effects:** These effects, while possible over a range of frequencies, are even rarer than items 1 to 4 above. The perception of a barely audible click, buzz or hiss, from

[12]Generally, these effects are seen only in association with high power industrial uses of RF or with medical applications (to which this standard does not apply).

pulsed radar type signals in a very quiet environment, based on real-world exposures, is not adverse to health.

6) **Low-level effects:** Despite more than 50 years of RF research, low-level biological effects have not been established. No theoretical mechanism has been established that supports the existence of any effect characterized by trivial heating other than microwave hearing. Moreover, the relevance of reported low-level effects to health remains speculative and such effects are not useful for standard setting.

C.2 Basic restrictions (BR) and maximum permissible exposure (MPE)

C.2.1 Basic restrictions: 3 kHz–5 MHz

The term *basic restriction* (BR) refers to those restrictions that are based on established adverse health effects. The BRs and MPEs at frequencies between 3 kHz and 5 MHz are established to limit adverse reactions (painful or aversive) due to excitation of nerve and muscle, i.e., "electrostimulation." The rationale for these BRs and MPEs, including adverse reaction thresholds, probability and safety factors, and induction models, are documented in IEEE Std C95.6-2002. An upper frequency limit on electrostimulation occurs with continuous sinusoidal waveforms at 100 kHz, below which electrostimulation thresholds are lower than thermal perception thresholds, and above which the opposite is true, i.e., heating effects will exhibit a lower threshold than electrostimulation effects (Chatterjee et al. [R22]), (Dalziel and Mansfield [B31]). However, for pulsed waveforms of low duty factor, electrostimulation thresholds may remain below thermal thresholds to significantly higher frequencies (Reilly [R929], [B113]). Electrostimulation thresholds have been experimentally demonstrated up to frequencies of several MHz. Consequently, methods for determining compliance in this standard for pulsed or non-sinusoidal waveforms are specified to include frequencies up to 5 MHz.

Basic restrictions of Table 1 refer to the electric field induced within the biological medium. Table 1 defines BRs in the frequency range of 3 kHz to 5 MHz. These restrictions have been developed to minimize adverse electrostimulation with an adequate safety factor, as described in IEEE C95.6-2002.

For purposes of this standard, adverse effect levels are those that result in an adverse reaction (see Clause 3). In this frequency range, this standard was developed with respect to established mechanisms of biological effects in humans from electric and magnetic field exposures as described in IEEE C95.6-2002. It does not apply to exposures encountered during medical procedures. The defined exposure limits do not necessarily protect against interference of medical devices or problems involving metallic implants (see C.7.2).

C.2.2 Basic restrictions: 100 kHz–3 GHz

C.2.2.1 Basic restrictions for whole-body exposure

Basic restrictions for frequencies between 100 kHz and 3 GHz are expressed in terms of the SAR (see Table 6). Such restrictions are derived with consideration of adverse effect thresholds associated with body tissue heating, their possible distribution among the population, and safety factors. At frequencies between 100 kHz and 5 MHz, the basic restrictions of both Table 1 and Table 6 must be applied.

The weight of the scientific evidence continues to support the determination made in IEEE Std C95.1, 1999 Edition [B70] that 4 W/kg is the threshold for potentially adverse health effects for short-term exposures of animals. Consistent with the philosophy of the prior standard, a safety factor of ten (10) has been applied to this threshold yielding an SAR of 0.4 W/kg averaged over the whole body, which is reaffirmed protective under almost all environmental conditions. Significantly, this level is also consistent with the weight of the scientific evidence showing no adverse effects in laboratory animals following long term exposure up to 2 years (lifetime

exposure). The weight of the scientific evidence is based on the literature described in Annex B and summarized in B.2. This basic restriction is considered protective for all human exposure and the derivation of the resulting limits is described in detail in B.5.

Within the committee that drafted this standard, a strong scientific argument, based on the biological effects database for potentially adverse effects was made for a single tier standard at 0.4 kg WBA SAR. The upper tier is considered protective for all with an acceptable margin of safety. Nevertheless, similar to IEEE Std C95.1, 1999 Edition [B70] a lower tier, with an additional margin of safety is included. The upper tier in this standard applies to persons in controlled environments; the lower tier, with an extra margin of safety, applies to all other individuals.

Since publication of ANSI C95.1-1982 [B69], significant advances have been made in our knowledge of the biological effects of RF exposure. This increased level of knowledge strengthens the basis for and confidence in the statement that the MPEs provided in this standard are protective against established adverse health effects with an adequate margin of safety. Nonetheless, because of the inherent limitations of the biological effects data base, these MPEs are presented as upper limits of exposure. While the weight of scientific evidence supports the conclusion that no measurable risk is associated with RF exposures less than the upper tier of this standard, it is impossible to scientifically prove absolute safety (the null hypothesis). The lower tier thus recognizes public concerns, serves as an action level above which implementation of an RF safety program is required, helps account for uncertainties in laboratory data and exposure assessment, and supports the process of harmonization with other standards, e.g., the NCRP recommendations [B95] and the ICNIRP [B62] guidelines. While exposures slightly in excess of the MPEs are not necessarily harmful, such exposures are not desirable and should be avoided. Wherever RF exposures can exceed the Action Levels of this standard, steps should be taken to ensure that the MPEs will not be exceeded.

Arguments supporting the lower tier are:

a) It is traditional to afford the general public a greater margin of safety. The general public includes, but is not limited to, children, pregnant women, the aged and infirm, individuals with impaired thermoregulatory systems, individuals equipped with electronic medical devices, and persons using medications that may result in poor thermoregulatory system performance.

b) This approach is consistent with the previous IEEE Std C95.1 standard and most other health and safety standards for RF exposure.

c) It is traditional to warn individuals of exposures to potentially harmful agents, and to implement safety measures to mitigate the hazards. Therefore the lower tier can be a useful criterion, or “action level,” for determining when RF “awareness” communication is required and above which other elements of an RF safety program shall be implemented. RF “awareness” is particularly important for protecting against accidental excessive exposures.

d) Exposure standards such as IEEE Std C95.1 traditionally have been used as the basis for *environmental limits* (limits for the general environment whether people are there or not) through a lower tier that incorporates a larger margin of safety.

A significant change in the present standard is the reduction of the frequency range over which WBA SAR is deemed to be the basic restriction. In the previous standard, WBA SAR was specified as a basic restriction up to a frequency of 6 GHz; in this standard, the upper frequency is 3 GHz. This frequency reduction is based on the following observations:

a) The depth of penetration of RF fields becomes progressively smaller as the frequency is increased with a consequent increased deposition of energy closer to the skin surface.

b) The thermal load imposed on the body by a fixed WBA SAR from RF exposure at higher frequencies becomes less uniformly distributed throughout the body mass and more concentrated near the surface.

c) The bulk of *in vivo* biological effect studies in the frequency range where the WBA SAR value of 4 W/kg is used as the threshold for potentially adverse effects has involved small laboratory animals.

Deep body penetration of the RF energy will almost always occur at the microwave frequencies used in this research.

d) Adjusting downward the highest frequency at which WBA SAR is the most meaningful dosimetric parameter helps to better recognize the spatially different manner in which thermal loads are applied to the body at higher frequencies. While the absorbed RF energy associated with exposures at high microwave frequencies is concentrated near the body's surface, resulting in localized SARs that may be substantial, the shift downward of the maximum frequency at which WBA SAR is applicable helps emphasize a belief that the power density MPEs are extraordinarily conservative (perhaps, even more so than had been previously thought).

The depth of penetration (skin depth) can be defined as the distance at which the field strengths or current densities are e^{-1} (0.368) of their surface value or, for purposes of this standard, where the power density is e^{-2} (0.135) of the surface value. As the frequency falls, the depth of penetration increases. Maximum energy absorption will occur when the body is aligned with the E-field vector, with the longest dimension of the body being ~0.4 of the free space wavelength (a condition known as resonance). The subject of penetration depth with frequency is treated thoroughly in the Radio Frequency Radiation Dosimetry Handbook [R901] for planar models, prolate spheroidal models, and several other, more complex models.

Depth of penetration is a function of the electrical properties of tissues, frequency, and physical shape of the body (e.g., curvature of various body parts can affect focusing of RF fields beneath the outer surface). Figure C.1 illustrates the depth of penetration for a planar slab model of muscle tissue exposed to plane wave RF fields over the frequency range of 10 MHz to 10 GHz. Figure C.1 indicates depths of penetration of about 2.1 cm at 2 GHz, 1.5 cm at 3 GHz, 0.6 cm at 6 GHz, and 0.27 cm at 10 GHz. The mass of the body in which most of the absorbed RF energy will be deposited can be estimated by simply multiplying the frontal surface area of the body (about 0.9 m^2) by the depth of penetration expressed in meters. If this mass is expressed as a percentage of the entire body mass (70 kg), the much smaller fraction of the body absorbing the incident energy can be seen in Figure C.1. This percentage changes from 18.6% of the total body mass at 3 GHz to 7.5% at 6 GHz to 3.7% at 10 GHz. At 3 GHz, more than twice as much of the body mass absorbs most of the energy as compared with 6 GHz and more than 5 times as much of the body mass absorbs energy compared with 10 GHz.

In addition to the above considerations, it is informative to note that the depth of penetration at 3 GHz approximates the dimension of a cube representing the 10 g tissue averaging mass used in this standard (see also C.7.5). A 10 g cube of tissue is approximately 2.15 cm on a side. Hence, the penetration depth at 3 GHz is approximately the size of the cube; at 2 GHz, the depth of penetration is almost exactly the same (2.1 cm) as the size of the 10 g cube. The relevance of this observation is that at much higher frequencies, there will be greater non-uniformity in the deposition of RF energy within the outer tissue averaging mass dimensions. In the 2 to 3 GHz frequency range, there will be more uniform spatial distribution of absorbed energy within the tissue averaging mass near the outer surface of the body.

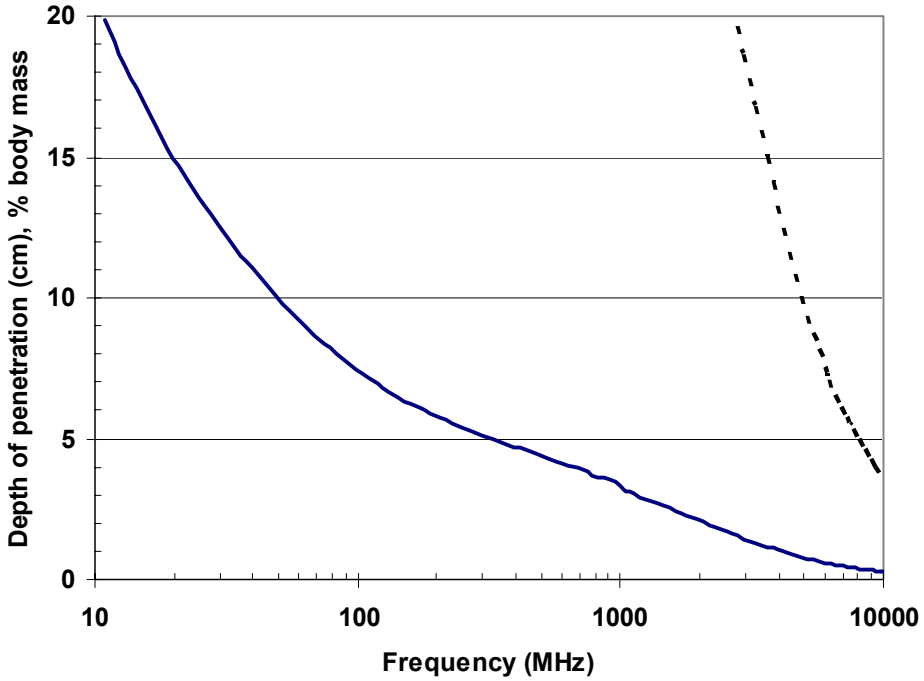

Figure C.1—Calculated depth of penetration (solid line) and percent of body mass (broken line) in which most of the incident RF energy is absorbed as a function of frequency. Calculations are for muscle-equivalent material and are based on a planar slab model.

Based on these observations, the use of whole-body-average SAR becomes less relevant when used above 3 GHz. Above 3 GHz, the relevant dosimetric parameter becomes incident power density.

C.2.2.2 Basic restrictions: localized exposure

These restrictions are established to protect against an excessive temperature rise in any part of the body that might result from localized or non-uniform exposure. The BRs for the upper tier are considered protective for all individuals. To meet the BRs, exposure shall not result in a peak spatial-average SAR that exceeds 10 W/kg as averaged over any 10 g of tissue (defined as a tissue volume in the shape of a cube)[13], except for the extremities and the pinnae where the peak spatial-average SAR shall not exceed 20 W/kg, as averaged over any 10 g of tissue (defined as a tissue volume in the shape of a cube). These levels have been reduced for the lower tier by a factor of 5. The limits for the lower tier include a peak spatial-average SAR of 2 W/kg as averaged over any 10 g of tissue (defined as a tissue volume in the shape of a cube), except for the extremities and the pinnae where the peak spatial-average SAR shall not exceed 4 W/kg, as averaged over any 10 g of tissue (defined as a tissue volume in the shape of a cube).

The rationale for changing the peak spatial-average SAR and averaging volume from that of IEEE C95.1, 1999 Edition [B70] was in part due to the desire of ICES to harmonize the BRs with those in the ICNIRP guidelines where scientifically justified. The revised limits are also based on recent theoretical biophysical research and thermophysiological data showing the inability of RF energy to cause significant local temperature increases in small tissue volumes for inducing adverse health effects (see C.2.2.2.1 and C.7.5). Consequently, the revised limits prevent adverse local temperature rises in various tissues in humans.

[13] The volume of the cube is approximately 10 cm^3.

C.2.2.2.1 Rationale for changing the values and averaging mass of the peak spatial average SAR

This preface to this clause emphasizes once again that this standard protects against all established adverse health effects from RF exposure to the whole body or to localized areas of the human body. Other parts of this standard document the conclusion that all established adverse health effects associated with RF exposures above 100 kHz are due to significant increases in the core body temperature or to temperature increases in localized areas of the body such as the lens of the eye. As explained elsewhere, exposure at the whole body SAR limit (0.4 W/kg) is protective against core body temperature increases of more than a small fraction of 1 ºC because this SAR limit is 10 times less than that needed to increase core temperature by 1 ºC in rats and monkeys (cf. B.5.2). Furthermore, temperature increases in human beings are limited to still smaller amounts because the human thermoregulatory system is more efficient than that of laboratory animals. As explained below, localized exposure at the upper limit (10 W/kg averaged over 10 g of tissue) is protective against all adverse effects including those occurring in the fetus and testes, the two targets identified as most sensitive to thermal damage. The threshold temperature increase for adverse effects in the fetus and testes is about 2 ºC (see B.6.1.1 and B.6.1.2). Potentially adverse effects in the brain apparently require higher temperature increases than those known to cause adverse effects in the testes and developing organism (Sharma and Hoopes [R1082]).

C.2.2.2.1.1 Change from dosimetry-based to biologically-based rationale

As described in more detail in C.7.6, the peak spatial-average SAR limits in ANSI C95.1-1982 and IEEE Std C95.1, 1999 Edition [B70] were based on dosimetry considerations. The 8 W/kg and 1.6 W/kg limits were determined from the 20:1 ratio between the peak spatial-average SAR and WBA SAR in experimental data available in the late 1970's (ANSI [B6]) (IEEE Std C95.1, 1999 Edition [B70]). The 1 g averaging mass was consistent with data limited by the resolution of thermographic measurements at the time. Recent advances in numerical calculations have shown that the ratio of peak spatial-average SAR to WBA SAR for a 1 g averaging mass can be much higher, with reported values of more than 100:1 (Bernardi et al. [R1109]). The committee, however, considered it inappropriate to relax the peak spatial-average SAR limits to 40 W/kg and 8 W/kg for this revision and instead discussed alternatives, one of which was to examine the basis of the ICNIRP peak spatial-average SAR limit. In an ICNIRP statement [B61], a 10 gram averaging mass was recommended, "because of the very inhomogeneous spatial distribution of energy absorbed inside the head, together with concerns about possible localized heating of the eye and other parts of the head with equivalent mass." The committee agrees that the biologically based ICNIRP rationale is more appropriate than the purely dosimetry based rationale in ANSI C95.1-1982 [B69] and IEEE Std C95.1, 1999 Edition [B70]. Furthermore, the limit of 10 W/kg averaged over 10 g is supported by results from animal experiments (Guy et al. [R698]), (Kramar et al. [R1001]), (Elder [R1099]) showing that this limit is 10 times below the SAR threshold for cataracts in humans, which is estimated to be 100 W/kg deposited in the eyeball, a mass of about 10 g. When considering tissue-averaging mass, a 10 g averaging mass is suitable for frequencies less than 3 GHz, the range where this revised standard recommends the use of SAR as the basic restriction (see C.7.5 for details). In addition to these scientific bases for revising the peak spatially averaged SAR in IEEE C95.1, 1999 Edition [B70], this change was also influenced by the desire of ICES to harmonize the basic restrictions with ICNIRP guidelines where scientifically justified. The limits in the ICNIRP guidelines have been considered adequate for health protection by many health authorities and independent review groups (see B.2) and have been adopted by more than 35 countries. Although harmonization itself is not a scientific rationale for setting the limits, the widespread adoption of the ICNIRP guidelines as recommended by the World Health Organization demonstrates scientific consensus on RF safety limits. In summary, the scientific judgment of this committee, as expressed above, is calibrated by and in agreement with the views of other independent expert groups.

C.2.2.2.1.2 Temperature increase in the eye and brain due to localized RF exposure

This subclause addresses concerns about temperature increases in the eye and brain from an exposure of 10 W/kg averaged over 10 g and the potential for such temperature increases to cause adverse health effects.

On 1 May 2003, the National Radiological Protection Board (NRPB) of the United Kingdom released a consultation document titled "Proposals for limiting exposure to electromagnetic fields (0 Hz to 300 GHz) [B103]." In this proposal, the NRPB discussed a possible rationale for lowering the occupational partial-body SAR limit from 10 W/kg averaged over 10 g of tissue to 5 W/kg averaged over 10 g, because the available modeling data indicated that the temperature rise in the eye and brain may exceed 1 °C. Subcommittee 4 formed a task group to analyze the modeling papers cited in the NRBP proposal and the more recent papers on this subject. The results of the analysis of the modeling data on the eye and brain are discussed below.

Eye temperature: In the NRPB document [B102], the data for the human eye were taken from two theoretical papers (Hirata et al. [R946], [R1074]). The 1999 conference proceeding paper reported that 0.36 W/kg could introduce a 0.14 °C rise in the eye. Based on this result, the NRPB concluded that "Studies of heating in the eye suggest that an SAR of 1 W/kg averaged over the eye, may lead to a temperature rise of 0.4 °C in the region of the lens." At 10 W/kg, the temperature rise in the eye would be 4 °C. This study, however, was based on an analysis of an isolated eyeball model without the presence of the head. The authors recognized the simplicity of their first model and made corrections in their subsequent study to include the head. A comparison of the results at 1.9 GHz in these two studies shows that the high temperature increase of 4 °C in the first crude model was reduced to 1.2 °C by improvements in the model (see Table C.1). The table includes results from two more recent papers from Hirata's laboratory showing temperature increases ranging from 0.94 °C at 900 MHz to 2.4 °C at 6 GHz. It is noted that the maximum eye temperature increase in Table C.1 is below the temperature threshold (41 °C) for cataracts. The models in Table C.1 did not take into account thermoregulatory mechanisms, therefore the results probably overestimated temperature rise. Further work on the eye models used in these theoretical studies will make them more useful for standard development. An important goal of future research would be the validation of data from physiologically realistic models with data from live animals. Therefore, the available eye modeling data must be interpreted with care and with consideration of the results from animal studies summarized below.

Table C.1—Maximum increase in eye temperature calculated from thermal models for RF exposures (0.9–6 GHz) of 10 W/kg averaged over 10 g (all values are estimated from data from Hirata's laboratory)

[Reference]: Comment	Frequency (GHz)	ΔT (°C)
(Hirata et al. [R1074]): isolated eyeball	1.9	4
(Hirata et al. [R946]): human eye model thermally isolated from head	1 1.9 6	1.1 1.2 2.2
(Hirata et al. [R999]): human eye model thermally isolated from head	0.9 1.9 6	0.94 1.3 2.4
(Hirata [R1135]): blood flow in retina, choroid and sclera included	0.9 1.5 1.9	1.7 1.7 1.7

a) The statement that the eye cannot effectively dissipate heat due to limited blood vascular systems is frequently mentioned, but Carpenter et al. [R988] took exception to this statement based on the following simple experiment. "If the temperature at the posterior pole of the lens in an anesthetized rabbit is measured prior to and during microwave irradiation, it may be found to rise perhaps 5 °C in the course of a 15-minute exposure. If a lethal dose of anesthetic is then injected intravenously, the heart will stop beating, whereupon the intraocular temperature will rapidly rise another 10 °C, thus

indicating that the vascular system is capable of handling at least two-thirds of the thermal stress which radiation imposes on the eye" (Carpenter et al. [R988], p. 354).

b) In the thermal analysis paper by Emery et al. [R1139], the eye blood flow rate (5% iris, 22% ciliary and 72% choroids, sclera and retina) had to be set at 1.7 cm^3/min at 100 mW/cm^2, 2.7 cm^3/min at 200 mW/cm^2 and 4 cm^3/min at 300 mW/cm^2 in order to match the experimental measurement of temperature rise in anesthetized rabbit eyes. Without the blood flow included, the calculated temperature increases were much higher than the measured values.

c) Kojima et al. [R1125] showed intraocular temperatures in rabbits were significantly higher (2-9 °C, when exposed to 300 mW/cm^2 for up to 60 min) in the group with general anesthesia than in the group without anesthesia, apparently due to impairment in blood flow due to anesthesia. These results imply that the results of RF exposure experiments describing lens opacities in animals under anesthesia must be interpreted with great care.

In summary, based on numerical modeling, an exposure of 10 W/kg averaged over 10 g will produce maximum temperature increases in the human eye well below the temperature threshold (41 °C) for cataracts in rabbits. Furthermore, based on animal studies, an exposure of this magnitude is 10 times below the SAR threshold for cataracts. For these reasons, a peak spatial-average SAR of 10 W/kg averaged over 10 g is adequate for protection from adverse effects on the eye such as cataracts.

Brain temperature: The NRPB proposal quoted the results of five papers (Van Leeuwen et al. [R711]) (Bernardi et al. [R725]) (Wainwright [R984]) (Wang and Fujiwara [R987]) (Gandhi et al. [R1105]), describing thermal models of the brain. Among them, the NRPB study (Wainwright [R984]) gave the highest temperature rise. Therefore, NRPB stated "in order to limit the temperature in all parts of the brain to 38 °C (corresponding to a temperature rise of 1 °C above baseline) the SAR in the head, averaged over 10 g, should not exceed about 6 W/kg."

All five papers plus 4 additional new papers were analyzed (Hirata et al. [R1076]), (Yioultsis et al. [R1083]) (Hirata and Shiozawa [R1084]) (Bernardi et al. [R1106]). Wainwright [R984] of NRPB reported that the highest calculated value of brain temperature increase was 1.6 °C when exposed to 10 W/kg averaged over 10 g tissue; an increase of 1.2 °C was reported by van Leeuwen et al. [R711]. Gandhi et al. [R1105] and Wang and Fujiwara [R987] showed 0.5 to 0.6 °C increase with the same exposure. Bernardi et al. [R725] reported a 1.2 °C increase. NRPB recognized some of the uncertainties indicated by the range of the modeling data relating temperature rise with localized SAR. Because our analysis identified additional uncertainties, we agree with NRPB that more dosimetry research is needed to determine the validity of the modeling data.

Table C.2 summarizes the analysis of 9 papers. The values in the rightmost column were calculated from the model data at either 835/900 or 1500/1800 MHz, whichever gave the greater temperature increase. When the peak spatial-average SAR is 10 W/kg averaged over 10 g of head tissue, four papers show that the brain temperature increase is greater than 1 °C (Van Leeuwen et al. [R711]) (Bernardi et al. [R725]) (Wainwright [R984]) (Yioultsis et al. [R1083]). The highest temperature rise of 1.64 ºC was reported by Wainwright [R984], although the conclusion in his paper states: "This study seems to confirm that such exposure (ICNIRP exposure limit 10 W/kg) is unlikely to cause temperature in the brain to rise by more than 1 ºC above the normal body core temperature." Responding to an inquiry from the SC4 Editorial Committee concerning the inconsistent data, Wainwright in 2004 indicated that artifacts in the original MRI-derived model led to a situation whereby a few elements of muscle tissue were misidentified as brain. The incorrect value of 1.64 °C was revised to 1.22 °C. In their recent paper, Bernardi et al. [R1106] calculated smaller temperature changes for a model that incorporated antenna patterns of modern mobile phones. As shown in Table C.2, the temperature change in the new results were less than half of the earlier values obtained with other antennas (Bernardi et al. [R725]). Two other papers (Van Leeuwen et al. [R711]), (Yioultsis et al. [R1083]) showed that brain temperature rise can be higher than 1 °C. However, the majority of the papers (Wang and Fujiwara [R987]), (Hirata et al. [R1076]), (Hirata and Shiozawa [R1084]), (Gandhi and Kang [R1105]), (Bernardi et al. [R1106]) reported temperature increases usually below 1 °C in the brain. As shown in Wang

and Fujiwara [R987], Hirata et al. [R1076], and Hirata and Shiozawa [R1084], the peak temperature rise in the brain due to 10 W/kg per 10 g exposure ranges from 0.567 to 1.25 °C. However, the head tissue peak SAR outside the brain will be higher than the upper limit. In this context, it is important to note that a human brain temperature greater than 40 °C, that is, a temperature more than 3 °C above a baseline body temperature of 37 °C is required for any histopathologic damage to occur (see summary below).

In March 2004, following a thorough review of current scientific knowledge, including the recently published modeling studies described above, and an extensive consultation exercise, the Board of NRPB concluded there was neither scientific justification nor any practical merit in recommending new restrictions that are close to those of ICNIRP but differ from them (see Pasour [B107]). The Board, therefore, recommended the adoption in the UK of the ICNIRP guidelines for limiting exposure to electromagnetic fields between 0 and 300 GHz, instead of lowering the occupational exposure limit to 5 W/kg as proposed in May 2003 based on the limited modeling data available at that time (see NRPB [B102]).

Table C.2—Correlation of the SAR_{max} (10g) in the whole head with the maximum temperature rise in the brain (SAR head–ΔT brain)

Reference	835/900 MHz		1500/1800 MHz		
	SAR_{max} (W/kg)–10 g	ΔT (ºC)	SAR_{max} (W/kg)–10 g	ΔT (ºC)	ΔT_{max} (ºC) @ 10 W/kg SAR_{max} (10 g)
(Wang and Fujiwara [R987])	0.92	0.053	0.59	0.045	0.763
(Van Leeuwen et al. [R711])	0.91	0.117	–	–	1.286
(Wainwright [R984])	1.43	0.201	2.43	0.398	1.22[a]
(Bernardi et al. [R725])	1.08	0.13	–	–	1.204[b]
(Gandhi et al. [R1105])	2.00	0.103	2.00	0.068	0.515
(Bernardi et al. [R1106])	1.19	0.061	0.87	0.036	0.513
(Yioultsis et al. [R1083])	2.072	0.331	0.591	0.079	1.597
(Hirata et al. [R1076])	1.31	0.154	2.41	0.166	0.836[c] (avg)
(Hirata and Shiozawa [R1084])	1.62	0.132	1.42	0.108	0.721[c] (avg)
[a]Due to an error in tissue classification in [R984], the temperature increase of 1.64 °C did not occur in the brain; the revised brain temperature increase is 1.22 °C (see text above).					
[b]Same authors published a paper one year later showing that the brain temperature increase is less than 1 °C (see [R1106] in table).					
[c]Averaged values provided by Akimasa Hirata.					

In summary, interpretation of the temperature data from modeling studies of the brain and eye must include consideration of the following limitations of the models: 1) the adequacy of physiological blood flow in many of the numerical model studies has not been verified, 2) none of the results for brain and eye have been validated in live animals and humans, and 3) the results from independent laboratories varied over a wide

range. Until these limitations can be resolved, thermal models are useful but in and of themselves are not sufficient for safety standard development. Animal studies have shown that temperature elevations of less than 2 °C produce no adverse effect on the embryo or testes, the two most thermally sensitive organs (Edwards et al, [R1081]); even higher temperatures are required to produce adverse effects in the brain (Sharma and Hoopes [R1082]). Although modeling data do not exist for all cases, localized SAR of 10 W/kg averaged over 10 g of tissue gave calculated temperature elevations ranging from about 0.5–1.6 °C in the brain (see Table C.2), values below those known to cause adverse effects in most sensitive organs. This analysis supports the conclusion that this standard does not allow exposures that would cause a) developmental effects in embryos because the required threshold is a temperature increase of 2–2.5 °C (Edwards et al. [R1082]) or b) sterility due to thermal damage to sperm because the minimum long-term temperature increase required is greater than 2 °C above an initial testicular temperature of 35 °C, the upper end of the range in normal human testicular temperatures (see B.6.1.2). Furthermore, the upper tier limit for localized exposure is protective against cataracts because the threshold temperature for lens opacities is 41 °C (Elder [R1099]) and is protective against potentially adverse effects in the central nervous system as shown by the following information. A number of animal studies that investigated effects of localized hyperthermia on the brain and spinal cord of laboratory animals are summarized in tables in Sminia et al. [B124], and in Sneed and Stea [B125]. A review of these studies indicates that the lowest brain temperature associated with contrast enhancement on computer tomography images (an indicator of BBB breakdown) was 40.3 °C for 30 min (Fike et al. [B43]). This temperature was caused by localized heating in the dog brain by a microwave antenna inserted into the frontal white matter. Other investigators concluded that higher brain temperatures and exposure times (>41 °C for 4 h) are associated with breakdown of the rat blood brain barrier (Sharma and Hoopes [R1082]). A study of human cancer patients given whole body hyperthermia treatment showed that the critical thermal maximum temperature was 41.6-42 °C for 45 min to 8 h (Sharma and Hoopes [R1082]) (see also Bull et al. [B20]). These results support the conclusion that the upper tier is protective against potentially adverse effects in the human central nervous system. Temperatures exceeding 40 °C of the whole human brain are required to cause nausea, disorientation, apathy, delirium and other reversible effects (Sharma and Hoopes [R1082]). No adverse effects were observed in other physiological systems (cardiac, hepatic and renal systems) following whole body hyperthermia treatment (39–39.5 °C for 3 or 6 h and 39.5–40 °C for 6 h, see Kraybill et al. [B80]).

IEEE standards are based on currently available knowledge; if any new adverse effect is established which would require a change in the standard, the standard can be promptly revised by amendments. The committee continues its efforts to monitor RF bioeffects research for the next standard revision, including studies on potentially adverse CNS functional effects at a peak spatial average SAR of 10 W/kg in 10 grams of tissue. Currently, there are dosimetry studies in progress to identify the relationship between temperature rise and peak spatial average SAR.

C.2.2.2.2 Rationale for extending the definition of “extremities” to include those portions of the arms and legs distal from the elbows and knees, respectively

IEEE Std C95.1, 1999 Edition [B70] relaxed the exposure limits for the hands, wrist, feet and ankles. These higher local SARs were permitted because of (1) the relatively high surface-to-volume ratios of these parts of the body, (2) the common experience of relatively large temperature excursions in these parts of the body that normally occur without apparent adverse effects, and (3) the lack of critical physiological/biochemical function when compared with vital organs. Compliance difficulties have arisen in determining the dividing line between the wrist and forearm and ankle and lower leg. This standard solves this problem by extending the relaxed exposure limits to include the forearms and lower legs. The three justifications listed above also apply to these limbs and this change removes the ambiguity of establishing compliance.

C.2.2.2.3 Rationale for applying the peak spatial-average SAR values for the extremities to the pinna

The rationale for applying the same peak spatial-average SAR values to the extremities and the pinna is briefly explained in IEEE Std C95.1b-2004 [B71] and is explained in more detail below. For purposes of regulating exposure to RF energy, the pinna (auricle of the external ear) is subjected to the same SAR limits as the extremities of the human body, i.e., hands, feet, wrists, and ankles and limbs. The projecting part of the ear lying outside of the head captures sound pressure waves and guides them into the external auditory meatus. The pinnae consist of skin, cartilage, fat, nerves, blood vessels, and muscle tissues, a composition similar to that of the extremities. The temperature of the pinnae usually lies between room temperature and body core temperature. Under thermoneutral conditions, the temperature of human skin usually falls within the range 32.0–35.0 °C. However, the pinnae, being a thin appendage, will normally have a somewhat cooler surface temperature (e.g., ~30 °C, see Guyton and Hall [B50]).

During use of a handheld mobile phone, a pinna may be pressed against the head and an increase in its surface temperature may occur, largely because surface heat loss by convective cooling is impeded. In addition, thermal conduction of heat generated within the device may raise pinna temperature, but calculations and limited experimental measurements indicate that absorption of RF energy has a minimal effect on pinna temperature. The temperature effect on human pinna would vary significantly from model to model of mobile phones because of differences in the heat generated by various devices. The contribution of the phone to an increase in pinna temperature is principally due to thermal conduction from the device, not from RF absorption. Joyner et al. [B78] reported that cheek temperature near an active mobile phone might increase by 1.7 to 4.5 °C relative to the opposite cheek. Bernardi et al. [R725] calculated a maximum pinna temperature increase from RF energy absorption of 0.23 °C after 80 minutes and an additional increase of ~1.0 °C after 15 minutes from heat conducted from the phone to the ear.

Temperature increases in the pinna from heat generated in the device and from RF absorption are not harmful even if imposed on an initial pinna temperature that is close to body core temperature. Thermal tolerance of skin and cartilage is well above that of the brain, for which the limiting temperature is 41.8 °C (as used in whole body hyperthermia treatment, see Bull et al. [B20]), (Sharma and Hoopes [R1082]). Also, during lengthy telephone use, convective heat transfer by the blood will stabilize pinna temperature. Even in hot environments or after exercise, an additional increase of 1–2 °C from use of a mobile phone would result in pinna temperatures that are well below the level (~42–45 °C) at which cellular injury or pain will occur.

C.2.3 Basic restrictions: 3 GHz–300 GHz

Basic restrictions (BRs) are established for incident power density of RF fields at frequencies between 3 GHz and 300 GHz. (The MPEs are equivalent to the BRs in this frequency range.) The BRs are derived with consideration of adverse effects thresholds, population groups (i.e., workers and the general public), and safety factors. The BRs described in 4.3 are considered protective for all human exposure. They were established after the thorough review and consideration of the literature described in Annex B and summarized in B.2. The derivation of the resulting values and their rationale are described in this Annex.

For purposes of assessing compliance with the BR and MPE at frequencies between 3 GHz–30 GHz, the power density is spatially averaged over any contiguous area corresponding to 100 λ^2, where λ is the free space wavelength of the RF field in centimeters. For frequencies greater than 30 GHz, the power density is spatially averaged over any contiguous area 100 cm^2, not to exceed a maximum power density of 1000 W/m^2 in any one square centimeter as determined by a calculation or a conventional field measurement.

C.2.4 MPE: 100 kHz–300 GHz

Inspection of Table 8 and Table 9 illustrates another significant change in this standard compared with IEEE Std C95.1, 1999 Edition. Specifically, the MPE for the lower tier has a different frequency dependence than the MPE for the upper tier for frequencies above 300 MHz. This change in the MPE is currently based on one published dosimetry research paper that presented a theoretical prediction that the WBA SAR for small children, resulting from exposure at the MPE for the lower tier of the previous standard, could potentially exceed the 0.08 W/kg BR in the 1–3 GHz frequency range (Dimbylow [R1085]).

In this study, using an improved human model and FDTD methods, the WBA SAR was computed for several different size children as well as an adult from approximately 70 MHz to 3 GHz (Dimbylow [R1085]). Similar data for the adult only using an alternative human model but also using the FDTD modeling method (Mason et al. [B86]) can be used for comparing nominal consistency between the two studies.

Figure C.2 illustrates how these two data sets compare. Two important observations are apparent. The two methods are in good agreement with only a 5.3% difference between the two independently obtained values at 1.4 GHz. Secondly, and importantly, both studies reveal a WBA SAR up to more than two times the Radio Frequency Radiation Dosimetry Handbook (Durney et al. [R901]) value upon which the previous MPEs were derived. When the newly calculated WBA SAR values for small children are examined (Dimbylow [R1085]), it becomes apparent that when exposed at the previous MPE, WBA SAR values, depending on the frequency, could exceed 0.08 W/kg by approximately a factor of two. This observation only holds for the smallest of children but means that the previous lower tier MPE was likely inconsistent with the stated objective of the standard to limit WBA SAR to no more than 0.08 W/kg. Due to other inherent conservatism in the previous standard, however, while the WBA SARs for adults determined in the new dosimetry data are also greater than previously assumed, the WBA SARs still comply with the stated objective of not exceeding the WBA value of 0.4 W/kg in adults.

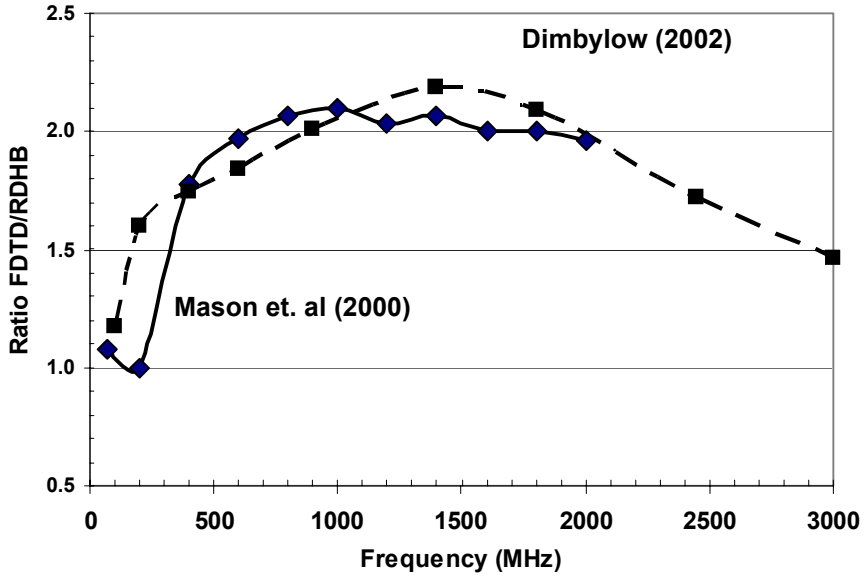

Figure C.2—Comparison of computed adult WBA SAR from two studies (Dimbylow [R1085], Mason et al. [B86]) relative to values from the Radio Frequency Radiation Dosimetry Handbook (RDHB) (Durney et al. [R901]) as a function of frequency

An alternative way of viewing these more recent dosimetry findings is to examine their implications relative to the safety factor inherent to the derivation of the MPE. For example, implicit safety factors of 10 and 50 have been discussed relative to WBA SAR in previous editions of this standard for the upper and lower tiers, respectively. Based on the more accurate WBA SAR values now available, the ratio of the resulting WBA SAR to the presumed threshold for potentially adverse effects can be calculated and the corresponding safety factor plotted as a function of frequency. Figure C.3 illustrates this analysis for the adult as well as 1, 5, and 10 year old children.

For the limits in IEEE Std C95.1, 1999 Edition, Figure C.3 shows that the SAR based safety factor is generally greater than 50 at most frequencies, but in the 1–3 GHz frequency range, may become less than 50; the smallest safety factor is approximately 25 for a 1-year-old infant. At other frequencies, the safety factor may be as great as almost 250 for adults and as much as 100 for 1 year old children. There was considerable debate within ICES Subcommittee 4 as to whether such a finding was of sufficient biological significance to require modifying the MPE to account for the new theoretical results. It was ultimately decided, however, that in the interest of internal consistency, it was better to revise the lower tier MPEs rather than change the stated safety factor from 50 to 25. No change in the MPE for the upper tier (individuals in controlled environments) was deemed necessary on the basis of this analysis of the more recent dosimetry data. As is a theme recurrent in all of the deliberations in preparation of this standard, there is no substantiated scientific or clinical evidence indicating that there is an adverse effect to anyone of exposures at the upper tier limits.

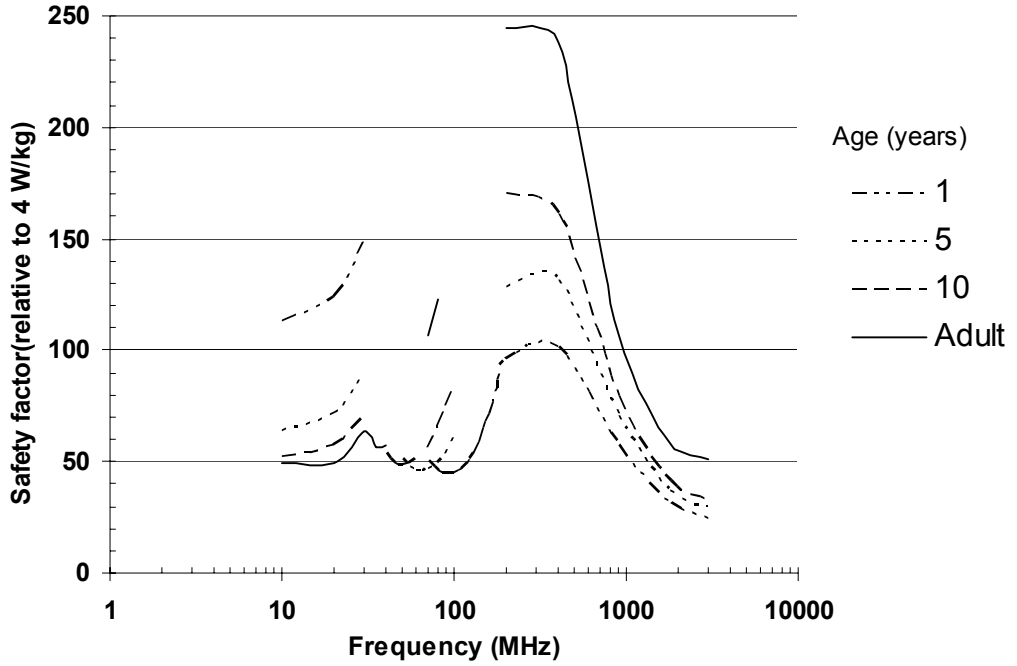

Figure C.3—Calculated ratio (ordinate) of the WBA SAR to 4 W/kg (the threshold for potentially adverse effects) for adults and 1-, 5-, and 10-year-old children, expressed as the SAR based safety factor. The ratio is based on the MPE in IEEE Std C95.1, 1999 Edition [B70] for uncontrolled environments and the FDTD calculations of WBA SAR predicted by Dimbylow [R1085]. The curves have been drawn through the data points contained in the Dimbylow paper. SAR values were not calculated for all frequencies.

C.3 Adverse effect levels

C.3.1 Induced and contact current

The electric field limits at low frequencies in Table 4 are primarily dictated by the following objectives:

a) Limiting induced currents in the ankles during free-field exposure to limit local SAR.

b) Lowering the probability of inducing large body currents when conducting objects are contacted, which could result in localized heating of the hand when grasping or touching an object in fields having a frequency above 100 kHz.

c) Lowering the probability of painful electric shock when conducting objects are contacted, which could result in painful electrostimulation in fields having a frequency below 100 kHz.

The limits on induced RF currents are based on two different considerations. First, currents are limited to a level that protects against RF burns due to excessively high current densities in small areas of tissue while the subject is free standing in high-strength fields. At 100 kHz, for example, the level taken from Chatterjee et al. [R22], Lin [R390], Rogers [R425], and Dalziel and Mansfield [B31] is 100 mA for the upper tier if measured through one foot, and 200 mA if measured through both feet. For the lower tier, the corresponding values at 100 kHz are 45 mA and 90 mA for one or both feet, respectively[14]. For exposures of persons in a controlled environment, e.g., for an occupational exposure, a value of 100 mA is applicable to contact situations, similar to a grasping contact with the hand, and 50 mA for touch contacts. For the lower tier, the touch contact current restriction is 16.7 mA. Contact areas of 15 and 1 cm^2 are assumed for the grasp and touch contacts, respectively. Grip contact is assumed to apply in situations where personnel are trained to make grasping contact and to avoid touch contacts with energized metallic objects that present the possibility of painful contact. Otherwise, a touch contact is to be assumed. The specified current limits will not result in localized SARs in the hands, wrist, forearms, feet, ankles, or lower legs that exceed 20 W/kg, but may be perceived if protective clothing, such as insulated gloves, is not worn.

For frequencies between 3 kHz and 100 kHz, the induced current is limited (Table 5) to reduce the probability of adverse reactions when currents exceed the electrostimulation perception threshold for grasping or touch contact with energized objects (see Chatterjee et al. [B26]). The contact current limits are based on human measurements as noted above. For grip contacts by adults at frequencies above 100 kHz, the median perception current is approximately 250 mA-(rms) based on thermal perception. Based on statistical data for continuous contacts (Reilly [R929]), 100 mA with grip contact current is estimated to be painful to approximately 1% of adult subjects. For frequencies between 3 kHz and 100 kHz, the rms contact current limits for the lower tier are specified as

$I = 1.00f$ mA (f is expressed in kHz)

Thresholds for perception and pain are considerably lower if contact is made with a finger touch, rather than with a grip. For this condition, the limit is specified as

$I = 0.5f$ mA (f is expressed in kHz)

For the general public, the method of contact cannot be assured. Consequently, a touch contact is assumed. The MPE contact current for the lower tier is specified as

$I = 0.167f$ mA (f is expressed in kHz)

[14] The limits for the induced current for the general public are reduced by a factor of 5, which corresponds to a factor of $\sqrt{5}$ (the safety factor) reduction in the SAR in the extremities (SAR ∞ I^2).

Subclause 4.1.2.4 specifies contact current for cases in which the exposure waveform is not sinusoidal. For waveforms with large peak transients, or for pulsed waveforms of low duty factor, the frequency at which electrostimulation effects dominate over thermal effects can be extended substantially above 100 kHz. In such cases, 4.1.2.4 specifies criteria in which the electrostimulation limits must be evaluated to a maximum frequency of 5 MHz. For the sole purpose of determining compliance of non-sinusoidal waveforms with 4.1.2.4, the preceding three equations are to be evaluated to a maximum frequency of 5000 kHz. This extension, which allows one to determine a peak current electrostimulation limit above 100 kHz, does not obviate the need to also comply with rms criteria stated elsewhere in this standard.

Generally, individuals will not be aware of the presence of induced currents in various objects illuminated with RF fields. Inadvertent contact by an individual with such objects could lead to startle reactions or small burns that, while not hazardous per se, could lead to an accident. To reduce the probability of such startle reactions, two measures have been taken in this standard.

1) The contact current limit is based on laboratory data on perception of currents at different frequencies in humans (see Chatterjee et al. [B26] and Dalziel and Mansfield [B31]). These data indicate that perception thresholds, at any given frequency, depend on the type of contact made with the conducting object; touching contact generally results in lower current perception thresholds than grasping contact by a factor as great as ten. Accordingly, the current limits in Table 5 limit the current for grasping contact to 10 mA at 10 kHz. In the frequency range of 0.1 to 110 MHz, the current perception thresholds are related to the sensation of heating and become relatively constant with increasing frequency. In this frequency range, the grasp contact current is limited to 100 mA.
2) In some environments, the transient discharge phenomenon associated with initiating or breaking contact with energized conductors can lead to easily perceived shock effects even though the steady-state current flow, after complete contact is established, is within the limits prescribed in this standard. These effects are more directly related to the energy contained in the transient discharge and, consequently, measures of the open-circuit voltage and short-circuit current on the energized object may be better indicators of the potential for momentary shock effects. Contact with conducting objects in RF environments can result in a spark discharge as the contact is made or broken. Because the spark terminates in a very small region of the skin, the current density tends to be high. Significant heating of the highly localized tissue volume can occur, particularly when conveyed to skin in an area where the stratum corneum is dry and thin (i.e., other than on palmar or plantar surfaces). At the threshold for spark discharge, the typical effect is perception, and at somewhat higher intensities, a startle response is possible. More intense spark discharges can inflict noticeable skin damage (RF burn) and pain, typically in a small area. These effects are much more pronounced and can occur at relatively low levels when one makes light, single-point contact with the object rather than rapid, grasping contact with the full hand.

C.3.2 Spark discharge (from static fields into the GHz region)

The spark discharge phenomenon (microshock), which occurs whenever voltages are sufficient to cause dielectric breakdown of air across a gap, is quite complex. Repetitive discharges from RF sources may dissipate enough energy in a small volume of skin to cause localized RF burns to the skin. The current carried by the spark is a function of both the voltage between a conducting object and the human body and the relevant impedances. These are the equivalent source impedance of the object and the impedance of the person, of which skin resistance is a critical feature. To quantify conditions for spark discharges, a systematic set of measurements of voltage and current is needed. For these to have general applicability, measurements must be performed over a wide range of source impedances, potentials, and frequencies.

To initiate a spark discharge, a minimum voltage must exist between the object and the person. At 60 Hz, the minimum peak voltage supporting a spark discharge is 500 V on dry skin and 330 V on damp skin, or on surfaces where the corneal layer of skin has been removed (Reilly [R929]). For frequencies in the MHz

range, the breakdown potential across metallic electrode gaps is reduced by about 15 - 20%, suggesting that a similarly lower spark discharge threshold with human skin contact might be possible. At frequencies of several GHz, much more substantial reductions in breakdown potential can occur (see Craggs [B30]).

The US Navy uses a voltage criterion of 140 V (rms) in RF fields to define a potentially hazardous situation that could cause a person pain, visible skin damage, or an involuntary muscle reaction (NAVSEA [B93]). While this criterion is probably protective against adverse spark discharge effects, it may be unduly conservative in some instances, particularly when the effective impedance of the discharging object is high.

To date, only a limited amount of data has been collected in studies of RF spark discharge. In the HF region (2-30 MHz), data were recently collected for the US Navy (Pasour [B107]). These data suggest that in shipboard environments the 140 V (rms) criterion is a practical, conservative voltage threshold for spark discharges of sufficient intensity to cause a startle response. However, if it can be shown for specific situations via measurements that a higher open circuit voltage can be tolerated without surface arcing and an attendant RF burn, then the 140 V (rms) criterion may be exceeded. The voltage is measured across a resistance of about 10 k to ground. Although there is a rather large variation in the voltage threshold from object to object, most of the data on thresholds of perception due to spark discharges fall in the range of 150-200 V (rms). For grasping contacts in the ship environment, contact currents in the wrist were measured in correspondence with the threshold voltage for spark discharge perception. These corresponding contact currents ranged from approximately 300 to 500 mA (rms). It has been suggested that both open-circuit voltage and conducted current may be needed to set criteria for RF burn hazards (Reilly [R929]).

C.3.3 Specific absorption rate and temperature

C.3.3.1 Relationship between temperature increase and SAR

The time rate of temperature change (dT/dt in ºC/s) in tissue exposed to RF energy can be determined by the equation

$$dT/dt = (SAR + M - K - C)/c$$

where SAR is the rate of absorbed RF energy, M is the metabolic heating rate, K is the rate of heat loss due to thermal conduction, C is the rate of heat loss due to convection (blood flow), each expressed in W/kg, and c is the specific heat capacity expressed in J/kg·°C. The above equation can be simplified by assuming that a steady-state condition exists in the tissue prior to exposure, that is, $M = K + C$. SAR can be expressed as

$$SAR = c\,(dT/dt).$$

For high water content tissues, an SAR of 58.6 W/kg is related to a tissue temperature increase of about 1 °C/min.

This equation, under an adiabatic condition, shows that SAR is proportional to the rate of change of temperature in a biological sample. This relationship is the basis for several methods of SAR determination in animals and other biological samples (NCRP [B94]). It should be further noted that this equation is a simplified approach to tissue heating by electromagnetic fields since the interaction of the tissue with the field can also result in changes to M, K, and C. However, the equation is useful because it describes the general parameters of heat burden on the body even though the details may be quite complicated.

The studies that provided the evidence for a threshold SAR of about 4 W/kg for behavioral effects (work stoppage) in rodents and non-human primates show that exposures for about one hour at the threshold SAR are associated with an increase in body temperature of about 1 °C (see Annex B.5.2.1). These experiments also demonstrated that SAR is a better predictor of biological effect than power density (NCRP [B95]).

In addition to the complicated dependence of SAR on frequency, polarization of applied field, and the dielectric properties, size, and shape of the exposed object, the relation between SAR and temperature increase is further complicated by the heat transfer characteristics of the exposed object to its environment.

The environmental factors include air flow, ambient temperature, humidity, insulation, etc. At resonance, for example, SAR and temperature elevation will be maximal but at frequencies below and above resonance, SAR and temperature elevation will decrease. Due to their much larger surface-area-to-volume ratio, mice dissipate heat much more readily than larger animals, such as rats, and require higher SARs to produce similar elevations in body temperature. Raising or lowering the ambient temperature and increasing air flow will affect the temperature of objects and thereby affect the relation between SAR and temperature increase (see B.5). For these reasons, both expert judgment and an awareness of the weight of scientific evidence are required to interpret results in the literature and to extrapolate any suggestive experimental findings to potential adverse human health effects.

Interestingly, experiments with biological samples can be designed to show that the effects observed are due to an increase in temperature and not associated with the SAR. Two examples of experiments, one on cataracts and the other on nerve conduction, demonstrated that intentional cooling will lessen the effects of RF exposure. Cataracts did not develop in rabbits given a cataractogenic exposure level when the animals were partially immersed in cold water. This prevented the temperature of the lens of the eye to rise to the minimum temperature (~41 °C) required to cause a cataract (Kramar et al. [R947]). In the second study, nerve conduction in isolated neurons (an effect known to be temperature sensitive), did not change at SARs up to 1500 W/kg (CW) or up to 220 kW/kg (PW), when the sample was kept at its normal temperature by cooling techniques (Chou and Guy [R643]). These experiments show that the causative factor for the biological responses was due to the rise in temperature and not the RF energy per se. It is conceivable that RF exposure limits for some frequency ranges in a future revision of IEEE Std C95.1 will be based on an elevation in temperature and not SAR.

C.3.3.2 Levels at which increased temperature causes adverse effects

Exposures equivalent to the MPE in the body resonance range result in energy deposition, averaged over the entire body mass for any 6 min period of about 144 J/kg or less. This SA corresponds to an SAR of about 0.4 W/kg or less, as spatially averaged over the entire body mass. This WBA SAR is equivalent to about 1/3rd of the resting metabolic heat production of an average human adult. This level is completely benign; it will not increase the core body temperature by a measurable amount under almost all environmental conditions. Exposure to RF fields is but one of several potential sources of energy input to the human body. Body temperature regularly depends on sources of heat input such as exposure to the sun, physical labor, exercise, and ambient temperature. The resulting temperature is dependent on heat dissipation capability, which in turn is affected by clothing, humidity, air flow, etc.

The database that has been and continues to be developed allows for an examination of whether there is any frequency dependent or modulation dependent RF effect. To date no effects that are useful for standard development have been established in the frequency range above 100 kHz, other than those associated with a thermal response. Therefore, the literature database supports only a thermal mechanism as the explanation for effects of RF energy. Thermal mechanism implies that there are no modulation dependent effects, and no such modulation specific effects have been substantiated. The limits in this standard are intended to protect against adverse effects on the functioning of the human body that would be caused by elevating body core and/or local tissue temperatures to an unsafe level.

In order for thermal damage to occur, human skin would have to be heated at 43 °C for 10–12 h (Moritz and Henriques [B91]). On the other hand, for brief (3–10 s) thermal stimulation of small areas of the skin, the pricking pain threshold of ~45 °C is much lower than the threshold (for the same time) for skin damage, which occurs at 55 to 60 °C (Hardy et al. [B53]). All of these thresholds are modified by the surface area and region stimulated, initial skin temperature, moisture on the skin, and exposure duration. The time required to produce a full thickness burn in human skin ranges from 100 min at 45 °C to ~5 s at 60 °C (Moritz and Henriques [B91]).

Dewhirst et al. [R1080] summarized time/temperature thresholds for thermal injury to the spinal cord (rat, mouse, and dog) and brain tissue (rat, mouse, dog and cat). In both cases there appears to be a clear temperature threshold across species of 43–44 °C for the initiation of significant damage. Murine data (Hume et al. [B60]) suggest that thresholds for thermal injury may differ for different tissue types. The testes and brain

may be more sensitive to heat than other tissues, e.g., the intestines and skin, although specific end points (necrosis vs. function vs. appearance) may account for part of these differences. Careful analyses of the available data reveal a remarkable similarity in the sensitivity of individual tissues across species. Unfortunately, no data for human tissues (other than skin) are available for comparison with the animal data. Nevertheless, based on the thermal sensitivity of human cells *in vitro* and the sensitivity of animal tissues across species, one can conclude that it is very unlikely that human tissues are more thermally sensitive than those of other species (Dewhirst et al. [R1080]).

In addition, no verified reports exist of injury to intact human beings or of adverse effects on the health of human beings who have been exposed to electromagnetic fields within the limits of frequency and SAR specified by previous standards, including ASA C95.1-1966 [B12], ANSI C95.1-1982 [B6], and the IEEE Std C95.1, 1999 Edition [B70]. Table C.3 lists established critical temperature levels (produced by RF energy or other types of heating) in various species, organs or tissues leading to adverse biological effects.

Table C.3—Established critical temperature levels (produced by RF energy or other types of heating) in various species, organs or tissues leading to adverse biological effects

Endpoint	Species/organ/tissue	Threshold (ºC and SAR (W/kg))	Exposure duration	Reference number
Heat stroke	Human (core temperature) Human (brain temperature)	>42 ºC ≥ 40.5 ºC	$T \times t$ Varies $T \times t$	(Bynum [B22]) (Cabanac [B24])
CNS deterioration	Human (CNS)	42–43 ºC	$T \times t$	(Bynum [B22])
Skin necrosis Skin necrosis Full thickness burn Full thickness burn Pricking pain	Human Human Human Human Human	43 ºC 55-50 ºC 45 ºC 60 ºC 45 ºC	10–12 h 3–10 s 100 min 5 s 3–10 s	(Dewhirst et al. [R1080])
Thermal injury	Rat, mouse, dog, cat (spinal cord, brain)	43–44 ºC	1 to 80 min	(Dewhirst et al. [R1080])
Fetal abnormalities	Rat (whole body)	2–2.5 ºC increase	Tens of minutes up to 1 h	(Edwards et al. [R1081])
Behavioral disruption	Rat (whole-body) Monkey (whole-body)	1 ºC increase, 4 W/kg	40–60 min	(de Lorge [R232], [R233]), (D'Andrea et al. [R269])
Cataract	Rabbit (eye)	>41 ºC (>150 W/kg)	>30 min	(Kramar et al. [R654]), (Guy et al. [R698]), (Carpenter et al. [R941])
Convulsions	Mouse	T_{re} = 44 ºC		(Wright [B139])
Increase in BBB permeability	Rat	>40 ºC brain temperature (>4 W/kg WBA SAR)	4 h	(Merritt et al. [R402]), (Finnie et al. [R841], [R851]), (Sharma and Hoopes [R1082])

C.3.3.2.1 Whole body exposure

Human core temperature can be as low as 36 °C in the early morning and as high as 40 °C during exercise or environmental stress (Adair and Black [R1091]). The core temperature in humans is generally stable within the range of 36.5 to 37.5 °C at most environmental temperatures encountered; however, skin surface temperature is directly related to the environmental temperature (including radiant heat). Sensations of heat or cold, as well as feelings of comfort and discomfort, are primarily related to skin surface temperature and skin hydration. Humans have very sensitive behavioral and autonomic mechanisms to maintain both core and surface temperatures. Failure of temperature regulation is described by heat related disorders including heat cramps, heat exhaustion, and heat stroke, and may occur at any core temperature within the range of 39 to 47 °C (see C.3.3.3.1.1).

Studies of human beings deliberately exposed to RF energy are rare and most of those reported involve localized RF exposure. In volunteers undergoing magnetic resonance imaging (MRI), when the SARs = 2.7 to ~6.0 W/kg for 30 min, core body temperature (tympanic) could rise as much as 0.4ºC. This was observed to be a direct function of the SAR. Increases in local skin temperature, local skin blood flow, sweating, and heart rate were found also to be SAR-related, but negligible (Shellock et al. [R182], [R183], [R816]). As the frequency of localized RF exposure increases, wavelength decreases and the RF energy is absorbed closer to the surface of the body. In laboratory studies of volunteers undergoing 45-min RF exposure at normalized peak SARs equal to 6.0 to 15.4 W/kg in controlled thermal environments, core body temperature (esophageal) remained stable within 0.1 °C of the equilibrated level. Metabolic heat production (*M*) changed little in the resting subjects (Adair et al. [R137], [R639], [R660], [R782], [R792], [R1102]), (Adair [R874]). Individual physiological responses (skin temperatures, sweating rate, skin blood flow) were a function of ambient temperature (T_a=24, 28, 31 °C), frequency (450, 2450 MHz), and field strength (when the subjects were exposed at 180 and 240 W/m² at 450 MHz, or 270, 350, 500, and 700 W/m² at 2450 MHz). Corresponding normalized peak SARs at 2450 MHz were 6.0. 7.7, 11.2, and 15.4 W/kg, the highest being well outside guidelines of IEEE Std C95.1, 1999 Edition [B70].

For whole body exposure, the maximal absorption of RF energy occurs when the long axis of the body is parallel to the electric field vector (E-polarization) and the longest dimension of the body is about 0.4 of the free space wavelength (resonant frequency) (Durney et al. [R901]). RF exposure of non-human primates at resonance yields somewhat less efficient thermoregulation than does exposure to sub-resonant or supra-resonant frequencies (Adair et al. [R137]) (Krupp [R241]) (Lotz [R247]) (Lotz and Saxton [R92]). Although the threshold for a reduction in metabolic heat production (*M*) may be lower at resonance, the magnitude of the response change may be less for a given SAR than at non-resonance and the body temperature may rise. However, the hyperthermia is modest and well regulated. The situation is similar to that of humans during exercise (Adair [R874]). Some have expressed concern that human exposure at resonance may pose a greater hazard than exposure at other frequencies. Experiments recently completed, where seated adults undergo 45-min whole-body RF exposures at resonance (100 MHz), demonstrate that autonomic heat loss mechanisms (blood flow and sweating) are rapidly mobilized to dissipate heat generated deep in the body. No increase in core temperature occurred, even at a power density that is 8 times the limits of the IEEE Std C95.1, 1999 Edition [B70] at 100 MHz (Adair et al. [R1102]).

C.3.3.2.2 Localized exposure

If a non-human primate undergoes localized exposure at 2450 MHz (either to the head or trunk), the magnitude of the change in *M* reflects the total absorbed energy, as though it were integrated over the whole body (Adair [R1]). If an animal is exposed to RF energy at SARs greater than those that reduce *M* to the resting level, thermoregulation will be accomplished by mobilization of the next response in the thermoregulatory hierarchy, i.e., changes in vasomotor state or conductance, including blood flow (ACGIH [B1]) (Adair [R1]) (Candas et al. [R317]) (Lotz and Saxton [R91]). Experimental partial-body far-field exposures of human volunteers have been conducted at 450 and 2450 MHz for several field strengths in controlled environments (ACGIH [B1]) (Adair et al. [R660], [R782], [R792]). Even though the exposures covered only the dorsal aspects of the head, trunk and upper arms, increased local skin temperatures provoked strong heat loss

responses of increased skin blood flow and sweating, thereby ensuring a stable core temperature. Complementary whole-body exposures at these frequencies have not been conducted. The necessity for very large anechoic chambers and extremely high power RF sources to achieve whole-body plane-wave exposures prohibits such experiments.

During both experimental and clinical MRI procedures, part of the body (e.g., knee, head, or trunk) is often exposed to complex electromagnetic fields including RF fields. Shellock [R184] investigated the possibility that high RF 'hot spots' may generate thermal “hot spots.” During MRI, RF energy is mainly absorbed by peripheral tissues allowing the use of thermography to record patterns of skin heating. The study found no evidence for thermal “hot spots” on the dorsal skin of human subjects undergoing 45-min MRI scans at a WBA SAR of 3.2 W/kg. Instead there appeared to be a smearing effect of the temperature as the thermal load was distributed across the skin surface. Several studies have involved MRI procedures of the head, brain, and cornea through use of a send/receive head coil at local SARs as high as 3.1 W/kg, and imaging of the spine, abdomen, or scrotum through use of a body coil at local SARs of up to 4.0 W/kg (Shellock and Crues [B117]) (Shellock et al. [B118], [B119], [B120], [R182], [R183]) (Shellock [R184]). In general, localized temperature increases, including that of the cornea, were modest and not deleterious.

Since the 1930’s, thermal physiologists have studied the mechanisms of heat production and heat loss in the human body as they change during whole-body and/or localized heating. Such research intensified in the 1960’s as experimental techniques and measurement devices became more sophisticated and refined. Of particular interest were changes in vasomotor adjustments (blood flow) and evaporative adjustments (sweating) during either localized heating or robust exercise of individual limbs. This research generated some temperature threshold information for response change. The most valuable information was that 1) a core temperature of 37 °C will initiate sweating in an exercising person or a person exposed to a warm environment, and 2) an abrupt increase in regional blood flow will occur when the local tissue temperature reaches 42 to 43 °C.

Cunningham [R878] built a temperature-controlled skin applicator (flow calorimeter) to measure the relationship between localized skin temperature (forearm or hand) and changes in skin blood flow (SkBF). In these experiments SkBF remained low and stable [~1 mL / 100 cm^2·min] until skin temperature (T_{sk}) reached 42 °C, at which point SkBF rose abruptly and continued to rise until T_{sk} = 45 °C [15 to 20 mL / 100 cm^2·min], where T_{sk} is calculated by this equation across a range of 15 to 20 mL. These results were confirmed by immersion of the hand in a temperature-controlled water bath, for which a thermal model was developed (Stolwijk [B127]).

It is more difficult to measure blood flow (BF) changes in deep tissues, such as muscle. Lehmann [B82] pioneered the use of localized diathermy applicators (900 and 2456 MHz) to heat sub-surface tissues. Sekins et al. [R1119] devised innovative techniques to monitor BF at depth in muscle tissues via clearance of locally-injected $xenon^{133}$ when a skin-cooled 915 MHz diathermy device, placed on the thigh skin of 15 human volunteers, was energized. Temperatures under this applicator were recorded at 5 tissue depths with non-perturbing probes that were introduced (under local anesthesia) through fine catheters. Convective cooling of the skin surface allowed highly controlled RF energy deposition in the muscle tissue below the applicator. The report (Sekins et al. [R1119]) confirmed the threshold for a rapid increase in muscle BF at 42–43 °C. Other findings included the occurrence of gradients of local BF in fat and muscle at specific skin depths, accumulation of sufficient physiological data for accurate modeling, and establishment of appropriate conditions for efficient treatment of restricted tumors located well below the skin surface. A high incidence of heat intolerance occurs in multiple sclerosis, where it is particularly noticeable (at some level in up to 85% of the patients).

Multiple sclerosis is a disease of the nervous system characterized by a patchy loss of the myelin surrounding nerve fibers. This loss affects the transmission of nerve impulses and produces the symptoms of the disease. The demyelinated nerves are heat sensitive, and small increases in temperature lead to a worsening of clinical symptoms such as muscle weakness and visual blurring. The magnitude of temperature elevation sufficient to induce this unfavorable reaction can be very small, perhaps as small as a few tenths of a degree.

The exacerbation of symptoms is temporary, producing no actual tissue damage, and generally is rapidly reversed when the source of the increased temperature is removed. Home air conditioning is frequently prescribed for patients with multiple sclerosis (if they do not have it already).

After an extensive series of animal experiments in which histopathology of many organs has been performed, there have been no reports that chronic RF exposure causes demyelinaton. There is no evidence that chronic exposure to RF fields causes multiple sclerosis or any of the above clinical conditions.

C.3.3.2.3 Sensitive tissues and organs

Although some information on this topic appears in C.2.2.2.1, a more comprehensive discussion is presented here. The extent to which biological cells are killed by heat depends on both the temperature applied and the duration of exposure at that temperature. The extent of killing can depend on the development of thermotolerance, i.e., a situation where additional cell killing at the same temperature over additional time becomes much less efficient. In clinical hyperthermia treatments, it is useful to normalize the time-at-temperature data to a common unit that may be applied to various heating regimes. An approach to accomplish this is to determine a "thermal isoeffective dose," by which one time-temperature combination can be compared with another. In this method, time-temperature data are converted to an equivalent number of minutes at 43 °C. This temperature is close to the point of discontinuity (break point) of functions in many Arrhenius plots of survival versus time data for many different temperatures (Dewey [B33]). The equation for converting one time-temperature combination to another is:

$$CEM_{43} = tR^{[43-(T)]}$$

where

CEM_{43} is the cumulative equivalent minutes at 43 °C,

t is time (min),

T is average temperature (ºC) during the time interval t, and

R is the number of minutes required to compensate for a 1.0 °C temperature change above or below the break point.

Sapareto and Dewey's method (Dewey [B33]) assumes that $R = 0.25$ below the break point, which is consistent with much rodent data. This value indicates that the time to achieve an isoeffect at a defined temperature is increased by a factor of 4 for each degree drop below the break point. On the other hand, above the break point $R = 0.43$ for rodent cells, indicating that the time to achieve an isoeffect is increased only by a factor of 2.2 for each degree rise above the break point. Dewhirst et al. [R1080] note that based on in vitro data, the break point on Arrhenius plots is slightly higher for human (43.5 °C) than for rodent cells (43.0 °C). However, *in situ*, there is very little human data available apart from a few measurements of thermally induced skin necrosis (Beuttner [B19]) (Hardy et al. [B53]) (Moritz and Henriques [B91]). Most of the available data have been collected from experiments on mice, rats, and rabbits, with some data from dogs and pigs. Since the characteristics of porcine skin are quite similar to those of humans, future work on the thermal sensitivity of skin might be best conducted on pigs.

Hyperthermia, in terms of CEM 43 °C at various durations from <1 min to >80 min, reveals the thermal sensitivity of many animal tissues (Dewhirst et al. [R1080]). Based on histopathological analysis, testicular and brain tissues appear to be the most sensitive to thermal insult for exposures of short duration. Changes in blood brain barrier (BBB) function can also be significant. Bone marrow, kidney, and spleen show minor changes of an acute nature after exposure to elevated temperatures. Hyperthermia of longer duration (up to 40 min) exacerbates effects on the brain and BBB, produces minor morphological effects on the cornea, retina, and eyelid, and may damage the prostate and rectum. Still longer exposures (up to 80 min) can impair the function of peripheral nerves, damage additional parts of the eye (sclera, choroid, lens, anterior chamber and ciliary body) and impact the liver, muscle, skin, and fat. Exposures longer than 80 min produce signifi-

cant damage to most of the tissues in the body in rabbits, dogs, and pigs. Evidently, rodents do not survive CEM 43 °C exposures of durations much longer than 40 min.

C.3.3.3 Relevance of information from classical heat stress studies

C.3.3.3.1 Levels at which health or a physiological function are adversely affected

Hyperthermia refers to the general condition where body temperatures are above normal. An elevated core temperature increases metabolism and certain other functions, such as heart rate, respiration, and nerve conduction velocity. Central nervous system function deteriorates at temperatures above 42 to 43 °C and convulsions may occur. At this temperature, protein denaturation may begin and cells may be damaged by this mechanism. This is particularly dangerous for the brain, since lost neurons are not replaced. Thermoregulatory responses of sweating and vasodilatation cease at about 43 °C, after which body temperatures may rise very rapidly if external cooling is not imposed. Other events that occur at this temperature level include elevated enzyme activity levels, confusion or unconsciousness, and damage to the heart and kidneys. The conditions just described characterize heat stroke, a true hazard to human beings.

Any factor that either reduces heat loss or increases heat gain will predispose to heat stroke. Three main factors have been identified that predispose to the breakdown of heat loss mechanisms. These include a) dehydration, which perturbs the cutaneous circulation and sweat secretion; b) poor acclimatization to heat; and c) poor physical fitness. Other factors that have been identified as potentially contributing to the problem include alcoholism, chronic illness, fatigue, lack of sleep, obesity, and restrictive clothing.

The three main factors involved in the etiology of heat stroke are elevated body temperature, metabolic acidosis, and hypoxia, as discussed in the following subclauses.

C.3.3.3.1.1 Elevated body temperature

Body temperatures that are sufficient to produce heat stroke and cause death are not identical. Some patients have died with a rectal temperature of 40 °C while others that were admitted to hospital with rectal temperatures as high as 47 °C have survived. It has been generally accepted that core body temperatures of 42 °C and above are incompatible with life because protein denaturation begins at about this level. It appears more accurate to consider the combination of elevated body temperature and exposure duration as the cause of tissue damage, leading to the multiple system effects that characterize heat stroke. Bynum et al. [B22] have defined this combination as the Critical Thermal Maximum (CTM), a concept that explains the various clinical symptoms seen in heat stroke victims with a wide range of core temperatures.

The CTM may be adjusted by the several factors known to influence heat tolerance. For example, the CTM may be raised by heat acclimation, fitness, or high motivation; it may be lowered by dehydration or exercise or a rapid rate of temperature increase. The concept of an adjustable CTM fits well with the knowledge that, although a specific critical temperature can be defined for a species, this does not necessarily predict the death of an individual.

The concept of the CTM, either in terms of the absolute level of temperature alone or temperature combined with time, is widely accepted. For animals, it is the level of heat load that prevents escape from the thermal threat. For humans, it is the combination of exposure time with elevated temperature that results in either subclinical (one value) or clinical (another value) injuries. It has been reported that mice develop convulsions and lose their righting reflex at a rectal temperature ~44 °C (Wright [B139]).

In heat stroke, disturbances of the CNS are always present and the level of consciousness is often depressed. The symptoms include coma, sleep, or delirium. Pathology after heat-induced death shows edema in the brain tissue and meninges with a flattening of the brain convolutions, facts that infer that the temperature of the CNS tissue is critical to the occurrence of heat stroke. Thus, the defense (maintenance) of brain temperature seems to be of paramount importance. Whether the brain temperature decreases to a lower temperature

than core temperature during heat stroke is unknown, especially under prolonged steady-state conditions when the thermoregulatory system fails. Those patients who have survived heat stroke with core temperatures of 45–47 °C have had neurological complications or permanent deficits. Selective brain cooling has been demonstrated in several animal species (gazelle, goat, sheep, and dog) by counter current cooling of arterial blood as it passes through the carotid rete in the cavernous sinuses. Humans do not possess a carotid rete, and there is no comparable mechanism for significant brain cooling, despite contentions by Cabanac [B24] that such a mechanism exists.

C.3.3.3.1.2 Metabolic acidosis

Data on the metabolic status of heat stroke patients is variable for many reasons. There is no standard procedure for attending physicians to follow and complications of timing, specific circumstances, and individual variation all play a role. Both metabolic acidosis and respiratory alkalosis are commonly found. Most often, acute respiratory alkalosis occurs, precipitated by heat-induced hyperventilation. This is replaced quickly by metabolic acidosis, the progress of which reflects the severity of preceding physical exertion, dehydration, hypotension, and tissue hypoxia, all of which promote the development of lactic acidosis.

A related concern is potassium balance. Hypokalemia (low serum potassium) can be prevalent in the early stage of treatment for heat stroke, especially during rehydration and body cooling. It is accepted that heat-induced hyperventilation decreases P_{CO_2} (partial pressure of carbon dioxide) and the resulting alkalosis shifts K^+ into the intracellular compartment, thus potentiating hypokalemia. With the appearance of acidosis and a sudden drop in plasma pH, the serum potassium is elevated, a condition called hyperkalemia. It is of interest that natives of Asian countries, such as Indonesia, where the average diet is composed largely of rice (which contains very little potassium), will be prone to hypokalemia as a first step in the pathogenic process that leads to heat stroke.

C.3.3.3.1.3 Hypoxia

Tissue hypoxia has been targeted as an operating factor in heat stroke. However, data from laboratory and the clinic are not necessarily in agreement. For example, anesthetized dogs heated to a rectal temperature of 42 °C showed no change in cerebral blood flow, oxygen consumption, or glucose consumption (Shibolet [B121]). On the other hand, clinical data on 233 heat stroke patients during the 1982 pilgrimage to Mecca indicated that 40% were hypoxic with normal or low arterial P_{O_2}. Hypoxia with metabolic acidosis was found to be associated with the highest mortality as compared with the overall mortality of 9.5% during the 1982 pilgrimage (Mustafa et al. [B92]).

C.3.3.3.2 Additional factors in heat stroke

C.3.3.3.2.1 Endotoxin involvement

Endotoxin has been detected in the plasma of patients and experimental animals with heat stroke. It has been suggested that the failing liver in heat stroke is unable to clear the blood of endotoxins that originate from intestinal bacteria. If the gut of dogs is sterilized before the animals are exposed to heat, mortality from heat stroke is significantly reduced (Bynum et al. [B23]). This result implies that endotoxemia of intestinal origin was sufficiently severe to contribute to the fatal outcome.

Butkow et al. [B21] studied lethal heat stress in rabbits. They found that rabbits pretreated with antibiotics and then exposed to heat had a slower increase in core temperature than did control rabbits. At a rectal temperature of 42.5 °C, all control rabbits had endotoxin in their plasma, but only 1 of 6 animals pretreated with antibiotics had detectable endotoxemia. Mortality in the pretreated animals was reduced significantly. This finding confirms that the endotoxin originated from gram-negative bacteria in the gut.

Again, it must be mentioned that no verified reports exist of injury to human beings, or of adverse effects on the health of human beings, who have been exposed to electromagnetic fields within the limits of frequency

and SAR specified by previous standards, including ASA C95.1-1966 [B12], ANSI C95.1-1982 [B6], and IEEE Std C95.1, 1999 Edition [B70].

C.3.3.3.2.2 Effects on evoked potentials

Britt et al. [B18] developed a whole body hyperthermia model for the cat that featured a cardiopulmonary bypass circuit with a heat exchanger. This circuit allowed core and brain temperatures to be clamped at specific levels. They studied the effects of systematic elevations of core and brain temperatures (from 36 to 45 °C) on changes in brain function of anesthetized cats. They measured evoked potentials (brainstem auditory, somatosensory, and visual), core and brain temperatures, heart rate, arterial pressure, hematocrit, blood gases, O_2 and CO_2 exchange. They found that both amplitude and latency of evoked potentials decreased as temperature was increased to a "critical" value at which the latencies increased and the amplitudes continued to diminish. For auditory evoked responses and somatosensory evoked responses, the critical temperature was ~42.5 °C. For visual evoked responses, the latencies of component waves decreased as temperature increased with little change in waveform until a "critical" temperature was reached (~41.9 °C) at which latencies increased. Heating the brain to 42.3 to 44.0 °C resulted in complete loss of the waveform without recovery after cooling.

Other studies (Lyons et al. [B83]) using ultrasound- or microwave-induced heating of normal brain tissue in cats showed cytological evidence of damage after heating at 42.2 to 42.5 °C for 50 min. Thus, neurons began to show deleterious physiological changes within or near the same critical range shown to alter assorted evoked potentials.

C.3.3.4 Levels at which behavior is adversely affected

Research conducted during the past three decades has shown that exposure of laboratory animals to RF energy can cause a variety of behavioral changes. These changes range from subtle effects such as perception of microwave pulse-induced sound to behavioral disruption and complete cessation of behavioral performance due to increased temperature. Thermoregulatory behaviors have been investigated. Studies that have evaluated the effects of microwave exposure on the performance of well-learned operant tasks have previously been the primary avenue for establishing the relationship between SAR and behavioral performance. In these studies, performance disruption (or complete work stoppage) was evaluated by first establishing a stable behavioral performance and then determining the effects of RF exposure on the baseline performance. Typically, the effect observed has been a decreased rate of responding or decreased reaction time, although occasionally increased rates of responding and reaction time have been observed. A key factor, adding to the value of this protocol, is that the exposures of the laboratory animals and human subjects to the RF fields occur while they are performing the behavioral task.

One of the first demonstrations of behavioral disruption during microwave exposure was conducted by de Lorge [R329] with rhesus monkeys trained on an observing task, which is similar to vigilance behavior in humans. This experiment demonstrated that disruption of observing behavior was associated with a rectal temperature increase of 1 ºC or more during microwave exposure. This temperature increase was highly correlated with a WBA SAR near 4 W/kg. This protocol has proven to be one of the most sensitive and repeatable measures of potentially harmful biological effects due to RF exposure.

The disruption of a highly demanding operant task is a statistically reliable endpoint that is associated with WBA SARs in a narrow range between 3.2 and 8.4 W/kg. This is the case for a broad range of carrier frequencies (225 MHz to 5.8 GHz), species (rodents to rhesus monkeys), and exposure parameters (near- and far-field, CW- and pulse-modulated). The time-averaged power densities associated with these thresholds of disruption ranged (by calculation or measurement) from 80 to 1400 W/m². RF fields can serve as either positive or negative reinforcers over this SAR range and can disrupt both simple and more complex behaviors associated with cognitive capabilities. Thermal changes seem to account for most of the reported behavioral effects of absorbed RF energy across the limited frequency range explored. Those studies that report disrup-

tion of behavioral performance during acute RF exposure also involve tissue heating, mild heat stress, and alternate behaviors that are thermoregulatory in nature.

Because the threshold for disruption of ongoing behavior in nonhuman primates always exceeded a WBA SAR of 3.2 to 4 W/kg (D'Andrea et al. [R231], [R269]), (de Lorge [R329], [R330], [R331]), the value of 4 W/kg has again been adopted as the working threshold for unfavorable biological effects in human beings in the frequency range from 100 kHz to 3 GHz. This information provides a scientific database from which protective exposure standards can be derived.

Behavioral studies have been very useful in pinpointing those characteristics of RF fields that control the SAR, thereby corroborating analytical and dosimetric predictions (D'Andrea et al. [R269]), (Schrot and Hawkins [B115]). Many thermal effects controlled by frequency-dependent energy absorption, animal shape and size, and the presence of local electrical "hot spots" in the animal have been investigated with behavioral tests. In most cases, a simple test protocol has been followed to (1) establish a stable behavioral baseline of performance and then (2) determine the effects of RF exposure on this performance baseline. Generally speaking, the effect of RF exposure and concomitant rise in body temperature has simply been a reduction in behavioral response. Stern [R915] and others have pointed out that the reduction of response of a learned task may not necessarily imply a hazardous effect, but may simply reflect the animal's attempts to engage in other behaviors (e.g., escape, cooling off). These are responses that are thermoregulatory in nature and incompatible with learned behaviors such as lever pressing for food pellets on a prescribed schedule.

A short-term RF exposure can produce a thermal burden in an organism that may cause behavioral and other effects, some of which may be harmful. Justesen [R905] has described several classes of behavioral effects for such exposures that include perception, aversion, work perturbation, work stoppage, endurance, and convulsions. The combination of intensity and duration of exposure is the assumed basis for these effects; as the one or both increases, the effect advances beyond the threshold of perception, through intermediate steps, to an extreme thermal insult, grand mal seizures and finally death. In this respect, exposure to a RF field differs little from exposure to conventional sources of thermal energy or inhospitable thermal environments.

There has been a great expansion of the RF database since IEEE Std C95.1, 1999 Edition [B70] was published. An extensive review of the literature revealed once again that the most sensitive measures of potentially harmful biological effects were based on the disruption of ongoing behavior associated with an increase of body temperature in the presence of RF electromagnetic fields (D'Andrea et al. [R231], [R269]), (de Lorge [R329]], [R330]), (de Lorge and Ezell [R331]). Because of the paucity of reliable behavioral data from chronic exposures, the committee focused on evidence of behavioral disruption under acute exposures, even if these were of a transient and fully reversible nature.

Behavioral changes have also been reported following low-level chronic microwave exposure. For example, D'Andrea et al. [R31] exposed rats intermittently to 2450 MHz microwaves at a power density of 5 W/m^2 for 90 days and reported changes in time-related lever pressing behavior. However, a replication experiment reported different effects and failed to replicate the initial lever pressing findings (DeWitt et al. [R37]). Neither of these experiments replicated earlier findings reported by Rudnev et al. [R912] and Shandala et al. [R433]. One can only conclude that these experiments were below the threshold for reliable effects to be observed and, therefore, they cannot be used for setting safety standards. Another study at 25 W/m^2, reported effects that were statistically reliable, but this study was never replicated (D'Andrea et al. [R32]). The few biological effects reported subsequent to chronic microwave exposure (Lovely et al. [R908]) such as reduced food intake in exposed rats, cannot by itself be viewed as adverse to the health of the exposed laboratory animal. Moreover, none of the above reported biological effects during or subsequent to chronic, low level exposure has been independently replicated. For these reasons, it is implausible to use the results of the very few low level chronic exposure studies on animal behavior to define thresholds for hazards to humans from exposures to RF fields. Extrapolation to human beings of thresholds of reversible changes in animal behavior, while useful as an interim basis for standard-setting, must eventually be superseded by reliable data for the species in question, *homo sapiens*.

A consensus of the Committee is that the literature is still supportive of the 4 W/kg criterion and that WBA SARs below 4 W/kg have not been associated with biological or physiological effects that demonstrably constitute a hazard for humans. Adoption of this 4 W/kg level in the frequency range of 100 kHz to 3 GHz was based on the determination of a threshold for disruption of ongoing behavior in laboratory animals including nonhuman primates, and agreement that this is an indicator for unfavorable effects in human beings. For comparison, human metabolic heat production at a level of 4 W/kg results from a moderate activity level (e.g., house-cleaning or driving a truck), and falls well within the normal range of human thermoregulatory ability.

C.3.3.4.1 Levels at which other effects are adverse

As indicated above, the threshold SAR to produce adverse behavioral effects in laboratory animals is near 4 W/kg. Other adverse effects have been reported at higher SARs; a comprehensive list of these effects showing species, frequency, time of exposure, ambient temperature, etc., would be too lengthy to be discussed here. A few examples are described in this section (see Table C.4). Death (50% mortality) of mice and rats was observed, respectively, after exposures at 42 and 18 W/kg (estimated SARs based on reported power density) for a 4 h exposure at 20 °C at 2450 MHz (Berman et al. [R227]). For comparison, another paper (Petin et al. [R1131]) reported survival times of about an hour or so at ~14 W/kg for rats and ~30 W/kg for mice at 7 GHz. The threshold for teratogenic effects after exposures at 27.12 MHz is near 11 W/kg (Brown-Woodman et al. [R19]), (Lary et al. [R81]), [R373], [R374]) while the threshold for memory deficits is 10 W/kg at 600 MHz (Mickley et al. [R810]), (Mickley and Cobb [R811]). Multiple effects including bradycardia were reported after exposures at 2450 MHz and 6.5 W/kg (Phillips et al. [R417]). At 2450 MHz an SAR of 5.6 W/kg produced temporary sterility in rats (Berman et al. [R307]). Reduced fetal weight was reported in offspring born to rats exposed during pregnancy at 6 GHz and an SAR of 7.3 W/kg (Jensh [R360]). In another study at 970 MHz, an exposure at an SAR of 4.8 W/kg during gestation caused reduced weight gain in the pregnant rats and lower fetal weight in the offspring (Berman et al. [R228]). All of these effects can be attributed to the thermalizing effects of sustained whole-body RF exposure.

Table C.4—Adverse biological effects produced by RF exposure greater than 4 W/kg

Endpoint	Species	Frequency (MHz)	Threshold (W/kg)	Exposure duration	Reference number
Death Death	Mouse Rat Mouse Rat	2450 7000	42 18 30 14	4 h 4 h 50–70 min 60–100 min	(Berman et al. [R227]) (Petin et al. [R1131])
Birth defects	Rat	27.12	~11	3 min (42.2 °C) 10–40 min (41.5 °C) 26–32 min (43 °C) 120 min (41.5 °C)	(Brown-Woodman et al. [R19]) (Lary et al. [R81]) (Lary et al. [R373]) (Lary et al. [R374])
Memory deficit	Rat	600	10	20 min	(Mickley et al. [R810]), (Mickley and Cobb [R811])
Reduced fetal weight	Rat Rat	970 6000	4.8 7.3	22 h/d, days 1–19 of gestation 6140 min during 12–14 d of gestation	(Berman et al. [R228]) (Jensh [R360])
Fertility (temporary sterility in male rats)	Rat	2450	5.6	4 h/d, 5 d/week, 4 week	(Berman et al. [R307])
Bradycardia	Rat	2450	6.5	30 min	(Phillips et al. [R417])
Reduced weight gain in pregnant rats (heat stress)	Rat	970	4.8	22 h/d, days 1–19 of pregnancy	(Berman et al. [R228])

An established adverse effect of localized RF exposure is cataracts. Threshold conditions for lens opacities in the rabbit eye are SARs ≥ 150 W/kg for ≥ 30 min causing temperatures ≥ 41 °C in or near the lens (Kramar et al. [R947]), after exposures at 2450 MHz.

C.3.3.5 Levels associated with uncomfortable or painful sensations

C.3.3.5.1 Thermal stimulation

RF energy of millimeter wavelength (30 to 300 GHz) is deposited in the skin, and is therefore most effective in evoking sensations. In fact, RF energy at a frequency near 100 GHz has been shown to be as effective as infrared radiation for evoking warmth sensations, even though infrared is the natural stimulus for such sensations (Blick et al. [R615]). To evoke pain, RF exposure must raise the surface temperature of the skin by 10 to 13 °C, depending on the duration of exposure. Very rapid heating evokes pain at lower temperatures of approximately 43–44 °C, than does slower heating at higher temperatures of approximately 44–46 °C. At frequencies above 100 GHz, power densities greater than 5000 W/m² will produce such surface temperatures. At lower frequencies, RF energy is less efficient in raising skin temperature, as the absorption is spread over greater depths (volumes) of tissue. Below 6 GHz, it takes approximately 20 times as much incident power density as at 100 GHz to produce equivalent heating.

Over most of the frequency range in which protection against adverse effects is associated with heating (100 kHz to 300 GHz), exposure under normal circumstances at the MPE for the lower tier cannot even be perceived. For exposures near 100 GHz in the controlled environment, the MPE (100 W/m²) can only be perceived by individuals who are carefully attending to their skin temperature, and who have been alerted to the onset of the RF exposure. At 30 GHz and below, 100 W/m² is imperceptible under any circumstances (Blick et al. [R615]). For the upper tier, since higher power densities up to 1000 W/m^2 over an area up to 0.01 m^2 (100 cm^2) is allowed, perception should be possible under such localized exposure (cf. Blick et al., [R615]), which reports for 10 s exposures at 94 GHz, a perception threshold of 45 W/m^2 over a stimulus area of 327 cm^2. Even in the millimeter wavelength range, extended exposures at the MPE are unlikely to elevate skin temperature by as much as 1 °C. RF exposures at lower frequencies (<30 GHz) are much less effective in heating the skin.

C.3.3.5.2 Human response to thermal environments and equivalent RF exposure

Another interesting insight to human response to RF exposure can be gleaned from an examination of how individuals react to warm environments and how they express their degree of satisfaction with the environment in terms of thermal comfort. While not related to a biological hazard associated with RF exposure, the perception of comfort has been studied in human populations for years to characterize thermal environments in which people can perform optimally (Fang et al. [B39]) (Fanger [B40]) (Gonzales and Gagge [B47]) (Meese et al. [B89]) (Tham [B128]) (Wyon [B140], [B141], [B142]) (Wyon et al. [B143]). These studies have resulted in standards by which environments can be evaluated relative to the statistical response of large populations in terms of a scale that expresses the perception of comfort for given sets of conditions involving ambient air temperature, relative humidity, air speed, the metabolic rate of the subjects, the thermal insulation effect of clothing, etc. A widely recognized American Society of Heating, Refrigeration, and Air-Conditioning Engineers (ASHRAE) standard (ASHRAE-55 [B14]), first created early in the 20th century, was updated in 1992 to incorporate the most recent work on thermal comfort. The standard recommends thermal environmental conditions to achieve comfort indoors in all types of buildings.

Other predictive models have been developed that encompass thermal comfort based on different indices of how comfort is expressed among the population. In addition to ASHRAE 55 [B14], a standard used primarily outside the United States has been developed by the International Standards Organization (ISO-7730 [B74]). In the ISO standard, predictive mean vote (PMV) is an empirical function derived from the physics of heat transfer and the thermal responses of people in climate chamber tests. PMV establishes a thermal strain based on environmental conditions and attaches a comfort vote to that amount of strain. If the environmental conditions combined with the activity and clothing of the person being modeled produce a PMV within the range of -0.5 to +0.5, then the ISO comfort zone recommendation is met.

Today, software tools exist that permit convenient exercise of these kinds of thermal comfort models (Fountain and Huizenga [B44]) and that are employed widely for designing heating and air conditioning systems for the workplace. The ASHRAE *Thermal Comfort Tool* [B14] was used to examine how RF energy absorption, expressed as equivalent metabolic rates, might be equivalent to the perception of thermal comfort for a range of environmental temperatures. The model was exercised to compute the percentage of a large population of individuals that would rate a thermal environment as comfortable or uncomfortable. In particular, a thermally comfortable condition consisting of an ambient dry bulb temperature of 24.2°C, 50% relative humidity, and with an air speed of 0.1 m/s was established for a 70 kg person standing at rest with a metabolic rate of 1.2 mets (equivalent to 105 W)[15] and dressed in summer attire with a clo[16] rating of 0.5. The model may be used to predict the percentage of subjects that would express dissatisfaction with thermal comfort condition, based simply on raising the ambient air temperature. Additionally, the model can exam-

[15]The met is the unit used to express the metabolic rate per unit DuBois skin surface area. The met is defined as the metabolic rate of a sedentary person (seated, quiet), 1 met = 58.2 W/m^2 = 50 k cal/(h m^2). A normal healthy man has a maximum energy capacity of $\approx M_{act}$ = 12 met at age 20. Typical metabolic heat generation for various activities ranges from 0.7 to 8.7 met.

[16]It is traditional to express clothing insulation in terms of the "clo." The symbol "I" is used instead of the symbol "R" (radiative heat loss from the outer surface of a clothed body). The relationship between the two is $R = 0.115\ I$ or, 1 clo is equivalent to 0.155 m^2•kW. Garment insulation values range from 0.01 to 0.48, or greater.

ine the additional metabolic load that would cause the same predicted percentages but with the ambient temperature at the initial and comfortable value of 24.2 °C. Finally, the increased thermal load due to metabolic activity can be expressed as an equivalent SAR in W/kg under the assumption that the thermal loading on the body from metabolic activity would be similar to that imposed by whole-body RF energy absorption.

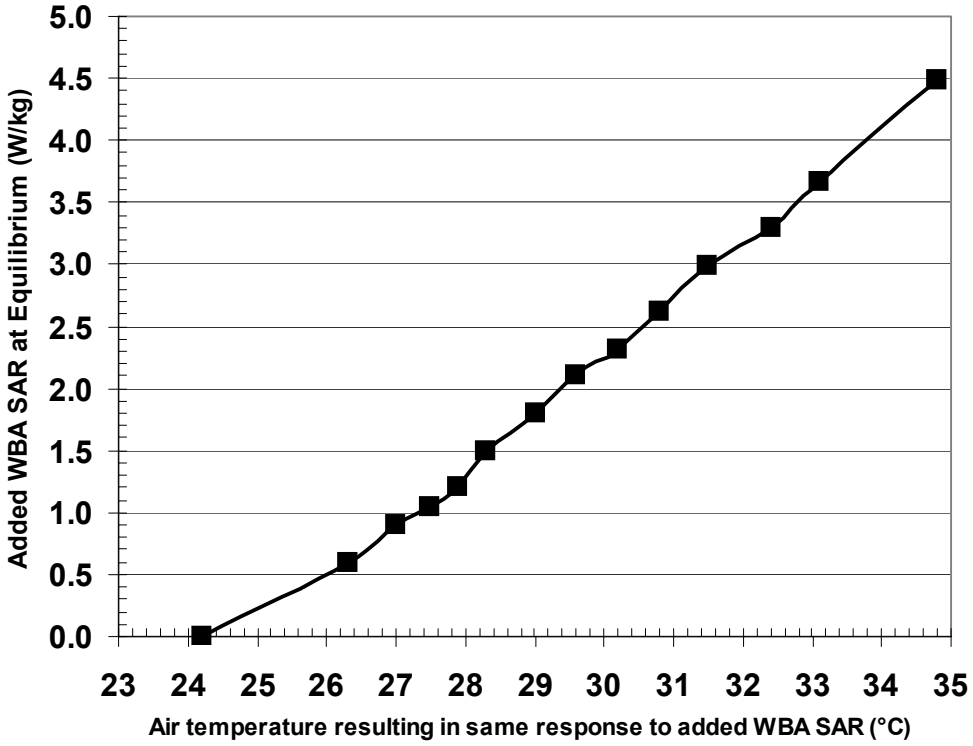

Figure C.4—Estimated added RF thermal load (W/kg) at rest and in a thermally comfortable environment to produce an equivalent discomfort response at rest to elevated ambient air temperature

Figure C.4 illustrates the results of this exercise wherein the additional thermal load on the body is correlated to an equivalent ambient air temperature. For example, an added load comparable to an SAR of 1 W/kg is expected to elicit a similar response in people as increasing the ambient air temperature from 24.2 °C to about 27.5 °C. This suggests that the extra heat burden of 1 W/kg would be perceived approximately the same as a 3.5 °C increase in the environmental temperature. In a similar fashion, an added SAR of 0.4 W/kg would be comparable to how an increase of about 1.5 °C in the ambient temperature would feel.

The concept of how RF energy absorption might be related to conditions of thermal comfort has been described (Berglund [R900]) wherein the RF energy dissipated in the body is compared to an equivalent metabolic rate. While the well noted behavioral response of laboratory animals to RF exposure has often been correlated to an increase in core body temperature, the response is likely related to a sensation of thermal discomfort in the animals and an urge to escape. This phenomenon has also been studied from a perspective not substantially different from the human thermal comfort responses discussed above. For example, data have been obtained (Adair and Adams [R292]) that demonstrate how squirrel monkeys will preferentially select a particular lower ambient temperature when subjected to differing levels of RF exposure. In one case (Adair and Adams [R292]), an incident power density of 20 mW/cm^2 at 2.45 GHz resulted in a preferred decrease in environmental temperature of 3 ºC, compared with the ambient temperature with

no exposure. This exposure is comparable to a WBA SAR of about 2 W/kg in the squirrel monkey (Durney et al. [R901]). From Figure C.4, for humans, an applied WBA SAR of 2 W/kg could be estimated to be comparable to an increase in ambient temperature of between 4 ºC and 5 ºC, a value not drastically different from the animal data.

These data offer further support that in the RF range the BR of the upper tier of this standard (a WBA SAR of 0.4 W/kg) is relatively benign from the perspective of thermal sensation, even when evaluation in the context of thermal comfort models that are based on extensive empirical human response data. When taken in concert with the analysis of the effect of 0.4 W/kg on human core temperatures in a wide range of ambient thermal conditions and with the human RF exposure studies discussed elsewhere, the results of the thermal comfort analysis add strength to the proposition stated early in this standard that there is strong scientific justification for the claim that exposures at the upper tier should be protective for all.

C.4 Stimulatory effects at frequencies from 3 kHz to 5 MHz

C.4.1 Relationships among in situ electric field, total current, and contact area

At frequencies between 3 kHz and 5 MHz electrostimulation of excitable cells is an important mechanism. Tables 1 through Table 5 list basic restrictions (BRs) and maximum permissible exposure values (MPEs) for electric and magnetic fields, and external electric fields, in addition to contact and induced current limits, are provided for this frequency range in Tables 1 Table 5. These limits are extensions of those published in IEEE Std C95.6-2002.

The excitation process of nerve and muscle is initiated by adequate depolarization of the cellular membrane from its resting potential. The fundamental force for membrane polarization (or depolarization) is the *in-situ* electric field, E_i, external to the excitable cell. Membrane depolarization effects are maximal when the internal field is oriented with the long axis of the excitable cell that bends or terminates within the induced field, such as with receptors or motor neuron end plates (Reilly [R929], [B111]). The electric field metric is preferred over current density, J. The two units are related by $J = \sigma E$, where s is the conductivity of the medium. However, the conversion introduces an additional parameter (σ), about which there may be some uncertainty in an applied situation. The calculation of E_i is less sensitive to assumptions of tissue conductivities compared to internal current density. Consequently, it is preferable to express basic restrictions associated with nerve and muscle electrostimulation effects in terms of E_i.

C.4.2 Strength-duration and strength-frequency relationships

The waveform of E_i is of critical importance in establishing the threshold of an electrical stimulus. Relevant waveform factors are expressed in this standard as strength-duration (S-D) and strength-frequency (S-F) functions, which express the relationship between the magnitude of the stimulus and its frequency or "phase duration." Asymptotic forms of these functions have one limb with a minimum plateau ("rheobase"), and another limb in which thresholds either rise in inverse proportion to phase duration for S-D curves, or in proportion to frequency for S-F curves. The connection point between these two limbs is expressed as an S-D time constant, τ_e, or an S-F frequency constant, f_e (above which stimulation becomes independent of frequency).

These parameters are related by $f_e = 1/(2\tau_e)$, as determined using a model of myelinated nerve (Reilly [R929], [B111]). Because of the nonlinear electrodynamics of excitable tissue, the relationship differs from that for linear systems, for which the relationship $t = 1/(2\pi f)$ would be anticipated.

Excitation thresholds can be represented by S-F and S-D curves for all excitable tissue. However, the constants τ_e and f_e differ significantly for the type of tissue. Values of f_e are greatest for nerve excitation (both

motor and sensory), are about ten times less for direct stimulation of muscle, and are roughly another factor of ten less for stimulation of synaptic processes in the central nervous system. Corresponding values of τ_e are inversely related to f_e. The parametric relationships in the basic restrictions of Table 1 reflect these properties for the different tissue types listed there as explained elsewhere (Reilly [R929], [B111]).

The *in situ* electric field, E_i, which is induced by external electric and magnetic fields, can be calculated through appropriate induction models to determine whether basic restrictions are met. However, with contact currents, it is often more convenient to specify the maximum permissible exposure in terms of the applied current, *I*. With current applied to electrodes contacting the skin, the lowest electrostimulation thresholds are usually determined by excitation of cutaneous sensory receptors, although peripheral motor neurons may be involved at somewhat higher levels. Perception thresholds for contact currents increase with the size of the electrode, which affects the current density (and the electric field) in the biological medium near the electrode (Reilly [R929]). Consequently, contact current thresholds are associated with the particular size of the contact area. In Table 5 of this standard, contact current limits are given for both touch and grasping contacts. The assumed contact area is 1 cm^2 for touch and 15 cm^2 for grasp contacts.

C.4.3 Spatial averaging of the in situ electric field

Basic restrictions (see Table 1) are specified in terms of the *in situ* electric field, E_i. In a practical sense, E_i may be determined from the potential difference across a small distance, divided by the distance. This calculation yields the average field over the measurement distance. In determining compliance, one must specify the spatial extent over which the field is to be averaged. The *in situ* field may vary locally at interfaces having differing conductivity. It is generally easier to comply with basic restrictions as the averaging distance is increased. An averaging distance should be neither overly restrictive nor permissive.

The biological significance of the averaging distance was explored with a myelinated nerve model (Reilly and Diamant [B112]). This theoretical study examined how the excitation threshold varies as a function of the spatial variability of a field surrounding a myelinated nerve fiber, and the errors in determining the field with different dimensions of spatial averaging. An averaging distance of 5 mm was found to be a reasonable choice for measurements or calculations of the *in situ* field in determining compliance with basic restrictions.

C.5 Averaging time

IEEE C95.1 standards have historically used a one-tenth of an hour (six-minute) averaging time. Since the whole-body thermal time constant is known to be an hour or more, 6 min corresponds to a time constant for partial body heating. This estimate has its origin in the earliest C95.1 standard (ASA C95.1-1966 [B12]) and was derived from an estimate of the thermal time constant of small objects like a human eyeball. Thus the MPE is conservative for short periods of time less than 6 min. By using 6 min in the main (resonance) frequency range (100 kHz to 3 GHz) for both the whole-body and localized exposures, the MPEs are extra-conservative.

As stated in the definition of *averaging time* (see Note), for an exposure time less than T_{avg} (the averaging time) the MPE in terms of the usual power entity is an inverse function of exposure time. On the other hand, as stated therein, one could alternatively express an MPE in terms of an SA (specific absorption) value or energy flux expressed in J/m^2. This alternative is also discussed in C.6.1.

Beginning in 1991, a concomitant of the concept of a lower tier was an increase of averaging time from 6 minutes to 30 minutes. This reflects earlier judgments (e.g., Commonwealth of Massachusetts, NCRP) that controversy on adequacy of limits existed only for long-term exposure times $>> T_{avg}$ and not for short-term exposure times ($t < T_{avg}$) where the potential hazards are acute thermal effects like a burn. If the same SA limit is adopted to protect against burns etc. for both tiers then necessarily T_{avg} must be 30 minutes for the

lower tier vs. 6-minutes for the upper tier. This is the case in the main human resonance range of 3–5000 MHz for E-field exposure and 100–5000 MHz for H-field exposure. At lower frequencies the averaging time is ramped down to 6 minutes for f <1.34 MHz for E-field exposure and for f <30 MHz for H-field exposure. These differences reflect a more conservative treatment of E-field at low frequencies.

Above 3 GHz, the exposure in human tissue is quasi-optical. At still higher frequencies, above 15 GHz, it is known that the RF energy penetration depth is much less than 1 cm (see C.2.2.1), and that the thermal time constants drop to seconds as the infrared range is approached. Consequently, the MPE for the upper tier specifies continuous functions for the field limits and averaging times as the frequency increases to the upper limit of 300 GHz. ANSI Z136.1-2000 [B7], the laser safety standard, has an averaging time (effective) of 10 seconds at 300 GHz for both small area exposure, where the MPE is approximately 1000 W/m^2, and large area exposure where the MPE is 100 W/m^2. The laser standard is also conservative for large areas by not increasing the averaging time to keep the same energy-density limit for short exposure durations. Both this standard, IEC-60825-1 [B65], and the ICNIRP laser safety guidelines [B63] have only one tier at 300 GHz. Therefore, this standard is in agreement at 300 GHz with the laser standards, which have world-wide recognition and acceptance.

In the late 1980's there were concerns expressed that the averaging time at frequencies from 10 to 30 GHz was too large and did not take into account penetration depth, which begins a rapid decline above 5 GHz. A caveat relating to exposure of the eyes and testes was therefore inserted in the localized exposure limits, which were relaxed to values above whole-body values. It was put forward that if the averaging time could be corrected to correspond more realistically to the thermal time constant dependence on frequency (and penetration depth), there would no longer be a need for the caveat.

In the 1990's, Foster et al. [R672] carried out extensive thermal modeling. Though quasi-one-dimensional, the results agreed with experimental data with appropriate adjustment of the blood-cooling (convection) constant. These results and their relation to the high-frequency ramp in IEEE Std C95.1, 1999 Edition were reviewed at two extensive workshops sponsored by the U.S. Air Force: one took place in January 1997 and one in August 1999 (USAFRL Digest [B130]).

Other relevant findings were presented at various workshops, conferences and in publications. One important finding, by Walters et al. [R713], was an observed time delay of tens of seconds before mm-wave exposure affected any increase in blood cooling. This supports the assumption of non-enhanced blood cooling constants in thermal modeling applied to the ramp problem. The new averaging times included in this standard (with a two-step variation above 3 GHz) provide for shorter averaging times at the upper end of the frequency range covered by this standard and are consistent with the laser safety guidelines at 300 GHz (ANSI Z136.1-2000 [B7]) (ICNIRP [B63]) (see Figure C.5).

Shown in Figure C.5 in bold is the ramped averaging time for the controlled environment (upper tier) as specified in Table 8. A similar ramp for the averaging time for the lower tier MPE is not shown in Figure C.5 but is listed in Table 9. These ramps were derived on the basis of the same energy density (the product of the MPE and the averaging time (W/m^2 × min) for both tiers for short exposure times, i.e., $t < T_{avg}$.

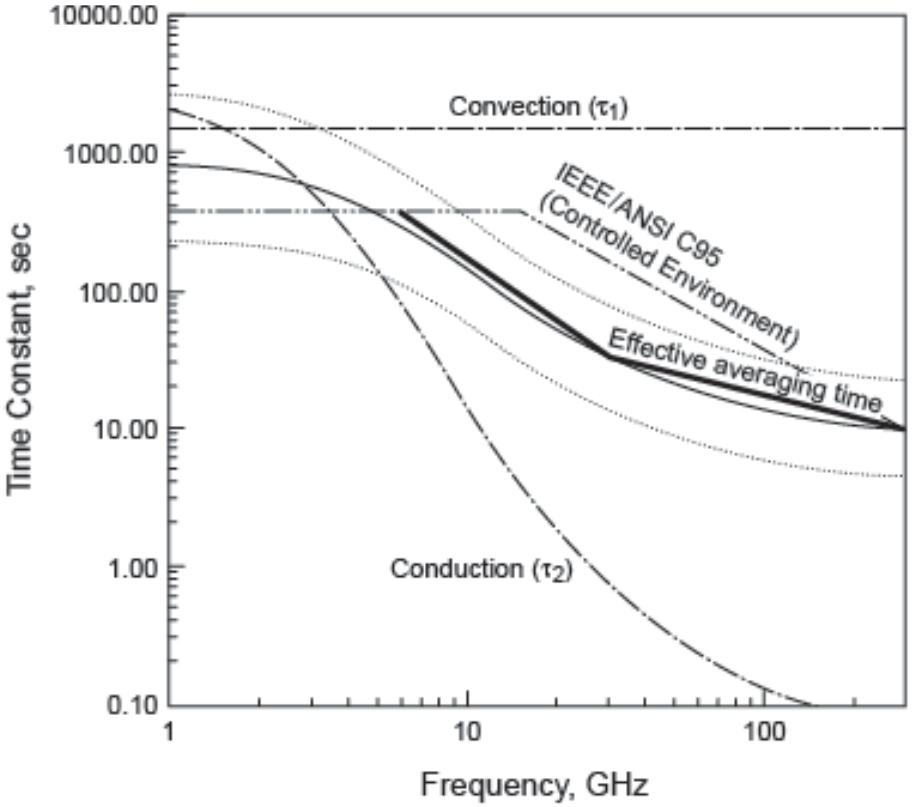

Figure C.5—The new averaging times included in this standard (a two-step variation) provide for shorter values at the upper end of the spectrum and are consistent with other standards and guidelines at 300 GHz

The averaging time in the frequency region where electrostimulation is the dominant mechanism is based on different mechanisms than for thermal effects discussed above. As explained in C95.6-2002 integration time constants for electrostimulation with repeated or sinusoidal waveforms can be as great as 0.2 s.

C.6 Safety factors and uncertainty factors

C.6.1 Safety factor

This standard, and its companion, C95.6-2002, address guidelines for the electromagnetic spectrum below 300 GHz in frequency. At low frequencies MPEs and basic restrictions are stated in terms of fields, current densities and currents. At higher frequencies (up to 3 GHz) the MPEs and basic restrictions are stated in terms of densities of power and energy. Above 3 GHz the basic restrictions and MPEs are stated in terms of power density. These match the terms in ANSI Z136.1-2000 [B7], although in the laser standard they are named as irradiance (W/m^2) and radiant exposure (J/m^2). ANSI Z136.1-2000 [B7] covers the wavelength range of 1 mm (300 GHz) to 180 nm (ultraviolet).

Safety factors and their rationales are different for frequencies below 5 MHz where the adverse effect being minimized is electrostimulation, and for the frequency range above 100 kHz where the adverse effects being protected against are related to heating. In the transition region of 100 kHz to 5 MHz both types of effects are protected against through separate sets of limits (MPEs and BRs).

The term "safety factor" is commonly interpreted to be the ratio of an exposure level causing an adverse effect to the corresponding allowable exposure limit. Consequently, the development of a safety factor presupposes the selection of a hazard threshold (HT) and identification of uncertainty parameters. Comparison of two safety factors of numeric ratios in fields vs. power density would be meaningless. To ensure that such comparisons are always meaningful, the safety factor is always expressed in terms of dB, where the dB value equals 10 times the $\log_{10}$ of a ratio of powers but 20 times the $\log_{10}$ of a ratio of fields or currents. Thus a safety factor of 20 dB relates to a ratio of 100 in power and a ratio of 10 in fields.

C.6.1.1 Minimization of adverse effects associated with electrostimulation (3 kHz to 5 MHz)

At frequencies below 5 MHz, a relevant hazard is associated with painful or aversive electrostimulation. Because the nature of adverse effect is different for electrostimulation (frequencies below 5 MHz) from those for heating above 100 kHz the nature of and rationale for a safety factor is different. At these low frequencies, exposure measurements require an averaging time of 0.2 s for rms metrics, and peak measurements require instantaneous values. The estimated safety factor in terms of currents or fields is between 3 and 10 (10 to 20 dB) in the worst case even though for many situations and people the safety factor is considerably greater. The upper tier in the standard, which is applicable to exposures in controlled environments (such as with certain occupational exposures), incorporates a lower safety factor that approaches a minimum of unity even though in most cases the safety factor is considerably greater. The tolerance of a margin of safety that can approach 1, meaning no margin of safety, is justified for the upper tier below 100 kHz because of the less serious nature of the adverse effect, i.e., a sensation, and the general awareness of workers in occupational situations.

The safety factors for special exposure situations, such as peak (short pulse) limits and contact and induced currents in the limbs, are often related to the safety factors incorporated in the BRs or MPEs for fields. It is believed that this factor is of the order of at least 10 dB in general.

In physiotherapy electrostimulation is used for beneficial medical purposes. The dose-response relationship for frequencies less than 100 kHz, is best presented in terms of the *in situ* electric field and time constant appearing in the so-called strength-duration curves for mono-phasic pulses of current. From this one can derive the related hazard threshold (HT) value for a sinusoidal current or field. (Although not commonly shown, in principle one can derive a corresponding HT curve in terms of power and energy). Because of the differing nature of the electrostimulation effect, the practical relevance of a time constant is less direct than with thermal effects. The time constant corresponds to the inverse of the frequency above which the HT threshold is believed to increase linearly with frequency.

While the lower tier is protective against electrostimulation for frequencies below 100 kHz, the upper tier allows as much as 3 times (9.5 dB) higher exposure in terms of electric field strength so that there is a small but finite probability, based on an assumed statistical spread in stimulation thresholds among people, that a person in a controlled environment (upper tier) could experience sensation or even pain at the limit. Thus, with regard to electrostimulation, the term *minimize* adverse effects is used throughout this standard. This small potential for a safety factor of unity is considered acceptable in a controlled environment where such stimulation can be anticipated by the individual and there is no lasting adverse effect, and where the exposure is brief and can be terminated by movement of the individual. The difference in safety factor for the two tiers is tolerated for exposure to the main body including the brain but a substantial safety factor exists for both tiers with regard to heart stimulation and minimum safety factor, when exposure of non critical body parts, such as the hands, feet, wrists, and ankles, are involved.

The basic restrictions of Table 1 refer to the electric field induced within the biological medium. Table 1 defines basic restrictions in the frequency range of 3 kHz to 5 MHz. These restrictions have been developed to minimize adverse electrostimulation with an adequate safety factor, as described in IEEE Std C95.6-2002.

A safety factor multiplier of $F_S = 0.333$ allows for protection of (possibly) exceptionally sensitive individuals, uncertainties concerning threshold effects due to pathological conditions or drug treatment, uncertainties

in reaction thresholds, and uncertainties in the induction models. In the case of the hands, wrists, feet, and ankles, $F_S = 1$ for the general public in recognition of the narrow cross sections and preponderance of low conductivity tissue that tend to enhance the *in situ* E-field in these areas in comparison with other areas of the body. Because these regions lack critical function when compared with the vital organs, a greater localized electric field is permitted. In the case of the controlled environment, $F_S = 1$ for all of the reaction types except for excitation of the brain or heart under the assumption that a small probability of discomfort is acceptable in the controlled environment for some mechanisms, but that excitation of the brain or heart is unacceptable for all individuals. $F_S = 1$ can be justified in some cases where short-term reactions are immediately apparent to the exposed individual because they can remove themselves from the environment, modify their activities, or can take other action to avoid the exposure entirely.

If $F_S = 0.333$ is to be compared with that applied at higher frequencies in this standard, it should be noted that a multiplier of 0.333 applied to the magnitude of the induced field is equivalent to a multiplier of 0.333^2 in SAR because SAR is proportional to the square of the induced field.

C.6.1.2 Protection against effects associated with heating (100 kHz–300 GHz)

Above 100 kHz exposures are assessed as to potential heating effects and with reference to an averaging time, with the standard values varying with frequency and at some frequencies with tier designation. Exposures of duration shorter than the averaging time are short-term exposures. In this case the adverse effects being avoided are burns and other potential damage from overheating of tissue. For longer exposure durations considerably greater than the averaging time, the adverse effect being protected against, based on an exhaustive evaluation of both the low- and high-level exposure literature, is the most sensitive effect seen in animals and extrapolated to humans; this adverse effect is behavioral change. The safety factor in terms of SAR or SA for these moderately long duration exposures has been estimated to be in the range of 10 to 50 in power (10 to 17 dB) for the upper tier BRs and MPEs. At frequencies where the predominant interaction mechanism is tissue heating, the lower tier for the BRs and MPEs provides no demonstrable increase in protection but is based on greater safety factor to address public concerns and uncertainties in exposure assessment, serve as a surrogate for environmental limits or for purposes of harmonizing with other standards and guidelines. More importantly, the lower tier is recommended as an action level to implement elements of an RF safety program to protect against ever exceeding exposures above the upper tier of limits.

Above 100 kHz, when assessing the heating effects of short duration exposures (less than the averaging time) the BRs and MPEs are essentially related to energy, i.e., specific absorption (SA) or energy density. One can, however, continue to use the BRs and MPEs expressed in power terms, i.e., SAR or power density or equivalent fields, while specifically recognizing their time dependence. The safety factor in this short duration regime is believed to be at least as large as in the long-duration regime. Note that since the limits are the same, in energy terms, for both tiers in the short duration regime, the safety factor is the same for both tiers.

In explaining any standard for safe exposure to electromagnetic energy, the basic exposure diagram, Figure C.6, is helpful. This is a log-log plot of power and energy entities versus time for a hazard threshold curve. Also shown is a lower exposure limit curve, either an MPE or basic restriction. These data generally apply to exposures to electromagnetic energy at frequencies between 100 kHz and 300 GHz where the hazards have been demonstrated to be related to thermal phenomena. For most laser standards and microwave standards the hazard threshold curve has two branches. One is a long-term exposure boundary described by a constant power density (or SAR) and the other is a short-term exposure boundary described by a constant energy density (or SA). The two branches merge around a time interpreted as a thermal time constant (see Figure C.6). Correspondingly the MPE or basic restriction curve has a similar shape but lower by a factor simplistically called the *safety factor*. Thus the *safety factor* is the ratio of HT to MPE expressed numerically or in decibels (dB). At ultraviolet and blue light frequencies *cumulative* photobiological (quantum) effects and hazards exist for which the HT curve is one of constant energy or radiant exposure and for which a suitable exposure limit is one limiting radiant exposure or total absorbed energy (SA). No such cumulative effects have been recognized or demonstrated at frequencies below 300 GHz.

At the lower laser frequencies and at frequencies below 300 GHz it has been universally recognized (Baranski and Czerski [B16]) (Minin [B90]) that the hazard threshold (HT) is real and that at exposures below (or suitably below by a finite amount) the HT there is no hazard. This is because the biological effects of exposure to EM fields or energy are known to be deterministic and not intrinsically stochastic (probability based) in nature, as is believed to apply for ionizing radiation. Above 100 kHz the long-term hazard to humans, as extrapolated from animal experiments, is associated with heating and not electrostimulation. The selected threshold of 4 W/kg is based primarily on behavioral disruption data in laboratory animals of several different species exposed acutely to RF fields. The short-term hazard uses burns or the pain preceding a burn as its basis. For very short exposure times, and relatively unique exposure conditions, "high peak power" effects such as the microwave auditory effect can occur. This is not considered, per se, an adverse effect and is actually very difficult for an exposed person to discern. This position is consistent with the judgment in both the microwave and laser regimes, that mere sensation, e.g., warmth or auditory, is not a hazard.

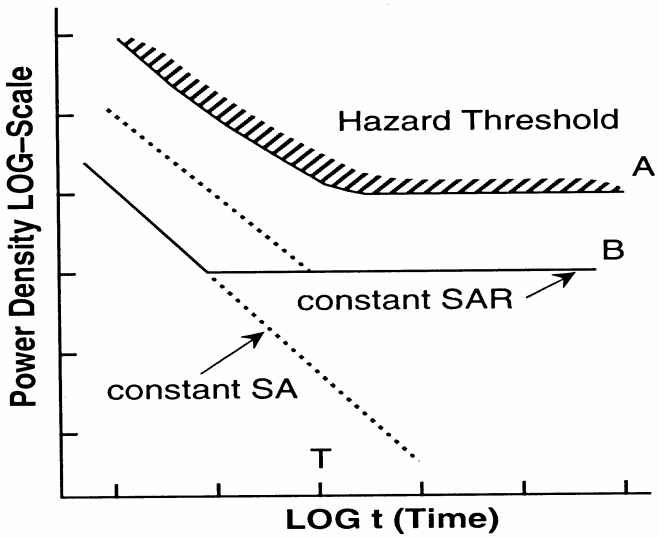

Figure C.6—Thresholds for various effects and hazards expressed as a function of time

Above 100 kHz the lower MPE curve (or basic restriction) associated with heating effects has a somewhat similar shape as the HT curve. The point of intersection of the constant SAR and constant SA branches, which is at a time called the averaging time, may or may not be exactly below the corresponding thermal time constant which in Figure C.6 is denoted *T*. If the safety factor is defined as the ratio of the HT curve ordinate in Figure C.6 to the MPE curve ordinate at some time *t*, then it is clear that the safety factor can be different in the short-term range of exposure time and the long-term range of exposure time. Thus the classical MPE in the microwave range with a six minute averaging time is believed to exhibit a higher safety factor for short exposure times ($t < T$) than for longer exposure times ($t > T$). This is particularly the case when whole-body exposure is considered, since the thermal time constant for the whole body is considered to be about an hour (Tell and Harlan [R256]).

If the inflection points of both curves A and B in Figure C.6 are the same, which is equivalent to saying that if the averaging time for the lower MPE curve is equal to the thermal time constant in the HT curve, then the safety factor is the same at all exposure times and is the same whether the entities are expressed in terms of SAR or SA. If, however, the averaging time and the thermal time constant are different, i.e., as most commonly happens the averaging time is considerably smaller than the corresponding thermal time constant, then the safety factor drops from a larger value at time below the averaging time to a smaller value for times

greater than the thermal time constant. Most of the time safety factor is addressed in the long-term exposure regime and these considerations are moot. In practice, most cases of overexposure and injury (burns) occur as a result of short-term exposures. This is to be expected since most people are not immobile for long periods of time (for many minutes). For short durations below the averaging time, and high SAR values above the long term HT curve threshold, the safety factor also finds expression as the ratio of time where the HT curve assumes the value of the SAR exposure to the time where the MPE curve assumes that SAR value. As an example, in the simplest case where averaging time and thermal time constant are equal, if the long term safety factor is 10 in SAR or 10 dB, then for short exposure durations the ratio of the HT and MPE abscissas in time is also 10.

In Figure C.6 the ordinate is a power entity so that the short-duration branch of the MPE curve and also (presumably) the HT curve is one of constant energy and is a straight line on a log-log plot expressing the fact that SAR ~1/*t*. If the curves are re-plotted in terms of the E field associated with the power entities, then the short-term branch is a line with a different slope reflecting the fact that $E \sim t^{-1/2}$. When exploring strength-duration curves at low frequencies, the plots are different, i.e., $E \sim t^{-1}$.

Although difficult to quantify, it is believed that the basic (upper) tier of the MPE of this standard for long-term exposure incorporates at least a safety factor of 10 and probably considerably more if the remarkable tolerance in human studies (Adair et al. [R660]) is accepted as generally valid. The lower tier extending upward from the resonance frequency range around 30 to 300 MHz incorporates an extra safety factor, for the purpose of ensuring a larger *margin of safety*, defined below, when the standard is a surrogate for an environmental limit. It is noted that although the extra safety factor is incorporated for long-term exposure, the safety factor is the same for both tiers for short-term exposure, where SA is the limiting entity. This is the result of the application of a longer averaging time, i.e., 30 minutes, in the resonance range for the lower tier as compared with the 6 minutes specified for the upper tier.

Localized exposure can tolerate a much larger SAR, SA, etc., than can a whole body exposure. Thus a concomitant of the limit on basic restriction is that local values of SAR, and by linkage SA, can be higher by a factor of 20 with the same safety factor presumed to exist for whole-body exposure. In this standard formulas exist to translate these limits into local MPEs. Although these are approximate formulas, it is believed that the original safety factors are preserved in the process.

The MPEs in this standard are well matched, albeit conservatively, to the laser MPEs at 300 GHz (USAFRL [B130]). Because at high frequencies the principal hazard becomes one of burns from small area beams, and because thermal time constants decrease with increasing frequency and decreasing penetration depth, the averaging time is decreased appropriately with frequency to maintain the same order of safety factor (see C.5 and the related publication by Riu and Foster [R672]). Because this standard does not provide a relaxation of the MPE at 300 GHz as the beam area decreases, but ANSI Z136.1-2000 [B7] allows a tenfold relaxation, this standard is actually more conservative at 300 GHz than ANSI Z136.1-2000 [B7], i.e., the RF standard incorporates an extra safety factor at 300 GHz.

C.6.1.3 Equivalence between RF exposure and metabolic rate

The notion of finding an equivalence between RF energy absorption and metabolic activity of humans was addressed 26 years ago (Tell and Harlan [R256]) but is still of interest today in exploring practical thermal impacts of RF exposures. An informative insight to the expected thermophysiological impact of RF energy absorption within the body is provided by studying the projected core temperature of soldiers in warm environments (Givoni and Goldman [B46]) at different work rates expressed in W/kg. Figure C.7 presents the results of applying empirical formulas derived from human data representing the equilibrium value of rectal temperature, typically obtained within about one hour of work activity, depending on the ambient temperature. Rectal temperature rises as work begins and eventually reaches a nominal plateau value shown in Figure C.7 as long as the ambient thermal conditions are not so severe as to stress the human thermoregulatory system beyond its ability to maintain normothermia. Equilibrium core temperature is seen to increase with increased work loads and increased ambient temperature. At very high air temperatures, the core tem-

perature rises rapidly and does not reach a plateau. These values are predicted based on an empirical model obtained under conditions less than the highest ambient temperatures plotted in Figure C.7 and likely do not accurately represent core temperatures when an equilibrium value could never be achieved due to extraordinary environmental circumstances.

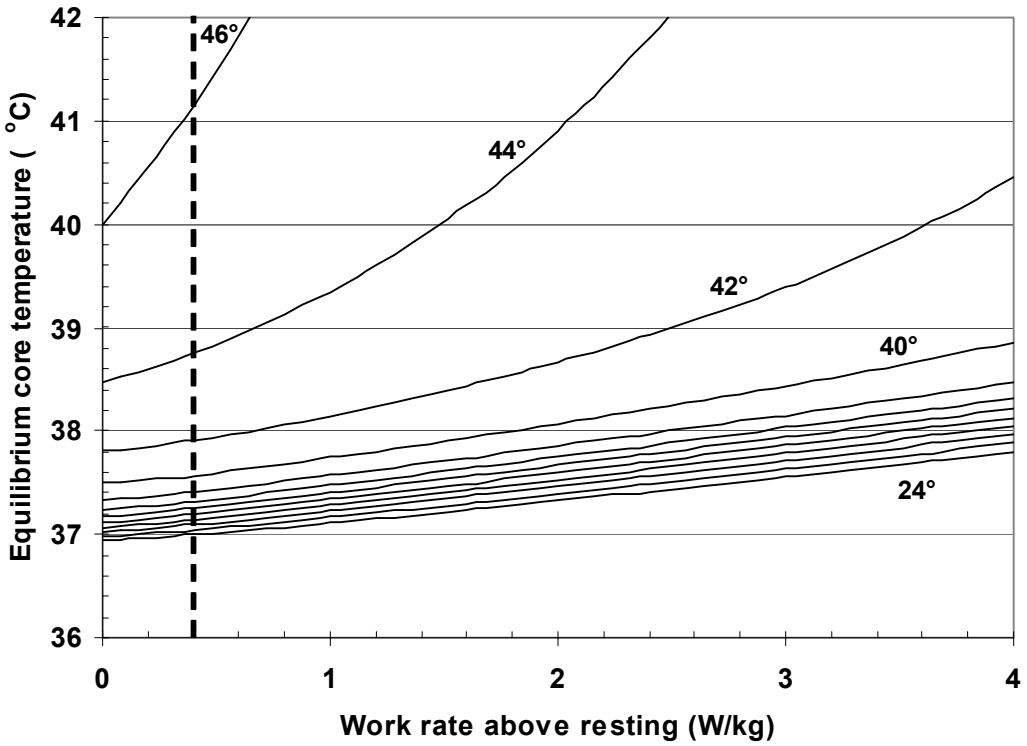

Figure C.7—Projected equilibrium core temperature as a function of additional workload above resting metabolic rate for a range of ambient air temperatures (24 °C to 46 °C) derived from empirical data obtained on army soldiers

It is of interest to note that when the added work load is equivalent to a whole body average RF energy absorption rate of 0.4 W/kg, the BR of the upper tier of this standard, the core body temperature rises only very modestly, less than 0.5 °C, for a range of environmental temperatures up to about 40 °C (104 °F). This observation is consistent with human RF exposure studies wherein exposures substantially exceeding the upper tier MPE resulted in only very minimal increases in core body temperature (Adair et al. [R639], [R782], [R875], [R1102]) (Adair and Black [R1091]), and adds support to the contention that the upper tier of BRs (100 kHz to 3 GHz) in this standard should be protective for all humans.

C.6.2 Uncertainty parameters

In the above discussion, except when discussing the statistical spread for electrostimulation, it has been assumed that both the HT and the corresponding MPE are crisp entities with definite values. In reality, there are many uncertainties that modify the meaning of safety factor and its dependability in practice. Figure C.8 shows a range of uncertainty above and below both the HT and MPE curves. In the most general view these uncertainties, ur_n, could be different below and above a curve and also different as a function of time.

The uncertainties in the HT curve include the following:

a) Measurement and other errors in the scientific database and statistical variation among experimental subjects/and or samples.

b) Lack of detailed knowledge of dosimetry in experimental or epidemiological data.

c) Variation in absorption with change of size, position, orientation and consideration of localized exposure and non-uniform fields

d) Effects of environmental factors like temperature, humidity, air flow, insulation, etc.

e) Statistical variation among people as to thresholds for tolerance of electrostimulation or heating under various conditions.

f) Extrapolation of experimental data from animals to humans. This uncertainty may be small when dealing with electrostimulation or local heating but it may be large when dealing with complicated higher-level phenomena in animals, such as behavioral effects. It should be encouraging, however, to note that humans have generally a far superior system of thermoregulation than most animals.

g) It has been long recognized (NRC [B101]) that individual scientists exercise different judgments in similar exercises of extrapolation of data. These are called value judgments and contribute to uncertainty but often in the conservative direction.

h) In practice there will be errors in determining exposure, which contribute to a compliance error Δu_c.

i) Finally, based upon the value judgments of a wide range of experts, there is the final agreement and the selection by consensus of a definite number for the MPE, in which there is recognition of some margin of safety, beyond allowance for uncertainties.

Figure C.8 shows what the actual minimum margin of safety is if all uncertainties are in the wrong or undesirable direction. This minimum is unlikely, so that equating margin of safety and safety factor is generally a reasonable action.

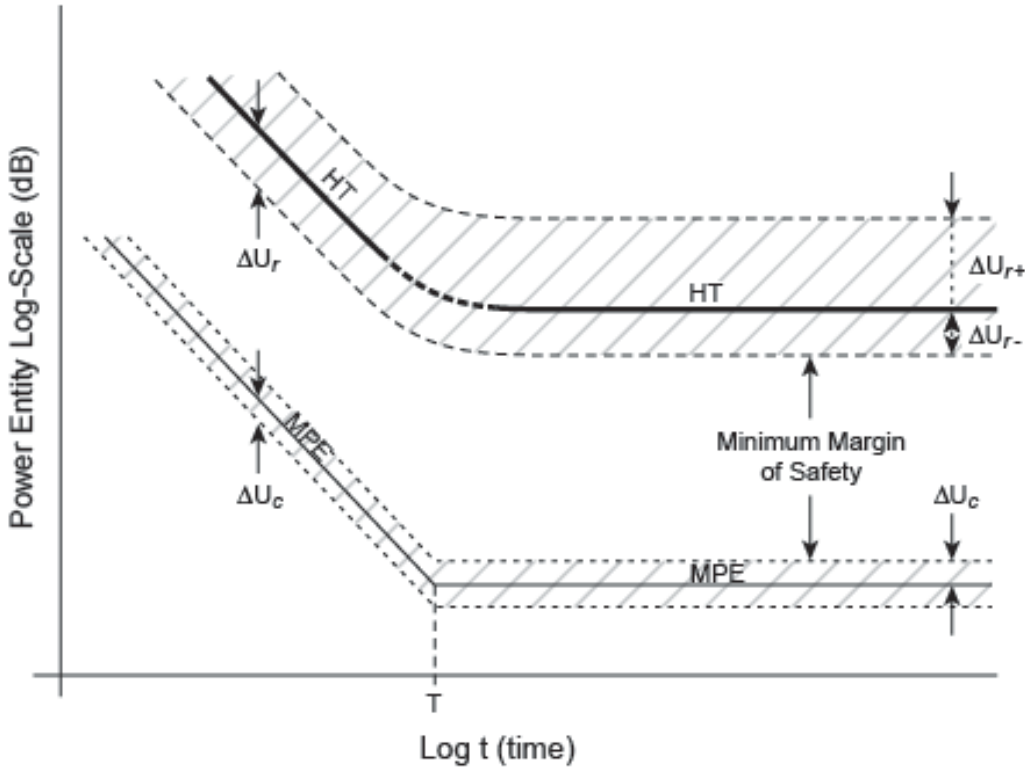

Figure C.8—A hypothetical exposure diagram illustrating uncertainty factors and resulting minimum margin of safety. This diagram is based on power. A similar diagram could be based on current density or internal electric field.

C.6.3 Conclusions

There is no substantiated evidence of illness or injury resulting from exposure to electromagnetic energy in the RF range when the exposures are within the limits of this standard. The experience of RF burns is well known to the occupational RF and medical communities, and is a principal hazard to be protected against by compliance with this standard. Transient electrical sensations, even those that are painful, are sometimes experienced by electrical workers; these are made improbable by compliance with this standard. Over all, the standard incorporates a reasonably large margin of safety. An RF safety program shall be employed for those potentially exposed above the lower tier. Indeed, the standard may be considered especially conservative, since the safety factors are applied against perception phenomena (electrostimulation, behavioral disruption) which are far less serious than reversible tissue damage and any permanent pathology that would occur at exposure levels much higher than those for perception phenomena.

C.7 Special considerations

C.7.1 Recognition of whole-body resonance

As is true of ANSI C95.1-1982 [B6] and IEEE C95.1, 1999 Edition [B70], the MPEs in this standard are based on recommendations of field strength or of plane-wave-equivalent power densities of incident fields. These limits are based on well established findings that the body, as a whole, exhibits frequency-dependent

rates of absorbing electromagnetic energy (Durney [B35]) (Durney et al. [B34], [R901]), (Gandhi [B45]), (Barber [R843]). Whole-body-averaged SARs approach maximal values when the long axis of a body is parallel to the E-field vector and is four tenths of a wavelength of the incident field. Maximal absorption occurs at a frequency near 70 MHz for Standard Man (height = 175 cm) and results in an approximate seven-fold increase of absorption relative to that of standard man in a 2450 MHz field (Gandhi and Chatterjee [R345]), (Durney et al. [R901]). In consideration of this dependency, recommended MPEs of field strength have been reduced across the range of frequencies in which human bodies, from infants to large adults, exhibit whole-body resonance. The whole-body resonance values for the range of human body size become relatively flat for frequencies in the range of about 1 to 3 GHz. The recommended MPEs have been developed to reflect this dependency of whole-body resonance and SAR on frequency to 3 GHz. Above 3 GHz, the absorption is quasi-optical and body resonance considerations do not apply. The limit increases through a transition phase up to the quasi-static region because of the relationship of skin thickness to the penetration depth of RF energy at the higher frequencies. At higher frequencies, above about 15–30 GHz, it is known that penetration depth is much less than 1 cm and thermal time constants drop to seconds as the infrared range is approached. Consequently, the recommended MPEs at 300 GHz, are consistent with the MPE at a wavelength of 1 mm as specified in ANSI Z136.1-2000 [B7] and IEC 60825-1 [B65].

C.7.2 Non-uniform exposure fields

From a dosimetry viewpoint, an important description of an exposure field is whether it is uniform or non-uniform. Uniform fields are those having a locally plane-wave character, i.e., the electric field vector is perpendicular to the magnetic field vector, and they are both perpendicular to the direction of propagation. Another characteristic of uniform fields is that the electric and the magnetic fields are interrelated by a constant, which is referred to as the characteristic impedance. Uniform fields exist in the far-field region of a radiating source (antenna) that is free from reflections from objects and the ground. The far-field region is commonly assumed to begin at a distance of about $2D^2/\lambda$ from the antenna, where D is the greatest dimension of the antenna and λ is the wavelength. At locations close to the source, exposure fields are usually non-uniform, their electric and magnetic field polarizations are not well defined and the field strengths may vary in an oscillatory fashion with distance. In addition, the ratio of electric to magnetic field strengths at these locations is spatially dependent.

In situations where ungrounded or poorly grounded conducting objects are located near a radiating source, RF energy from the source induces electric charges or currents on the object. The amount of the induced current depends on the physical characteristics of the object (size, shape, orientation with respect to the source) and the frequency of the incident field. This current produces its own electric and magnetic fields in close proximity to the object. The produced fields, which are generally reactive, interact with the incident field and may result in enhanced electric and/or magnetic fields close to the object surface. The enhanced fields are non-uniform and generally decrease to the ambient levels in the surrounding areas within very short distances from the object.

Exposure evaluation is an important step for performing risk assessments. Determination of exposure fields can be done using a theoretical estimation, e.g., as described in IEEE Std C95.3-2002, or an appropriate instrument. However, it is generally difficult to predict non-uniform exposure fields by theoretical methods. The reliable way to determine actual levels of these fields is by measurement.

C.7.3 Near vs. far-field exposures and SAR

Depending on the distance from an RF source, a person can be exposed to RF energy in the near or far field. Even in the far field, RF energy absorption in tissues is a complex function of many variables (Chou et al. [R726]). The absorption will generally produce a non-uniform induced RF field distribution within the object, regardless of the external exposure field uniformity. The far field typically begins at a distance of $2D^2/\lambda$ from the radiating source, where D is the longest dimension of the radiating structure and λ is the wavelength in air. In the far field, with the exception of polarization, SAR is independent of source configu-

ration (there is no interaction or "coupling" between the source and the object). However, in the near field (closer than $2D^2/\lambda$), the body may couple to the ambient RF field in such a way that the resulting SAR (whole-body-average and local) are not related to the strength of the unperturbed fields in the same way that they are when in the far field and may be significantly affected, as well, by the relative sizes and shapes of both the RF field source and the body, e.g., an operator's position relative to an RF dielectric heater or heat sealer (Stuchly and Lecuyer [R221]). Kuster and Balzano [R175] have shown that in the immediate vicinity of resonant RF current sources, such as a hand-held mobile telephone, the SAR in an exposed homogenous model is primarily associated with the current induced by the RF magnetic field. Peak SAR in the head, for example, is dependent on the distance between the RF source and tissue. Therefore positioning is critical in determining the peak SAR associated with the RF exposure from a mobile phone or other device that is positioned at or very near the surface of the body. A special concern is exposures taking place in the reactive near field of a source which is typically taken to be a distance equivalent to $\lambda/2\pi$, or approximately one-sixth of the incident field wavelength (CENELEC [B25]). Table C.5 below summarizes these distances within which the resulting whole body SAR may not follow a direct relationship with the incident plane wave equivalent power density or the square of the electric or magnetic field strength. Nonetheless, WBA SARs are not expected to exceed those values associated with the same plane wave equivalent power densities at these distances. When the exposure occurs in the reactive near field of a source, compliance with this standard can be determined by ensuring that *both* the electric and magnetic field components do not exceed the corresponding MPEs. In some cases, however, alternative measures, such as induced body current, may be more useful, e.g., when characterizing exposure associated with dielectric heaters (heat sealers). For a more accurate assessment of the actual WBA SAR, a direct assessment, be it analysis or measurement may be necessary.

Table C.5—Estimated reactive near-field region of RF field sources within which SAR assessment may be necessary

Freq (MHz)	$\lambda/2\pi$ (cm)	$\lambda/2\pi$ (in)	$\lambda/2\pi$ (ft)
30	159.15	62.66	5.22
100	47.75	18.80	1.57
200	23.87	9.40	0.78
300	15.92	6.27	0.52
500	9.55	3.76	0.31
750	6.37	2.51	0.21
800	5.97	2.35	0.20
900	5.31	2.09	0.17
1800	2.65	1.04	0.09
2450	1.95	0.77	0.06

Because of the relatively simple dosimetry for far field RF exposure, field strength or power density in the space to be occupied by a person, without the person present, is usually measured for comparison with the derived MPE as specified in Table 8 and Table 9. When exposure takes place in the reactive near field of a source, SAR assessment, in contrast with the simpler task of measurement or analysis of the fields in air, may be needed to accurately determine compliance; while WBA SAR should be conservatively estimated in the reactive near field by determining the electric and magnetic fields, local SAR may not. If one can show

compliance with field or power density measurements, no further SAR measurement is needed. However, if SAR measurements show that the basic restriction is met, the MPE may be exceeded. For example, McCoy et al. [B87] have shown that inside a car, the SAR in a back seat passenger exposed to VHF or UHF fields from a trunk-mount antenna is within the SAR limit, but the field intensity in the back seat exceeds the MPE. This situation complies with the standard because the basic restrictions have been met.

C.7.4 Spatial considerations (peak vs. whole-body average values)

Under conditions of non-uniform illumination it is possible that the average field exposure over the whole body does not exceed the MPE, but still results in excessive localized heating. To accommodate these circumstances, the MPEs include requirements that limit the localized field exposure. These caveats, expressed via limits in the extent of spatial averaging area, are specified in the notes for Table 8 and Table 9.

For frequencies of 3–30 GHz the whole-body averaging area decreases as the square of the free space wavelength, from 10 000 cm^2 to 100 cm^2. These areas represent nominal values for the human body cross section and the surface area of the human face or hand, respectively.

The transition frequency at which whole-body averaging is not used starts at 3 GHz. The rationale for the selection of this transition frequency begins with the observation that the penetration depth in human tissue at 3 GHz is approximately 2 cm. The localized SAR (spatial peak-10 g average) is calculated over 10 grams of tissue, which is represented by a cube approximately 2.15 cm on a side. For frequencies above 3 GHz, where the penetration depth is small, incident power density is the basic restriction.

For frequencies greater than 30 GHz, most of the energy is deposited in the skin. Therefore, the averaging area remains constant at 100 cm^2. This value is logically consistent with ANSI Z136.1-2000 [B7], in which section 8.4.2 of that standard specifies the MPE for large area skin exposure for the far infrared. In addition, the human thermal aversion response will normally prevent overexposure of the cornea to millimeter wave fields.

The choice of 1 cm^2 for the spatial peak averaging area was influenced by several factors. First, there is general agreement with other guidelines and standards including ICNIRP [B63] and ANSI Z136.1-2000 [B7]. Second, the surface area of the cornea is approximately 1 cm^2. Lastly, this is a practical limit for spot size at 5 cm or 3 probe diameters (which ever is greater) from an RF source for assessing compliance with the MPE to avoid undesirable coupling between the probe and the source (see IEEE Std C95.3-2002).

C.7.5 Tissue averaging mass considerations

The extensive review of both the low level and high level RF biological effects literature has established that RF exposure results in adverse health effects only when the exposure results in a detrimental temperature increase. SAR has been used as a surrogate for the expected temperature rise, particularly for localized exposure. However, calculated or measured values of the SAR averaged over a particular volume do not always correlate with temperature rise. Heat transport and the resulting temperature depend on the size of the region absorbing energy and area blood flow among other factors. When a small region is heated, it rapidly transfers heat to cooler surrounding regions and its temperature does not rise appreciably. On the other hand, when a large volume is heated, the rapid local transfer of heat tends to produce a uniformly elevated temperature throughout. These observations support the use of a volume-averaged SAR if the volume is chosen small enough to avoid excessive temperature gradients over its extent and yet large enough to obtain an average SAR that corresponds well to the actual temperature increase throughout the volume.

Non-uniform SAR distributions can be expected to occur more readily at higher frequencies where it is possible to produce SARs that can vary significantly over a distance of several centimeters to less than a millimeter, comparable to the scale of anatomical features in tissue. Non-uniform exposures generally occur

for sources that are close to the body, but they also can occur when the sources are at a distance for frequencies where the penetration depth is small.

A localized high SAR value produces localized heating and a localized increase in temperature. The magnitude of the temperature rise in a small region of body tissue is determined by the localized SAR, thermal properties of the tissue, diffusion of heat from warmer to cooler regions, and removal of heat by circulating blood, which acts as a heat reservoir at body temperature. Biophysical models for temperature distribution in a tissue heated by a localized source of RF energy have established that even without the significant cooling effect of blood flow, thermal diffusion is highly effective in reducing localized temperature differences at equilibrium, during the transient period following sudden onset of exposure, and for short duration exposures. Blood perfusion of living tissue further reduces temperature differences by a significant amount.

These considerations are supported by calculations and measurements that have been made using tissue models (Riu and Foster [R672]) (Van Leeuwen et al. [R711]) (Wang and Fujiwara [R987]) for the particular case of exposures of the head near antennas operating at approximately 0.9 GHz or 1.8 GHz. In one anatomically detailed analysis, temperature increases were always less than 0.12 ºC in the brain for an SAR of 0.91 W/kg averaged over a cubic volume with a mass of 10 g (Van Leeuwen et al. [R711]).[17] This maximum temperature occurred superficially in the skin of the skull; temperature increases elsewhere in the head were lower. In comparison to this average SAR of 0.91 W/kg, the maximum SAR for any 1 mm^3 volume was 4.0 W/kg (Van Leeuwen et al. [R711]). These values illustrate two important features: (1) SARs in tissue do not have an extreme range as seen in the ratio of less than 5 for the highest SAR to the average SAR over a 10 g volume (which contains approximately 10,000 one mm cubes); (2) the temperature in a small volume resulting from an RF exposure with a relatively high SAR cannot be increased significantly compared to the temperature of nearby tissue regions unless the average SAR was so high that it caused generalized heating of all of the tissues in the vicinity. Average SARs of that magnitude would not be permitted by other requirements of the standard.

Because the depth of penetration of RF energy decreases as frequency increases (see C.2.2.1), depth of penetration provides a reasonable reference for the volume that can be heated at a particular frequency. The depth of penetration at 3 GHz in muscle and some other tissues is approximately 2 cm. This dimension provides a natural and convenient dividing point between “low frequencies” for which heating is uniform over anatomical regions of a few centimeters or more, and the “high frequencies” for which heating is limited to the superficial layers (skin) and is highly non-uniform in depth for any anatomical region. A cube with 2.15 cm sides has a volume of 10 cm^3, which, at frequencies of 3 GHz and below, is large enough to obtain an average SAR that assures a relatively uniform temperature over the volume. As frequency increases progressively above 3 GHz, a 10 cm^3 cubic volume is less suitable for averaging the SAR because of the temperature gradients that arise in correspondence to reduced penetration depth. For tissues with densities close to the density of water, 10^3 kg m^{-3} (1g cm^{-3}), a 10 cm^3 averaging volume contains approximately 10 g of tissue. Since absorption of RF energy requires a mass of tissue rather than an (empty) geometric volume, the requirements for averaging volume are expressed in terms of a 10 g tissue mass. Other standards have chosen a 10 g tissue mass based on rationales that are consistent with the foregoing discussion.

C.7.6 Historical perspective on the evolution of the lower tier

This subclause provides a perspective on the development of the lower tier basic restrictions (BRs) for whole-body and localized exposure. The 1982 and earlier ANSI RF exposure standards had a single tier of exposure limits. The BRs in ANSI C95.1-1982 [B6] were based on research demonstrating a whole body average (WBA) SAR threshold for behavioral disruption in laboratory animals of nominally 4 W/kg. A safety factor of 10 was applied to yield a WBA BR of 0.4 W/kg. This BR was considered to be a conserva-

[17]The same maximum temperature increase was correlated to a calculated SAR of 1.53 W/kg when averaged over a 1 g cubic volume. Both a 1 g and 10 g averaging volume are adequate to limit excessive localized SAR.

tive limit, given the far greater thermoregulatory capacity of humans vs. the laboratory animal species studied. For localized exposure, the magnitude of the spatial peak SAR limit was derived from the WBA limit based on studies showing the ratio of peak-to-WBA SAR to be about 20:1 (ANSI [B6]). A tissue mass of one gram was chosen as the mass over which the spatial peak SAR value was averaged because, at the time, 1 cm^3 (~1 g) was the approximate resolution of the best available dosimetry derived from thermographic measurements. Based upon these decisions, the spatial peak SAR in ANSI C95.1-1982 [B6] was set at 8 W/kg (a value 20 times the WBA limit of 0.4 W/kg) as averaged over any one gram of tissue. Thus, the 1982 standard specified a single tier of exposure limits incorporating a safety factor of 10 that was believed to be protective of all persons in the population.

The measurement of WBA SAR in the studies supporting the 1982 standard was quite accurate. Furthermore, WBA SAR represented the dosimetric quantity most meaningfully related to behavioral disruption, the effect still believed today to be the most sensitive biological indicator of potentially adverse health effects. Behavioral disruption in rats and non-human primates exposed to RF energy was often associated with a core body temperature increase of about 1 °C above normal. The relationship between the threshold RF exposure level and body temperature was not emphasized in the 1982 standard, however, due to the contentious issue of thermal versus non-thermal effects. Even so, it was recognized that humans have a significantly greater thermoregulatory capacity compared to laboratory animals and, for this reason, the limits were judged conservative and protective against uncertainties in the extrapolation of animal data to human beings.

The framers of ANSI C95.1-1982 [B6] recognized that local hot spots of energy absorption likely existed in the exposed animals (and in exposed humans as well). They further presumed that, by limiting the WBA SAR, protection for any contribution to behavioral disruption, possibly due to the higher localized SAR levels, was provided. At the time, a complete understanding of the magnitude and spatial distribution of peak SAR values to arrive at useful RF protection guides for partial-body exposure was considered unnecessary for the following reason: If the BR of 0.4 W/kg and its attendant spatial peak SARs up to 8 W/kg is the safe limit for whole-body exposure, then the reasonable conclusion is that 8 W/kg is a safe limit for partial body (localized) exposure only.

By applying an additional safety factor of 5 to the original BRs, a second lower tier for people in “uncontrolled environments” was included in IEEE-C95.1, 1999 Edition [B70], specifically, 0.08 W/kg for whole-body and 1.6 W/kg averaged over 1 g tissue for partial-body exposure. Thus, the lower tier incorporated a safety factor of 50. The committee that developed the 1991 standard and its subsequent 1999 Edition concluded that an additional safety factor was justified only for exposures in uncontrolled environments and then only for exposures that were penetrating (i.e., resonant frequency exposure) or associated with complicating factors like effects from contacting metal objects.

Some background information on how the safety factor of 5 was selected is warranted. First, the committee determined that an additional factor of 10 was likely excessive and a factor of 2 not sufficiently differentiating from the upper tier. Second, the committee was influenced by the 1986 NCRP report [B95] that recommended a general public exposure limit incorporating a safety factor of 5. The NCRP rationale was based on continuous exposure of the public compared with workers, that is, on a weekly basis, the public is exposed for 168 h compared with 40 h for workers (168/40 = 4.2, a value rounded to 5). IEEE-C95.1, 1999 Edition [B70] maintained the original 10x safety factor for people in “controlled environments” (upper tier). (A “controlled environment” is an environment requiring RF exposure controls in contrast to an “uncontrolled environment” in which no controls are judged to be necessary.).

With the advent of more precise and high resolution dosimetry from experiments in animals and human beings, it became clear that peak to average SAR ratios during RF exposures are often of the order of 100:1 (Bernardi et al. [R1109]). This insight suggests that existing spatial peak limits derived under the previous rationale using a 20:1 ratio might have been set significantly higher. Thus, new dosimetric data provide additional evidence that the standard is conservative with respect to the spatial peak limits.

Further, contemporary dosimetric and state-of-the-art thermophysiological modeling that incorporates FDTD and realistic human and animal models (Mason et al. [B86]), (Bernardi et al. [R1109]) has shown that earlier experiments, from which SAR was derived from simplistic simulations using prolate spheroids, may have underestimated values by 2 times or more (Durney et al. [B34]). Such findings could imply that safety factors assumed for BRs based upon these data might have been half of what was initially thought (ANSI [B6]), (NCRP [B95]). However, the lack of credible scientific and medical reports showing adverse health effects from RF exposure at or below similar occupational exposure limits in past standards lends support to the protective nature of these limits.

A topic of extensive discussion during preparation of this revision was the data for children relating to WBA SARs in the 2–3 GHz range (Dimbylow [R1085]). These data, based on computational modeling, indicate that the BRs for children may be exceeded, i.e., the safety factor would be less than 50 in the 2–3 GHz range. The Committee's discussions focused on the already inherently conservative BRs and whether there was a need to change these to accommodate the recent dosimetric data. For example, the NRPB, when considering the implications of the same dosimetry data on possible modifications of the ICNIRP guidelines, concluded that "…given the uncertainties in the science, there appears to be neither scientific justification nor, considering harmonization of approaches to exposure guidelines, any practical merit in proposing new restrictions that are close to those of ICNIRP but differ from them" (NRPB [B102]). Despite similar arguments, this discussion resulted in the consensus within the Committee to change the limits in the lower tier to preserve the 50-fold safety factor.

Finally, the Committee understands that while safety factors have historically been defined in terms of SAR reduction factors, they may also be characterized by the degree to which they limit any temperature elevations in the body as a whole or in specific organs or tissues (Bernardi et al. [R1109]). In summary, the MPEs in this revised standard are derived from prolate spheroidal models as in ANSI C95.1-1982 [B6]. Within the scientific uncertainties associated with the complex subject of RF dosimetry, the MPEs represent reasonable estimates of exposure values that will yield SARs that do not exceed the BRs recommended in this standard. However, it is important to recognize the crucial role of deep body and tissue temperatures in evaluating the significance of RF exposures and appreciate that future revisions of this standard are likely to focus more on local tissue temperature limitations rather than ratios of peak to WBA SARs or other similar dosimetric constructs.

C.7.7 Exposure to electric fields, person not in reach of grounded objects

When an exposed individual is not within reach of a grounded conducting object, such as with a worker in an insulated bucket, the maximum exposure limits in Table 4 may not apply. In such cases, the magnitude of contact current and spark discharges will be determined by the potential difference between the individual and the touched object, and their capacitances. This standard specifies adherence to the limits of Table 4 for the general public. However, the limits of Table 4 may be exceeded in controlled environments in which workers are not within reach of grounded conducting objects. This standard does not provide a specific recommendation at this time for this situation owing to the lack of information (and research) on this issue. Moreover, there have been no definitive studies on the RF current and RF voltage at low frequencies (e.g., <10 MHz) induced and conducted in the metallic infrastructure (pipes, wires, towers, etc.) common in modern society. Therefore judgment on potential contact currents from the metallic infrastructure will require studies separate from measurement of free-space radiation fields.

C.7.8 Adverse environmental conditions

C.7.8.1 Zones of physiological response

Environmental engineers characterize the thermal environment in terms of operative temperature (T_0), which is defined as the average of the mean radiant and ambient air temperatures, weighted by their respective heat transfer coefficients. In the operative temperature range from 23 to 27 °C for normally

clothed (0.6 clo), sedentary people, there is no body cooling or heating and no increase in evaporative heat loss. In this zone, each person has a neutral temperature where the environment feels neither hot nor cold. If the ambient temperature exceeds the upper limit of the operative temperature range, increases in blood flow occur to maintain a constant core temperature. An RF source in the environment can contribute to the thermal load on the body in different amounts, depending on the frequency, field strength, distance from the person, and many other variables. If, in spite of greatly increased blood flow, the core temperature begins to rise above 37 °C, the second line of defense, regulatory sweating, is mobilized to provide evaporative cooling of the skin. As long as evaporative cooling maintains the required heat loss, the body is in the zone of evaporative regulation. However, any environmental factor that affects the evaporation of water from the skin affects thermoregulation. For example, increased ambient water vapor pressure, reduced air movement, and added clothing all affect the upper limit of evaporative heat loss. When this cooling is inadequate, the person is in the zone of body heating.

C.7.8.2 Environmental parameters

Psychrometrics deals with thermodynamic properties of moist air and uses these properties to analyze the thermal environment. Several of the parameters used to describe the thermal environment are psychrometric and include air temperature, wet-bulb temperature, dew-point temperature, water vapor pressure, total atmospheric pressure, relative humidity, and humidity ratio. Two important parameters that can be measured are air velocity and mean radiant temperature. The most important calculated parameter is mean radiant temperature, a key variable in making thermal calculations for the human body. The mean radiant temperature, T_r, is the uniform blackbody surface temperature with which a person (also assumed to be a blackbody) exchanges the same heat by radiation R as in the actual environment. The operative temperature, T_o is the uniform temperature of an imaginary enclosure with which a person exchanges the same dry heat by radiation and convection ($R + C$) as in the actual environment. Another definition of T_o is an average of T_r and T_a (where T_a is the ambient temperature) weighted by their respective transfer coefficients. Details of all these environmental parameters and how they are used may be found in any "ASHRAE Handbook—Fundamentals" (cf. the 1993 Edition [B13]).

C.7.8.2.1 Empirical indices

There are two important empirical indices that appear in psychrometric charts. Effective Temperature (ET or T_{eff}) has been the best known and most widely used thermal index. It combines the effect of dry-bulb and wet-bulb temperatures with air velocity to yield equal sensations of warmth or cold. This scale overemphasizes the effect of humidity in cooler and neutral conditions, underemphasizes its effect in warm conditions, and fails to account for air velocity in hot-humid conditions. The humid operative temperature T_{oh} for a subject wearing 1 clo of insulation coincides closely with the ET scale for heat loss by sweating. The second important empirical index is Wet-Bulb Globe Temperature (WBGT), which is used as a weighted average of the dry-bulb, a naturally convected wet-bulb and globe temperature. This index includes the combined effect of low temperature radiant heat, solar radiation, and air movement (see C.7.8.2.3).

C.7.8.2.2 The effective temperature scale (ET*)

The revised Effective Temperature (ET*) is the dry-bulb temperature of a uniform enclosure at 50% RH in which people have the same net heat exchange by radiation, convection, and evaporation as they do in varying humidity of the test environment. The ET* scale assumes clothing at 0.6 clo, air movement (still) at 0.2 m/s, a time of exposure 1 h, and a sedentary activity level (≈1 met; 58.2 W/m^2). The varying zones of physiological regulation for this standard combination are shown in the accompanying psychrometric chart (see Figure C.9). Thermal neutrality and comfort occur for sedentary subjects when regulatory sweating is zero and when the residual skin wettedness w is near 0.06. The upper limit of regulation occurs when w ≈ 1.0.

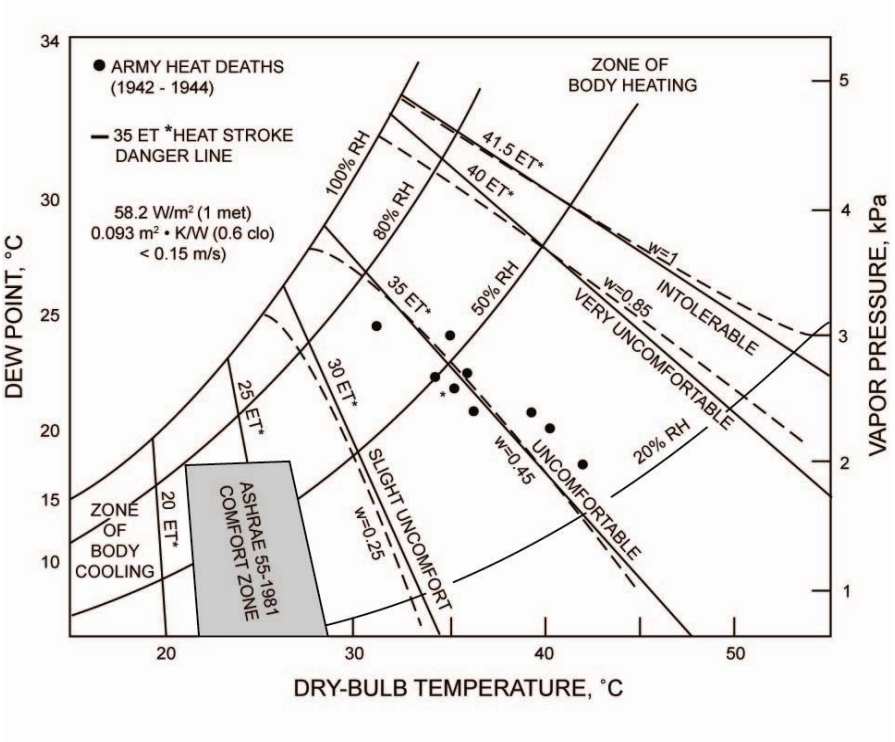

Figure C.9—A standard psychrometric chart that delineates the zone of thermal comfort (shaded), zones of body heating and cooling in terms of dew point, ambient vapor pressure and dry-bulb temperature. ET* defines the limits of thermal comfort (ASHRAE [B13]).

In the psychrometric chart, for the same standard conditions, isotherms have been drawn for 20 ET* in the zone of body cooling, 23.5 ET* for the threshold line of neutrality, 25 ET* as comfortable, 35 ET* as uncomfortable, 40 ET* as very uncomfortable, and 45 ET* as the limit where evaporative regulation fails. Above the 41 ET* level, body heating increases. The chart also plots dew point temperature and ambient vapor pressure against dry-bulb temperature. The ASHRAE comfort zone lies between ET* 22 °C and ET* 27 °C. The danger line for heat stroke roughly coincides with ET* 34 to 36 °C for w = 0.40 to 0.50. ET* loci are lines of constant physiological strain on the human thermoregulatory system and correspond to constant levels of thermal discomfort. The heat stroke deaths noted near the ET* 35 to 36 °C are from data that are based on military files for U.S. soldiers assigned to sedentary duties in Midwest Army camps. These data closely parallel the physiological strain in active, healthy men with prolonged sweating at $w = 0.5$.

C.7.8.2.3 Wet bulb globe temperature (WBGT)

The WBGT is an environmental heat stress index that combined dry-bulb temperature T_{db}, a naturally ventilated (not aspirated) wet-bulb temperature T_{nwb}, and black globe temperature T_g, according to the relation:

$$WBGT = 0.7\ T_{nwb} + 0.2\ T_g + 0.1\ T_{db}$$

This form of the equation is usually used where solar radiation is present. The naturally ventilated wet-bulb thermometer is left exposed to the sunlight, but the air temperature T_a sensor is left shaded. In enclosed environments, this equation is simplified by dropping the T_a term and using a 0.3 weighting factor for T_g.

The black globe thermometer is responsive to air temperature, mean radiant temperature, and air movement, while the naturally ventilated wet-bulb thermometer responds to air humidity, air movement, radiant

temperature, and air temperature. Thus, WBGT is a function of all four environmental factors affecting human environmental heat stress.

The WBGT index is widely used for estimating the heat stress potential of industrial environments. In the United States, NIOSH developed a criteria document for a heat-stress limiting standard (see NIOSH [B98]). Figure C.10 is a graphical summary of the permissible heat exposure limits for both acclimatized and unacclimatized workers. These limits are expressed as working time per hour as specified for various WBGT levels. The values in the figure apply to normal permeable clothing (0.6 clo) and have to be adjusted if workers wear heavy or partly vapor permeable clothing, e.g., RF protective suits. Persons wearing chem-bio clothing or body armor require an upward adjustment in WBGT of 6 °C to compensate for reduced evaporative cooling.

The concept of the Heat Stress Index (HSI) was originally proposed by Belding and Hatch [B17]. This rational index (HSI) is the ratio of the total evaporative heat loss E_{sk} required for thermal equilibrium (the sum of metabolism plus dry heat load) to the maximum evaporative heat load E_{max} possible for the environment, multiplied by 100, for steady-state conditions and with skin temperature held constant at 35 °C. The ratio E_{sk}/E_{max} equals skin wettedness. When HSI >100, body heating occurs; when HSI <0, body cooling occurs. Belding and Hatch [B17] limited E_{max} to 700 W/m², which corresponds to a sweat rate of ~280 mg/(s × m²). When skin temperature is constant, loci of constant HSI coincide with lines of constant ET* on a psychrometric chart. Table C.6 describes physiological factors associated with HSI values.

Table C.6—Evaluation of Heat Stress Index

Heat Stress Index	Physiological and Hygienic Implications of 8-h Exposures to Various Heat Stresses
0	No thermal response
10 20 30	Mild to moderate heat strain. If job involves higher intellectual functions, dexterity, or alertness, subtle to substantial decrements in performance may be expected. In performing heavy physical work, little decrement is expected, unless ability of individuals to perform such work under no thermal stress is marginal.
40 50 60	Severe heat strain involving a threat to health unless men are physically fit. Break-in period required for men not previously acclimatized. Some decrement in performance of physical work is to be expected. Medical selection of personnel desirable, because these conditions are unsuitable for those with cardiovascular or respiratory impairment or with chronic dermatitis. These working conditions are also unsuitable for activities requiring sustained mental effort.
70 80 90	Very severe heat strain. Only a small percentage of the population may be expected to qualify for this work. Personnel should be selected: a) by trial on the job (after acclimatization), and b) by medical examination. Special measures are needed to assure adequate water and salt intake. Amelioration of working conditions by any feasible means is highly desirable, and may be expected to decrease the health hazard while increasing job efficiency. Slight "indisposition," which in most jobs would be insufficient to affect performance, may render workers unfit for this exposure.
100	The maximum strain tolerated daily by fit, acclimatized young men.

C.7.9 High work loads

The WBGT index may also be used to predict the permissible heat exposure limits as shown in Figure C.10 for different continuous and intermittent work loads imposed upon a worker. The National Institute of Occupational Safety and Health (NIOSH) developed a criteria document for the limitation of heat stress in workers (NIOSH [B98]).

Table C.7 provides ceiling limits and recommended alert limits for heat unacclimatized workers (standard mass) for 5 levels of metabolic heat production and 2 clo values (normal permeable clothing = 0.6 clo, and chem-bio protective clothing or an anti-G suit = 2.0 clo). The table demonstrates clearly the effects of insulation on human heat tolerance and the role played by increased metabolic heat production.

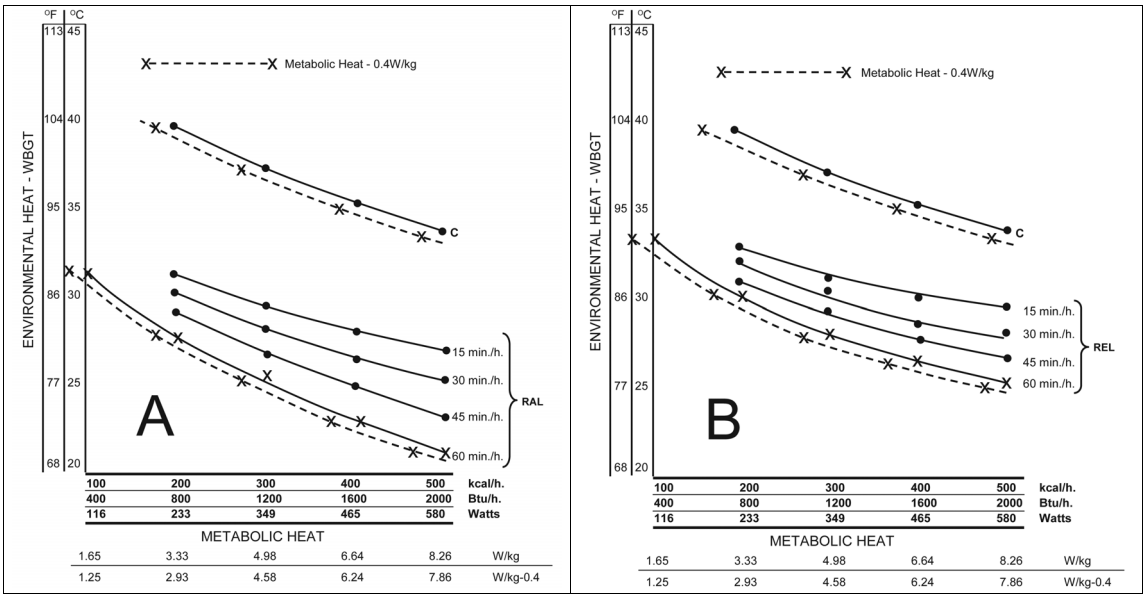

Figure C.10—NIOSH recommended heat-stress alert limits for unacclimatized workers (Panel A) and exposure limits for heat-acclimatized workers (Panel B) as modified to include the SAR criterion of 0.4 W/kg adopted for many RF exposure guidelines for humans (dashed lines). C = ceiling limit, RAL = recommended alert limit, REL = recommended exposure limit. Recommendations are for "standard workers," i.e., 70 kg mass and 1.8 m^2 surface area of the body. Figures reproduced from NIOSH [B98].

Table C.7—NIOSH recommended heat stress alert and ceiling limits for workers[a] who are unacclimatized to heat

		0.6 *clo*		2.0 *clo*	
M_{max} (W/kg)	met	Ceiling (°C)	Alert (°C)	Ceiling (°C)	Alert (°C)
1.65	1.18				
3.33	2.38	39.0	27.5	33.0	21.5
4.98	3.56	37.5	25.0	31.5	19.0
6.64	4.74	35.0	23.0	29.0	17.0
8.28	5.91	33.0	21.5	27.0	16.5
Data from NIOSH Publication 86-113, 1986 [B98].					
[a]Standard worker of 70 kg body mass and 1.8 m² body surface area.					

C.7.10 Presence of medical devices or metallic implants

Whenever an RF field impinges on a metallic object, re-radiated fields are produced around it. This phenomenon can redistribute the energy of the incident field to produce peak SAR concentrations and elevated temperatures in tissues adjacent to certain parts of the object. For example, in some industrial accidents where very high RF fields were involved, the only tissue damages noted were skin burns around wrist watches and rings. Peak SAR concentrations can also occur around metallic objects implanted *inside* the body, such as orthopedic plates, screws, wires and pins.

In general, the peak SAR concentrations induced around metallic objects that are carried on or within the body are relatively modest and would not be expected to cause any harmful tissue temperature rise for RF exposures at the MPE. Determining the exact impact of a metallic implant on localized RF tissue heating would for many cases require complex electromagnetic and thermal modeling, which is normally beyond the capabilities of individuals or organizations seeking to show or enforce compliance with this standard. Research is currently underway to develop simple guidelines for assessing the impact of implants, but until this information is available, the following advice may provide some useful guidance:

1) The frequency of exposure, the shape of the implant and its orientation with respect to the polarization of the *in situ* field will all affect SAR distributions around the implant.
2) Linear implants that are oriented parallel to the *in situ* E-field produce resonant field enhancements around their tips when their length is around one third of the field wavelength in the tissue.
3) A loop shaped metal implant which is oriented normal to the *in situ* H field may produce enhanced SAR in any gap in the loop.
4) Metal plates screwed on to bones that lie directly beneath the skin may enhance SAR in the skin at microwave frequencies due to constructive interference.
5) The re-radiated fields around an implant tend to decay very quickly in a lossy dielectric tissue environment.
6) Field enhancements can occur around any sharp point in the implant, though these are often so localized that their influence is not noticeable in a 10 g averaging mass.
7) An implant is a passive re-radiator, and in itself cannot *create* additional RF energy absorption. Thus the overall RF heating in the vicinity of the implant will generally remain about the same. One possible exception to this rule is the case of a large implant in one leg (e.g., a metal rod in

the tibia), which by providing a lower impedance conductance path diverts additional current flow to that leg for exposure frequencies around and below whole body resonance frequencies.

8) The potential for excessive localized SARs around an implant is only realistic in parts of the body where *in situ* fields are already relatively high. Thus implants located in parts of the body which are relatively well shielded will not be problematic.

9) From a thermal perspective, the implant may act as a heat sink. Temperature variations around the localized parts of the implant due to SAR enhancements will tend to be equalized by heat conduction through the implant. Furthermore, by displacing blood perfused tissue, the metallic implant may actually reduce the surrounding tissue temperature.

10) Some implants are located in a thermal environment where efficient heat transfer mechanisms will greatly mitigate any localized heating around parts of the implant. For example, the temperature of an arterial stent is strongly controlled by the convective heat transfer of the arterial blood flow passing through it. Metal plates located close to the skin (e.g., plates on the outside of the cranium) are another example.

11) Metal dental fillings are not normally regarded as problematic, since any localized heating associated with exposures below the MPE would be trivial compared with the other thermal loads routinely experienced in the mouth, such as hot coffee.

Another concern resulting from RF exposure is electromagnetic interference with the operation of active implantable medical devices (such as implantable pacemakers, implantable defibrillators, implantable neurostimulators and infusion pumps, etc.). Sufficiently high electromagnetic fields and/or modulations in the bandpass of these devices may inappropriately interfere with their intended operation. While laboratory studies demonstrate that EMI effects are possible under test conditions, verified reports of significant EMI appear to be uncommon in real practice. Recommendations from the US FDA CDRH [B131], Health Canada [B54], the UK MHRA [B132], and the Japanese Ministry of Public Management, Home Affairs, Posts and Telecommunications [B77] suggest keeping a minimal separation distance between mobile phones and implantable cardiac devices of between 15 to 22 cm, but contain no specific restrictions and make no further recommendations. Any doubts about the susceptibility of such medical electronic devices should be referred back to the patient's medical practitioner and/or device manufacturer.

C.7.11 Influences of medications

Drugs can influence the effects of RF exposure in two ways: 1) by directly affecting thermal regulation, and 2) by decreasing awareness of being exposed. Many drugs do both. Drugs, known as pyretics, actually cause an increase in body temperature, resulting in a feverish condition. This would add to the overall heat load on a person being over exposed to RF energy, and if sufficiently intense, there is the potential that the drug alone would lead to increased tissue damage. Any increase in temperature could be additive. Other drugs, such as opiates and some hormones are capable of raising the body temperature, but to a lesser degree than the pyretics. Alcohol does not cause a temperature elevation directly, but does diminish the body's ability to regulate body temperature. Many drugs affect alertness and, therefore, can interfere with one's ability to sense the heating resulting from being overexposed to an RF field. These drugs include, but are not necessarily limited to, anesthetics, antihistamines, sedatives, alcohol, tranquilizers, and many psychoactive drugs.

It must be noted that no adverse effect of RF exposure to individuals under the influence of any of the above drugs would be possible unless the RF exposure was significantly above the MPE. At levels at or below the MPE, RF fields would not adversely affect any individual whether or not they use such drugs.

C.7.12 Pregnancy

A question that needs to be addressed concerns the potential for harm if a pregnant woman were to be exposed to RF energy at the higher level specified in this standard, i.e., the controlled environment limit of 0.4 W/kg. Consideration of this question clearly must be related to the frequency to which the individual is

exposed, with secondary considerations related to exposure characteristics. The depth of penetration, and whether or not RF energy even reaches the fetus or embryo, is directly related to the frequency. The exposure characteristics include field strength, near vs. far field, potential for regions of peak SAR (electrical hotspots), ambient conditions, workload, and possibly the stage of pregnancy. The following considerations, many of which are based on geometry and/or physiology, are considered relevant to the question.

Frequency is clearly the most important variable because it relates to depth of penetration of the RF energy below the skin surface. RF energy absorption can be characterized with reasonable accuracy in a homogeneous, planar tissue slab of known permittivity. However, a human body is highly irregular in shape, size, and composition and thus RF energy will be absorbed in a highly non-uniform fashion, even for the ideal case of exposure to a uniform plane wave. Whole-body exposures in a uniform far field are taken as worst case situations: consideration is given first to frequencies above 2 GHz and then to frequencies below 2 GHz. As is well documented, for exposures of adult humans at frequencies above 2 GHz, the predominant energy absorption is almost totally within 2 cm or less from the skin surface, and only those surfaces that are directly exposed will receive measurable levels of RF energy. At the controlled environment level in IEEE Std C95.1, 1999 Edition [B70], even at millimeter wave frequencies (where the averaging time is reduced), surface heating will be minimal. This is true for all persons, including pregnant women. Even if the abdomen of a pregnant woman close to full term were exposed directly to RF energy at the controlled level (0.4 W/kg) at frequencies above 2 GHz, insufficient RF energy will be absorbed to cause a temperature increase in the fetus.

There are, however, no predictions provided specifically for women in various stages of pregnancy. Some information, probably of low utility, may be found in the differences between Figures 6.6 and 6.7 in the 4th edition of the RF Dosimetry Handbook [R901] (figures not reproduced here). These figures represent calculated planewave average SAR in prolate spheroidal models of an “average woman” for whom the resonance peaks in E- and H-polarizations are greater than for a “large woman.” It is significant that, apart from anecdotal data and a single modeling study of a pregnant woman (Fleming and Joyner [R1068]) (see B.7.2), no controlled laboratory data of human beings exposed to RF energy at or near their resonant frequencies have been available to assess the utility of these MPEs.

The results of a recent study (Adair et al. [R1102]) are reassuring with regard to the ability of human volunteers to maintain a normal body temperature during controlled 100-MHz CW exposures of the whole body at field strengths up to 8 times the upper tier MPE in this standard. A frequency of 100 MHz is close to resonance for seated human adults. Seven subject volunteers, including one woman, were seated 2.5 m in front of a dipole antenna within an anechoic chamber. Dosimetry was conducted on a human model to determine both regional and whole-body SAR.

Each subject served in 12 test sessions during which the ambient temperature T_a was controlled at one of three levels (24, 28, and 31 °C). Three field strengths (40, 60, and 80 W/m²) were studied at each T_a in addition to T_a controls (no RF exposure). A standard protocol was always followed (30 min equilibration to T_a, 45-min RF or sham exposure, 10 min re-equilibration). Physiological responses of heat production and heat loss were measured continuously. These included core body temperature (measured in the esophagus at the level of the heart), skin temperatures at 6 sites, metabolic heat production, local sweat rate at 2 sites, and local skin blood flow at 3 or 4 sites. Because theoretical dosimetry indicated high RF energy absorption in the legs, ankle skin temperature was also measured. Derived measures included heart rate, respiration rate, and total body weight loss. Judgments of thermal sensation and thermal comfort were obtained 4 times during each test.

The results of the tests under each test condition, both for individual subjects and for group means, showed no change in metabolism. There was also little or no change in local skin temperatures, including those sites on the subject's back that were exposed directly. The temperature of the ankle skin rose up to 4 °C in some subjects at 80 W/m², especially during tests conducted at T_a = 31 °C. This increase was linearly related to power density for all T_a. During the 45-min period of RF exposure esophageal temperature changed little (ranging from 0.13 to 0.15 °C) due to the increased sweating and skin blood flow that were T_a dependent.

Since individual skin temperatures (except for the ankle) changed hardly at all, it is clear that the physiological heat loss responses of increased blood flow and sweating must have been stimulated by thermoreceptors deep in the body, not by those located in the skin. These results indicate that thermoregulation will proceed normally when humans are exposed to RF energy at close to the resonant frequency even though the individual may not sense the presence of the RF field. This observation and the fact that little change occurred in core body temperature even at levels 8 times the MPE for a controlled environment, indicate the improbability of a thermal hazard to either a pregnant woman or to the embryo/fetus when the mother is exposed within allowed limits. Other studies involving localized RF exposure of human volunteers to 450 and 2450 MHz at or above the controlled MPEs, also support this finding (Adair et al. [R639], [R660], [R782], [R792]).

It is important to discuss why the experiment with human volunteers exposed to 100 MHz was not conducted at levels as low as 10 W/m^2, the C95.1 limit for controlled environments (Adair et al. [R1102]). As originally planned, the experiment included an exposure level at twice the limit (20 W/m^2) but this level was not used upon finding minimal changes in physiological responses at four times the limit. Further, the data show that exposure at a level eight times the limit for controlled environments is essentially benign in terms of impaired thermoregulation. Thus, for women in the workplace, the C95.1 limit protects against increases in maternal body temperature that might otherwise lead to heat-induced abnormalities in the fetus. The demonstration in the literature that a threshold maternal temperature elevation to ~39 °C, a rise of ~2 °C above normal, is associated with a significant increase in the incidence of heat-induced defects in the human fetus (Edwards et al. [R1081]), supports the conclusion of absence of risk to the embryo/fetus upon exposure of the parent to RF energy at the MPE and basic restrictions of this standard.

As mentioned above, the study with human volunteers exposed at 100 MHz also addressed thermal sensation and thermal comfort, which could be important considerations for a pregnant woman. A growing deterioration in thermal comfort was evident, as was an awareness of increased sweating, at the higher exposure levels (6 and 8 times the controlled limit) in the warmest environment (31 ºC) (Adair et al. [R1102]). The results from this short-term exposure study indicate that the C95.1 RF exposure limits assure thermal comfort under almost all extremes of environmental conditions.

Regarding dosimetry, SARs have been calculated, using simple models of a pregnant woman, for exposures in the 80 to 1500 MHz frequency range. Exposure of the model at the upper tier BR (0.4 W/kg) resulted in SARs in the fetus greater than three times higher (0.27 versus 0.08 W/kg) than the BR for the lower tier (Fleming and Joyner [R1068]). Since these calculations have not been independently confirmed, extended to physiological-based models or validated in animal models, the results have not been used to derive the limits recommended in this standard. In the study with metal detectors, the SAR in a model based on a pregnant woman in the 34th gestational week and exposed to devices placed directly on the abdomen, was 60,000 times less than the limit of 0.08 W/kg (Kainz et al. [R1100]).

Subclause B.6 includes a summary of the human reproductive studies of workers exposed to electromagnetic fields emitted by VDTs, MRI devices, RF heat sealers, medical diathermy units and radar. The weight of the scientific evidence of these studies does not provide support for human reproductive effects occurring in workplaces having RF-emitting devices. To create the potential for RF-induced defects in the human fetus, the exposure level would have to be much greater than the adverse effect threshold of 4 W/kg or lower RF levels coupled with extreme ambient conditions (i.e., high temperature, high humidity and low air flow), where the result is a rise in body temperature of ~2 ºC above normal. In addition to the adequacy of the MPE in this RF exposure standard to protect against a 2 ºC rise, another safeguard protects workers against an increase in body temperature of this magnitude. The ACGIH (American Conference of Governmental Industrial Hygienists) limits body core temperature of unacclimatized workers to 38 ºC (ACGIH [B1]). Also, the results of animal studies are in good agreement with the human threshold (~2 ºC) for fetal defects. A maternal temperature increase of ~2 to 2.5 ºC was associated with abnormalities in the offspring of laboratory animals (Edwards et al. [R1081]). In summary, the basic restrictions in this standard protect against adverse effects for both pregnant women and the fetus.

C.7.13 Use of mobile telephones by children

Concern about the use of mobile phones by children was documented in the 2000 report of the Independent Expert Group on Mobile Phones (IEGMP) entitled "Mobile Phones and Health" [B73] and the NRPB report on "Mobile Phones and Health 2004" [B105]. The latter report stated that: "… children might be more vulnerable to any effects arising from the use of mobile phones because of their developing nervous system, the greater absorption of energy in the tissues of the head, and a longer lifetime of exposure." From the scientific point of view, there is no evidence to support the need for a special precautionary approach for children or adults. At the time, the IEGMP quotation reflected accurately the absence of published health effects studies in the RF database involving children as subjects. In this regard, the RF database is similar to most health effects databases for other physical and chemical agents. In the absence of data on children, risk assessments are based on studies of experimental animals that serve as surrogates for human exposure. For example, birth defects (terata) are investigated in offspring of pregnant animals exposed during gestation to chemical and physical agents. A review of the extensive RF database shows a number of studies involving RF exposure during gestation through young adulthood that are considered to be relevant to the use of mobile phones by children (B.6.1). Health endpoints in these studies included development, CNS structure and function including cognition, brain cancer, and teratogenesis. The IEGMP, however, in making its risk assessment regarding the use of mobile phones by children, did not demonstrate that it gave appropriate weight to this relevant literature on the biological effects of RF exposure on developing laboratory animals, particularly those studies that tested mobile phone signals.

The relevance of this literature is based on knowledge of the comparative development of the CNS in laboratory animals and human beings. All major brain structures in humans are also present in laboratory rodents and have somewhat similar functions. The sequence of brain development, in general, is comparable among species, although the timing is quite different. To different degrees, development of brain structures continues through early life, adolescence and young adulthood in primates, including human beings, and rodents (Rice and Barone [B113]). The database includes important long-term exposure studies of nonhuman primates in which the similarity of CNS development to that of humans is greater than that of rodents. In these studies, investigations of brain histology and neurobehavioral functions were evaluated following exposure *in utero* and during the first year of life. In order to emphasize studies of particular relevance for children's use of mobile phones, literature previously reviewed in B.6.1 is revisited here in the context of the IEGMP conclusions. Specifically, the RF literature addresses all three points cited by the IEGMP. The following discussion addresses each point in the following order: the developing nervous system, long-term exposure including lifetime exposure, and greater absorption of RF energy in the young.

C.7.13.1 Studies of RF exposure during nervous system development

Studies that have investigated the possibility of physical defects in the offspring of pregnant animals exposed to RF energy are important because the exposures occurred during the most sensitive *in utero* stages of CNS development and the results addressed the question of whether or not the head and brain developed normally. Some studies included almost continuous RF exposure throughout pregnancy.

Studies on possible teratogenic effects of RF exposure and other conditions causing heat stress in animal models have demonstrated that significant increases in the incidence of heat-induced abnormalities are observed at maternal temperature increases of approximately 2 to 2.5 ºC, mostly following exposures of tens of minutes up to one hour or so. Higher temperature elevations, of up to ~5 ºC, are effective at shorter exposure durations (Edwards et al. [R1081]). The effects observed included abnormalities of the head, which would be expected to have adverse effects on the CNS and later development (assuming that the defects did not prevent survival of the offspring). For example, high-intensity RF exposure (11 W/kg, whole-body average at 27.12 Hz) of the pregnant rat on day 9 of gestation caused encephalocele, microphthalmia and other defects in the head of fetus (Brown-Woodman et al. [R19]). By increasing the duration of RF exposure to elevate the maternal body temperature from 2.5 ºC (no abnormalities) to 5 ºC, the incidence of these defects increased. The authors noted that the teratogenicity of RF energy deposition is primarily related to hyperthermia because the RF-induced defects were similar to those obtained by heating rats on the same day of

gestation in a water bath (Brown-Woodman et al. [R19]). Two studies reported resorption effects in rats exposed to pulsed RF fields at 27.12 MHz and 2.8 W/kg (Brown-Woodman et al. [R18]) and to very low level CW RF fields (Tofani et al. [R129]). However, neither of these studies have been confirmed or replicated by an independent laboratory. Studies such as these, which are inconsistent with the weight of evidence indicating a thermal basis for teratogenesis in animals exposed to RF, are few in number.

In a series of six papers, teratogenesis and postnatal growth/neurobehavioral development in rats exposed to three frequencies were examined (Jensh et al. [R356], [R357], [R358], [R359]), (Jensh [R360], [R361]). Pregnant rats were exposed for about 20% of the total gestation period of 21 days. In the teratology studies at 3.6 W/kg (915 MHz), 3.6-5.2 W/kg (2450 MHz), and 7.3 W/kg (6000 MHz), no changes were observed in maternal body weight, resorptions, abnormality rate, litter size or fetal weight, with the exception of decreased fetal weight at 7.3 W/kg, well above the threshold for established adverse health effects (4 W/kg). Within four days of birth, four reflex tests were given (surface righting, air righting, auditory startle and visual placing). One physiological measure (eye opening) was observed. In addition, at 60 days of age, the rats were given six behavioral tests (shuttle box, water T-maze, open field, activity wheel, forelimb hanging and swimming). The endpoints examined were not affected after exposure at 3.6 W/kg (915 MHz). At a slightly higher SAR (3.6-5.2 W/kg at 2450 MHz), increased activity in the activity wheel and open field test was observed in the females (not in the males). Neither result in females was confirmed at 7.3 W/kg (6000 MHz); other changes were recorded at this SAR and frequency, i.e., increase in open field activity (males only), decreased endurance in water maze (females only), increased shuttle box activity (females only) and earlier eye opening. Other effects at 7.3 W/kg included decreased birth weight and postnatal growth to the fifth week of life. The results in these six papers are considered to be consistent with a threshold for neurobehavioral effects greater than 4 W/kg, the threshold for established adverse health effects. In a review of the six papers (Jensh [R646]), it was concluded that "…in the absence of a hyperthermic state, the microwave frequencies tested, which included frequencies used in cellular phones and microwave ovens, do not induce a consistent, significant increase in reproductive risk as assessed by classical morphologic and postnatal psychophysiologic parameters."

Following prenatal exposure or pre- plus postnatal exposure, 30- and 100-day-old rats were subjected to a neurobehavioral test battery, which included locomotor activity, startle to acoustic and air-puff stimuli, fore- and hind limb grip strength, negative geotaxis, reaction to thermal stimulation, and swimming endurance (Galvin et al. [R45]). The maximum fetal exposure was 4 W/kg (3 h/d from days 5–20 of gestation). The pre- plus postnatally exposed group had less swimming endurance at 30, but not 100, days. The only other behavioral effects, an increase in the air-puff startle response at 30 days of age and a decrease at 100 days of age, were limited to prenatally exposed females (not males). The fetus could have received up to 4 W/kg for 3 h/d from days 5–20 of gestation. After birth, the pups were exposed from 2–20 days of age at SARs ranging from 16.5 W/kg at 2 days of age to 5.5 W/kg at 20 days of age. Thus, these limited neurobehavioral results occurred in animals exposed at and above the threshold for established adverse health effects (4 W/kg). In the RF-exposed groups, the observation that the 30-day-old rats (males and females), but not 100-day-old rats, were heavier is not consistent with the weight of evidence in the RF database (Berman et al. [R228], [R538]), (Jensh [R360]), (Berman and Carter [R537]).

Young mice were evaluated for development on 1, 5, 10, 12, 15, and 17 days of age following *in utero* exposure at 16.5 W/kg for 100 min on days 6–17 of gestation. The tests used to determine differences in the developmental age of mice in the exposed and sham-exposed groups included body weight, brain weight, bone lengths, and urine concentrating ability. There were no changes except for lower body weight on day 1 and lower brain weight on days 10, 12, and 17 (Berman et al. [R538]). These changes, which are indicative of a delay in postnatal development, were observed at an exposure level more than four times the threshold for established adverse health effects (4 W/kg).

Rat brain development was investigated histologically at 15, 20, 30, and 40 days of age following prenatal and postnatal exposure (3 h/d, 2450 MHz) from day four of gestation to 40 days of age (except for two days) (Inouye et al. [R781]). The *in utero* exposure of 1.76 W/kg to the pregnant animals occurred on days 4–21 of gestation. In offspring aged 15–40 days, the brain SAR ranged from 19 to 9.5 W/kg. The brain development

markers were the cortical architecture of the cerebral cortex and hippocampal formation, the germinal layer along the lateral ventricles, myelination of corpus callosum, and the external germinal layer of the cerebellar cortex. In addition, in 40-day old rats, quantitative measurements of neurons were made, i.e., spine density of the pyramidal cells in cortex. Other endpoints included the density of the Purkinje cells and the extent of the Purkinje cell layer in the cerebellum. This extensive investigation of mammalian brain development following exposure of the rat prenatally- and postnatally to SARs almost five times greater than the threshold for established adverse health effects found no histological changes in the developing rat brain (Inouye et al. [R781]).

A transient decrease in Purkinje cells in the cerebellum in RF-exposed rats could not be confirmed in nonhuman primates (squirrel monkeys) by the same laboratory (Albert and Sherif [R299]), (Albert et al. [R300]). The primates were exposed at 2450 MHz and 3.4 W/kg (3 h/d, 5 days/week), from the 35th day of pregnancy until birth with exposure of the infants continuing until they were 9.5 months of age. No difference was found in body mass, brain weight, brain volume or total number and density of Purkinje cells in the cerebellum (Albert et al. [R300]) of the exposed animals compared with sham exposed animals.

In an investigation of the effects of RF exposure during most of the gestational period on the development of the mammalian brain in the 18-day old fetus, pregnant rats were exposed continuously (24 h/d, 7 days/week) from days 2–18 of gestation at 0.4 W/kg (2.45 GHz). No microncephalous was found in the exposed group and there was no change in fetal brain weight or DNA, RNA and protein content of the brain (Merritt et al. [R404]). The authors concluded that brain organogenesis was not affected by almost continuous exposure during the gestational period of CNS development at an SAR equal to the upper tier BR (0.4 W/kg).

Prenatal exposure of rats to mobile phone signals, at a level approximating the general public limit to fields from base stations of the GSM digital mobile-phone technology, had no effect on cognitive function in adulthood. The animals were exposed continuously during pregnancy at low SARs ranging from 0.0175–0.075 W/kg. The offspring were tested as adults (11–12 weeks of age) for learning deficits. No measurable cognitive deficits were observed (Bornhausen and Scheingraber [R746]).

C.7.13.2 Studies of other physiological changes possible after long-term RF exposure

C.7.13.2.1 CNS effects

In addition to the studies described above in which animals were exposed *in utero* and during early life for extended durations, there are several long-term exposure studies involving lifetime (chronic) 2-year exposures; some of these studies included prenatal exposure.

Histopathological analysis of the brain and other CNS tissues was a special focus of three lifetime RF exposure studies in rats, which included exposure of the animals during gestation (Adey et al. [R677], [R727]), (Anderson et al. [R1120]). In two long-term brain cancer studies, the heads of rats were exposed to RF levels chosen to simulate maximal exposure to the human head during use of a mobile phone (Adey et al. [R677], [R727]); the measured peak brain SAR ranged from 1.8–2.3 W/kg as the animal aged and gained weight. The mobile phone frequency was 836.55 MHz with North American Digital Cellular (NADC) TDMA modulation in one study and frequency modulation (FM) (also called “analog”) in the other. Some pregnant animals were treated with ethylnitrosourea (ENU) to induce CNS tumors in the offspring. RF exposure (2 h/d) began on gestational day 19 and continued until weaning at 21 days of age. Exposure (2 h/d, 4 days/week) resumed at 31 or 35 days of age and continued for 22 months. The study examined both spontaneous tumorigenicity in the CNS and the incidence of ENU-induced CNS tumors. In both studies, “lifetime exposure,” that is, mobile phone simulated exposure from late gestation through 24 months of age, did not increase the incidence of either spontaneous primary CNS tumors or ENU-induced CNS tumors. In the third study, animals were first exposed *in utero* (2 h/d, 7 days/week, 1.6 GHz) at 0.16 W/kg (fetal brain average) from gestational day 19 to 23 days of age. At 35 days of age, the exposure resumed at 0.16 and 1.6 W/kg (brain average) and continued for two years. At the end of the lifetime exposure, there was no evidence of increased number of tumors in any major organ or tissue, including the brain and CNS tissues (Anderson et al. [R1120]). The results of these

three long-term exposure studies provide no support for the hypothesis that the tested forms of RF energy act as a carcinogen or a cancer promoter in CNS tissues, including the brain, when RF exposure occurred during critical periods of CNS development in the fetus, as well as throughout young and adult life.

C.7.13.2.2 Blood-brain-barrier, body weight and other biological studies

Another lifetime study examined blood brain barrier permeability in mice exposed for 1 h/d, 5 days/week, for two years at four SAR levels (0.25, 1, 2, and 4 W/kg). RF exposure commenced in 8-week-old animals, an age that is at or near their reproductive age. At all SAR levels, the mobile telephone-type signal (900 MHz, GSM) produced no significant disruption to the integrity of the BBB (Finnie et al. [R851]). These results are consistent with the weight of evidence showing that changes in the BBB are induced by exposures above 4 W/kg causing significant elevation in brain temperature (D'Andrea et al. [R1089]) (see B.6.3). Thus, the function of the BBB to allow passage of the molecules necessary for metabolism but to protect the brain from foreign toxic substances should not be affected within the limits of internationally accepted standards.

A sensitive and reliable indicator of toxicity is body weight. Research has shown that fetal body weight is not affected at SARs below 4 W/kg even by almost continuous exposure during *in utero* development to RF fields in the 900-MHz range of mobile phones. For example, an investigation with 20-day rat fetuses following almost continuous 970-MHz exposure during gestation (22 h/d during days 1–19) showed a decrease (12%) in fetal body weight at 4.8 W/kg but no effect at 2.4 and 0.07 W/kg (Berman et al. [R228]). In a related study, no change in fetal weight was seen in 22-day rat fetuses following exposure of pregnant rats at 3.6 W/kg (915 MHz) for 6 h/d during days 1–21 of gestation (Jensh et al. [R356]). At a higher frequency (6000 MHz), an exposure of 7.3 W/kg for about 20% of the gestational period was sufficiently intense to decrease fetal body weight (Jensh [R360]). Another study reporting reduced weight of fetal rats after exposure at 2450 MHz and 6 W/kg for 100 min per day during the days 6–15 of gestation (Berman and Carter [R537]) supports the conclusion that this effect can be caused by SARs greater than the threshold for established adverse health effects (4 W/kg).

In a long-term study of primates, squirrel monkeys were exposed at 2450 MHz to three SARs (0.034, 0.34 and 3.4 W/kg) for 3 h/d, 5 days/week beginning the second trimester of pregnancy (Kaplan et al. [R363]). Mothers and offspring were exposed for an additional 6 months after parturition and the offspring were exposed for another 6 months. In the offspring, a wide array of endpoints were measured including growth rate, EEG, biochemistry (urinary epinephrine and norepinephrine and blood cortisol), hematology (lymphocyte counts), and five tests of behavioral development (righting, orienting, climb down, climb up and directed locomotion). No significant changes were found except for an effect on one behavioral test at the highest SAR (3.4 W/kg); however, there were very few animals in this group available for the behavioral test due to a high mortality rate. It is noted that the high mortality rate was not replicated by the same laboratory in a follow-up study (Kaplan et al. [R363]). Exposure *in utero* plus 12 months of exposure after birth at SARs less than (0.034 W/kg) the lower tier limit and near (3.4 W/kg) the adverse effect level did not affect neurobehavioral function of nonhuman primates (Kaplan et al. [R363]).

C.7.13.3 Question of possible greater RF energy absorption in young animals

The question of whether similar RF exposures result in more energy being absorbed in tissues of young animals compared with those of adults is moot when discussing the published literature because the SARs in fetal and young animals were measured or calculated and reported. The literature provides the SAR levels, including whole-body SARs in some studies and/or peak brain SARs in other studies, that are associated with either the reported effects or the absence of effects in fetal and young animals (as well as in exposures of adults).

The related dosimetric question of whether exposures to the head and brain tissues of children using a mobile telephone handset are significantly greater than those for an adult using the same handset has been addressed in a number of research papers, (Gandhi and Kang [R1126]) (Hadjem et al. [R1129]) (Martinez-

Burdalo et al. [R979]) (Schonborn et al. [R1127]) (Wang and Fujiwara [R1128]) (Gandhi et al. [R644]) (Anderson [R1103]) (Bit-Babik et al. [R1130]). The consensus from the more recent studies is that the size and shape of children's heads do not cause a significant difference in SAR compared with the adult for exposed tissues of the head.

Rather than using a comprehensive review of the literature in the RF database as described for the development of this standard, the Health Council of the Netherlands considered a different approach in assessing children's use of mobile phones. Their approach was based on whether or not developmental arguments could be found, i.e., is there reason to believe that the heads of children are more susceptible to the electromagnetic fields emitted by mobile telephones than those of adults? That report states that no major changes in head development occur after the second year of life that might point to a difference in electromagnetic susceptibility between children and adults (van Rongen et al. [R1123]).

C.7.13.4 Summary

This review identified many important laboratory animal studies that are relevant to possible health effects in children using mobile phones, or otherwise exposed to RF energy. The weight of evidence of these studies supports the conclusion that decreased birth weight, teratogenic effects, changes in brain histology, and effects on neurobehavioral function in laboratory animals exposed *in utero* and in early life, that is, exposure during the periods of CNS development, do not occur unless the RF exposure is >4 W/kg, resulting in a significant temperature increase above normal body temperature. The literature for the developing animal, as a surrogate for the developing human, does not provide support for the hypothesis that the developing or young person is more sensitive than adults to RF exposure. This conclusion is in agreement with the 2004 report from the Health Council of the Netherlands, which states that there is "…no reason for recommending limiting the use of mobile phones by children" (van Rongen et al. [R1123]). Compared with adults, the size and shape of the child's head do not cause a significant difference in SAR of exposed tissues of the head. As a final note, advice from the U.S. FDA [B42] includes the statement that "The scientific evidence does not show a danger to users of wireless phones, including children and teenagers." Thus, the FDA statement, the overall results of dosimetric studies of children versus adult heads, the conclusion that no major changes in head development occur after the second year of life that might point to a difference in electromagnetic susceptibility between children and adults, and an extensive review of the biological literature, are all in general agreement that the application of the precautionary approach to the use of mobile phones by children lacks scientific basis. Two recent studies have found no effect on RF exposure from mobile phones on cognitive function in children (Preece et al. [R1141]), (Haarala et al. [R1142]).

C.7.14 Macular degeneration

The question of whether or not a person suffering from macular degeneration would be at increased risk from a temperature increase from exposure where the local SAR is below the basic restriction for spatial peak-average SAR was considered. The etiology of macular degeneration is not established; the disease appears to be age-related and most likely has a genetic basis. There is no known causative effect for macular degeneration produced by temperature elevation. In fact, laser-induced temperature elevation is frequently used to treat the wet form of macular degeneration. Therefore, exposures below the spatial peak-average SAR of this standard (2 W/kg and 10 W/kg for the lower and upper tier, respectively) should not be considered problematic for the production of or worsening of macular degeneration.

Annex D

(informative)

Practical applications—examples

D.1 Introduction

Often there are situations where determining compliance with this standard is difficult and not always straightforward. This annex focuses on those portions of the standard that have traditionally been problematic for interpretation and implementation. However, this annex is not a substitute for the more detailed measurement guidance that can be found in other resources such as IEEE Std C95.3-2002.

Generally, determining compliance can be accomplished in the following two ways:

a) Theoretical analyses

b) Measurement

In most cases, these methods are complementary. Theoretical analyses should be done (when possible) prior to taking measurements. Usually theoretical analyses prove to be the most accurate (and conservative) approach in far field compliance evaluations. However, there are some situations where such analyses are not possible or are not an adequate or complete approach. For example, in near field situations (where fields may be non-uniform or high induced currents may be present) it is extremely difficult to determine analytically the levels that may be present. Also, measurement may be the only feasible method for assessing energized objects, determining contact current potentials, and characterizing environments with multiple sources.

The user of this standard should remember that this standard relates to permissible exposure, not emissions. As such, analysis and/or measurement results that indicate levels in excess of the MPEs do not necessarily imply that persons will actually be exposed to such levels. This can depend on the exact environment and on the radiation protection program, if one is associated with that particular RF environment. See IEEE Std C95.7-2005 for recommended guidelines for establishing RF safety programs.

D.1.1 Characterizing exposure to non-uniform fields

D.1.1.1 Practical constraints

Exposure to non-uniform fields may be characterized as exposure to fields over a specified volume of space, in which there exists a highly localized area of relatively intense RF energy. Non-uniform fields may be due to 1) the superposition of RF fields caused by reflections that result in localized standing waves; 2) narrow beams produced by highly directional antennas or radiating structures; or 3) the near field region of a radiating structure. In all cases, the fields may be characterized by very rapid changes in field strength with distance. Localized exposures result from exposure to non-uniform fields leading to non-uniform SAR distributions with high spatial peak SAR values (non-uniform energy absorption). Localized exposures can also result from the exposure to a non-uniform field, with the exposure dependent on the size and orientation of the person in the field. Non-uniform fields can result in localized exposures in excess of the MPE.

In the reactive near-field region, there is no simple relationship between the E and H fields (the impedance (E/H) will differ from 377 ohms). The linear decrease in field strength with distance and the decrease in power density with distance squared that is characteristic of the far field do not apply in the near field region.

The reactive near field contains stored RF energy rather than radiated RF energy and the fields often vary rapidly with distance. Issues that should be considered are:

a) **MPEs:** The MPEs are based on the assumption of uniform exposure and are expressed in terms of field strengths or plane-wave equivalent power density of the incident field, i.e., the electric and magnetic field strengths that correspond to a plane-wave field with the same values and uniformly distributed in planes transverse to the direction of propagation.

b) **Field perturbations:** Objects located near sources may strongly affect the nature of the fields. For example, placing a probe near a source or standing near a source while carrying out measurements may change the characteristics of the fields considerably.

Measurements to determine adherence to the recommended MPEs should take into account the fact that a number of factors influence the response of measurement probes to the field. These factors include:

a) Variation of probe impedance with proximity to nearby reflective surfaces;

b) Capacitive coupling between the probe and the field source; and

c) Non-uniform illumination of the sensing elements that make up the probe.

Maintaining adequate separation distance between the probe elements and the field source can eliminate the influence of each of these factors, which otherwise could result in erroneous field strength measurements. Accordingly, measurements should be made at a distance no closer than three probe-diameters between the center of the probe and any object, or 20 cm—whichever is greater. When assessing whole-body-average exposure, a minimum measurement distance of 3 probe diameters or 20 cm, which ever is greater, is recommended in IEEE Std C95.3-2002.

For practical measures of compliance with the standard, the average of a series of ten field strength measurements performed in a vertical line with uniform spacing starting from about 20 cm above ground level up to a height of 2 m is deemed sufficient (spatial averaging). Additional field strength data obtained at smaller spacing than 20 cm, e.g., as obtained through the use of data logging or spatial averaging equipment, are acceptable and will provide more detail on the spatial distribution of the fields. However, the measurement spacing should be held constant so as to obtain a true spatial average.

If the results of the measured spatial average do not exceed the MPEs, then each measurement value must also be compared to the criteria for limiting spatial peak values of field strength. If any of the individual spatial values exceed the calculated spatial peak value, then the exposure does not comply with the MPE. However, non compliance with the spatial average or spatial peak values may be overturned by demonstrating compliance with the basic restrictions using other analytical methods (e.g., dosimetry models). These are typically much more complex to obtain: so it is usually easier to first test for compliance by measurement.

D.1.1.2 Applying the peak power density limits

As indicated in 4.4, the peak power density limits apply to exposures to pulsed RF fields at frequencies in the range of 100 kHz to 300 GHz. The limits are as follows:

a) For exposures to pulsed RF fields, in the range of 100 kHz to 300 GHz, the peak (temporal) value of the MPE in terms of the E field is 100 kV/m.

b) For exposures to pulsed RF fields in the range of 100 kHz to 300 GHz, peak pulse power densities are limited only by the use of time averaging and the limit on peak E field, with the following exception: the total incident energy density during any one-tenth second period within the averaging time shall not exceed one-fifth of the total energy density permitted during the entire averaging time for a continuous field (1/5 of 144 J/kg), i.e.,

$$\sum_{0}^{0.1s} (S_{\mathrm{pk}} \times \tau) \le \frac{MPE_{\mathrm{avg}} \times T_{avg}}{5} \le 28.8 \mathrm{J/kg}$$

where

τ is the pulse width
MPE is the plane-wave equivalent power density given in column 4 of Table 8 and Table 9, and
T_{avg} is the averaging time given in column 5 of Table 8 and Table 9.

A maximum of five pulses with pulse durations less than 100 ms is permitted during any period equal to the averaging time. If there are more than five pulses during the averaging time, or if the pulse duration is more than 100 ms, normal averaging time calculations apply.

D.1.1.3 Examples

D.1.1.3.1 Extremely low pulse repetition rate source

Determine whether or not the peak-power limits for the controlled environment are exceeded for a radar with the following characteristics:

Frequency	10 GHz
pulse width (T)	10 ms
pulse repetition frequency (prf)	0.004 pulses/s (1 pulse every 250 s)
peak RF power density	1 200 000 W/m^2

Solution: In order to comply with the peak power limits, both the peak electric field criterion and the energy density criterion must be satisfied.

Peak electric-field strength criterion:

$$E_{peak} = (377 \times S)^{1/2} = (377 \times 1\ 200\ 000)^{1/2} = 21\ 270 \text{ V/m} < 100 \text{ kV/m}$$

Energy density criterion:

$$(1/5)[MPE_{tavg}\ (\text{W/m}^2)\quad (T_{avg})] = (1/5)(100\ \text{W/m}^2\quad 360\ \text{s}) = 7200\ \text{J/m}^2$$

In order to comply, the exposure must meet both the energy density and the peak electric field criteria.

But,

$$\int S(t)\,dt = (1\ 200\ 000\ \text{W/m}^2)(10\ \text{ms}) = 12\ 000\ \text{J/m}^2 > 7200\ \text{J/m}^2$$

Although the exposure meets the peak electric-field strength criterion it does not meet the energy density criterion and, therefore, does not meet the peak power limitations for the controlled environment.

D.1.1.3.2 Conventional radar

Determine whether or not the peak-power limits for the controlled environment are exceeded for a radar with the following characteristics:

pulse width (T_w)	10 s
pulse repetition frequency (prf)	1200 pulses/s (Hz)
beam width (θ)	2 degrees
antenna rotation (360°)	6 revolutions/min (r/min)
peak power density	300 000 W/m^2
Frequency	9.4 GHz

Solution: The pulse width (T_{br}) of a single burst of RF pulses (associated with rotation of the beam) is

T_{br} = (60 s/6 revolutions) (2°/360°) = 55.6 ms

The 55.6 ms exposure (while the beam sweeps by) will consist of approximately (0.0556 s)(1200 pulses/s) = 66.7 pulses of RF energy, each pulse lasting 10 μs. However, since there will be more than five 55.6 ms bursts during any 6 min interval, normal averaging-time rules apply, i.e.,

S_{avg} = (300 000 W/m^2)(1200 Hz)(10 μs)(2°/360°) = 20 W/m^2

<MPE (100 W/m^2)

Therefore, the system complies with this criterion.

D.1.1.3.3 Non-sinusoidal waveform

Previously, a pulse of microwave energy was considered. In each example, the pulse width was significantly longer than the time between each complete oscillation of the microwave frequency. In this section, an example of how to assess compliance when the pulse is a non-sinusoidal waveform is provided.

For example, consider a square wave pulse with a fundamental frequency of 10 kHz. The phase duration t_p (defined as the time between zero crossings of a waveform having zero mean) of this pulse is 0.05 milliseconds (ms). To assess compliance, it is necessary to first test for compliance with the RMS MPE. This is essentially no different than the previous example. Then test for compliance with either the peak field restriction or the Fourier component restriction.

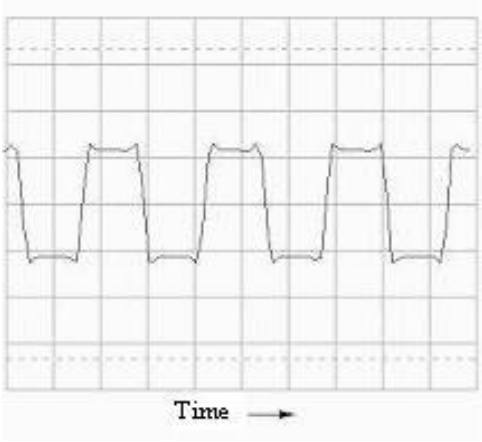

Figure D.1—Square wave in the time domain

Peak field. The external B and E field strengths are limited by the pulse rise time (since real pulses are never square) expressed as the time rate of change of the B or E field (dB/dt or dE/dt). For this simple case, the frequency (f) is 10 kHz, so the applicable MPE is 0.165 mT (rms) (from Table 2) for the B field and 1.842 kV/m (rms) for E field.

$$\dot{B}_p = \sqrt{2} MPE_B (2\pi f)$$

dB/dt (peak) = $\sqrt{2} \times 0.000615$ T×2×π×10,000 Hz = 54.7 T/s

$$\dot{E}_p = \sqrt{2} MPE_E(2\pi f)$$

dE/dt (peak) = $\sqrt{2} \times 1842$ kV/m×2×π×10,000 Hz = 163 000 kV/m/s

Fourier component. The B or E field strength of each Fourier component of the square wave is divided by the MPE at each component frequency and summed. This summation must be less than unity to comply with this restriction. In this example, the fundamental frequency component is 10 kHz, the 3rd harmonic is 30 kHz, the 5th is 50 kHz, the 7th is 70 kHz, the 9th is 90 kHz, the 11th is 110 kHz, and the 13th is 130 kHz. Notice that the even harmonics of a square wave function are null. A spectrum analyzer may be employed to measure the field strength of each Fourier comment out to 5 MHz.

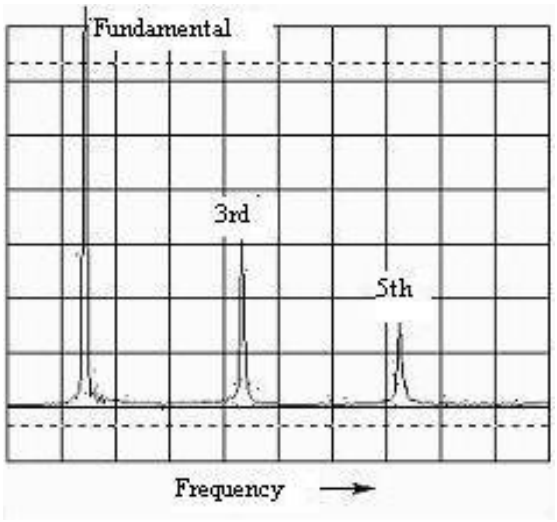

Figure D.2—Square wave in the frequency domain (Fourier spectrum)

A_0 = 100 A/m; A_3 = 50 A/m; A_5 = 10 A/m; A_7 = 5 A/m; A_9 = 1 A/m;
A_{11} = 0.5 A/m; A_{13} = 0.1 A/m

Also
MPE = 490 A/m from 10 kHz to 100 kHz
MPE = 445 A/m at 110 kHz
MPE = 377 A/m at 130 kHz

Therefore, since
100/490 + 50/490 + 10/490 + 5/490 + 1/490 + 0.5/445 + 0.1/377 = 0.33 <1,

the MPE is not exceeded.

D.1.1.4 27 MHz heat sealing application

Table 1—RF heat-sealing equipment typically operates in the 27 MHz ISM band. MPEs are obtained from Tables 8 and 9. Induced current measurements are especially important because of the relative proximity to the RF source compared with the wavelength of the fields (the free-space wavelength at 27 MHz is approximately 10 meters). From Annex C, Table C.5, the reactive near field is estimated to extend out 1.6 meters from the source. From Table 8, the MPE at 27 MHz is 68 V/m and 0.6 A/m for the electric field and magnetic field, respectively. A whole-body measurement at the operator location provided the following values (which are typical):

Table D.1—Measured electric and magnetic field strength at various anatomical positions of a heat-sealer operator

	Location	Electric field strength (E) (V/m)	$\|E\|^2$ $(V/m)^2$	Magnetic field strength (H) (A/m)	$\|H\|^2$ $(A/m)^2$
1.	Foot	22	484	0.08	0.0064
2.	Ankle	34	1156	0.23	0.0529
3.	Calf	47	2209	0.7	0.49
4.	Knee	58	3364	0.97	0.9409
5.	Thigh	69	4761	0.82	0.6724
6.	Groin	75	5625	0.59	0.3481
7.	Belly	81	6561	0.36	0.1296
8.	Chest	75	5625	0.14	0.0196
9.	Shoulder	66	4356	0.1	0.01
10.	Head	58	3364	0.1	0.01
	Average	61.2	3751	0.52	0.27

The whole-body average value is determined based on the square of the field strength for both electric and magnetic field components. The average electric field strength squared (3751 V^2/m^2) does not exceed the corresponding MPE of 4624 V^2/m^2. The average magnetic field strength squared (0.27 A^2/m^2) does not exceed the corresponding MPE of 0.36 A^2/m^2. In this controlled environment application, the squares of the peak values observed (81 V/m and 0.97 A/m) do not exceed 20 times the square of the MPEs, i.e.,

$81^2 < 20 \times 68^2$ (6561 <92,480)

$0.97^2 < 20 \times 0.6^2$ (0.94 <7.2)

Not withstanding the compliance with the MPE for the electric field, the 27 MHz data in Figure 1 indicate that for a field/MPE ratio greater than 16%, induced currents must be measured and for values beyond 8%, touch currents should be considered. In this example, the ratio (61.2/68) is 90%, indicating that both induced current and touch current measurements are also required.

D.1.1.5 Evaluating polarization dependent exposures

The exposure limits of this standard are conservative for several reasons, one of which is the assumption that all exposures are such that the incident electric field is polarized with the long axis of the body. This condition leads to maximum RF energy absorption but may not be realistic for a particular exposure scenario. While this standard provides no tables or charts that show how WBA SAR varies with polarization of the incident field, this information can be obtained from other sources (see Durney [B34]). It may be possible to assess compliance with the BRs of this standard through an evaluation of the WBA SAR that would be associated with the polarization of the exposure field, assuming that it is known. For example, in some cases, the principal exposure may be caused by an RF field that is not polarized with the long axis of the body and, hence, the resulting SAR may be substantially less than that value resulting from optimum polarization. If the particular exposure situation is such that it can be assured that non-optimum polarization exists during

the exposure, then RF fields that may exceed the MPEs specified in this standard may be applied after a careful analysis of the dosimetry using as a reference the data contained in Durney [B34].

D.2 Multi-frequency exposures (exposures to multiple sources)

D.2.1 Field strength and power density

When multiple sources are introduced into an environment, it becomes necessary to address the sources interdependently since each source will contribute some percentage of the MPE toward the total exposure at a fixed location. The sum of the ratios of the exposure from each source (expressed as a plane-wave equivalent power density) to the corresponding MPE for the frequency of each source is evaluated. The exposure complies with the MPE if the sum of the ratios is less than unity, i.e.,

$$\sum_{i-1}^{n} \frac{exposure}{MPE_i} < 1$$

NOTE—Although the MPEs in Table 8 and Table 9 are be expressed in terms of field strength (E and H) and power density, the exposures and the corresponding MPEs must be expressed in terms of power density in the above summation or in terms of the field strength squared.

Example: Measurements were made in a controlled environment at a point near several induction heaters (IH) and dielectric heaters (DH). The values shown in columns 2, 3, and 6 in the table below represent the measured frequency and the electric and magnetic field strengths as averaged over an area equivalent to the vertical cross section of an adult. $S_{\text{E-pwe}}$, $MPE_{\text{E-pwe}}$, $S_{\text{H-pwe}}$ and $MPE_{\text{H-pwe}}$ are the E- and H-field plane wave equivalent power densities and MPEs, respectively.

Table D.2—Results of measurements of electric and magnetic fields over the vertical cross section of an adult

Source (i)	f (MHz)	E (V/m)	$S_{\text{E-pwe}}$ (W/m^2)	$MPE_{\text{E-pwe}}$ (W/m^2)	H (A/m)	$S_{\text{H-pwe}}$ (W/m^2)	$MPE_{\text{H-pwe}}$ (W/m^2)	Duty-factor (%)
DH_1	27.5	90	21.5	11.9	0.1	03.8	13.2	20
DH_2	7.5	283	212	160	0.2	15.1	1,780	60
DH_3	3.5	592	930	735	0.4	60.3	8, 160	45
IH_1	0.4	15	0.6	1000	8.0	24 100	625,000	100
IH_2	0.9	21	1.2	1000	4.0	6030	123,500	100
IH_3	8.04	30	2.4	140	0.2	15.1	1,550	100
NOTE—Power densities are the calculated plane-wave equivalent.								

Solution: In order to ensure compliance with the MPE for a controlled environment, the sum of the ratios of the time averaged squares of the measured electric field strength to the corresponding squares of the MPE, and the sum of the ratios of the time-averaged squares of the measured magnetic field strength to the corresponding squares of the MPE, should not exceed unity. That is:

$$\sum_{i=1}^{i=6} \frac{S_{E_i}(duty\ factor)}{MPE_{E_i}} < 1$$

and

$$\sum_{i=1}^{i=6} \frac{S_{H_i}(duty\ factor)}{MPE_{H_i}} < 1$$

For this example

$$\sum_{i=1}^{i=6} \frac{S_{E_i}(duty\ factor)}{MPE_{E_i}} = \frac{2.15 \times 0.2}{1.19} + \frac{21.2 \times 0.6}{16} + \frac{93 \times 0.45}{73.5} + \frac{0.06}{100} + \frac{0.12}{100} + \frac{0.24}{14} = 1.74 > 1$$

and

$$\sum_{i=1}^{i=6} \frac{S_{H_i}(duty\ factor)}{MPE_{H_i}} = \frac{0.38 \times 0.2}{13.2} + \frac{1.51 \times 0.6}{178} + \frac{6.03 \times 0.45}{816} + \frac{2410}{62,\ 500} + \frac{603}{12,\ 350} + \frac{1.51}{155} < 1$$

In order to comply with the MPE for the controlled environment, both summations must be less than unity. Although the second summation corresponding to the magnetic field strength measurements is less than unity, the first summation of electric field strength measurements exceeds unity—therefore the exposure exceeds the MPE for the controlled environment.

D.3 Induced and contact current

D.3.1 Induced current

A similar procedure is applied to the case where induced or contact current is associated with more than one source. In this case,

For frequencies <100 kHz:

$$\sum_{i=1}^{i=n} \frac{(induced\ current)_i}{MPE_i} < 1$$

For frequencies ≥ 100 kHz:

$$\sum_{i=1}^{i=n} \frac{(induced\ current)_i^2}{MPE_i^2} < 1$$

where MPE_i represents the induced current MPE for the i^{th} source.

Example: The measured induced currents shown in the table below correspond to those expected in an individual working in the vicinity of several sinusoidal sources. Determine whether or not the exposure exceeds the induced current MPE for the controlled environment.

Table D.3—Induced current measurements in an exposed worker

Source	Frequency (MHz)	Induced current (mA)	MPE_I (mA)
S_1	0.006	3.2	6
S_2	0.070	56.3	70
S_3	2.0	49.6	100

Solution:

$$\sum_{i=1}^{i=n} \frac{(induced\ current)_i}{MPE_i} = \frac{3.2}{6} + \frac{56.3}{70} = 1.34 > 1$$

The summation for sources <100 kHz exceeds 1 and, therefore, the exposure exceeds the induced current MPE for the controlled environment. However, the induced current from the table above does not exceed the induced current MPE. The MPEs for frequencies <100 kHz are designed to protect against effects associated with electrostimulation, while the MPEs for frequencies >100 kHz are designed to protect against effects associated with heating, (See Annex C.2.1.)

NOTE—See 4.1.4.2 for non-sinusoidal current waveform applications.

D.4 Measurement requirements

D.4.1 Field measurements

In general, measurements of both electric and magnetic fields are required when the measurement location is too close to the emitting source to be in the far field or when the location is in the near vicinity of a re-radiating (reflecting) source.

The far field of a simple antenna is generally defined as starting at a distance of five wavelengths from the antenna or, in the case of an antenna with a parabolic reflector, at a distance of ten times the diameter of the reflector. For an antenna with multiple elements, the radiation pattern of the antenna can be considered to be fully formed at a distance of ten times the maximum element spacing. Most commonly, measurements of both field components are not required at frequencies above 100 MHz (wavelength three meters) unless multiple emitters are involved or standing waves are produced by the presence of re-radiators.

When metallic (conducting) surfaces are immersed in an RF field, currents are induced in those surfaces which, in turn, produce electric and magnetic fields that combine with, and are out of phase with the primary field in a complex manner and produce near field radiation near the metallic surface. Accurate depiction of exposure to determine compliance with exposure standards therefore requires the measurement of both field components. Absent a focusing effect (which might be produced by a pair of orthogonally-related conducting surfaces), the total absorbed energy is no greater than would be experienced in the absence of the reflecting object.

D.4.2 Induced current measurements

In some cases induced current may best indicate exposure. For example, when RF exposure must be determined in the near field of an emitter or re-radiating object, measurement of induced current in the subject is likely to provide a more realistic determination of compliance with the standard than measurement of field strength. Field strength in the near vicinity of the radiator or re-radiator may be very high and drop off rapidly with distance, but the coupling of the human body with these localized RF fields is likely to be very small, resulting in only minor absorption. In addition, locations where the distribution of electromagnetic energy exhibits a complex pattern, compliance with the pertinent standard for maximum permitted exposure may be better determined by measuring the induced current in the subject than by measuring field strength. This condition may occur particularly in locations where multiple emitters, utilizing a variety of frequencies and at different locations, are producing the total exposure environment.

D.4.2.1 Conditions in which induced current measurements are not required

In addition to field strength limits, this standard specifies limits for induced and contact currents. Intuitively, one may conclude that, at some level of electric field strength, induced currents in the human body cannot exceed the standard, thus making unnecessary current measurements to show compliance with the standard. Employing the work of Gandhi et al. [R346] and Tofani, et al. [R575], calculations have been made of the threshold field strengths below which induced current need not be made. Results of those calculations have been included in the standard as percent of maximum permitted electric field strength versus frequency. See Figure 1 and Figure 2 in clause 4.

Annex E

(informative)

Glossary

For the convenience of the reader, this glossary contains terms that are used in this standard and are defined in the *The Authoritative Dictionary of IEEE Standards Terms* [B72].

E.1 conductivity (σ): The ratio of the conduction-current density in a medium to the electric field strength. The SI unit of conductivity is the siemen per meter (S/m).

E.2 current density (J): The ratio of the current flowing though a given cross sectional area to the value of the cross-sectional area. The SI unit of current density is the ampere per square meter (A/m^2).

E.3 decibel (dB): A standard unit for expressing the ratio between two parameters using logarithms to the base 10. Decibels provide a convenient format to express voltages or powers that range several orders of magnitude for a given system.

NOTE—With P_1 and P_2 designating two amounts of power, and n the number of decibels denoting their ratio:

$$n = 10\log_{10}\frac{P_1}{P_2}$$

When the conditions are such that ratios of currents or voltages (or analogous quantities in other disciplines) are the square roots of the corresponding power ratios, "decibel" is expressed as

$$n = 20\log_{10}\frac{V_1}{V_2}$$

E.4 duty factor: The ratio of pulse duration (pulse width) to the pulse period of a periodic pulse train. A duty factor of 1.0 corresponds to continuous-wave (CW) operation.

E.5 electric field strength (E): At a given point, the magnitude (modulus) of the vector limit of the quotient of the force that a small stationary charge at that point will experience to the charge as the charge approaches zero in a macroscopic sense. The SI unit of electric field strength is the volt per meter (V/m).

E.6 electromagnetic field: A time-varying field associated with the electric or magnetic forces and described by Maxwell's equations.

E.7 electromagnetic energy (W): The flow of energy consisting of orthogonally oscillating electric and magnetic fields lying transverse to the direction of propagation. The SI unit of energy is the joule (J).

E.8 energy density (electromagnetic field): The electromagnetic energy crossing an infinitesimal area divided by that area. The SI unit of surface energy density is the joule per square meter (J/m^2).

NOTE—The equivalent term at optical wavelengths is called "radiant exposure."

E.9 far-field region: That region of the field of an antenna where the angular field distribution is essentially independent of the distance from the antenna. In this region (also called the free space region), the field has a predominantly plane-wave character, i.e., locally uniform distributions of electric field strength and magnetic field strength in planes transverse to the direction of propagation.

E.10 magnetic field strength (H): The magnitude of the magnetic field vector. For time harmonic fields in a medium with linear and isotropic magnetic properties, H is equal to the ratio of the magnitude of the magnetic flux density B to the magnetic permeability of the medium μ, i.e., H = B/μ. The SI unit of magnetic field strength is the ampere per meter (A/m).

E.11 magnetic flux density (B): A vector quantity that describes the force per unit charge on a moving infinitesimal charge at a given point in space $\boldsymbol{F/q = v \times B}$, where $\boldsymbol{F}$ is the vector force acting on the particle, $\boldsymbol{q}$ is the charge on the particle, $\boldsymbol{v}$ is the velocity of the particle, and $\boldsymbol{B}$ is the magnetic-flux density. The SI unit of magnetic flux density is the tesla (T); 1 T = 10^4 gauss.

E.12 penetration depth: For a plane electromagnetic wave incident on the boundary of a medium, the distance from the boundary of the medium to the point at which the field strengths or induced current densities have been reduced to 1/e (~36.8%) of their initial boundary value in the medium.

E.13 permeability (μ): The ratio of the magnetic flux density to the magnetic field strength at a point. The SI unit of permeability is the henry per meter (H/m).

E.14 permittivity (ε_r): The ratio of the electric flux density in a medium to the electric field strength at a point. The permittivity of biological tissues is frequency dependent and may be a complex quantity. The SI unit of permittivity is the farad per meter (F/m).

E.15 root-mean-square (rms) value (of a periodic function): A mathematical operation on a series of measurements (or a temporal sequence of data) in which the square root of the arithmetic mean of the squares of the measurements of data is taken. For a time-varying function Y with a period T, the rms value of Y is

$$Y_{rms} = \left[\frac{1}{T}\int_{a}^{a+T} y^2 \mathrm{dt}\right]^{\frac{1}{2}}$$

where a is any value of time t.

E.16 wavelength (λ): Of a monochromatic wave, the distance between two points of corresponding phase of two consecutive cycles in the direction of propagation. The wavelength (λ) of an electromagnetic wave is related to the frequency (f) and velocity (v) by the expression $v = f\lambda$. In free space the velocity of an electromagnetic wave is equal to the speed of light, i.e., approximately 3×10^8 m/s. The SI unit of wavelength is the meter (m).

Annex F

(informative)

Literature database

This annex contains papers from the International EMF Project (IEEE/WHO) database that are cited in this standard.[18] Following each citation is the IEEE accession number (the number of the citation as it appears in the IEEE/WHO database). All other bibliographical references are listed in Annex G. Not all of the papers in the IEEE/WHO database are cited in this standard; many were categorized as "peripheral," e.g., papers reporting the results of field measurements around various RF sources, and were not considered relevant for developing the rationale for this revision

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Annex G

(informative)

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Exposure to high frequency electromagnetic fields, biological effects and health consequences (100 kHz-300 GHz)

Exposure to high frequency electromagnetic fields, biological effects and health consequences (100 kHz-300 GHz)

Review of
the scientific evidence on dosimetry, biological effects, epidemiological observations, and health consequences concerning exposure to high frequency electromagnetic fields (100 kHz to 300 GHz)

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ICNIRP 16/2009

International Commission on Non-Ionizing Radiation Protection

ICNIRP Cataloguing in Publication Data

Review of the scientific evidence on dosimetry, biological effects, epidemiological observations, and health consequences concerning exposure to high frequency electromagnetic fields (100 kHz to 300 GHz).

ICNIRP 16/2009

1. Electromagnetic fields 2. Biological effects 3. Non-Ionizing Radiation
4. RF
I. Review

ISBN 978-3-934994-10-2

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Preface

International Commission on Non-Ionizing Radiation Protection

The International Commission on Non-Ionizing Radiation Protection (ICNIRP) is an independent scientific organization whose aims are to provide guidance and advice on the health hazards of non-ionizing radiation exposure.

ICNIRP was established to advance non-ionizing radiation protection for the benefit of people and the environment. It develops international guidelines on limits of exposure to non-ionizing radiations which are independent and science based; provides science based guidance and recommendations on protection from non-ionizing radiation exposure; establishes principles of non-ionizing radiation protection for formulating international and national protection programs.

ICNIRP is a non-governmental organization in non-ionizing radiation in formal relations with the World Health Organization and the International Labour Office. It maintains a close liaison and working relationship with all international bodies engaged in the field of non-ionizing radiation protection, and interacts with radiation protection professionals worldwide through its close collaboration with the International Radiation Protection Association and its national societies.

Work is conducted in four standing committees - on Epidemiology, Biology, Physics and Optical Radiation - and in conjunction with appropriate international and national health and research organizations as well as universities and other academic institutions.

Preface

During the preparation of this document, the composition of the ICNIRP was as follows:

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Preface

FOREWORD

This document addresses the current scientific evidence concerning exposure to high frequency electromagnetic fields (EMF) and the resulting consequences for health. The following review was conducted by the ICNIRP Standing Committees in cooperation with its Consulting Members. It covers all scientific aspects relevant in this area which include numerical dosimetry, measurements, biological laboratory investigations in vitro and in vivo, as well as epidemiological findings. This review was motivated by the needs of the World Health Organization's International EMF Project and ICNIRP's own agenda of reviewing its guidance and advice on the health hazards of EMF exposure. Since the 1998 publication of the ICNIRP guidelines on limiting exposure to electromagnetic fields, there have been important studies published, that need detailed analysis and discussion to determine their implications for health.

This review only addresses high frequency EMFs from 100 kHz to 300 GHz. It aims at providing input to the respective health risk assessment currently undertaken by the World Health Organization (WHO). A similar review of the scientific evidence in the static and low frequency fields was published by ICNIRP in 2003.

Both reviews will form the basis for a thorough reevaluation of ICNIRP's science-based guidance on limiting exposure to electromagnetic fields.

The effort put into this review by the ICNIRP Standing Committees was supported by many external experts who provided very helpful comments. ICNIRP wishes to thank these scientists sincerely for their support.

The Editors

Preface

ACKNOWLEDGEMENT

The support received from the World Health Organization for the development and production of this review "Exposure to High Frequency Fields, Biological Effects and Health Consequences (100 kHz -300 GHz)" is gratefully acknowledged.

The comments received during the review process by the following experts are gratefully acknowledged: Jørgen Bach-Andersen, Aalborg University, Denmark; Jürgen H Bernhardt, ICNIRP Consulting Expert; Simon Bouffler, HPA, United Kingdom; Jutta Brix, Bavarian Ministry for Environment, Germany; Lawrie Challis, University of Nottingham, United Kingdom; Rodney Croft, Swinburne University of Technology, Australia; Peter Dimbylow, HPA, United Kingdom; Patrick Haggard, University College London, United Kingdom; Päivi Heikkinen, National Public Health Institute, Finland; Wolfgang Kainz, Food and Drug Administration, United States of America; Dariusz Leszczynski, STUK, Finland; Maria Lönn, Karolinska Institute, Sweden; Carmela Marino, ENEA, Italy; Kenneth McLeod, Binghamton University (SUNY), United States of America, Georg Neubauer, Austrian Institute of Technology, Austria; Maria Rosaria Scarfi, CNR-IREA, Italy; Gernot Schmid, Austrian Institute of Technology, Austria; Murielle Taxille, IMS Laboratory, France; Evi Vogel, Bavarian Ministry for Environment, Germany; Joe Wiart, France Telecom Orange Labs RD, France; Zenon Sienkiewicz, HPA, United Kingdom.

The support received from Mona Bittar, Karolinska Institute, Sweden is gratefully acknowledged.

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I. Dosimetry of high frequency electromagnetic fields (100 kHz to 300 GHz)

ICNIRP Standing Committee III and Task Group – Physics and Engineering

Allen S, Bassen H, D´Inzeo G, Hirata A, Jokela K, Lin J, Mann S, Matthes R, Roy C, Taki M, Wang J, and Watanabe S

I.1. SUMMARY

I.1.1. Sources

The electromagnetic environment consists of natural radiation and man-made electromagnetic fields that are produced either intentionally or as by-products of the use of electrical devices and systems.

The natural electromagnetic environment originates from terrestrial and extraterrestrial sources such as electrical discharges in the earth's atmosphere and radiation from sun and space. Characteristic of natural fields is a very broadband spectrum where random high peak transients or bursts arise over the noise-like continuum background. This natural background is orders of magnitude below local field levels produced by man-made RF-sources considered here. The everyday use of devices and systems emitting radio frequency (RF) electromagnetic fields is continuously increasing. Sources generating high levels of electromagnetic fields are typically found in medical applications and at certain workplaces. Medical devices used for magnetic resonance imaging, diathermy, hyperthermia, various kinds of RF ablation, surgery, and diagnoses may cause high levels of electromagnetic fields at the patients position or locally inside the patient's body. In addition, some of these medical applications may produce high fields at certain workspaces.

For broadcasting high RF power is generally required to maximize the area of coverage. Close to the antennas electric field strengths can reach several hundred volts per meter. Even higher values can be found close to occupational sources used for processing of various materials by heating and sometimes by formation of plasma discharge in the material. In many such applications RF-safety problems arise because RF- power is high and it may be difficult to enclose the field-generating electrodes and processing space inside a good electromagnetic shield. Sources used by the general public e.g. for wireless communication, data transmission or food processing generate comparably much lower fields at the position of the user. But this may also depend on the behavior of the user especially concerning the distance to the source.

Cellular mobile communication networks cause on average low levels of electromagnetic fields in areas accessible to the general public. Handsets and cell phones, however, might cause significantly higher peak levels of exposure during use.

Electronic article surveillance (EAS) systems and radio frequency identification devices (RFID) operate at many different frequencies within the RF band. Inside some EAS gates electromagnetic fields could get close to the existing exposure limits. In general these systems cause only low fields in the environment.

Radars produce high power main beams only a few degrees wide and usually not accessible during operation. In addition radar antennas typically rotate and signals are pulsed, leading to a reduction in average exposure.

In recent years specialized exposure systems have been designed for laboratory studies. The main purpose of exposure systems is to provide a highly defined electromagnetic exposure to the study subject. This includes all exposure parameters and their variation over time and space. In addition exposure systems for laboratory studies need to fulfill certain criteria in order to prevent or at least minimize any non electromagnetic fields (EMF) exposure related interference of the system itself with the study subject.

I.1.2. Measurement

Given the disparity in the type and nature of the sources, a wide range of approaches is used to evaluate exposure. There are many factors that affect instrumentation and its use in evaluating exposure for a variety of purposes; consequently, there will be particular needs associated with specific tasks.

Both narrow-band (frequency selective) and broad-band instruments can be used for assessing exposure to RF fields. In selecting instrumentation it is necessary to consider a number of key factors that include the response time of the instrument, peak power limitations of the sensor, polarization aspects of the field, dynamic range, response to the characteristics of the signal(s) being measured, including the detailed frequency spectrum content and aspects of time variations, modulation and harmonics and the capability to measure in near and far-fields depending on the circumstances of the field measurement. Moreover, appropriate calibration of the instruments using realistic signals as reference should be performed, i.e. using actual modulation rather than continuous wave (CW) signals for devices intended to measure modulated signals. Potential interference from out of band signals should also be considered.

For external measurements there are essentially three methods that are used to measure electric and magnetic fields and these are portable survey instrumentation, spectrum analyzers and personal exposure monitors.

Portable RF measurement instrumentation provide a relatively simple and convenient means for measuring electric and magnetic field strengths to assess compliance with exposure guidelines. In most cases only instruments with shaped frequency response should be used for that purpose. (It is a type of broadband instrument that is specially designed to have RF field sensors with detection sensitivity that varies as a function of frequency.) The limitations inherent in broadband instrumentation of relative spectral insensitivity, slow response time, and the lack of information on the frequencies of measured fields can be overcome by narrowband measurements, such as spectrum analyzers. There are many parameters that have to be set carefully when using a spectrum analyzer in order to obtain a reading of the desired signal.

In recent years, telecommunications systems have been developed that separate different transmitted signals on the basis of waveform orthogonality rather than in terms of frequency and/or time. Many signals are therefore transmitted at the same time within the same bandwidth meaning that even a spectrum analyzer cannot separate them. Such systems include the existing 3G cellular systems, which use CDMA (Code Division Multiple Access). In order to identify the individual signals associated with such systems, it is necessary to use specialized equipment able to correlate with all of the possible signal patterns and thereby identify the power level and source of each individual signal present.

For studies of health effects on people exposed to RF fields it is clearly important to have meaningful estimates of exposure over time. In the past, personal exposure assessments have been made using exposure data obtained from spot measurements. More recently, instruments have been developed to enable exposure estimates to be made using personal exposure monitors worn on the body. The type of monitor has been dependent on the environment in which people are exposed. Workers on antenna sites have worn pocket-sized devices that are relatively inexpensive whereas more sensitive instruments have been developed to capture relatively low level exposures of the general population over a range of frequency bands used for telecommunications. The characteristics of these types of device is to carry out data logging over periods of activity that sample field strength periodically and store the results for subsequent downloading. While personal monitoring may be very useful for categorizing exposure of groups of people for epidemiological studies, the perturbation of the impressed field by the body may result in considerable uncertainty. The field strength recorded by a body worn instrument may differ from that recorded by the same instrument in the same position with the body absent by up to 10-15 dB close to body resonance frequencies (few 10s of MHz), depending on the direction of incidence and the polarization of the radiation. The accuracy of personal monitors will also be limited in situations where the field strengths are non-uniform over the body.

In addition to the measurement of external electric and magnetic fields, in some circumstances it is possible to measure currents induced as a result of exposure to RF fields. There are two main types of body current meter. Transformer clamps measure the currents flowing through limbs while foot current meters measure the current flowing through the feet to the ground. Meters are also available for measuring contact current as a result of a person contacting conducting objects.

There are various factors that contribute to the derivation of the expanded uncertainty budget of any of the described measurement procedures. In addition to the uncertainty in the calibration procedures, there are other measurement factors that will affect the overall uncertainty when using RF field instrumentation in

particular situations. These will include temperature and drift effects, resolution of the display, issues related to the relative location of the RF source and the measurement probe, positioning of the sensor, nature of polarization, perturbation of measurement by people and the degree of repeatability. The overall uncertainty may be much larger than the calibration uncertainty but may be reduced by adopting approaches to minimize the uncertainty on some of the foregoing factors.

Computational techniques are appropriate in some circumstances and discussion and references are provided.

I.1.3. Interaction mechanisms

Radio-frequency exposure of biological systems is usually specified in terms of such physical characteristics as modulation (continuous wave or pulsed), incident electric-field and magnetic-field strengths, incident power density (when appropriate), source frequency, type and zone of exposure (near or far field), and duration of exposure. The coupling of RF energy into biological systems may be quantified by the induced electric and magnetic fields, power deposition, energy absorption, and the distribution and penetration into biological tissues. These quantities are all functions of its relationship to the physical configuration and dimension of the biological body. A complicating factor is that exposure of the whole body to a given field strength could have outcomes far different for partial body or localized exposure at the same strength. The spatially averaged field strength, depending on the region of space over which the fields are averaged, may vary widely for a given body. Current understanding is that induced fields are the primary cause for biological effect of RF exposure, regardless of the mechanism. Thus, to achieve a quantitative understanding of biological response, dosimetric quantities such as SAR, induced electric field, and current density, must be quantified and correlated with the observed phenomenon. It is noteworthy that dosimetric quantities and their determinations are tissue-type dependent, and require a region of specific tissue mass for averaging, and for correlation with any induced biological response. Thus, a smaller averaging region is scientifically more relevant and precise. It is emphasized that the sensitivity and resolution of present-day computational algorithms and resources, and experimental measurement devices and techniques, can provide accurate dosimetric values with a spatial resolution on the order of 1-mm in dimension or better.

The established biophysical mechanisms underlying the interaction of RF radiation with cells, tissues and entire bodies include ionization potential, induced charge and dipole relaxation, enhanced attraction between cells for pearl-chains formation and other RF-induced force effects, microwave auditory phenomenon, and thermal effects as manifested in tissue temperature elevations. It should be noted that the low energy photons of RF radiation are too weak to affect ionization or cause significant damage to biological molecules such as DNA, under ordinary circumstances.

Polar molecules such as water and other cellular components of biological materials can translate and rotate in response to an applied sinusoidal electric field. The translation and rotation is impeded by inertia and by viscous forces. Since reorientation of polar molecules does not occur instantaneously, this gives rise to a time-dependent behavior known as the relaxation process in biological tissues. Under the influence of RF electric fields at frequencies up to 100 MHz molecules and cells would rearrange and form chains along the direction of the field. A threshold electric field strength between 2 and 10 $kV \cdot m^{-1}$ is needed to produce the non-thermal effect which depends on frequency, cell or particle size, and pulsing parameters of the applied field. Both pulsed and CW fields are known to produce the pearl-chain effect, with a time constant that appears to be proportional to E^{-2}. In addition to alignment of cells and larger molecules, other RF fields-induced effects such as shape changes and electroporation or permeabilization of cells have been documented. However, the reversible and irreversible changes in membranes require much stronger fields.

The microwave auditory effect occurs at a physiologically insignificant temperature rise. The minuscule but rapid rise in temperature as a result of the absorption of pulsed microwave energy launches an acoustic wave of pressure that travels to the cochlea, detected by the hair cells and relayed to the central auditory system for perception. For the size of human heads, the theory predicts frequencies between 7 and 15 kHz, which are clearly within the audible range of humans and have been verified experimentally. Peak amplitude of thermo-elastic pressure waves have been computed for spherical head models approximating the size of rats, cats, infant and adult humans exposed to 10 μs plane wave pulses at 1 $kW \cdot kg^{-1}$. The corresponding incident peak power density is about 5 to 20 $kW \cdot m^{-2}$ for frequencies between 915 and 2450 MHz and the induced peak pressures vary from approximately 350 to 1000 mPa. (The threshold pressure is 20 mPa for perception of sound at the cochlea by humans.)

Tissue heating is the most widely accepted mechanism of microwave radiation with biological systems. The effect can result from elevations of tissue temperature induced by RF energy deposited or absorbed in biological systems through local, partial-body or whole-body exposures. The bulk properties of complex permittivity and electrical conductivity cause the electric fields and currents induced to be absorbed and dissipated in cells and tissues of the human body. For a single pulse or brief application of RF energy, the exposure duration may not be long enough for significant conductive or convective heat transfer to contribute to tissue temperature rise. In this case, the time rate of rise in temperature is proportional to SAR. For longer exposure durations, RF energy-induced temperature rise depends on the animal or tissue target and their thermal regulatory behavior and active compensation process. For local or partial body exposures, if the amount of RF energy absorbed is excessive, rapid temperature rise and local tissue damage can occur. Under moderate conditions, a temperature rise on the order of 1°C in humans and laboratory animals can result from an SAR input of 4 $W \cdot kg^{-1}$. However, this temperature rise falls within the normal range of human thermoregulatory capacity.

Under ambient environmental conditions where the temperature and humidity are already elevated, the same SAR could produce body temperatures that reach well beyond normal levels permitted by the 1°C increment, and it could precipitate undesired heat-stress-related responses. The central premise of the exposure guidelines to protect exposed subjects against temperature increases could be eclipsed, breaching the temperature threshold for induction of adverse thermal effects.

Lastly, while a mechanism(s) must be involved in giving rise to biological effects from RF exposure, it is possible that because of their complexity and the limitations of our scientific knowledge some mechanism(s) responsible for producing a significant effect(s) may still be awaiting discovery or identification.

I.1.4. Dosimetry

Dosimetry plays an important role in risk evaluation of human exposure to RF fields, e.g., evaluation of SAR, induced field and current density. It is important to carefully select appropriate methods of dosimetry in each case. It is also highly recommended to validate the dosimetry by comparing with the results obtained with other methods.

A phantom, a surrogate of a human body, is used for experimental dosimetry of a human body exposed to RF fields. The phantom has equivalent electrical properties of those of the human body. Various materials have been developed to realize the electrical properties.

One of the most recent advances in RF dosimetry is availability of numerical voxel models. Realistic numerical human models are developed with medical diagnostic data, i.e., magnetic resonance imaging (MRI), computer tomography (CT), etc. meter Present finite difference time domain (FDTD) calculations using the voxel models provide millimeter-order SAR distribution. It is noted that the detailed SAR distributions derived from the voxel models are generally consistent with the basic SAR characteristics previously obtained with more coarse or simple human models.

In the frequency range from 100 kHz to 110 MHz, induced electric field and current, and contact current should be quantified in order to evaluate the effects of shocks and burns. Several numerical methods have been used to evaluate the detailed information in the voxel human models. It is however noted that the procedure of the spatial averaging can significantly affect the evaluation.

Theoretical analysis using simple human models, such as a dielectric spheroid, shows general characteristics of SAR inside the human body, including whole-body resonance. From the 1970s, method of moments (MoM) calculations with relatively coarse block human models demonstrated various characteristics of human-body SAR and helped to establish the rationale of the reference levels of RF safety guidelines. Since the 1990s, FDTD calculations with millimeter resolution block models have contributed towards the development of RF dosimetry. These FDTD calculations show whole-body SAR characteristics similar to those obtained from MoM calculations but with wider variations of spatial averaged local SAR. The differences of the shape and structure of the voxel models and of the procedure of spatial averaging of the local SAR over 1 g or 10 g are important causes of this variation. Also the finite element method is used extensively in commercially available software to resolve sub millimeter induced currents, electric and magnetic fields and SAR at lower frequencies.

Detailed SAR distribution in a human head exposed to the near-field of a cellular phone has been derived from FDTD calculations. It is found that the antenna current distribution is one of the important factors to determine the SAR distribution and the position of the maximum local SAR.

SAR distribution inside a human body or a laboratory animal has also been evaluated experimentally. Phantoms have usually been used for experimental dosimetry of human exposure while animal cadavers have been used for dosimetry in laboratory studies. Measurement procedures with an electric field probe have been standardized for compliance tests of cellular phones to RF safety guidelines requiring high reproducibility. Experimental dosimetry based on temperature measurement has also been conducted.

Temperature elevation has been evaluated as a factor in inducing adverse health effects due to exposure to RF fields. Numerical simulation techniques using voxel human models have been developed to include complex thermal properties of a human body. Time constants of temperature elevation at locally-exposed region depend on the blood-flow convection and heat conduction while the time constant of body-core temperature due to the whole-body exposure is also affected by thermoregulatory response which results in longer time constants compared with those of partial-body exposure.

Temperature elevation of tissues associated with the localized exposure of the human head to near field of a cellular phone has been studied. The eye has been extensively investigated using various models for the temperature simulation. It has been found that tissue thermal properties influence greatly temperature elevation inside the eye. Temperature elevation in other organs of the head is an issue of equal importance. Indeed there exists good correlation between peak spatial-average SAR and maximum temperature elevation in the head. It is also clear that the presence of the handset and the battery causes temperature elevation in the skin greater than that from RF energy.

The age dependence aspect is also of relevance for dosimetry and risk assessment. It is found that the permittivity and conductivity of tissues are higher for young rats than for adult ones. Recent studies using realistic whole-body voxel models of children suggest that the whole-body averaged SAR can be higher for children than for adults. However, significant differences in SAR average over 10 g due to a cellular phone have not been found between child and adult head models in a multi-laboratory collaboration study, although some research suggest the possibility of significant increase of the child head SAR. It remains possible that the distribution of absorption within the child and adult head may be different. Pregnant female voxel models have also been developed recently. Although most of the calculated SAR of the fetus or embryo models are similar or lower than that of the mother, temperature simulation is required for a more comprehensive risk assessment of RF exposure of fetuses and embryos.

Metal objects implanted in a human body can cause enhancement of local SAR around the objects although RF exposure guidelines often do not address such situations as well as malfunction of medical implanted devices. Numerical dosimetry has revealed that the enhancement of the SAR due to the metal objects is limited to a very small area around the tip or corner of the metal objects.

Above 10 GHz, a direct relationship exists between the temperature elevation and the incident power density. The power absorption is localized within the skin and some thresholds of thermal sensation have been estimated based on present data. However, more detailed dosimetry as well as the measurement of electrical properties at millimeter-wave frequencies is needed to better evaluate safety of millimeter-wave exposure.

Micro-dosimetry is the quantitative study of the spatial and temporal distributions of electromagnetic fields imparted in cellular and sub-cellular biological structures and their relationship to biological effects. Although marked field discontinuities exist at microscopic level of cell membrane, micro-thermal heating due to RF exposure is negligible. Methodologies for micro-dosimetry have been developed for microscopic dielectric theory and biochemical process, as well as the interaction of fields with biological materials, e.g., electric field manipulation of cells and electroporation.

An evaluation of uncertainty in RF dosimetry is necessary for appropriate risk assessment. While international standards exist for the evaluation of uncertainty in the maximum local SAR values for compliance tests of cellular phones, procedures to evaluate the uncertainty of the numerical dosimetry have not been established. The representativeness of the human anatomic voxel models in use is also a limitation for risk assessment. Accurate and repeatable dosimetry is essential in developing laboratory exposure systems.

I.2. PHYSICAL CHARACTERISTICS

I.2.1. Introduction

High frequency electromagnetic fields are parts of the electromagnetic spectrum between the low frequency and the optical part of the spectrum. As this part of the spectrum is used for broadcasting and telecommunication, it is termed radio frequency (RF). The RF spectrum is defined in the frequency range between 9 kHz and 300 GHz. In this review only frequencies above 100 kHz are considered.

Electromagnetic fields in this frequency range have natural or man made origin. They may have a continuous sinusoidal waveform, but more often they have a complex amplitude distribution over time. For broadcast or telecommunication purposes for example they are modulated or pulsed.

I.2.2. Quantities and units

High frequency electromagnetic fields are quantified in terms of the electric field strength **E**, expressed as volts per meter ($V \cdot m^{-1}$) and magnetic field strengths **H**, expressed as amperes per meter ($A \cdot m^{-1}$). **E** and **H** are vector fields[1]. In the far field of an antenna, the high frequency electromagnetic field is often quantified in terms of power flux density S, expressed in units of watt per square meter ($W \cdot m^{-2}$).

For the purpose of radiation protection physical quantities to describe sources and field properties as well as the interaction of such fields with biological systems are needed to quantify the exposure of the human body to non-ionizing radiation and to estimate the absorbed energy and its distribution inside the body (dosimetric quantities).

A dosimetric measure that has been widely adopted is the specific absorption rate (SAR), defined as the time derivative of the incremental energy δW, absorbed by or dissipated in an incremental mass, δm, contained in a volume element, δV, of a given density ρ:

[1] The ratio E/H is called the intrinsic impedance and for free space it has the value of 377 ohms.

$$SAR = \frac{\delta}{\delta t}\left(\frac{\delta W}{\delta m}\right) = \frac{\delta}{\delta t}\left(\frac{\delta W}{\rho \delta V}\right) \qquad \textit{Eqn. 2.2.1}$$

The SAR is expressed in watt per kilogram ($W \cdot kg^{-1}$).

Table I. 2.1.: Quantities and units used in the radiofrequency band

Quantity	Symbol	Unit	Symbol
Conductivity	σ	Siemens per meter	$S \cdot m^{-1}$
Permittivity	ε	Farad per meter	$F \cdot m^{-1}$
Current	I	Ampere	A
Current density	J	Ampere per square meter	$A \cdot m^{-2}$
Electric field strength	E	Volt per meter	$V \cdot m^{-1}$
Power density	S	Watt per square meter	$W \cdot m^{-2}$
Frequency	f	Hertz	Hz
Impedance	Z	Ohm	Ω
Magnetic field strength	H	Ampere per meter	$A \cdot m^{-1}$
Propagation constant	k	per meter	m^{-1}
Specific absorption	SA	Joule per kilogram	$J \cdot kg^{-1}$
Specific absorption rate	SAR	Watt per kilogram	$W \cdot kg^{-1}$
Wavelength	λ	Meter	m

I.3. SOURCES AND EXPOSURES

I.3.1. Introduction

The man-made electromagnetic environment consists of electromagnetic fields that are produced either intentionally or as by-products of the use of electric devices. Man-made RF-sources considered here produce local field levels many orders of magnitude above the natural background. For all practical purposes of hazard assessment, therefore, the electromagnetic fields on the earth's surface arise from man-made sources.

Exposure quantities used in this chapter depend upon the exposure conditions. In the near field of a source, field strengths are quoted, whereas in the far field, where the plane wave model applies, power densities are quoted.

I.3.2. Natural high frequency fields

The natural electromagnetic environment originates from terrestrial and extraterrestrial sources such as electrical discharges in the earth’s atmosphere and radiation from sun and space (Figure I.3.1). Compared to man-made fields, natural fields are extremely small at radio-frequencies (RF). Characteristic of natural fields is a very broadband spectrum where random high peak transients or bursts arise over the noise-like continuum background.

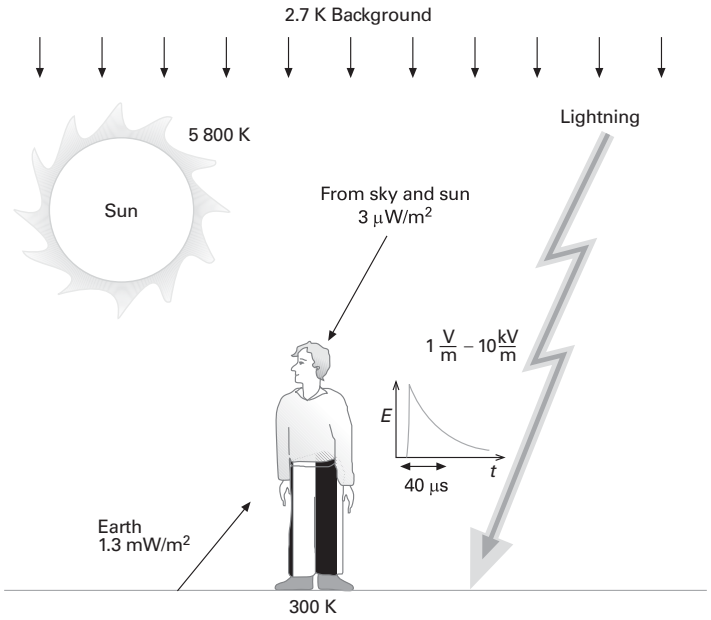

Figure I.3.1. : Terrestrial and extraterrestrial sources of radio-frequency radiation.

At lower radio frequencies, below 30 MHz, the background electromagnetic radiation is mainly due to lightning discharges during thunderstorms. In most cases it is a cloud to cloud flash but also more dangerous cloud to ground flashes are common. Satellite observations show that over land areas the annual number of lightning flashes varies from 2 to 50 km^{-2}, the maximum arising in the tropics (Cooray 2003). The intense current pulse (up to 100 kA) associated with the discharge generates a broadband electromagnetic pulse which propagates long distances in the waveguide composed of the conducting ionosphere and the surface of the earth. The intensity and spectrum of the pulse depends on the current of the lightning discharge, distance and electric properties of the earth. At a distance of a few hundred kilometers, typical peak electric field strength and the width of the main peak of the pulse may vary from 1 to 5 $V \cdot m^{-1}$ and 10 to 50 μs respectively. At a range of 30 km the typical peak value may range from 5 to 20 $V \cdot m^{-1}$ (Willett et al 1990). At short distances less than 100 m to the ground flash the peak electric field strength may exceed 10 $kV \cdot m^{-1}$. The main part of the spectral energy of lightning pulses is distributed below 100 kHz. In the frequency band from 0.2 MHz to 20 MHz the spectral energy decays as $1/f^2$ and much faster above 20 MHz (Willett et al 1990).

At high radio frequencies, above 30 MHz, the natural EM-fields originate from very broadband blackbody radiation from the warm earth and from extraterrestrial processes, mainly from the sun and the extraterrestrial microwave background radiation from the whole sky (Kraus 1986; Burke and Graham-Smith 1997). It should be noted that only at frequencies above 30 MHz and below 30 GHz do electromagnetic waves penetrate the atmosphere efficiently. Below 30 MHz the ionosphere reflects the radiation back to the space and above 30 GHz attenuation is high except in narrow frequency windows. The power density of the radiation component emitted by the warm surface of the ground at 300 K temperature (27 ^{o}C) is a few $mW \cdot m^{-2}$. The extraterrestrial radiation is approximately 1000 times smaller. It is of interest to note that the blackbody radiation from a person in the RF-band is approximately 3 $mW \cdot m^{-2}$.

I.3.3. Man-made fields

I.3.3.1. Telecommunications/Broadcasting

The basic goal of broadcasting is to distribute RF electromagnetic energy over large areas around the transmitter site. To maximize the area of coverage, high RF power is required. The radiated power in the MF band (300 kHz – 3 MHz) and the HF band (3 MHz - 30 MHz) may be as much as 600 kW while in the TV and FM radio bands (50 - 800 MHz) the power fed to a single antenna typically range from 10 to 50 kW, respectively. Indeed, the antennas of broadcast stations are the most powerful continuous sources of RF energy intentionally radiated into free space. Representative data on exposure levels are given in Table I.3.1 (Mantiply et al 1997; Jokela et al 1994; Mild 1981). The most critical group of exposed people consists of the construction and maintenance workers in the towers near energized antennas. The exposure to the general public is, in general, very small except to those people living in the immediate neighborhood of medium and short-wave stations.

Table I.3.1.: Electric field strength and induced current measured in the vicinity of broadcast antennas. MF and HF data were measured at ground level at different distances from the antenna. Most of the VHF and UHF measurements were carried out in the towers near the antennas**.**

Frequency range (MHz)	Average transmitter power (kW)	Modulation	E ($V\ m^{-1}$)	Body current (mA)	Distance and location	Reference
0.3 - 3 (MF)	1-50	AM	3- 800		1-100 m	Mantiply et. al 1997
	600	AM	40-500	10 -100	10-100 m	Jokela et al 1994
3-30 (HF)	-	AM	2 - 200		0-300 m	Mantiply et al 1997
	500	AM	35-120	50-400	5-100 m	Jokela et al 1994
30-300 (VHF)	4	FM	60-900		in tower	Mantiply et al 1997
	-	FM/AM (TV)	up to 430		in tower	Mantiply et al. 1997
	-	FM	300		10-15 cm from The RF cable	Hansson Mild 1981
	40	FM	20- 150		20 cm from the ladder	Jokela et al 1999
300-3000 (UHF)	30	DVB or FM/AM (TV)	up to 620		in tower	Hansson-Mild 1981
	16	DVB or FM/WM (TV)	up to 526		10 -20 cm from antenna	Jokela et al 1984

Medium- and short-wave stations

Short-wave and medium-wave broadcast stations (0,3 – 30 MHz) utilize the reflection of radio-waves from the conducting ionosphere. To reach distant targets very high powers and efficiently radiating large antenna structures are needed. The general public can be exposed to relatively high field strengths levels up to a distance of a few hundred meters from the antenna. In the antenna field open transmission lines used to feed large curtain type HF-antennas are another source of the exposure. In modern medium- and

short-wave broadcast stations the transmitter building as well as transmitters and transmission lines are normally well shielded against electromagnetic interference and leakage fields such that RF-exposure is not a problem inside the buildings.

A typical example of exposure conditions in medium and short-wave broadcast stations is data measured in the Pori (Finland) broadcasting station (Jokela et al 1994). The MF-antenna is a vertical monopole antenna with a height of 185 m, input power of 600 kW and frequency 963 kHz. The electric field measured at a height of 1 m was 500 $V \cdot m^{-1}$ at a distance of 10 m from the antenna decreasing to 90 $V \cdot m^{-1}$ at 40 m. At the same distances the total current flowing from the feet of a grounded person decreased from 140 mA to 30 mA.

For HF -transmission the most popular antenna is a large dipole curtain antenna which is comprised of an array of half-wavelength dipoles installed in front of a reflecting mesh. As a typical example consider the exposure environment in front of the 500 kW HF-curtain antenna operating at 21.55 MHz at the Pori broadcasting station. The maximal measured electric field and total current from a grounded person are found at a distance of 30 m from the antenna where the electric field strength is 90 $V \cdot m^{-1}$ (at 1 m height) and current is 400 mA. At a distance of 100 m there is a second maximum 35 $V \cdot m^{-1}$ and 75 mA. The electric field in front of large curtain antennas does not drop below 20 V m^{-1} until a distance of 150 -200 m is reached. On the other hand, the field strength in the immediate vicinity of the antenna is not extremely large because the transmitter power is distributed over a large antenna area and the radiated power is not effectively concentrated into the main lobe in the reactive near field.

FM and TV

People working in FM/TV towers near high power FM/TV broadcast antennas are exposed to intense electromagnetic fields in the frequency range of 50 to 800 MHz (Jokela and Puranen 1999; Hansson-Mild 1981). Even though the power to the antenna under work may be switched off the workers may need to climb through energized antennas because the broadcast distribution companies try to minimize breaks in the transmissions. The antennas consist typically of three or four vertical dipole array antennas installed on three or four sides of the tower. Input power to the whole antenna varies typically from 10 to 50 kW and the input power to one dipole from 50-500 W even though in USA as high power as 5 kW is not uncommon (Mantiply et al 1997). The nearest dipoles are the primary source of the exposure. The secondary source of the exposure comprises of currents induced in the metallic structures of the mast. Part of that current may also couple directly to the hands and legs which are in contact with ladders and other tower structures.

Because the FM and TV antennas have been designed to radiate a disc-like beam pointed slightly below the horizon, radiation towards vertical direction along the tower is much smaller than towards the main beam which is normally inaccessible. Typically the most hazardous area is confined to a distance of about 15 m from the dipoles. In USA, however, relatively high electric field strengths from 2 to 200 $V \cdot m^{-1}$ have been measured at ground level (Mantiply et al 1997). High levels are explained by the relatively low height of the antenna in the tower and down directed side-lobe of the antenna.

In the FM band measured fields varied from 60 to 900 $V \cdot m^{-1}$ (Hansson-Mild 1981; Mantiply et al 1997; Jokela and Puranen 1999.). In the VHF TV band the exposure is generally slightly lower than in the FM band, the order of 60 $V \cdot m^{-1}$, but close to the dipoles and metallic parts of the tower high values from 400 to 900 $V \cdot m^{-1}$ have been reported. Near UHF-TV antenna elements maximum electric field may exceed 600 $V \cdot m^{-1}$. It is, however, not clear how relevant these highest field strength values are for the assessment of exposure because they may have been measured too close to the metallic parts of the tower where the fields are very non-uniform. For realistic exposure analysis the fields should be measured at a distance greater than 20 cm and averaged in terms of E^2 or H^2 (Jokela 2007). When the distance is 30 - 50 cm the maximal field strengths seem to remain below 300 $V \cdot m^{-1}$ and 0.8 $A \cdot m^{-1}$. The averaged electric field, measured at a realistic distance, however, may still exceed 60 $V \cdot m^{-1}$ (10 $W \cdot m^{-2}$) at 100 MHz.

In many countries terrestrial digital video (DVB-T) and audio broadcast (DAB) have or are about to replace the existing analogue broadcast systems. Schubert et al (2007) have made measurements, at more than 300 identical points, in a ‘before’ and ‘after’ switchover in parts of Germany. Statistical analysis of

the measurement showed an increase in mean exposure in the center of the DVB-T starting areas which was mainly based on the increase in the radiated power at the transmitter stations. The maximal exposure value for analogue TV in the 'before' measurement was 0.9 mWm^{-2} and 6.5 mWm^{-2} in the 'after' measurement for DVB-T. A comparison of analogue FM radio and DAB showed that FM exposure was more than a factor of 10 higher. However, planned increase of DAB transmitter power to improve DAB indoor coverage will reduce this difference. Relatively high body average electric field up to 200 $V \cdot m^{-1}$ (100 $W \cdot m^{-2}$) has been measured in Finland inside a relatively small digital TV antenna. The increase is explained by the high power and small size of the antenna. If the size of the antenna remains the same as for analog UHF antennas the exposure is expected to remain the same (Jokela 2007).

Mobile and wireless communication technologies

The cellular mobile telephone industry has undergone rapid growth; in many countries the take-up rate is approaching and sometimes exceeding 100%. Wireless communication devices are used widely in all parts of modern society. Cellular mobile communication technologies have developed markedly since the early 1980s when analogue cellular radio systems were introduced in Europe. The development has proceeded through the generations described below.

1G Systems

The first generation of mobile telephones consisted of analog systems - typically operating at 450 MHz or 800/900 MHz - using frequency modulation. The Advanced Mobile Phone Standard (AMPS) was developed in the USA in the 1970s. The analog systems deployed during the 1980s in various part of the world were slightly different, namely, Nordic Mobile Telephony (NMT) mainly in the North European countries, Total Access Communication System (TACS) in some European countries, AMPS in the USA, and the Nippon Telegraph and Telephone (NTT) system in Japan. At present, the service has either stopped or is running at a low level of traffic, in most parts of the world. Apart from mobile handsets and base stations, analog systems also are used for cordless telephones. 1G provided mostly voice services.

2G Systems

2G refers to development of digital mobile communication systems (GSM or Global System for Mobile Communication) in the early 1990s. Globally, there are currently more than 1 billion users. There are a number of different systems. In Europe and parts of Asia and the Americas the GSM system is dominating. It features carrier frequencies at 900 and 1800 MHz (850 and 1900 MHz in USA). The bandwidth of each frequency channel is around 200 kHz, and a 9.6 Kbits/s data rate for encoded speech. It uses a time division multiple access (TDMA) technique - each user is 'on' for 4.615/8 = 0.58 milliseconds - then comes back periodically at a frequency of 217 Hz. The remaining 7/8 of the time is used for other users. So from the RF point of view it is a burst type of transmission. Apart from the access frequency of 217 Hz and its harmonics, there are various control and system signals giving rise to power variations at the frequency of 2 and 8 Hz. Japan developed its own TDMA system operating in the 1.5 GHz band. North American developed a version of a code division multiple access (CDMA) standard. This version is a so-called direct-sequence spread spectrum system where the users are 'on' simultaneously, but separated by different codes, which are 'spread' on the carrier to a wider bandwidth than dictated by the un-spread scheme. These systems carry voice, data and enable the sending of text messages

2.5G Systems

The popularity of the Internet and of personal computers created a need for higher data rates on wireless networks than available with 2G systems, which were designed mainly for voice applications. One of the systems that evolved was the general packet radio service (GPRS). The GPRS supports a data rate of up to 140.8kbit/s and is packet based rather than connection oriented. It is deployed in many places where GSM is used. GPRS achieves the higher data rates by combining several timeslots. Another system, Enhanced Data rates for GSM Evolution (EDGE) is an add-on enhancement for 2.5G GSM and GPRS networks and can carry data speeds up to 236.8 kbit/s for 4 timeslots with a theoretical maximum of 473.6 kbits/s for 8 timeslots. It meets the definition of a 3G system.

3G Systems

3G is the newest digital mobile communications technology, and is also known as UMTS in Europe. It operates at frequencies between 1900 and 2200 MHz. Mobile phones are no longer used simply for voice communications, users now require video games and playback, email access, internet browsing, video telephony, high speed data access and music downloads. Hence the requirement for 3G is higher data rates, which can be as high as 384 Kbits/s and up to 2 Mbits/s in indoor environments. The global standard for 3G wireless communications, IMT-2000, is a family of 3G standards adopted by of the International Telecommunications Union (ITU). It includes the universal mobile telecommunications system (UMTS) and wideband CDMA, or W-CDMA. The common feature is the use of spread spectrum as the dominant access scheme for multiple users. The first W-CDMA system was developed in Japan under the name FOMA (freedom of mobile multimedia access) however it is currently incompatible with standard UMTS.

CDMA-2000 is the North American version of the 3G system. It differs from UMTS mainly in the network architecture. CDMA-2000 uses one or more 1.25 MHz channels for each direction of transmissions. The specific frequency bands are 1885-2025 MHz and 2110-2200 MHz, for uplink (from user to base station) and downlink, respectively. W-CDMA (UMTS) uses a pair of 5-MHz channels, one in the 1900 MHz range for uplink and one in the 2100 MHz range for downlink. Thus, UMTS has wider bandwidth requirements. UMTS supports up to 2 Mbit/s data transfer rates, although rates can drop markedly in a heavily loaded site.

Beyond 3G

4G (or beyond 3G) is the tentative descriptor for the next system in the technology and for which research is already underway. For this generation the ITU has set goals of 100 Mbits/s for general environments and 1 Gbits/s (1000 Mbits/s) for indoors. IEEE 802.16 has been engaged in developing an air interface for combined fixed and mobile broadband wireless access to support platforms moving at vehicular speeds. The system is specified to operate in the 2 and 6 GHz licensed bands suitable for mobility.

Mobile telephony networks

The mobile phone network consists of a system of adjoining zones called 'cells'. Each cell has its own base station that sends and receives radio signals throughout its specified zone. Macrocells provide the main structure for the network and the base stations have power outputs of tens of watts and communicate with phones up to a few tens of kilometers distant (35 km in the case of GSM). Microcells are used to infill and improve the main network, especially where the volume of calls is high. The microcell base stations emit less power (a few watts) and have an effective range of a few hundred meters. Picocell base stations have a lower power again (typically a fraction of a watt) and provide very short-range communication, often being sited inside buildings. The RF wave used for communication is referred to as a carrier wave. The information it carries – speech, data, photos etc – is added to the carrier wave in a process known as modulation. The change from analog to digital technology, as described above, is to meet the demand for more data and faster transmission.

Henderson and Bangay (2006) reported the results of an exposure level survey of radiofrequency electromagnetic energy originating from mobile telephone base station antennas. Measurements of CDMA800, GSM900, GSM1800 and 3G (UMTS) signals were performed at distances ranging over 50m to 500m from sixty base stations in five Australian cities. The exposure levels from these mobile telecommunications base stations were very low. The highest recorded level from a single base station was $8.1 \cdot 10^{-4}$ $W \cdot m^{-2}$, (see Table I.3.2.).

Table I.3.2.: Measurements made at nominal distances from base station tower. Measurements units are W m^{-2}.

Technology	Measured powerflux density levels			
	50 m	**200 m**	**500 m**	**Maximum[1]**
CDMA (29 towers)	$2.7 \cdot 10^{-5}$	$3.3 \cdot 10^{-5}$	$5.9 \cdot 10^{-6}$	$8.1 \cdot 10^{-5}$
GSM900 (51 towers)	$3.3 \cdot 10^{-4}$	$2.6 \cdot 10^{-4}$	$2.3 \cdot 10^{-5}$	$7.1 \cdot 10^{-4}$
GSM1800 (12 towers)	$3.1 \cdot 10^{-4}$	$4.1 \cdot 10^{-5}$	$4.7 \cdot 10^{-6}$	$4.3 \cdot 10^{-4}$
3G (35 towers)	$4.1 \cdot 10^{-5}$	$5.6 \cdot 10^{-5}$	$7.6 \cdot 10^{-6}$	$1.4 \cdot 10^{-4}$
All mobile	$3.8 \cdot 10^{-4}$	$2.8 \cdot 10^{-4}$	$2.8 \cdot 10^{-5}$	$8.1 \cdot 10^{-4}$

[1]Maximum occurred at distances varying between 50 and 200 m.

Power density measurements were made in the vicinity of 20 randomly selected GSM microcells and picocells by Cooper et al (2006). The base stations employed a single antenna and between one and four transmitters. The antenna heights ranged between 2.5 m and 9 m and the total radiated power was in the range 1-5 W. Ninety-five percent of the data fell within two 'tramlines' separated by 21 dB. The average power density at a distance of 1m was about $2 \cdot 10^{-2}$ Wm^{-2} which decreased to about $3 \cdot 10^{-3}$ Wm^{-2} at 10 m and $2 \cdot 10^{-6}$ Wm^{-2} at 100 m. The 'tramlines' had a gradient of -10 dB up to a distance of 20 m and a gradient of -40 dB per decade to longer distances.

Mobile transmitters

Mobile transmitters are usually vehicle mounted and there are no physical restrictions to prevent the public approaching even to within touching distance of them. Passengers inside vehicles with roof mounted antennas will be partially shielded from the fields and in the case of antennas mounted at the rear of a car, separations from rear passengers are likely to exceed 60 cm. The far-field distances are only between about 2 and 4.3 cm, allowing field strengths calculations for exposure assessments at all but the closest distances.

Very close to the antenna of mobile telephones very high field strengths can be measured. It is important to note that although these field strengths are high, they are highly non-uniform reactive fields which do not give rise to the same level of induced currents and heating effects as equivalent plane waves. They also only give rise to exposure over very small regions of the body.

Handsets

3G mobile phones operate at lower power levels than both GSM and CDMA handsets. The maximum power from a 3G phone (2100 MHz) is 0.125 watts produced over a 5 MHz bandwidth, whereas GSM phones (900 and 1800 MHz) emit an average power of 0.25 and 0.125 watts over a 0.2 MHz bandwidth and CDMA handsets (800 MHz) have a maximum power of 1 watt. With adaptive power control technology, handsets operate at the lowest power necessary for good radio communications. Handsets are held against the head while a call is made. Typically, the distance from the antenna to the head is only about 2 cm or less. Therefore, the user is in the near-field of the source and simple field calculations are not appropriate to assess exposure.

Terrestrial trunked radio

Terrestrial trunked radio (TETRA) is a digital mobile radio standard, with some similarities to GSM, especially designed for professional users who need high reliability and security (i.e. emergency services and commercial organizations with mobile workforces or large vehicle fleets). The standard defines four

basic power classes – 1, 3, 10, and 30 W. The frequency bands recommended for use in Europe are 380-400, 410-430, 450-470 and 870-933 MHz. Vehicle mounted transmitters and hand portables have output powers of 3W and 1W respectively. Voice data are in timeslots 14.2 ms long and occur every 56.7 ms. This corresponds to a duty factor of 0.25 and a pulse frequency of 17.6 Hz. With this duty factor the average output powers will be 0.75 and 0.25 W.

Citizens band radio

Citizens band (CB) radio in the 27 MHz and 477 MHz band is used in some countries. Antennas are often mounted upon the bumpers of cars, on poles outside houses or on mobile handsets which are held close to the heads of users. Transmitters are permitted a maximum power of 4 W into a 50 Ω load. At close distances, the fields depend upon the precise length and structure of the antenna. Loading coils have a very great effect upon the near-fields of CB antennas with much stronger electric fields close to the shorter antennas. E-field strengths of 200 to 1350 $V \cdot m^{-1}$ have been measured 2 cm from low power mobile antennas (27-450 MHz, Allen, 1991). Although the field strengths are high, the relevance of such localized reactive fields for radiation protection is limited. In general the use of CB radio has fallen dramatically in recent years as the use of mobile phones and related technologies has increased.

Microwave communication links

Pairs of highly directive microwave dish antennas are used to provide line of sight communications links in a variety of applications including cellular telephony, public telecommunications, private business communications, and digital data links. Systems can usually transmit over large distances using only low power levels.

The frequencies used for microwave links are usually in the range 5 to 40 GHz and power levels range from less than 1 to a 8 W. Highly directive dish antennas are used; however, they also have many side lobes which may be the more significant in relation to public exposure but the power is usually at least 20 dB below that in the main beam.

The antennas are mounted upon towers or the tops of buildings with heights of at least 20 m, thus a typical main beam normally does not intercept the ground at distances of less than 230 m. With a radiated power of 8 W and a gain of 50 dB, the power density would be 2.4 $W \cdot m^{-2}$. Assuming a gain of 10 dB for a side lobe traveling directly downwards, the power density at 20 m from an 8 W antenna will be 0.064 $W \cdot m^{-2}$, under far-field conditions.

Satellite uplinks

Powerful and highly directive transmission systems are used to communicate between Earth stations and satellites which are usually in geostationary orbits. The antennas have very high gains ranging from 50 to 70 dB corresponding to very narrow main beam widths and operate at typical equivalent isotropic radiated powers from 50 MW to 350 GW. Therefore, in the main beam it would be possible to be exposed to power densities of a few hundred $W \cdot m^{-2}$. A 225 kW EIRP station at 2.38 GHz using a 64 m dish antenna gives a power density of 2.77 $W \cdot m^{-2}$ even at 100 km. However, the antennas are directed at satellites and of necessity nearby buildings and features have to be avoided; consequently exposure in the main lobe is most unlikely to arise under normal circumstances.

I.3.3.2. Medical applications

Diathermy and hyperthermia

The earliest therapeutic application of radiofrequency electromagnetic fields was in diathermy. Two types of diathermy are commonly used, short-wave (at 13.56 or 27.12 MHz) and microwave. Only a part of the patient's body is exposed to RF energy and exposure duration is limited (typically 15-30 minutes). However, exposure intensity is high and sufficient to cause the intended sustained increase in tissue temperature. Exposures to operators of short-wave diathermy devices may exceed 60 $V \cdot m^{-1}$ and/or 0.16

$A \cdot m^{-1}$ for operators standing in their normal positions (in front of the diathermy console) for some treatment regimes. Stronger fields are encountered close to the electrodes and cables (Stuchly et al 1982). In the "worst case", high exposure of staff may occur at distances less than 1.5-2 m (27.12 MHz) or 1 m (433 MHz and 2.45 GHz, Veit and Bernhardt, 1984). However, more information is needed to fully characterize RF exposures encountered by staff in the therapeutic environment (Shah and Farrow 2007). Electromagnetic energy has also been used in inducing hyperthermia for cancer therapy where the tumor temperature is elevated to the range of 43-45 ^{o}C (Falk and Issels 2001). The procedure is mostly used in conjunction with radiotherapy and chemotherapy since the ability of ionizing radiation to kill tumor cells and the anticancer action of drugs are enhanced by hyperthermia. Systems designed for local or regional hyperthermia operating at 13.56, 27,12, 433, 915 or 2450 MHz employ induction coils, interstitial antennas, dipole arrays or waveguide applicators (Lin 1999a, 2004; Pisa et al 2003). As in diathermy, the patient is exposed to intense fields for about 30 to 60 min during hyperthermia with 20 to 100 W of RF power. While the most significant side effect is a thermal burn on the skin or subcutaneous tissue, there is relatively little information on operator exposure.

Magnetic Resonance Imaging (MRI)

MRI is an imaging technique that employs strong static, gradient, and radiofrequency magnetic fields. It can image soft tissues - unobstructed by bone - with enhanced contrast. Moreover, the ability to provide images in numerous planes without requiring the repositioning of the patient has rendered MRI a very effective and important tool for soft tissue imaging. Indeed, it has become the radiological modality of choice for a great number of diagnostic procedures.

In a clinical MRI system operating at 1.5 T, because of its design, it is unlikely that radiological staff would be exposed to significant RF fields. Some newer open 0.7 T MRI systems allow medical personnel to perform interventional procedures on patients under MRI guidance. It is possible that their hands, heads or torsos may receive significant exposure under such conditions, especially for gradient fields (ICNIRP 2004; 2008). The gradient field is pulsed rapidly in time and is a function of the imaging technique and design of the MRI system. It is significant to note that the time rate of change of the gradient magnetic field is closely related to the strength of electric field induced inside the body.

Recently, the demand for increased spatial resolution and high signal-to-noise ratio (SNR) from MRI instruments has prompted the development of systems using much higher static magnetic fields (greater than 11 T). This development has led to the use of higher RF frequencies for MRI, which, in principle, not only can augment the amount of RF power deposition inside the patient’s body, but also increases the EMF exposure for workers using MRI equipment in the hospital environment and workers employed for supporting, servicing, developing and manufacturing this equipment. There has been particular interest in the exposure of the head, torso, and limbs to the gradient fields, which may be substantial under certain operational environments.

Typical exposure levels from electromagnetic fields for medical applications are summarized in Table I.3.3.

Table I.3.3.: Typical exposure levels from electromagnetic fields from medical applications

Source	Frequency	Distance	Exposure	Remarks
Shortwave diathermy	27.12 MHz	0.2 m 0.5 m 1 m	<1000 $V \cdot m^{-1}$ <500 $W \cdot m^{-2}$ <140 $V \cdot m^{-1}$ 100-1000 $V \cdot m^{-1}$	Staff exposed Patient, untreated body parts Staff
Microwave treatment	433 MHz 2450 MHz 433 MHz 2450 MHz	0.5 m 1 m 0.3-3 m	25 $W \cdot m^{-2}$ 10 $W \cdot m^{-2}$ 50-200 $V \cdot m^{-1}$ 20-140 $W \cdot m^{-2}$	Patient, untreated body parts Whole body average Frequency depending on the static field
Magnetic Resonance Imaging (MRI)	42-300 MHz	Within system	up to 2 W kg^{-1}	SAR refers to normal operational mode

RF ablation

Radiofrequency ablation is a technique that uses contact electrodes to deliver low frequency (500 – 750) voltages for a wide variety of medical therapies. For over a half century, an electrosurgical knife (electro surgery) has been used by surgeons to cut and cauterize tissues as a replacement for the scalpel.

Cardiac ablation uses a catheter electrode, inserted through a vein, in the heart, without requiring opening of the chest wall or heart. An RF generator with a power of about 50 watts is used to creating lesions on the inner wall of the heart for the treatment of various cardiac rhythm disorders. These disorders are due to abnormal cardiac rhythms (arrhythmias) as a result of abnormal electrical pathways in the heart muscle (Huang and Wilber 2000; Lin 2000a; Bernardi et al 2004).

Radiofrequency ablation (RFA) for cancer therapy is a new technique that uses heat to destroy tumors deep within the body. A small needle electrode is placed directly into the tumor. The electrode's high frequency voltages create intense heat that can reach the boiling point of water, killing cancerous cells. This technique has been used to destroy liver tumors as well as renal and breast tumors (Garbey et al 2008; Gervais et al 2009; Hui et al 2008). Similarly, small interstitial microwave antennas have been used in minimally invasive medical ablation techniques (Lin 2003).

RF Telemetry

RF telemetry transmitters encapsulated in a small pill have been used to monitor internal body temperature and other physiological parameters. In addition, pills with imaging cameras have been discussed and may be developed. These devices transmit at a variety of frequencies. Since the receiver is a few meters away (outside the body) total radiated power from the pills does not exceed a few milliwatts.

Devices that are planned for use in patients must pass the safety requirements of the countries where they are sold.

I.3.3.3. Industrial and domestic applications

Intense electric and magnetic fields are used for processing of various materials by heating and sometimes by formation of plasma discharge in the material. In many applications RF-safety problems are unavoidable because RF-power is high and it may be difficult to enclose the field-generating electrodes

and processing space inside a good electromagnetic shield. Consequently relatively intense stray fields and leakage radiation may arise in the vicinity of the electrodes. The manually operated older appliances in the workplaces are frequently more problematic than the new automatic appliances where the operator can control the device remotely. Consumer products such as microwave ovens are nowadays of little concern because as a rule the heating process is well shielded and the units have to meet product standards.

The main objective of this chapter is to present a brief review of high power RF sources used for material processing in industrial and domestic environments Those RF sources which produce high exposure are chosen for closer inspection. Illustrative data from various exposure situations are presented and problems with exposure assessment addressed.

Dielectric heating

High-Frequency dielectric heating is potentially one of the most important RF-exposure sources in the workplace (Mantiply et al 1997, AGNIR 2003). Dielectric heaters and sealers are intended to heat wood (glue dryers) or weld plastics (plastic sealers) by applying a strong radio-frequency electric field between two capacitive metal electrodes (ILO 1998). Plastic sealers operate at 27.12 MHz and less frequently at 40.68 MHz frequency, while glue dryers are generally operated at 13.56 MHz. The RF power varies from less than one kilowatt to tens of kilowatts for typical heat sealers, while for glue-dryers the maximum power may exceed 100 kW (ILO 1998). Most of this power is absorbed in the material to be processed, but some of the power is absorbed by the operator of the heater. The electric and magnetic fields are highly non-uniform, concentrating around the electrode.

HF dielectric heaters are used for other industrial applications such as food processing and paper making (Jones 1987). The RF power may be very high, for example 500 kW in paper making. Radiofrequency exposure is generally not as relevant as in the case of HF-sealers because the machines are well shielded and the presence of operators in the vicinity of the machine is not required due to automatic operation.

Absorption from plastic sealers and glue dryers is determined by many factors related to the appliance and work practices, such as RF-power, shielding of the electrode, thickness and dielectric properties of the material, grounding of the electrode, distance of the worker from the electrode and the duty factor. Duty factor DF ($t_{on}/(t_{on} + t_{off})$) varies typically from 0.07 to 0.5 for plastic sealers and from 0.3 to 0.8 for glue dryers. The distance to the electrodes is particularly critical because the reactive electric and magnetic near-field of the dielectric heaters decays rapidly as a function of distance. Many plastic sealers continue to be operated manually by a person standing or sitting during the heating. Semi-automatic or automatic sealers, where the operator has no need to be close to the electrode during the heating, are generally less problematic. Glue dryers are one example of this category.

Several surveys (Bini et al 1986; Joyner 1986; Conover 1992) show that the spatial maximum of the peak electric field produced by some plastic sealers in the position of the operator may exceed 1000 $V \cdot m^{-1}$, particularly at the position of hands. Values in excess of 100 $V \cdot m^{-1}$ are not uncommon (Wilen et al 2004).

Induction heating

Induction heaters use strong magnetic fields at power frequencies (50/60 Hz) and radio-frequencies for heating of conducting bodies. Heating is due to ohmic and magnetic losses. The former are associated with strong currents induced by the field in the work piece and the latter with direct interaction of the field with magnetic dipoles in the material. When the frequency increases, the current concentrates due to the skin effect on the surface of the work piece. Therefore RF induction heaters are most suitable for surface processing of relatively small work pieces. The frequency and power of RF heaters vary typically from 100 kHz to 3 MHz and from 1 to 100 kW, respectively. Depending on the localization of the heated volume, the field-generating coils may vary from small single-turn devices to larger multi-turn systems. In addition to magnetic fields, electric fields may also be relevant for the exposure at frequencies above several hundred kHz because the impedance of the coil increases as a function of frequency thereby generating high voltages along the coil.

Table 3.4 shows some representative exposure data in the position of the operator of RF induction heaters (AGNIR 2003; Cooper 2002). Magnetic field strength varied from 0.2 to 20 $A \cdot m^{-1}$ and electric field from 10 $V \cdot m^{-1}$ to 1600 $V \cdot m^{-1}$ in the position of head and torso.

Table I.3.4.: Measurements of electric and magnetic fields at the position of the operator of an induction heater (Cooper 2002, AGNIR 2003).

	Magnetic field strength ($A \cdot m^{-1}$)			Electric field strength ($V\ m^{-1}$)		
Frequency (kHz)	Head	Hands	Abdomen	Head	Hands	Abdomen
484	1,44	-	1,68	650	8175	500
743	0,88	0,72	0,40	160	213	32
394	1,52	12,88	5,44	168	840	70
300	0,24	0,24	0,24	16	16	8
630	1,28	0,80	0,80	35	35	23
785	14,64	9,92	0,72	929	310	36
715	18,00	-	6,72	1583	-	326
790	7,04	8,64	1,2	413	722	16
434	20,48	20,48	14,64	1192	1828	646
500	8,48	-	3,52	192	-	64

These values are in general agreement with the previous exposure surveys which showed that the exposure varied from 2 to 8000 $V \cdot m^{-1}$ and from 0.1 to 20 $A \cdot m^{-1}$ as Mantiply et al (1997) have reviewed.

Floderus et al (2002) measured relatively low values 2 $A \cdot m^{-1}$ and 0.3 $A \cdot m^{-1}$ in the vicinity of a 900 kHz hardening machine and a 1.25 MHz brazing machine, respectively. These were spot measurements at a distance of 0.5 m from the machine. The corresponding electric field strengths were 20 and 40 $V \cdot m^{-1}$. For a 400 kHz surface treatment machine they measured 4.8 $A \cdot m^{-1}$ and 160 $V \cdot m^{-1}$ at the same distance.

Estimated on the basis of electric and magnetic field strength alone, RF induction heating seems to produce exposures comparable to the exposures from dielectric RF heating (Mantiply et al 1997). However, based on exact dosimetry, the exposure is clearly lower because the coupling of the human body to the external fields is not as efficient as at higher frequencies. At the same external field level the current density and SAR arising from induction heater exposure are typically lower by a factor of 10 (current density) and 100 (SAR) than for the dielectric heater case.

Domestic induction heating

Domestic induction heating hobs (stoves or cook tops) have recently gained some popularity in Japan and European countries, even though they were introduced into the market some time ago. When electrically conducting materials are immersed in an alternating magnetic field, they can be heated as a result of eddy current losses (Joule effect). This heating technique has been applied mainly for industrial purposes, such as in metal furnaces, but it can also be used as a cooking tool. Aside from high-power (5-10 kW) equipment for commercial catering use, low-power (1-3 kW) induction heating hobs are produced as domestic kitchen appliances. Induction heating hobs operate at the intermediate frequencies of 20 to 50 kHz to take advantage of efficient energy usage and avoiding audible noise created by cooking utensils (pots, pans, and other containers) made of cast iron and stainless steel having high magnetic permeability (ICNIRP 2003; Litvak et al 2002; Wennberg 2001). More recent developments in induction heating hobs have enabled the use of aluminum cookware at higher frequencies (over 60 kHz) (Suzuki and Taki 2005).

The strength of the electric field in the vicinity of induction heating hobs is much lower (a few tens of volts per meter at a distance of 10 cm from the stove edge) than the strength of the magnetic field (Stuchly and Lecuyer 1987). A typical waveform of the magnetic field consists of a carrier wave (26.1 kHz), amplitude modulated at a frequency of 100 Hz (for 50 Hz power) or 120 Hz (for 60 Hz power). In general, the harmonic content of the amplitude modulation extends significantly higher, and the operating frequency depends on the output power setting. For a given power setting, the magnetic field strengths around the hob depend on the material and size of the utensils. The magnetic fields decrease rapidly with distance, and are characterized by the magnetic field distributions of a magnetic dipole or a current loop (Yamazaki 2004).

In practice, the magnetic field strength experienced by the user depends on the user's position, i.e., where the operator is likely to stand (IEC 62233), or whether a person is leaning over the top of the hob or not (Stuchly and Lecuyer 1987). Numerical calculations of induced current showed that only a part of the body of the operator, in particular the hands, are significantly exposed (Burais 1998; Suzuki and Taki 2005).

Plasma discharge equipment

Very intensive RF electric fields produce plasma discharge, which are used in semiconductor fabrication processes such as etching and sputtering. The operating frequency of the plasma discharge appliances is most commonly 13.56 MHz and the power ranges from a few hundred watts to kilowatts. Measurements (Cooper 2002) indicate that the exposure of the operator is relatively low, less than 10 V m^{-1} for distances greater than 10 cm and 0.07 A m^{-1} for distances greater than 30 cm from the discharge electrode. Higher field strengths were measured at shorter distances, but these much localized fields are not very relevant for the exposure assessment. In these conditions SAR and induced current density are much lower than in the case of uniform fields. Some units were found to operate at lower frequencies (0.38 and 0.14 MHz) but, taking into account the lower frequency, the exposure does not exceed the exposure from 13.56 MHz devices.

Microwave heating and drying

Microwave energy is used for heating and drying of many materials such as foods, building materials, paper, rubber, cloths, medical supplies and chemical mixtures (Osepchuk 2002). Generation of plasma in UV-curing is a novel rapidly expanding application. The most popular and well known use of microwave energy is the cooking and heating of food at home and in restaurants and cafe's. Most microwave heating devices operate at the frequency of 2.450 MHz but in some countries 915 MHz is also used.

Industrial microwave systems are most commonly compact batch ovens or large conveyer belts where the microwave power varies from 1 to 600 kW. Despite the large power, most systems are well shielded meeting the requirements of the product performance standards for microwave ovens (leakage radiation 50 W m^{-2} at 5 cm distance). Additionally, due to automatic or semiautomatic operations, operators need not stay in the vicinity of the microwave source.

Microwave levels are more likely to be a problem in mobile applications where high power microwaves are guided to the material to be heated through open applicators pressed toward the material surface, or by using small coaxial antennas drilled into the material. Asphalt processing and moisture-drying of buildings are a few examples. In moisture-drying the power density may well exceed 1000 W m^{-2} on the back surface of the wall being dried.

Microwave ovens

In the western world, up to 90 percent of the households own a microwave oven (Bangay and Zombolas 2003). Due to high microwave power, which typically varies from 500 to 1500 W, this consumer product is potentially hazardous. The present day domestic microwave ovens, however, have been designed and manufactured to satisfy stringent requirements set out in internationally approved product standard. The safety design of these standards aim is to reduce the leakage radiation well below 50 W·m^{-2} at a 5 cm distance and prevent generation of the microwave power when the door is open. Additional protection is

achieved with two independent safety switches which switch off the microwave power when the door is open. The design of the doors, such as the use of a filtering choke on the edges of the door, prevents excessive leakage of microwave radiation, even when visible heavy mechanical damage occurs.

Leakage radiation surveys in Germany, Canada and Australia (Vollmer 2004; Thansandote et al 2000; Bangay and Zombolas 2003; Matthes 1992) indicate that approximately 99 % of the ovens comply with the 50 $W \cdot m^{-2}$ limit. The power density follows approximately the square law as a function of distance, which means that actual exposure decreases from 50 $W \cdot m^{-2}$ to approximately 1.4 $W \cdot m^{-2}$ when the distance increases from 5 to 30 cm, which is the minimum practical distance from the oven. According to the measurements of Bangay and Zombolas (2003) the corresponding maximal local SAR values are 0.256 $W \cdot kg^{-1}$ and 0.0056 $W \cdot kg^{-1}$ (10 g average).

Electronic Article Surveillance (EAS)

Electronic Article Surveillance systems protect merchandise and other assets from theft. An EAS system is basically composed of three components:

- labels and hard tags - electronic sensors that are attached to merchandise;
- deactivators and detachers - used at the point of sale to electronically deactivate labels and detach reusable hard tags as items are purchased; and
- detectors that create a surveillance zone at exits or checkout aisles. In addition, systems that activate tags may sometimes be used in e.g. the retail industry.

The different technologies have been extensively reviewed by ICNIRP (2002) and are shown in Table I.3.5.

TableI.3.5.: Different EAS technologies

Category	Frequency range	Primary tag component
Acousto-Magnetic	40–132 kHz	Resonant Magnetostrictive
Radio Frequency (Swept RF)	1.8–10 MHz	Resonant LC Circuit
Microwave	902-928 MHz & 2400-2500 MHz	Diode

Measurement data for radio-frequency EAS systems in the frequency range from 8.8 to 10.2 MHz show that the magnetic flux density remains generally below 0.2 μT at a distance of 20 cm or more from the coil (Harris et al 2000). Table I.3.6 shows exposure data for magnetic type electronic article surveillance gates (EAS) measured inside the gate. The peak magnetic flux densities are maximal values measured at the indicated distance from the transmitter.

Table I.3.6.: Typical peak magnetic flux densities in the central area of magnetic type EAS gates.

Type	Reference.	Waveform	B (μT_{peak})	Distance from transmitter pylon (cm)
Acousto-Magnetic	Casamento (1999)	PW[a] 58 kHz	65	36
	Casamento (1999)	PW 58 kHz	62.2	36
	Casamento (1999)	PW 58 kHz	61.7	36
	Jokela et al(1998)	PW 58 kHz	17.4	62.5
	IEASMA (2000)	CW[b] 58 kHz [c]	52[c]	37.2

[a]Pulse modulated sinusoid

[b]Continuous Wave

[c] For a person located in the center of the gate. The maximum current density for the spinal cord averaged over 1 cm^2 is 72 mA m^{-2} (peak).

Radiofrequency identification (RFID)

The object of any RFID system is to carry data in suitable transponders, generally known as “tags,” and to retrieve those data by machine-readable means at a suitable time and place to satisfy particular application needs. Data within a tag may provide identification for an item in manufacture, goods in transit, a location, a vehicle, an animal or an individual.

A system requires, in addition to tags, “readers” for interrogating the tags and some means of communicating the data to a host computer or information management system.

Interrogator and reader units may be handheld, fixed or mounted on vehicles. Likewise, tags/transponders may be attached or embedded into various objects, or fixed to the ground.

Portable Data Capture systems are characterized by the use of portable data terminals with integral RFID readers. The hand-held readers/portable data terminals capture data that are then either transmitted to a host information management system via a radio frequency data communication (RFDC) link or held for delivery by line-linkage to the host on a batch processing basis.

Transfer of data between tags and a reader is by wireless communication. Two methods distinguish and categorize RFID systems, one based upon close proximity electromagnetic or inductive coupling and one based upon propagating electromagnetic waves.

The technology varies according to the required application:

- low frequency 124-135 kHz tags have been accepted for near-contact reading for applications such as access control, item identification and animal identification;
- high frequency (13.56 MHz) RFID originated from smart card technology. It offers a longer read range, typically one meter, and is being used more extensively in item management systems e.g. library systems;
- active tag technology uses 433 MHz for read ranges of up to 100 meters. The tags are used for asset tracking, with the tag signaling its presence by transmitting an identifier signal;
- the 860-960 MHz range is used for supply chain and logistics purposes. The actual band used is much narrower e.g. the European spectrum is 865-868 MHz;
- Microwave frequency (2.45 GHz) is used for logistics purposes, for factory automation applications and for active tag technology; and
- Microwave frequency (5.8 GHz) is for road traffic and road tolling systems, where active tag technology provides the range and the frequency provides fast data transfer rates.

I.3.3.4. Safety applications/navigation/radar

Radar

Radar systems mainly use microwave frequencies from 500 MHz up to around 15 GHz, although there are some systems operating up to 100 GHz. The signals produced differ from those of the other sources described in this chapter in that they are pulsed with very short duty cycles that give average powers relevant for radiation protection which are several orders of magnitude less than the peak powers.

The antennas used for radars produce main beams only a few degrees wide. In addition, many of the systems feature antennas whose direction is continuously varied by either rotating them in azimuth or varying their elevation by a nodding motion. Typically this rotation or nodding will reduce mean power by a factor of at least 100 and thus reduce root-mean-square (RMS) fields by a factor of 10. These considerations further reduce the likelihood of excessive exposure.

With stationary antennas, which represent the worst case, peak power flux densities of 10 MW m^{-2} may occur on the antenna axis up to a few meters from the source.

Acquisition and tracking

These antennas can either rotate to perform a scan or, if they lock on to a target, point in a particular direction for an appreciable length of time. Certain tracking radar systems can produce mean power densities greater than 100 $W \cdot m^{-2}$ at distances in excess of a kilometer, even after duty cycle correction. In the case of acquisition radar systems which rotate, the effect of rotation reduces the average power density by a factor of around 100.

Air traffic control

Air traffic control (ATC) radars are scanning devices which are used to track aircraft flights and control their landings at airports. They rotate through a full 360° arc and therefore produce relatively low mean power densities in any one direction. Also, the powers used tend to be slightly lower than with tracking radars. Measurements made in the vicinity of an ATC radar operating at 2.8 GHz with a peak output power of 650 kW gave power densities with the antenna stationary of less than 0.5 $W \cdot m^{-2}$ at 60 m and 20 $W \cdot m^{-2}$ at 19 m. With a rotating antenna, the mean power densities would be lower.

In an exposure survey of civilian airport radar workers in Australia it was found that, unless working on open waveguide slots, or within transmitter cabinets when high voltage arcing was occurring, personnel were, in general, not exposed to levels of radiation exceeding the specified limits (Joyner and Bangay 1986).

Ground penetrating radar (GPR)

Ground Penetrating Radar (GPR), surface penetrating radar, or subsurface radar are all names which refer to the same technique used to locate objects and (or) interfaces situated in a region not penetrable to the eyes. GPR is similar to the conventional free space radar used to detect backscattered radiation from a target to evaluate its position and velocity. GPR systems are made of a transmitting part (source and antenna) which transmits electromagnetic power to the region under investigation, and a receiving part which collects the reflected power and, through signal processing techniques, elaborates it to extract the requested information. The presence of the interface between the air where the antenna is located and the region under investigation, and its influence on the reflected signal, are the fundamental differences between GPR and conventional radar. GPR is used as an alternative technique to seismic methods, sonar, or other specific techniques, its main advantage over those techniques being the general purpose principles of operation and the use of remote, non-contacting transducers to radiate and receive the electromagnetic energy. Moreover, it has the highest resolution in subsurface imaging of any geophysical method, approaching centimeters under the right conditions (Leon et al 1994).

The design of GPR systems is largely applications-oriented and the overall design philosophy, as well as the details, depends on the target type and the background medium. The bandwidth of the received signal is directly linked to the number of features (geological strata or buried objects) which will be resolved. Since penetration depth decreases with frequency, usually GPR systems work with frequencies less than 1 GHz. In long range investigations, frequencies as low as a few tens of MHz have been also used. On the other hand, resolution is higher for higher frequencies. Consequently, low frequency antennas (10-120 MHz) radiate long wave-length electromagnetic fields that can penetrate up to 50 meters or more in certain conditions, but are capable of resolving only very large subsurface features. In contrast, the penetration depth of a 900 MHz electromagnetic field is about one meter, and often less in typical ground conditions, but the generated reflections can resolve features down to a few centimeters in diameter (Carin 2001; Daniels 1996).

Generally, GPR systems use very narrow pulses (e.g. pulse duration of 1 ns) with low mean power (e.g. peak pulse power 50 W, mean pulse power 50 mW) and the received power is at least one order of magnitude below that transmitted. It should be noted that GPR systems, on the basis of the Federal Communications Commission (FCC) classification (FCC 2002), belong to the imaging system class; as a consequence, their transmitted power should conform to Table I.3.12, at least in the USA. Since antenna frequency, radiation pattern and radiated power strongly depend on the application, it is very difficult to define general exposure conditions with reference to GPR systems as a whole. In particular, to evaluate

the operator exposure to the GPR electromagnetic field, it must be considered that the operator will be in the near-field of the transmitting antenna, so that the exposure evaluation should be conducted considering the SAR according to the formula for multiple frequency exposure (ICNIRP 1998).

Marine radar

Marine radar equipment ranges from large installations on super tankers to the smaller mast mounted equipment used by yachts. Utilization of the systems is also variable with the larger installations of cross-channel ferries being operated continuously while the battery-powered equipment of small-boat radars is used only intermittently. Generally the powers are rather lower than other radar systems with peak powers of up to 30 kW and mean powers ranging from around 1 to 25 W. Under normal operating conditions with the antenna rotating, the average power density of the higher power systems within a meter of the turning circle of the radar system can be calculated to be less than 10 $W \cdot m^{-2}$.

I.3.3.5. New and emerging technologies

Wireless LANs

Simultaneous with cellular mobile communication, significant developments have taken place in the area of Wireless Local Area Networks (WLAN), with rather short range communication between an access point (a base station) and one or several users. WLANs are ad-hoc systems set up in the home, hotels, cafes, office buildings, airports, city parks, corporate and university campuses as hotspots, and usually are connected to the Internet. It allows mobility of data terminals in a well-defined area.

WLANs have been standardized through different standards such as the IEEE Standard family, (IEEE 802.11), or the European HIPERLAN standard (HIPERLAN2). The main features of the different extensions of the IEEE 802.11 Standard are summarized in Table I.3.7.

Table I.3.7.: Summary of the WLAN Standards family IEEE 802.11 and extensions

Standard	Description	Frequency	Data rate	Year
IEEE 802.11	Original standard, exploiting the ISM band	2.4 GHz	2 Mb/s	1997
IEEE 802.11b	Enhanced data rate in the ISM band	2.4 GHz	11 Mb/s	1999
IEEE 802.11a	Fastest version of the standard, exploiting the UNII band	5.7 GHz	54 Mb/s	1999
IEEE 802.11g	Same 802.11a speed, but in the ISM band	2.4 GHz	54 Mb/s	2003
IEEE 802.11h	Modification of 802.11a to ensure usability in Europe	5.7 GHz	54 Mb/s	2003

The IEEE 802.11 standard does not impose any limit on the maximum radiated power, because such limits, together with the available frequency bands, are decided by different regulatory bodies, such as FCC (2005) in the US and CEPT (2002, 2004) in Europe. The assigned frequency bands and allowed maximum radiated powers are summarized in Table I.3.8. The modulation schemes employed by WLANs include frequency hopping and direct sequence spread spectrum in the 2.4 GHz band and orthogonal frequency division multiplexing in both the 2.4 and 5 GHz band. WLAN transmissions are intermittent, which lead to power fluctuations at the stated data rates or higher. Therefore, time-averaged powers are lower and depend on the quantity of data being transmitted.

Table I.3.8.: Assigned frequency bands and allowed radiated powers for Wireless LANs

Frequency band [MHz]	USA (FCC)		Europe (CEPT)	
	Radiated power	EIRP	Radiated power	EIRP
2400 ÷ 2483.5	30 dBm	36 dBm	~	20 dBm
5150 ÷ 5250	17 dBm	23 dBm	~	23 dBm
5250 ÷ 5350	24 dBm	30 dBm	~	23 dBm
5470 ÷ 5725	24 dBm	30 dBm	~	30 dBm
5725 ÷ 5850	30 dBm	36 dBm	Unavailable frequency band	

Bluetooth

Short-range wireless connectivity is achieved using the Bluetooth cable replacement system, which operates around 2.45 GHz. Devices incorporating Bluetooth wireless technology include mobile phone headsets and computer accessories such as printers, keyboards, mice and personal digital assistants. This technology is being increasingly used in business and in the home.

The technology can support small networks, known as piconets, with a point-to-multipoint configuration. The communication is normally over very short ranges, from a few meters to tens of meters. Devices for these applications have very low output powers of only a few mW, about one hundred times lower than mobile phones. Power requirements are given as power levels at the antenna connector and three power classes are defined (see Table I.3.9 for technical details). The low power outputs will give rise to correspondingly low exposures.

Table I.3. 9.: Power classes for Bluetooth technology

Item	Power class 1	Power class 2	Power class 3
Maximum output power (P_{max})	100 mW (20 dBm)	2.5 mW (4 dBm)	1 mW (0 dBm)
Nominal output power	n/a	1 mW (0 dBm)	n/a
Minimum output power at maximum power setting	1 mW (0 dBm)	0.25 mW (–6 dBm)	n/a
Range of mandatory power control	- 4 dBm to P_{max}	n/a	n/a

DECT

Digital Enhanced Cordless Telecommunication (DECT) is a digital technology which originated in Europe, but has now been adopted worldwide. DECT technology is a flexible digital cordless access system for communications in home, office and public environments. DECT is mainly known for high quality voice communications, but it has widespread application like Internet access and internetworking with other fixed or wireless services. The DECT band is divided into 10 equal sub-bands. Within a frequency channel, transmit and receive channels are separated by time slots through a TDMA scheme. The technical data are summarized in Table I.3.10.

In Asia and especially China the Personal Handy-phone System (PHS) has been deployed. PHS is essentially a cordless phone like DECT but with the capability to handover from one cell to the next. The transmission power of the base station is around 500 mW and a range of up to several hundred meters. The PHS phone can support high speed wireless data transfer, internet access, text messaging and image transfer.

Table I.3.10.: DECT technology overview

DECT parameters	Range
Frequency band	1880 – 1900 MHz
Carrier spacing	1.728 MHz
Modulation	GFSK
Radio access	FDMA TDMA TDD
Number of time slots	24
Number of carriers	10
Total duplex channels	120
Bit rate	1.15 Mb/s
Maximum data rate	552 Kb/s
Frame duration	10 ms
Error detection	CRC
Speech coding	32 Kb/s ADPCM
Channel assignment	Dynamic channel selection
Mobility speed	20 km/hour
Peak power (average)	250 mW

Ultra-wide-band (UWB) technology

UWB technology is mainly used in imaging, sensing and communication systems (Kaiser et al 2007). Examples of imaging and sensing systems include vehicular radar, (GPR), through-wall sensing, and medical imaging, while communications systems include hand-held transceivers, sensor networks, Wireless Personal Area Networks (WPAN), etc.

The Federal Communication Commission of the United States (FCC) defines a UWB device as any device where the fractional bandwidth is greater than 0.20 or occupies 0.5 GHz of spectrum (FCC 2002). The effect of this definition is that UWB systems with a center frequency greater than 2.5 GHz need to have a –10 dB bandwidth of at least 500 MHz, while UWB systems operating with a center frequency below 2.5 GHz need to have a fractional bandwidth of at least 0.20.

Specifically, for UWB applications, FCC allows the use of the frequency range below 0.96 GHz, between 1.99 and 10.6 GHz, and between 22 and 29 GHz in the US. In particular, FCC has stipulated the following definitions: low frequency imaging systems are those whose –10 dB bandwidth is contained below 960 MHz. Mid-frequency imaging, consisting of through-wall imaging systems and surveillance systems, that operate with the –10 dB bandwidth within the frequency band 1990-10,600 MHz. High frequency imaging systems, equipment that operates exclusively indoors, and hand-held UWB devices that may operate anywhere, including outdoors and for peer-to-peer applications, that operate with a –10 dB bandwidth within the frequency band of 3100-10,600 MHz. Vehicular radar systems operate with the –10 dB bandwidth within the frequency band of 22-29 GHz and with a carrier frequency greater than 24.075 GHz.

The average emission limits for UWB systems, in terms of EIRP measured in dBm with 1 MHz resolution bandwidth, are given in Table I.3.11. It must be noted that the highest value in the table is -41.3 dBm/MHz, which corresponds to 75 nW/MHz.

Table I.3.11.: The average emission limits for UWB systems, in terms of EIRP measured in dBm with 1 MHz resolution bandwidth

Frequency band (MHz)	Imaging below 960 MHz	Imaging mid frequency	Imaging high frequency	Indoor applications	Hand held including outdoor	Vehicular radar
0.009-960	-41.3	-41.3	-41.3	-41.3	-41.3	-41.3
960-1610	-65.3	-53.3	-65.3	-75.3	-75.3	-75.3
1610-1990	-53.3	-51.3	-53.3	-53.3	-63.3	-61.3
1990-3100	-51.3	-41.3	-51.3	-51.3	-61.3	-61.3
3100-10600	-51.3	-41.3	-41.3	-41.3	-41.3	-61.3
10600-22000	-51.3	-51.3	-51.3	-51.3	-61.3	-61.3
22000-29000	-51.3	-51.3	-51.3	-51.3	-61.3	-41.3
Above 29000	-51.3	-51.3	-51.3	-51.3	-61.3	-51.3

With regard to imaging systems, UWB signals are appealing due to their low probability of interception, non interfering signal waveform, precision ranging and localization (Taylor 1995). In this application, UWB radar has the possibility to probe the motion of the internal organs of the human body with a remote non-contact approach (McEwan 1994). For example, a UWB radar was able to detect, non-invasively, the movements of the heart wall. In practice vocal cords, vessels, bowels, heart, lung, chest, bladder and fetus and any body part of adequate size can be monitored by a UWB radar. Recently UWB systems have been also used for breast tumor detection (Fear et al 2002).

In UWB systems, a radiating antenna could be placed very close to the human body (hand-held radio, wireless headphones, etc.); at the same time another radiating antenna could be broadcasting. Thus, in principle, both near field and far field human exposure can take place. Since the fields radiated by UWB systems are broadband or multiple frequency, according to ICNIRP guidelines, exposure assessment is based on the summation formula, in the frequency range of UWB systems, where the relevant dosimetric parameter is the SAR.

Finally, an aspect of UWB to be taken into account is that it is a form of broadband EM radiation, which can increase the level of background noise for radio communication services or overload receiver input. Communication systems such as cellular phones, WLANs, etc., often employ adaptive power control. When such systems find the quality of service is degrading, they ramp up their transmitter power to compensate. Thus, it is conceivable that an indirect consequence of UWB systems could be a rise in average SAR as cellular phones, etc. are caused to use increased powers.

Wireless transport of electrical energy

The concept of wireless-power transmission (WPT) from solar-power satellites (SPS) envisions the generation of electric power by solar energy in space for use on earth (NRC 2001; Lin 2002a, URSI 2007). The system would involve placing a constellation of solar power satellites in geostationary Earth orbits. Each satellite would provide between 1 and 6 GW of power to the ground, using a 2.45 or 5.8-GHz microwave beam (see Table I.3.12.). The power-receiving antenna (rectenna) on the ground would be a structure measuring 1.0 to 3.4 km in diameter. The higher (5.8 GHz) frequency has been proposed since it has a similar atmospheric transparency. Although, in principle, the higher frequency could involve a reduced size for the transmitting and receiving antennas, as can be seen from the table current designs have opted for larger transmitting antennas and smaller rectenna sites, but a larger power density on the ground to conserve land use, especially in Japan.

As can be seen from Table I.3.12, at the center of the microwave beam, where power densities would be maximum, the proposed power densities range from 23 to 180 $mW \cdot cm^{-2}$ (230 to 1800 $W \cdot m^{-2}$) above the rectenna. At 2.45 GHz, the power density is projected to be 1.0 $W \cdot m^{-2}$ at the perimeter of the rectenna. Beyond the perimeter of the rectenna site or 15 km, the side lobe peaks would be less than 0.1 $W \cdot m^{-2}$.

The danger of loss of control of highly focused beams may be minimized by tightly tuned phased array techniques and by automatic beam defocusing to disperse the power in the event it occurs. Defocusing would degrade the beam toward a more isotropic radiation pattern, which would give rise to even lower power density on the ground (Osepchuk 1996).

Near the center of the microwave beam, power densities would be extremely high. Except for maintenance personnel, human exposure would normally not be allowed at this location. In the case of occupationally required presence, protective measures such as glasses, gloves and garments could be used to reduce the exposure to a permissible level.

Table I.3.12.: Microwave parameters for wireless energy transmission from space power satellites

System parameters	NASA[1]	JAXA[2]	JAXA2
Frequency	2.45 GHz	5.8 GHz	5.8 GHz
Total transmitted power	6.72 GW	1.3 GW	1.3 GW
Maximum power density in beam	22,000 W m^{-2}	630 W m^{-2}	1,140 W m^{-2}
Minimum power density	2,200 W m^{-2}	63 W m^{-2}	114 W m^{-2}
Maximum power/element	185 W	0.95 W	1.7 W
Number of antenna elements	97 million	3,450 million	1,950 million
Transmit antenna size	1.0 km dia	2.6 km dia	1.93 km dia
Amplitude taper	10 dB Gaussian	10 dB Gaussian	10 dB Gaussian
Rectenna size	1.0 km dia	2.0 km dia	2.45 km dia
Max power density above rectenna	230 W m^{-2}	1,800 W m^{-2}	1,000 W m^{-2}

[1]National Aerospace Administration (NASA)
[2]Japan Aerospace Exploration Agency (JAXA)

I.3.4. Exposure systems for laboratory studies

In recent years the design of exposure systems for laboratory studies has been improved considerably. The main purpose of exposure systems is to provide a highly defined electromagnetic exposure to the study subject. These include all exposure parameters and their variation over time and space. In addition exposure systems for laboratory studies need to fulfill certain criteria in order to prevent or at least minimize any non EMF exposure related interference of the system itself with the study subject. Exposure systems must for example be controlled for temperature variations, they have to provide a live friendly environment (food, air, etc.), and they should not expose the study subject to other physical or chemical agents. In addition, there are biological factors, which influence the design and the performance of an exposure system that need to be known and considered. The requirements that exposure systems for RF laboratory studies need to fulfill have been described in the literature (Burkhardt and Kuster 2000; Kuster 1997; Valberg 1995; Guy et al 1999; Kuster and Schönborn 2000).

Polarization of the incident field has a strong influence both on coupling and on homogeneity of the induced field or SAR. In general there are three types of polarizations, E, H or K. They refer to the orientation of the electric field, magnetic field or direction of propagation with respect to the long dimension of the exposed object.

I.3.4.1. *In vitro* exposure systems

An important factor for the design of exposure systems for *in vitro* studies is the coupling between the incident electromagnetic field and the medium. The factors that influence this coupling have been widely discussed (Schuderer and Kuster 2003; Guy et al 1999; Schönborn et al 2001; Zhao 2005; Zhao and Wei 2005).

In this case E-polarization provides the weakest coupling. It has been calculated that for a 60 mm Petri dish the coupling factor increases by approximately one order of magnitude between 1 GHz and 2.5 GHz. It also increases by approximately one order of magnitude when medium height increases from 2 to 5 mm. Depending on the height of the medium, coupling efficiency for an E-polarized standing wave can be up to a factor of four higher than for a plane wave. SAR is highest at the bottom of the dish. Homogeneity of the spatial SAR distribution with respect to a cell monolayer at the bottom of the dish or flask is very good. For a 60 mm Petri dish a standard deviation of approximately 20% was calculated with only little variation with medium height or frequency. For cell suspensions, however, homogeneity is poor. H-polarization or K-polarization provides a much higher coupling efficiency, but homogeneity of SAR distribution is very weak.

In addition the meniscus, which forms at the walls of dishes or flasks at the air-liquid boundary, can significantly influence the SAR distribution in the medium (Schuderer and Kuster 2003; Guy et al 1999). For a cell monolayer at the bottom of the dish, SAR values are underestimated if the meniscus is not taken into account. The magnitude of this effect depends on medium height in the dish and on frequency. In a 35 mm Petri dish with a medium height of 2 mm the error of ignoring the meniscus at 1800MHz can be approx. 60 %. At much higher frequencies this error is reduced (Zhao and Wei 2005). If cells do not settle from the meniscus in cell suspensions very high inhomogeneity in the SAR distribution of more than 100% may result.

Generally, the placement of flasks within the scattering field of other flasks can result in significant changes of the conditions, even in cases in which the magnitude of the scattering field is small. The material used in the vicinity of the flasks can significantly alter the coupling as well.

Special attention must be paid to temperature control. For plastic flasks surrounded mainly by air, the thermal coupling between the medium and the temperature controlled environment is poor, and even SAR values much below 2 W kg^{-1} may result in an unacceptable temperature rises (Pickard et al 2000; Schönborn et al 2000).

For *in vitro* exposure systems different technical solutions have been chosen. They include wave guides, transverse electromagnetic (TEM) cells, RF chambers or wire patch cells. The characteristics of these exposure systems are quite different (Schönborn et al 2001).

TEM Cell

The most commonly used system in the past has been the TEM cell, since it is small, self-contained, and can easily be placed in commercial incubators. In addition the incident field is similar to a plane wave with only transversal electric and magnetic components. If only few dishes are used per cell, the homogeneity is excellent. In an improved design Nikoloski et al (2005) modified a TEM cell to hold four T25 flasks. They achieved an overall average SAR within the medium of 6.0 W kg^{-1} at 1 W input power with a standard deviation of less than 52%.

However, for larger numbers of dishes the inhomogeneity increases drastically, since the E field amplitude decreases rapidly toward the wall of the cell. Therefore, the TEM cell can only be recommended for studies with a very low number of dishes.

When using TEM cells for ultra wide band exposure, it should be considered that the flasks containing the cells may disturb the field due to refraction and distortion of the incident wave combined with the excitation of resonant modes within the flasks (Ji et al 2006).

RF Anechoic Chamber

In a RF chamber an array of flasks with a dimension of several wavelengths can be simultaneously exposed. K polarization is normally employed, due to the immense power requirements for E polarization.

In a more recent design (Lyama et al 2004) for a large scale *in vitro* study, up to 49 Petri dishes were exposed employing a horn antenna, a dielectric lens, and a culture case in an anechoic chamber. The average SAR was 0.175 W kg^{-1} per 1 W antenna input power with a standard deviation of 59%. There are

solutions to reduce the inhomogeneity of this design, for example by surrounding the flask with a matching box filled with liquid to the same level as the medium in the flasks.

Radial Transmission Line

The Radial Transmission Line (RTL) exposure system consists of a circular parallel plate applicator, driven at its center by a conical antenna and terminated radially by microwave absorbers or a matching load (Pickard et al 2000). An interesting feature of this system is that several dishes can be exposed at the same time and that it can be used for a wide frequency band. Moros et al (1999) positioned the flasks either directly on the metal bottom or on an aluminum oxide layer in the RTL.

Waveguide System

Waveguide systems have also been widely used in the past (Czerska et al 1992; Joyner et al1989). The flasks can be oriented in E, H or K polarization. If E polarization is employed to achieve low inhomogeneity for plated cells at the bottom of the flasks, it may be necessary to overcome the poor efficiency due to the weak coupling. One possibility that increases the efficiency by a factor of almost four is to terminate one end of the waveguide with a short circuiting plate as described in Schönborn et al (2000).

Optimized systems have been described (Schönborn 2000; Schuderer et al 2004a; Schuderer et al 2004b; Calabrese et al 2006). Depending on the type of cell culture, the frequency used and other factors, up to ten 35 mm Petri dishes are located inside a standard L-band waveguide (selected for the frequency used). The efficiency in a non resonant waveguide setting at 1.62 GHz was 1.6 W kg^{-1} per 1 W input power and an inhomogeneity of approximately ±30%. In a resonant design an efficiency of 50 W kg^{-1} per 1 W input power could be achieved with an inhomogeneity of again ±30%. In general, the efficiency of a tuned resonant system is higher than a non resonant one.

Another approach was to expose cells in a 60 mm Petri dish at the open end of a waveguide (Gajda et al 2002). Temperature was controlled by placing the Petri dish inside a 150 mm dish with circulating coolant water. The efficiency was 8.55 W kg^{-1} per 1 W input power and a standard deviation of SAR at the bottom of the Petri dish of 24 %.

Wire Patch Cell

The wire patch cell is basically a parallel plate resonator fed in the center of the plate, resulting in large E fields between the plates (Laval et al 2000). To reduce the inhomogeneity caused by the tangential E field, the Petri dishes with medium are placed inside larger Petri dishes filled with medium to the same height as in the smaller dishes. The efficiency reported in Laval et al (2000) is 0.6 W kg^{-1} per W input power at 900 MHz. The deviations from the mean value were within 12% when the evaluation was restricted to the area more than 3 mm away from the edge of each 35 mm Petri dish. This exposure system has been modified (Ardoino et al 2004) for experimental evaluations at 1800 MHz. The mean power efficiency was 1.25 W kg^{-1} per 1 W input power with a standard deviation of 15,2%.

I.3.4.2. *In vivo* exposure systems

In vivo exposure systems should in principle fulfill the same criteria as *in vitro* systems but with special consideration of the needs of animals, which may cause additional problems like animal movement for example. Free movement may have a huge impact on exposure homogeneity. Restraining of the animals may increase exposure homogeneity but cause unacceptable stress for the animal. In addition, animals can move even in the restraining holder, resulting in variation of exposure. It is important to evaluate inhomogeneity of exposure during the experiment taking into account the many factors affecting exposure (Kuster 2000). This includes limitations due to the animals' body and its associated dependence on coupling mechanisms of the electromagnetic fields. Details of dosimetric differences between laboratory animals and humans are described in II.6.7. Exposure systems need to provide a clearly defined SAR distribution within the experimental animal (Kuster et al 2006). Average SAR may be misleading in cases

where organ specific reactions are tested. Exposure systems for whole body and for partial body exposure have been developed and used to investigate biological effects from near field and far field exposure.

Whole-body exposure systems

TEM cells

TEM cells have been used for animal exposure (Ardoino et al 2005). The animals are usually restrained in holders. These systems operating at 900 MHz provide a mean whole body SAR in mice (24 g) of 0,38 W kg^{-1} per 1 W input power with a standard deviation of approximately 25 %.

Radial waveguide

Radial waveguides have been designed for whole-body exposure of different laboratory animals (Hansen 2003). Depending on the design up to 120 animals can be exposed simultaneously. Animals are typically not restrained, but can move freely in a small volume. In hamsters the typical medium whole-body SAR per 1 W input power was 1,7 mW kg^{-1} (±20%) at 383 MHz, 27,6 mW kg^{-1} (±30%) at 900 MHz, and 24,2 mW kg^{-1} (±30%) at 1800 MHz. The shielding factor of the system is better than 75 dB.

In a classical waveguide system (Chou et al 1984; Chao et al 1985; Chou and Guy 1987) with circular polarization at 24,50 MHz, a whole-body average SAR in mice of 3.6 W kg^{-1} per 1 W was reported. The absorption in animals varies considerably with body mass and orientation.

Ferris wheel

The Ferris wheel design consists of two parallel circular plates shorted around the perimeter to form a radial electromagnetic cavity fed at the center in order to excite a cylindrical TEM wave. This is a resonant system and needs an appropriate tuning. These systems have been characterized and optimized for the *in vivo* whole-body exposure of laboratory animals (e.g. mice) (Balzano et al 2000; Faraone 2006). The average whole-body SAR in mice in this system was 0.79 W kg^{-1} per 1 Watt. Over the selected range of body mass from 23 to 36 g and varying locations of the animals in the exposure system, the peak SAR variation was about 29%.

Reverberation chamber

Reverberation chamber exposure systems have been developed to overcome the body restraining to allow free movement (Kainz 2006; Jung et al 2008). This is a multimode resonant cavity exposure system. In this case the dosimetry is based on stochastic SAR values varying over time and space in a random manner.

Anechoic exposure chamber

Anechoic exposure chambers have been used for free moving as well as for restrained animals (Chou et al 1985; Chou and Guy 1987). Calorimetric measurements showed an efficiency of one system with respect to the whole body average SAR in mice ranging from 0.11 to 0.17 W kg^{-1} per 1 W with a standard error of 0.01 W kg^{-1} per 1 W depending on the orientation of the animal with respect to the electric field vector.

A system for the exposure of 100 free moving animals in a multi-generation study (Schelkshorn et al 2007; Tejero et al 2005) used a parabolic reflector with a diameter of 320 cm, to obtain a plane wave at a relative short distance. Results from a numerical simulation show that the plane wave condition has been fulfilled with a maximum phase deviation of 12° compared to an ideal plane wave. The standard deviation of the power density within the whole exposure volume, was 14.9% and 15,5 % for GSM and UMTS systems, respectively. Whole body SAR in the rats was 0002.3 mW kg^{-1} per 1 W with a standard deviation of 41 % at 900 MHz and 0002.5 W kg^{-1} per 1 W with a standard deviation of 45% at 1966 MHz.

A similar system (Wilson et al 2002) used a flared parallel plate waveguide to produce a TEM wave exposure to 18 animal cages located at the aperture plane. Average SAR efficiency with respect to whole body SAR of the free moving animals was 2 mW kg^{-1} per 1 W at 1.6 GHz.

In mice experiments an exposure chamber attached to a horn antenna (Wang et al 2002) was used. Mice could freely move inside a plastic container. This system had an efficiency with respect to the whole-body average SAR of 0.36 W kg^{-1} per 1 W and a variation of ± 0.09 W kg^{-1} at 2.45 GHz.

Partial-body exposure systems

Carousel systems

One example of local exposure systems is the carousel-type head exposure systems, although some body exposure occurs also (Schönborn et al 2004; Wake et al 2007a; Swicord et al 1999). A dipole or monopole antenna is located in the center of circularly arranged animal holders, like a carousel with his head toward the antenna. Target organ is rat brain. The ratio of brain average to whole-body average SAR is reported to be 5 and 9 in these systems operated at 900 MHz and 1.5 GHz, respectively. The ratio is much less than that of actual mobile phone exposures in humans. The efficiency of such exposure systems varies with animal mass and ranges from 5.3 W kg^{-1} per 1 W for animals weighting 70-120 g to 2,8 W kg^{-1} per 1 W for animals weighting more than 180 g, at a frequency of 1.6 GHz. SAR varied with animal movement in the restrainers by +15% to - 30% (Schönborn et al 2004). SAR efficiency also varies with frequency.

In another carousel design (Moros et al 1998; Moros et al 1999) average SAR in the brain of small rats as measured thermo-metrically was 0.85 ± 0.34 W kg^{-1} per 1 W at a frequency of 835.6 MHz.

Loop antenna

Several exposure systems using a tuned loop antenna close to the head of a restrained animal have been designed (Chou et al 1999; Leveque et al 2004; Lopresto et al 2007). They can provide peak SAR values inside the skull of well above 10 W kg^{-1} for 1 W. Simulations showed that the ratio of the maximum local SAR in the brain of a rat exposed with a loop antenna versus a human exposed by a GSM cell phone was 1.3 ±0.6. In the human head, 20% of the brain absorbs approximately 60% of the total power deposited in the brain, compared to approximately 35% of the total power absorbed by the same percentage of rat brain. Additional exposure data obtained by such systems are summarized in Table I.3.13.

Table I.3.13.

System	Frequency MHz	Target	Average W kg^{-1} per 1W	SD W kg^{--1} per 1 W	Ref
Loop antenna	837	brain	23.8	14.4	Chou et al 1999
		whole body	1.2	4.6	
	1957	brain	22.6	11.3	
		whole body	1.1	4.6	
	900	brain	6,8		Leveque et al 2004
	1800	cochlea	4.5	1.3	Lopresto et al 2007

I.3.4.3. Human exposure systems

Important characteristics that exposure systems for human laboratory studies have to fulfill, include well defined exposure parameters, blinded exposure, and no emission of other physical or chemical agents.

Partial-body exposure systems

There are several exposure systems used for human studies (Boutry 2008; Haarala et al 2007, Krause et al 2007; Loughran 2005; Regel et al 2006). Some studies employed modified commercial products of

mobile phones. The dosimetric analysis is based on a numerical approach which has been validated by measurements with phantoms. SAR distributions in brain are estimated for each exposure system in detail. The results show that the highly exposed part is limited and the location is different from phone to phone. It has been shown that the SAR distribution from different types of cell phones could vary by more than 15 dB (Kuster et al 2004).

As an alternative to the use of cell phones, antenna systems were developed that can comfortably be worn on the head all day long and even through the night (Bahr et al 2006). Those systems simulate the exposure from a standard mobile phone with integrated antenna. The efficiency at 900 MHz (GSM signal) was estimated to be 7.66 W kg^{-1} per 1 W, at 1966 MHz (WCDMA signal) it was 13.3 W kg^{-1} per 1 W. Similar dosimetric results were reported from integrated mobile phone antennas (Manteuffel et al 2001; Kivekäs et al 2004).

Whole-body exposure systems

In two studies on human well-being and cognitive performances (Health Council of the Netherlands 2004, Regel et al 2006) a far-field, whole-body exposure system was used emitting GSM- and UMTS-like signals of 1 V m^{-1} incident electric field strength. A base station antenna was located at a distance of 3 m from the subject sitting in an anechoic room. Numerical calculations by FDTD method revealed that the whole-body average SAR was 6.2 μW kg^{-1}, and that the average and peak (1 g average) SAR in brain was 11 and 73 μW kg^{-1}, respectively for 1 V m^{-1} incident electric field strength of UMTS signal at 2.1 GHz (Regel et al 2006).

I.4. RF MEASUREMENT

I.4.1. Introduction

RF sources give rise to electric and magnetic fields which can directly couple into people, inducing fields and currents in their bodies. The fields from sources can also couple into objects, which can then give rise to indirect exposure when people touch the objects and currents flow into their body at points of contact.

The presentation here is concerned only with measurements performed outside the body. The external measurable quantities include electric and magnetic field strength, induced current and, on occasion, temperature. Measurements made with instruments inside the body are discussed in Chapter I.6.

Given the disparity in the type and nature of the sources, a wide range of approaches is used to evaluate exposure. There are many factors that affect instrumentation and its use in evaluating exposure for a variety of purposes; consequently, there will be particular needs associated with specific tasks. However, there are some commonalities in approach that will be highlighted here.

The electric and magnetic field components of an electromagnetic field can vary throughout space and over time in terms of their magnitude and direction. A measurement aims to gain information about these quantities that is needed for a particular purpose. The aim here is to indicate the approaches that can be used to assess exposure to RF fields to evaluate compliance with guidelines, standards and regulations, or for personal exposure assessment for health related studies.

While it is often convenient to describe the time-domain characteristics of fields, the diversity of sources requires the assessment of sinusoidal, non-sinusoidal, pulse-modulated and wideband signals. The implications for measurements of these aspects of fields are also considered here.

I.4.2. Principles of measurements

The measurement equipment must suitably record the quantities to be measured with sufficient accuracy and precision with regard to the signal characteristics and the conditions under which the measurements

are made. The equipment must have a sensitivity and a frequency range suitable for the application and the measurement uncertainty must be considered.

The measurement results may be affected by environmental parameters such as temperature and humidity, the equipment itself, or interference. The latter may arise due to interactions with the operator, inadequate immunity of the equipment, including pick-up in its connecting cables, and the effect of other fields including the effect of the magnetic component in the measurement of an electric field and vice versa.

It is important that the behavior of the instrument as an entity is known insofar as its response to the characteristics of the signal(s) is being measured. The detailed frequency spectrum content and aspects of modulation and harmonics in the measured fields/currents must be taken into account. The calibration of an instrument should take into account the purpose for which it is to be used, e.g. calibration should be done using a GSM signal if an instrument is to be used for measuring GSM signals

Both narrow-band (frequency selective) and broad-band instruments can be used for assessing exposure to RF fields (Chapter I.4.4). In selecting instrumentation it is necessary to consider a number of key factors that include the response time of the instrument, peak power limitations of the sensor, polarization aspects of the field, dynamic range and the capability to measure in near- and far-fields depending on the circumstances of the field measurement.

Standardization bodies such as the International Electrotechnical Commission (IEC), the European Committee for Electrotechnical Standardization (CENELEC), and the Institute of Electrical and Electronics Engineers (IEEE) have devoted considerable efforts into developing technical standards for the assessment of EMF exposure. This has been to satisfy various needs, including product safety certification, occupational exposure legislation, and the desire to standardize the methods for making environmental measurements of electromagnetic fields. The documents are too numerous to list here and this remains a rapidly developing area. The reader is advised to consult the work of the above bodies to gain further perspective.

Guidance and suggestions for evaluating compliance with exposure guidelines have been given by the FCC in OET Bulletin 65 (FCC 1997a). The Bulletin provides advice in predicting and measuring field strengths. A supplement to the Bulletin has been published providing additional detailed information relevant to radio and television broadcast stations (FCC 1997b).

The US National Council on Radiation Protection and Measurements (NCRP) has published a report containing a practical guide to the determination of exposure to RF fields (NCRP 1993). The report outlines procedures for evaluating exposure. It also describes methods for performing practical measurements and computations of exposure specific to a number of different types of RF source.

In addition to the reports mentioned above, there are a number of monographs and technical notes produced by instrumentation manufacturers that provide advice on making measurements and using commercial products (Bitzer and Keller 1999; Kitchen 2001).

I.4.3. Characteristics of Electromagnetic Fields

I.4.3.1. General Considerations

The measurement of EM fields must account for numerous parameters including the following:

- The *power* of each field source and the field strength it produces at the location of interest. Relevant considerations are whether the source uses adaptive power control, produces intermittent transmissions, and whether it can produce multiple carriers.
- The *modulation* of the signal; that is, the time-dependent amplitude and frequency (or phase) changes of each carrier.
- Multipath propagation, wave contributions from the same source arriving at the measurement position via different reflected paths and adding constructively or destructively according to the path length in relation to the wavelength.

- *Fading of the signal*, as statistical variations in its amplitude over time due to multipath propagation between the source and measurement position.
- The *radiation pattern* generated by the source, which is the spatial distribution of the EM field with respect to the source. In the near-field, angular field distributions change greatly as a function of distance from the source. In the far-field, there should be no significant change in the angular field pattern with distance from the source, but reflecting objects in the far-field often make this assumption incorrect.
- The *frequency spectrum* of the source(s), as energy may be distributed over several decades of frequency. The latest ultra wideband (UWB) sources have energy spread over ranges as great as 3.1-10.6 GHz.
- The *impedance* of the field, which describes the amount of energy associated with the electric versus the magnetic field at each point of interest in space.
- The *polarization* of the field, which for a single frequency field, is the direction of the electric field vector and/or the magnetic field vector. The polarization may be constant in a particular direction (linear polarization) or rotating (elliptical polarization).
- The *direction of propagation* for a far-field source.
- The *spatial distribution* of fields as a function of location from the RF source.
- The *physical environment* between the source and measurement location, including the ground and other reflecting objects.

I.4.3.2. Measurements in the Far-Field Region

The far-field refers to a region far away from a single electromagnetic field source, as shown in Figure I.4.1. The electric (E) and magnetic (H) field distributions are essentially independent of the distance from the source. The field has a predominantly plane wave character, i.e., completely uniform distribution of electric field strength and magnetic field strength in a plane normal to the direction of propagation. The E and H-fields are perpendicular to each other and their magnitudes are related according o |E|/|H| = 377 Ω where 377 Ω is the characteristic impedance of free space. Problems can be encountered in any realistic measurement situation in the far-field of a radiating source.

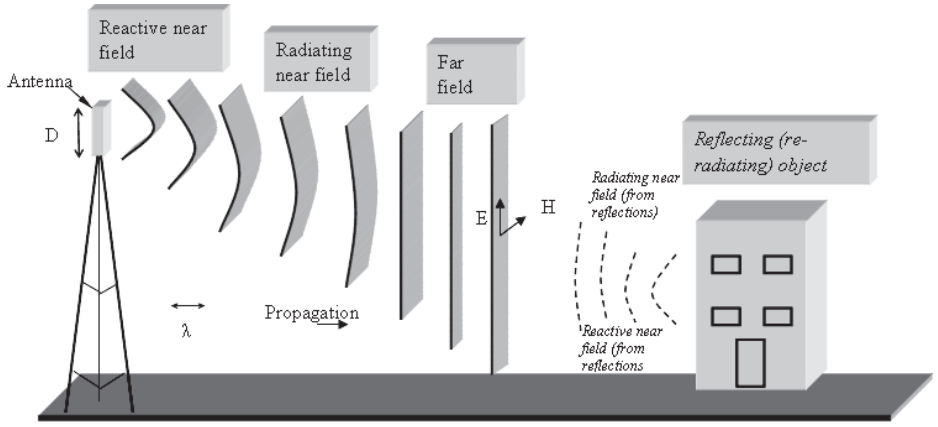

Figure I.4.1.: Near and Far-Field Nomenclature

Measurement issues for a single source at far-field.

When there is only a single source (the source can contain one or more frequencies) of EM fields,

measurements in the far-field of this source can often be performed with relatively simple instruments and techniques. Measurement of only one field, either electric or magnetic, is needed. In addition, high spatial resolution is usually not necessary, since the far-field does not have transverse spatial gradients. In the far-field of a single radiating source only one constant polarization is assumed to exist. This polarization can be linear or elliptical.

Spatial variations in the far-field

Under far-field conditions the electric and magnetic field strengths decrease in proportion to the distance from the source (inverse relationship). This relationship does not apply close to an electrically large radiator such as an antenna that is several wavelengths long, or a reflecting surface (e.g. exterior wall) that has dimensions that are large compared to a wavelength. Reflections cause constructive or destructive interference which causes a periodic variation in the magnitude and phase of the E and H-fields. The distances between maxima and minima are greater or equal to a half wavelength. Reflections of the incident fields occur whenever objects are anywhere near to the region where measurements are being performed and these can cause significant errors in the field strengths being measured. Reflecting objects include the measurement instrument (its housing and cables connected to it) and the ground or other objects in the region of interest. Also, the body of the operator can cause significant reflections.

Time variations of the far-field

Instrumentation must be able to make accurate measurements of fields with various time-varying characteristics. Variations in the far-field region occur due to the source characteristics and the nature of the environment. Under almost all circumstances, change can occur only in the amplitude and not in the frequency. The exception would be the presence of a very rapidly moving source causing a Doppler shift in frequency. Measurement issues associated with time varying field strengths can arise due to amplitude and frequency modulation. There are several types of time variations of a field e.g.

- Variations much shorter than the averaging time, due to modulation or the fast fading of signals due to multipath propagation.
- Certain sources such as air traffic control radars sweep their antenna beam as they scan a volume of space. These sources cause periodic variations in the field strengths at any point, and the changes occur over short periods of time (seconds).
- Slow variations that occur over periods longer than the averaging time at different times of the day.

Measurement issues for multiple sources

If multiple field sources exist, special procedures must be used. Multiple sources may include near-, and far-field conditions, with respect to the measurement instrument. Performing correct measurements requires consideration of frequency, polarization, modulation, and on and off times of each source.

Interference from other sources outside the frequency range that the instrument is designed to measure can greatly degrade measurement accuracy. This interference, called out-of-band interference, is important in areas where multiple signals are present. Signals outside of the instrument's designed useful frequency band, may produce readings greatly in excess of the actual field strength from the useful band signal. Caution should be exercised to ensure no strong fields exist outside the measurement range of the instrument.

I.4.3.3. Measurements in the Near-Field Region

There are two types of near fields: reactive and radiative (Figure I.4.1). The reactive near-field region contains stored non-radiating energy (quasi-static fields) and is located closest to a source of electromagnetic fields. The spatial distributions of the electric (E) and magnetic field (H) field are effectively independent of each other. The amplitudes and phases of both the electric and magnetic fields also vary greatly as a function of distance from the source. The ratio of the magnitudes of E- and H-fields departs from 377 □ and is not constant or easily calculated without detailed knowledge of the structure of

the EM source. Therefore, both E and H must be measured at every point of interest. The transition from a region where the spatial distributions for E and H are independent to one where they are correlated is gradual with increasing distance. For radiators that are small compared to a wavelength, the reactive near-field is taken as extending to

$$r = \lambda / 2\pi \qquad \textit{Eqn. 4.3.1}$$

For radiators that are not small with respect to a wavelength, it is taken as extending to

$$r = 0.62\sqrt{(D^3 / \lambda)} \qquad \textit{Eqn. 4.3.2}$$

The radiating near-field region is farther away from the source. The spatial distributions of E- and H-fields are well correlated in the radiating near-field region, but the far-field radiation pattern of a source is not yet fully formed and there are changes in the angular distribution of the E- and H-fields with increasing distance. This region is defined as beyond the reactive near-field region and extends to

$$r = 2D^2 / \lambda \qquad \textit{Eqn. 4.3.3}$$

In the above three equations:

r = distance from the geometric center of the radiating object
λ = wavelength
D = the largest linear dimension of the radiator

Issues of near-field measurement do not only apply close to a traditional radiating object, such as a transmitting antenna or mobile phone handset. A reflecting object that is in the far-field of a transmitter produces near-field "radiation". For example, a metallic object such as a structural beam in a building, an electrical cable behind a wall, or the ground is a re-radiating object that produces near-fields.

Measurement issues for a single source of near-fields

Spatial variations in the near-field

In the near-field the field strength (E or H) does not diminish in direct proportion to increasing distance from the source but more rapidly, as shown in equations 4.3.4 to 4.3.7 for a very small (infinitesimal) electric dipole. The electric field component varies with distance cubed very close to the dipole (source). Therefore, measurements of near-fields must be made at very frequent spatial intervals. Preliminary measurements must be made to estimate the spatial gradients that exist in the region of interest. Then final measurements can be performed to obtain accurate data.

$$E_r = \frac{I_0 h}{4\pi} e^{-jkr}\left(\frac{2\eta_0}{d^2} + \frac{2}{j\omega\varepsilon d^3}\right)\cos\theta \qquad \textit{Eqn. 4.3.4}$$

$$E_\theta = \frac{I_0 h}{4\pi} e^{-jkr}\left(\frac{j\omega\mu}{d} + \frac{1}{j\omega\varepsilon d^3} + \frac{\eta_0}{d^2}\right)\sin\theta \qquad \textit{Eqn. 4.3.5}$$

$$H_\phi = \frac{I_0 h}{4\pi} e^{-jkr}\left(\frac{jk}{d} + \frac{1}{d^2}\right)\sin\theta \qquad \textit{Eqn. 4.3.6}$$

where

$$k = \frac{2\pi}{\lambda} \qquad (\mathrm{m}^{-1}) \qquad \textit{Eqn. 4.3.7}$$

η_0 = impedance of free space (377 Ω)

ε = permittivity of free space (F m^{-1})

μ = permeability of free space (H m^{-1})

h = length of the dipole (m)

I_0 = antenna current (A)

ω = angular frequency (rad s^{-1})

λ = wavelength (m)

d = distance from center of dipole to the location of interest (m)

θ = angle between the axis of the dipole and the vector from the center of the dipole to the point r

Time variations in the near-field

When measuring time varying E- and H-fields, the factors that must be considered are identical to the factors discussed in this chapter for time variations in the far-field. These factors include changes in amplitude and frequency.

Perturbations of the near-field

Measurement instruments or other objects in the reactive near-field of a source can alter the field strengths and phases of E and H. For example, the presence of measurement personnel or an instrument at an arbitrary location in the reactive near-field of a source may change the E- and H-fields at any other nearby locations. Therefore, sensors that are used to measure fields in this region must be very small compared not only to the wavelength, but also to the field gradients.

I.4.4. Instrumentation

Traditionally, there have been two categories of instruments, namely broad- and narrowband (or frequency selective). The band refers to the frequency range that the instrument measures at a particular instant. A narrow bandwidth is one that is small with respect to the frequencies being measured and is such that two different sources can be distinguishly resolved.

Modern telecommunications systems have been developed that separate different transmitted signals on the basis of orthogonality of signals instead of frequency and/or time. Such systems include the current 3G cellular systems, which use CDMA (Code Division Multiple Access). Many signals are transmitted at the same time within the same bandwidth meaning that even a spectrum analyzer cannot separate them. In order to identify the individual signals associated with such systems, it is therefore necessary to use specialized equipment able to detect all possible signal patterns and thereby identify the power level and source of each individual signal.

There are essentially three classes of instruments used to measure external electric and magnetic fields, namely survey instrumentation, spectrum analyzers and personal exposure monitors. These types of equipment are described below, as are the instruments used to measure body current.

I.4.4.1. Broadband instrumentation for electric and magnetic fields

Portable RF measurement instrumentation, or “hazard survey meters”, provides a relatively simple and convenient means for measuring electric and magnetic field strength to assess compliance with exposure

guidelines. The desired characteristics of the meters, the principles of operation of different types of probe and calibration methods have been described in the literature (see Chapter I.4.2).

Most commercially available RF survey meters are broadband devices. A broadband electromagnetic field instrument is one that ideally measures the total field (both near- and far-fields) impinging on the instrument's sensors simultaneously regardless of modulation (amplitude and/or frequency) within the range specified by the manufacturer.

For the specific purpose of checking compliance with exposure standards, shaped frequency response instruments have been developed. They are specially designed to have RF field sensors with detection sensitivity that varies as a function of frequency. The displayed output from the instrument is a single number that is expressed as a percent of the limit from a specific frequency-dependent standard.

The major components of a broadband instrument, as shown in Figure I.4.2, are:

- *Field Sensor* – an antenna and detection device that produces a low frequency signal proportional to the magnitude of the total field strengths or the square of field strengths being measured. Usually the antenna is a dipole or loop that is small compared to the shortest wavelength of the field being measured.
- *Data link* – a resistive or metallic wire, or a fiber optic cable that carries the output of the field sensor to a display and data collection unit. The link cable usually is designed to prevent RF currents from flowing from the sensor to the display/data collection unit. Ideally the link is "transparent" to the RF field by being highly resistive at RF frequencies or being a fiber optic cable. Some broadband units do not have this data link, since the field sensor, signal conditioning and display parts are integrated into one unit that is small compared to a wavelength.
- *Data processing and display* – provides the signal processing, which can include filtering, amplification, summation, digitization, and a display to show the field strength, field-strength-squared, and other data. This unit may also perform signal averaging and storage and data transmission to a computer or other external computing device.

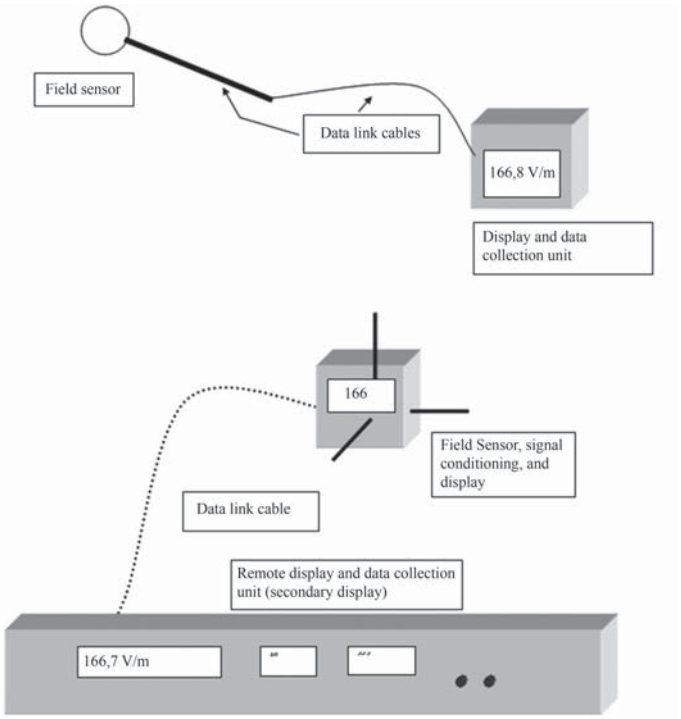

Figure I.4.2.: Schematic of a broadband hazard assessment instrument

The antenna and detector are generally contained within a hand-held probe connected either directly or *via* a flexible lead to a meter containing the processing electronics and display. The antenna in an electric field probe usually consists of one or more electric dipoles. Isotropic probes contain three mutually orthogonal dipoles and derive a vector summation of their outputs to give a response independent of probe orientation. The antennas in magnetic field probes are usually three mutually orthogonal loop or coil elements.

Detectors commonly used in commercially available probes are often diodes and thermocouples. Diodes are widely used since they are sensitive and can also tolerate relatively high field strengths without being overloaded; they are non-linear devices and in weak fields produce a rectified voltage proportional to the square of the incident field strength. In stronger fields, diodes operate out of the square-law region and processing electronics are required to compensate for the deviation. This can introduce imprecision in multiple-frequency environments and can affect the accuracy of measurements of time-averaged field strength when the fields are pulse modulated. Another potential source of error is the sensitivity of diodes to temperature variation.

Thermocouples detect temperature changes and produce a voltage proportional to the power deposited in the junctions of the device. Disadvantages of thermocouples include thermal drift, limited dynamic range, susceptibility to burnout in strong fields and their relative insensitivity.

I.4.4.2. Spectrum analyzers (narrowband instruments)

The limitations inherent in broadband instrumentation can be overcome by making narrowband measurements. A narrowband instrument is frequency selective and measures the electric or magnetic field strength from one or more sources in a “narrow” frequency band. This type of instruments capable of stepping in time through an entire frequency range of interest is called spectrum analyzers. They display and/or record the field strength versus frequency through the frequency range of interest. Some

spectrum analyzers are designed to display the field strength at each frequency as a percentage of the exposure limit in a specific frequency-dependent standard.

The instrument is designed to measure near-fields, the dimensions of the probe sensor should be a small fraction of a wavelength at the highest operating frequency. The sensor and other instrument components should not produce significant scattering of the incident electromagnetic fields.

The sensor response should be isotropic (independent of orientation), non directional, and not sensitive to the polarization of the fields to be measured. A sensor with a non isotropic response is useful if the polarization of the measured quantity (E or H) is known or if the sensor can be rotated to find the direction of polarization. The leads from the sensor to the meter should not interact significantly with the field or conduct RF current from the field to the sensor.

A spectrum analyzer generally employs a broadband antenna in conjunction with a narrowband tunable receiver that provides the frequency and amplitude of the signal to which it is tuned. Spectrum analyzers are tunable over a wide frequency range and they can be used to display the variation of amplitude over a specified portion of the spectrum. An example of the equipment showing antenna, spectrum analyzer and computer control is shown in Figure I.4.3. Most types of antennas used in conjunction with spectrum analyzers for narrowband measurements are not isotropic. Therefore, three measurements are required to determine the vector-summed resultant field strength if the direction of propagation and/or the frequency are unknown. The antennas also tend to be large, since they contain resonant elements, and this may give rise to perturbation in the near-field and prohibit measurements with high spatial resolution. Moreover, the antennas may couple with nearby dielectric bodies, including the operator, which complicates the measurement.

Figure I.4.3.: Typical spectrum analyzer measurement kit

There are many parameters that have to be carefully set when using a spectrum analyzer in order to obtain a reading of the signal, e.g. the RMS field strength. Some of these are as follows:

- *Frequency span* – this is the bandwidth over which the analyzer sweeps. The sweep is not continuous, but made in discrete frequency steps.
- *Resolution bandwidth* – the bandwidth with which the analyzer measures the field strength at a particular frequency. The measurement is typically made with a Gaussian filter. Insufficient resolution bandwidth may result in under-reading of the field strength.
- *Number of points* – the number of discrete frequencies at which measurements are made over the frequency span.
- *Dwell time* – the dwell time at any particular frequency is defined by the sweep time divided by the number of points.
- *Detector* –several types of detectors are provided in most spectrum analyzers for average or peak measurement.

A new generation of frequency-selective instruments has been developed for easy-to-perform frequency-selective measurements. These portable spectrum analyzers with tailor-made software measure multiple signals at different frequencies and then sum the results in the context of a given set of exposure

guidelines and assigning percentage contributions to different signals. In some cases the measuring antennas are placed on tripods in order to minimize interaction with the operator, while in other cases the entire instrument (probe and spectrum analyzer) is in a single hand-held unit.

I.4.4.3. Personal exposure monitors

For studies of human exposure it is important to have meaningful estimates of exposure over time. Personal exposure assessments have been made using exposure data obtained from spot measurements, taken at a point in time and space where a person may be present. Measurements are generally made of the electric field strengths and plane-wave equivalent power densities, and exposures are estimated based on time and motion investigations. More recently, instruments using personal exposure monitors worn on the body have been developed to enable exposure estimates. The type of monitor varies depending on the exposure environment. Workers on antenna sites have worn pocket-sized devices and more sensitive instruments have been developed to capture relatively low level exposures of the general population over a range of frequency bands used for telecommunications. The characteristics of these types of devices allow data logging over extended periods of time and activity.

While personal monitoring may be very useful for categorizing exposure of groups of people for epidemiological studies, the perturbation of the incident field by the body may result in considerable uncertainty.

I.4.4.4. Body current measurements

In addition to the measurement of external electric and magnetic fields, in some circumstances it is possible to measure currents induced as a result of exposure to RF fields. There are two main types of body current meter. Transformer clamps measure the currents flowing through limbs while foot current meters measure the current flowing through the feet to the ground. Meters are also available for measuring contact current as a result of a person contacting conducting objects.

Foot current meters

Current flowing between the feet and the ground can be measured using two parallel conducting plates, separated by a slab of dielectric material and short circuited via a small resistance. The individual stands on the upper plate and the lower plate is placed on the ground. The induced current is calculated by Ohm's law from the potential difference measured across the resistor using a voltmeter incorporating e.g. a diode detector. Alternatively the resistor and detector could be replaced by a thermocouple RF milliammeter connected in series with the two plates. Foot current meters may be appropriate for measurements at ground level but are of limited use if carrying out measurements above ground.

Current transformers

Clamp-on current transformers have the advantage over foot current meters in that they can be used in a greater range of environments. The clamp consists of a solenoid wound around a ferrite core and the current induced in the coil provides a direct measurement of current flowing through the region of interest in the body. Clamp-on instruments have been developed that can be worn and are generally placed around the wrist, ankle or neck (Blackwell 1990).

The meter display unit can be mounted either directly on the transformer or connected through a fiber-optic link to indicate the current flowing through the clamped limb. Current sensing in these units may be accomplished using either narrowband techniques such as spectrum analyzers or tuned receivers or broadband techniques using diode detection or thermal conversion. Instruments have been designed to provide true RMS indications.

The upper frequency response of ferrite-cored current transformers is around 250 MHz. Lighter air-cored transformers have been used to extend the upper frequency response of these instruments but they are significantly less sensitive than ferrite-cored devices.

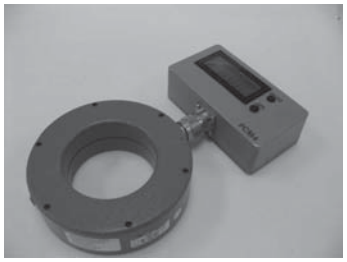

Figure I.4.6.: Example of a Personal Current Meter and Display

Contact current meters

RF contact current measurements are made to investigate the currents due to contact with metallic objects in RF fields, The current measurement device has to be inserted between the hand of the person and the conductive object. The measurement technique may consist of a metallic probe with a defined contact area to be held by hand at one end of the probe while the other end contacts the conductive object.

A clamp-on current transformer, as described above, can be used to measure the contact current which is flowing into the hand in contact with the conductive object. Other approaches are:

- the measurement of the potential difference across a non-inductive resistor of a few ohms connected in series between the object and the metallic probe held in hand
- use of a thermocouple milliammeter placed directly in series.

Commercial equipment has been developed where there is a potential for high currents that could give rise to RF burns. The principle of operation is to use an electrical network of resistors and capacitors which can simulate the body's equivalent impedance.

I.4.5. Calibration of external field measurement equipment

I.4.5.1. Introduction

A number of methods are used for calibrating RF survey probes. These may involve calibrating the probe under free field plane-wave conditions or placing the probe inside a uniform field generated by, e.g. a rectangular waveguide, TEM cell, anechoic chamber or, in the case of some magnetic field probes, Helmholtz coils. The facilities may be used to generate standard fields or use transfer standard probes whereby the field strength is first measured using a standard probe with known calibration traceable to national standards institutions, and then measured with the uncalibrated probe.

The accuracy achieved in a calibration facility is rarely reproduced in practical measurements outside the laboratory because of the following reasons.

- The calibration is usually performed under plane-wave or uniform-field conditions, however the probe may respond differently under realistic conditions where exposure may be in the near-field such that the field strength varies considerably over space. In the reactive near-field the probe may couple with the radiator and alter its emission characteristics.
- In some calibrations only the probe is immersed in the field, however in realistic situations the connecting lead and display unit are also positioned in the field.
- Measurements may be performed in the vicinity of dielectric or metallic scatterers and/or reflecting surfaces.
- In calibrations the probe is positioned in a mount designed for minimum perturbation of the incident field. During exposure assessments the probe is generally held by an individual whose body may couple to the antenna or act as a scattering object.

I.4.5.2. Factors for consideration

Apart from the effects of temperature and instrument stability drift over time there are a number of factors that can materially affect the accuracy of RF instruments.

- Frequency response – calibrations are ideally carried out at frequencies over which the instruments are to be used and at field strengths that are comparable with mid-range readings or above. The response should be reasonably flat over the design frequency range and in the range 1-3 dB.
- Linearity – a range of levels between 25% and 100% of full scale on each range should permit a good assessment of linearity.
- Out of band response – the potential effect of signals outside of the specified frequency response of an instrument need to be considered. Such signals which could originate from multiple sources or harmonics and can potentially affect any element of the instruments construction e.g. the sensor/detector, connecting cables and readout.
- Near-field response – where the instrument may be used in either high or low impedance fields encountered in near-fields, the response of an instrument designed for E or H should be appropriately evaluated to examine the response to the H or E-fields respectively. This can be achieved using mismatched TEM cells up to about 300 MHz.
- Modulation – the modulation characteristics of sources are important considerations particularly where pulsed modulation associated with digital equipment or with radar signals where peak to mean power duty factors may be in the order of 0.001.
- Isotropy – instruments using orthogonal arrays of sensors should be insensitive to the direction of propagation of the incident field however there will be some uncertainty in the sensitivity of individual antenna/detector elements that can be ascertained by rotation of the probe about the handle axis. Another aspect of the isotropic nature of probes can be assessed by rotating the probe handle through the electric field plane.
- Interference – the possibility for RF interference occurring with some component of the instrument should be considered, particularly if calibrations are carried out without all components of the equipment in the field.

I.4.5.3. Uncertainty budgets

In carrying out calibrations in facilities such as those referred to above, the effect of scattering objects and the conducting parts of the RF instrumentation being calibrated will disturb the incident field. In general it would be expected that the uncertainty should not exceed 2 dB and in some circumstances may be less. Uncertainty for TEM cell calibrations may be as little as 5%, but 10% is more typical. For GTEM cells, where the field strength cannot be simply calculated from the power and cell geometry, it is likely that a transfer standard field sensor will provide the lowest uncertainty for calibration.

In addition to the uncertainty in the calibration procedures, there are other measurement factors that will affect the overall uncertainty when using RF field instrumentation in particular situations. These will include temperature and drift effects, resolution of the display, issues related to the relative location of the RF source and the measurement probe, positioning of the sensor, nature of polarization, perturbation of measurement by people and the degree of repeatability. All of these will contribute to the derivation of the expanded uncertainty budget which may be much larger than the calibration uncertainty but may be reduced by adopting approaches to minimize the uncertainty on some of the foregoing factors.

I.5. MECHANISMS OF INTERACTION

I.5.1. RF exposure and coupling into biological systems

When a radio frequency electromagnetic field in air impinges on a biological body it is reflected, transmitted, refracted or scattered by the biological body; the refracted and scattered fields may proceed in directions different from that of the incident RF field. These phenomena are described and governed by the well-known Maxwell's equations of electromagnetic theory. The transmitted and refracted fields from the RF exposure induce electric and magnetic fields in the biological systems that interact with cells and tissues in a variety of ways, depending on the frequency, waveform, and strength of the induced fields and the energy deposited or absorbed in the biological systems. Thus, to achieve a biological response, the electric, magnetic or electromagnetic field must couple into and exert its influence on the biological system in some manner, regardless of what mechanism(s) may be accountable for the response.

Nevertheless, knowledge of the specific mechanism responsible for a given observed biological effect is of scientific interest because: (1) they facilitate understanding of the phenomenon, (2) they help in analyzing relationships among various observed biological effects in different experimental models and subjects, and (3) they serve as guides for comparison and extrapolation of experimental results from tissue to tissue, from tissue to animal, from animal to animal, from animal to human, and from human to human undergoing RF exposure. Therefore, it is important in assessing the health and safety risk of RF energy to determine not only the fields induced in biological tissues, but also the mechanisms underlying its biological interactions with cells, tissues and the human body. However, while a mechanism(s) must be involved in giving rise to biological effects from RF exposure, it is possible that because of their complexity and the limitations of our scientific knowledge some mechanism(s) responsible for producing a significant effect(s) may still be awaiting discovery or identification.

As mentioned in chapter I.4.3., radiation of RF electromagnetic energy is accomplished through the use of antennas, applicators, or radiators. The spatial distribution of RF energy from an antenna is directional and varies with distance from the antenna. At distances sufficiently far from an antenna so that the RF field distribution changes only with distance, not angle or orientation, the region is called a far field or radiation zone. At lesser distances, the energy distribution in the near field or zone is a function of both angle and distance. Moreover, the behavior of RF fields and their coupling and interaction with biological systems are very different in the near and far zones.

The demarcating boundary between near and far zones occurs at a conservative distance of $R = 2\ D^2\ \lambda^{-1}$, where D is the largest dimension of the antenna. Furthermore, the near zone can be divided into two subregions: the radiative region and the reactive region. In the radiative region, the region close to and within $2\ D^2\ \lambda^{-1}$, the radiated power varies with distance from the antenna. The vicinity of the antenna where the reactive components predominate is known as the reactive region. The precise extent of these regions varies for different antennas. For most antennas, the transition point between reactive and radiative regions occurs from 0.2 to $0.4\ D^2\ \lambda^{-1}$. For a short dipole antenna, the reactive component predominates to a distance of approximately $\lambda/2\pi$, where the radiative and reactive components are equal to each other. However, the outer limit is on the order of a few wavelengths or less in most cases (Lin 2000b; 2007).

At the lower radio frequency of 100 kHz, the wavelength in air is 3 km and the $\lambda/2\pi$ distance is about 477 m for the reactive and radiation fields to have equal amplitudes. In contrast, at 900 MHz, the wavelength in air is 33 cm and the $\lambda/2\pi$ distance is 5.3 cm, which comes very close to the $2\ D^2\ \lambda^{-1}$ distance of 6 cm for a 10-cm RF antenna operating at 900 MHz in air. Clearly, both near-zone reactive and far-zone radiative interactions are encountered in the vicinity of personal wireless telecommunication systems.

Some of the salient features of near zone field are: (1) RF electric and magnetic fields are decoupled, quasi-static, and are not uniform, (2) wave impedance varies from point to point, (3) beam width from the antenna is divergent and is small compared with the head or human body, especially for a small antenna, (4) the power varies less with distance from the antenna and (5) the power transfers back and forth may be nearly constant between the antenna and its surrounding medium.

In the far zone, RF fields are characterized as follows: (1) they have plane wave fronts and are independent of source configurations, (2) the radiated power decreases monotonically with distance from the antenna, and (3) the electric and magnetic fields are uniquely defined through the intrinsic impedance of the medium. Thus, a determination of the electric or magnetic field behavior is sufficient to characterize the exposure in terms of power density.

An important consideration in RF exposure is the coupling of RF fields and their distribution inside the body. This association is also valuable in human epidemiological investigations on the health effects of RF field usage. The coupling of RF electromagnetic energy into biological systems may be quantified by the induced electric and magnetic fields, power deposition, energy absorption, and their distribution and penetration into biological tissues. These quantities are all functions of the source and its frequency or wavelength, and their relationship to the physical configuration and dimension of the biological body. Furthermore, the coupling is more complicated in that the same exposure or incident field does not necessarily provide the same field inside biological systems of different species, size, or constitution. Additionally, the interaction of RF energy with biological systems depends on electric field polarization, especially for elongated bodies with a large height-to-width ratio.

It is emphasized that the quantity of induced field is the primary driving force underlying the interaction of electromagnetic energy with biological systems. The induced field in biological tissue is a function of body geometry, tissue property, and the exposure conditions. Moreover, determination of the induced field is important because: (1) it relates the field to specific responses of the body, (2) it facilitates understanding of biological phenomena, and (3) it applies to all mechanism of interaction. Once the induced field is known, quantities such as current density (J) and specific energy absorption rate (SAR) are related to it by simple conversion formulas. In this case, for an induced electric field E in $V \cdot m^{-1}$, the induced current density is given by

$$J(x,y,z) = \sigma(x,y,z)E(x,y,z) \qquad \textit{Eqn. 5.3.1}$$

where σ is the electrical conductivity ($S\ m^{-1}$) of biological tissue and SAR is given,

$$SAR(x,y,z) = \frac{\sigma(x,y,z)\left|E(x,y,z)\right|^2_{rms}}{\rho(x,y,z)} \quad \left[\frac{W}{kg}\right] \qquad \textit{Eqn. 5.3.2}$$

where ρ is the mass density of the tissue ($kg\ m^{-3}$).

At lower frequencies, e.g., 100 kHz or 10 MHz, where the wavelength of RF radiation is at least an order of magnitude longer than the dimensions of the human body, field behavior inside the body is characterized by near-zone reactive field and is quasi-static in character. The electric and magnetic fields become decoupled, and they act separately and additively inside tissue medium (Lin et al 1973; Lin 2000b; 2007). For all practical purposes, the induced fields can be obtained by combining the two independent quasi-static electric and magnetic solutions of the electromagnetic field theory. For example, an externally applied uniform electric field gives rise to a uniform induced electric field inside the body that is in the same direction, but reduced in strength by a factor inversely proportional to the dielectric constant and is independent of body size. The magnetically induced electric field amplitude inside the body is given by

$$E(x,y,z) = \omega B(x,y,z)r/2 = \pi f r \mu H(x,y,z) \qquad \textit{Eqn. 5.3.3}$$

where $f = \omega/2\pi$ is the frequency, μ is magnetic permeability, r is the equivalent radius of a region with homogeneous electrical conductivity, B is magnetic flux density, and H is the strength of the magnetic field component. A uniform magnetic field produces an internal electric field that increases in proportion with distance away from the body center. Thus, magnetically induced electric field, i.e., inductive coupling, would dominate inside a biological body except for tissue bodies that are 1 mm or less in size. A similar scenario exists in the near-zone-reactive region of all antennas and radiating systems. A case in point, the interaction of a cellular mobile telephone handset with the user's head is quasi-static in nature and inductive coupling of antenna-current-generated magnetic field dominates power deposition in the head.

I.5.2. Biophysical mechanisms of interaction

I.5.2.1. Ionization potential of RF fields

Electromagnetic energy may be thought of as being carried by photons or quanta. In this case, the energy (E) of a photon is given by E = *h*f, where *h* is the Planck's constant = $6.625 \cdot 10^{-34}$ J·s, and f is frequency in Hz. Note that 1 eV (electron volt) is equal to $1.602 \cdot 10^{-19}$ J, and the frequency of 1 eV photon is equal to $2.418 \cdot 10^{14}$ Hz. Therefore, the higher the frequency, the higher the energy per photon. A definite amount of photon energy is required to produce ionization by ejection or promotion of orbital electrons from atoms of the material through which an electromagnetic wave propagates. The minimum photon energies capable of producing ionization in water and in atomic carbon, hydrogen, nitrogen, and oxygen are between 10 and 25 eV. Inasmuch as these atoms constitute the basic elements of living organisms, 10 eV may be considered as the lower limit for ionization in biological systems.

A single photon of RF radiation has relatively low energy levels, less than $1.24 \cdot 10^{-5}$ eV; therefore it is not capable of ionization. Accordingly, electromagnetic radiation in the RF spectrum is regarded as non-ionizing radiation. The deleterious biological effects of such ionizing radiations as gamma- and x-rays that largely result from ionization taking place in biological cells and tissues are not produced by a single photon of RF radiation (Lin 1978). It is noted that for strong RF fields, simultaneous absorption of $8.06 \cdot 10^{5}$ or more low energy RF photons, could potentially produce ionization in biological materials, but the probability is small. The point is that RF radiation has low energy photons, therefore under ordinary circumstances, RF radiation is too weak to affect ionization or cause significant damage to biological molecules such as DNA, which is especially renowned for its repair mechanism.

I.5.2.2. Induced charge and dipole relaxation

Polar molecules such as water and other cellular components of biological materials can translate and rotate in response to an applied sinusoidal electric field. The translation and rotation is impeded by inertia and by viscous forces. Therefore, the orientation of polar molecules does not occur instantaneously, giving rise to a time-dependent behavior known as the relaxation process. Moreover, cells and tissue structures carry different electric charges. When subjected to a sudden electrical stimulation they require a finite time for charges to accumulate at the interfaces and to equilibrate. The accumulation of charges at the interfaces continues until a condition of equilibrium is re-established, leading to the relaxation phenomenon. Many types of relaxation processes can take place in biological tissues, owing to polar molecules and membrane charges.

When a dipole distribution is uniform, the positive charges of one dipole cancel the effect of the negative charges from another adjacent dipole. However, when the dipole distribution varies from point to point, this complete cancellation cannot occur. At an interface especially, the ends of the dipoles leave an uncancelled charge on the surface, which becomes an equivalent bound charge in the material. The relaxation process may therefore be illustrated by considering the response of bound charges to an applied electric field (Lin 2000b; Michaelson and Lin 1987). In this case, the dynamic force balance equation is given by

$$m\frac{d^2x}{dt^2} = qE - m\omega_s^2 x - m\nu\frac{dx}{dt} \qquad \textit{Eqn. 5.4.4}$$

where x is the displacement of a charged particle, E the applied electric field, ω_s is the characteristic frequency of the elastic, spring-mass system, ν is the particle collision frequency, and m and q are the mass and charge of the particle, respectively. The force exerted on the particle -- mass multiplied by particle acceleration on the left-hand side of equation (5.4.4), results from an electric driving force qE, an elastic restoring force in proportion to displacement x with elastic constant denoted as $m\ \omega_s^2$, and a retarding damping force proportional to velocity dx/dt with damping coefficient, $m\nu$.

After Fourier transformation and rearranging terms, equation (5.4.4) becomes

$$x(\omega) = [(q/m)E]/[\omega_s^2 - \omega^2 + j\omega\nu)] \qquad \textit{Eqn. 5.4.5}$$

Note that the equilibrium position for the charge (x = 0) represents local charge neutrality within the medium. When the charge is displaced from its equilibrium position, a dipole is established between the charge itself and the „hole" that is left behind and bound in the molecular and membrane structure. A dipole moment p is formed by the charge q times the displacement x. For a medium with volume-bound charge density ρ, the total dipole moment per unit volume or polarization P is

$$P = \rho p = [\rho(q^2/m)E]/[\omega_s^2 - \omega^2 + j\omega\nu)] \qquad \textit{Eqn. 5.4.6}$$

The electric flux density D may be expressed in terms of the electric field E and polarization P as

$$\vec{D} = \varepsilon_0\vec{E} + \vec{P} \qquad \textit{Eqn. 5.4.7}$$

For isotropic media, the permittivity may be related to D by the expression D = εE. These relations together with equation (5.4.6), give an equation for the permittivity,

$$\varepsilon(\omega) = \varepsilon_0[1 + (\omega_p^2)/(\omega_s^2 - \omega^2 + j\omega\nu)] \qquad \textit{Eqn. 5.4.8}$$

Where

$$\omega_p^2 = \rho q^2/m\varepsilon_0 \qquad \textit{Eqn. 5.4.9}$$

and ε_0 is the vacuum or free-space permittivity. Clearly, ε is a complex quantity and can be denoted by

$$\varepsilon = \varepsilon' - j\varepsilon'' \qquad \textit{Eqn. 5.4.10}$$

where ε′ and ε″ are the real and imaginary parts of the permittivity and can be obtained by equating the real and imaginary parts of equations (5.4.8) and (5.4.10). The relationship between electrical conductivity σ and ε″ is derived from Maxwell's equations and it is

$$\sigma = \omega\varepsilon'' \qquad \textit{Eqn. 5.4.11}$$

The velocity of bound charge motion v = dx/dt can be obtained from equation (5.4.5), such that

$$v(\omega) = [(q/m)E]/[\nu - j(\omega_s^2 - \omega^2)/\omega] \qquad \textit{Eqn. 5.4.12}$$

The finite velocity of charge motion in the material media indicates that the particle cannot respond instantaneously to a suddenly applied electric field. This time-delay phenomenon gives rise to a frequency-dependent behavior of charge displacement leading to changes in permittivity with frequency or the relaxation mechanism of interaction of electromagnetic radiation with biological systems. It is noteworthy that the same conclusions are reached by performing the inverse Fourier transforms of equations (5.4.8) and (5.4.12) and examining the phenomenon in the time domain. Note that the dependence of permittivity on source and characteristic frequencies ω, ω_p, and ω_s suggests that the charge displacement and motion given by equations (5.4.5) and (5.4.12), respectively, can also be resonant in nature.

I.5.2.3. Enhanced attraction between cells for pearl-chain formation

Molecules and cells under the influence of RF electric fields at frequencies up to 100 MHz would rearrange and form chains along the direction of the field. This phenomenon has been observed by many investigators and often referred to as the pearl-chain effect (Schwan 1982; Takashima and Schwan 1985). Pearl chains have been formed with biological materials such as erythrocytes or bacterial suspensions.

Under the influence of RF electric fields, electrical charges tend to accumulate on opposite cell surfaces to form induced dipoles, whose orientation changes with oscillations of the field. A dipole–dipole attraction occurs in the process. The attractive forces between the dipoles are enhanced when the cells are in close proximity to each other. The dipoles then align in the direction of the applied electric field and form chains of many cells or molecules. These chains are mostly single-stranded but they can be multi-stranded as well.

The pearl-chain effect has been extensively investigated, both experimentally and theoretically (Sher et al 1970; Schwan 1982; Takashima and Schwan 1985). It has been shown that, for frequencies up to about 100 MHz, the threshold electric field strength needed to produce the effect depends on frequency, cell or particle size, and pulsing parameters of the applied field. At higher frequencies, the induced dipoles have insufficient time to follow the oscillating field to change their directions. At low frequencies, the threshold is proportional to the 0.5 power of frequency, but it is nearly independent of frequency above 1 MHz. At 70 MHz, the threshold is around 10 $kV \cdot m^{-1}$ and it decreases markedly below 100 kHz to about 2 $kV \cdot m^{-1}$ at 500 Hz for an approximately 2.2 μm albumin coated silicon particle. The threshold field using a variety of particles with different sizes, shapes, and compositions indicates that particle properties do not significantly influence the threshold field strength. However, the threshold field has been demonstrated to be proportional to $R^{-1.5}$ on the particle size, where R is the radius of the particle.

Both pulsed (single or multiple pulses) and continuous wave (CW) fields are known to produce the pearl-chain effect, with a time constant that appears to be proportional to E^{-2}, where E is the field strength. A minimum amount of energy -- proportional to τE^2, where τ and E are the minimum pulse width and threshold field strength, respectively, is required to overcome the Brownian forces associated with random motion. Note that the minimum average field strength required of pulsed fields to produce pearl chains is equal to the minimum average field strength for CW fields, suggesting that pulsed field is no more effective than CW fields in inducing the pearl-chain effect. On the basis that the pearl-chain effect can be produced by a single pulse without a significant temperature rise, the pearl-chain effect is regarded as being caused by forces induced by RF electric field, not by a biologically significant temperature elevation (Sher et al 1970; Takashima and Schwan 1985).

The rotation of non-spherical cells - typical biological cells in a circularly polarized electric field - is a related electric-field induced, nonthermal effect with a high threshold field strength about 10 $kV \cdot m^{-1}$, depending on the cell and at frequencies up to the GHz range (Holzapfel et al 1982; Saito et al 1966).

I.5.2.4. Other RF fields-induced force effects

In addition to alignment of cells and larger molecules, other RF fields-induced effects such as shape changes and electroporation or permeabilization of cells have been documented (Gehl 2003; Weaver 1993). However, the mechanisms responsible for reversible and irreversible changes in membranes require much stronger fields. For example, millisecond wide pulses of up to 100 kV m^{-1} are required for permeabilization of cells using frequencies from 50 to 500 kHz.

I.5.2.5. Microwave auditory phenomenon

The microwave auditory phenomenon or microwave hearing effect pertains to the hearing of short-pulse, modulated microwave energy at high peak power by humans and laboratory animals (Lin 1980; 1990; 2007b). It involves electromagnetic waves whose frequency ranges from hundreds of MHz to tens of GHz. Experimental and theoretical studies have shown that the microwave auditory phenomenon does not arise from an interaction of microwave pulses directly with the auditory nerves or neurons along the auditory neurophysiological pathways of the central nervous system. Instead, the microwave pulse, upon absorption by soft tissues in the head, launches a thermoelastic wave of acoustic pressure that travels by bone conduction to the inner ear. There, it activates the cochlear receptors via the same process involved for normal hearing. The effect can arise, for example, at an incident energy density threshold of 400 $mJ \cdot m^{-2}$ for a single, 10-μs-wide pulse of 2450 MHz microwave energy, incident on the head of a human subject at an SAR threshold of 1.6 $kW \cdot kg^{-1}$. A single microwave pulse can be perceived as an acoustic

click or knocking sound, and a train of microwave pulses to the head can be sensed as a buzz or audible tune, with a pitch corresponding to the pulse repetition rate.

The microwave auditory effect is a biological effect of microwave radiation that occurs at a physiologically insignificant temperature rise with a known mechanism of interaction: the thermoelastic theory. Analyzes have shown that the minuscule, but rapid (~μs) rise in temperature (~10^{-6} °C) as a result of the absorption of pulsed microwave energy, creates a thermoelastic expansion of the soft tissue matter, which then launches an acoustic wave of pressure that travels to the cochlea, detected by the hair cells and relayed to the central auditory system for perception. In addition to the expected dependence of sound pressure on the strength of microwave pulses, the theoretical prediction and experimental measurements have shown a sound pressure or loudness that initially increases with pulse width and after reaching a peak value, and then, with further increases in pulse width, it starts to oscillate toward a lower pressure. Moreover, the induced sound frequency exhibits an acoustically resonant behavior and depends on head size. For example, the fundamental sound frequency or pitch varies inversely with the head radius: the smaller the radius, the higher the frequency. For rat-size heads, it predicts acoustic frequencies of 25 to 35 kHz in the ultrasonic range, which rats can easily hear. For the size of human heads, the theory predicts frequencies between 7 and 15 kHz, which are clearly within the audible range of humans and have been verified experimentally. Peak amplitudes of thermoelastic pressure waves have been computed for spherical head models approximating the size of rats, cats, infant and adult humans and exposed to 10 μs plane wave pulses at 1 kW·kg^{-1}. The corresponding incident peak power density is about 5 to 20 kW·m^{-2} for frequencies between 915 and 2450 MHz and the induced peak pressures were found to vary from approximately 350 to 1000 mPa. (The threshold pressure is 20 mPa for perception of sound at the cochlea by humans.)

I.5.2.6. Thermal effect and temperature elevation

Tissue heating is the most widely accepted mechanism of microwave radiation with biological systems. Obviously, RF energy is the driving force for any temperature elevation associated biological response. These effects can result from elevations of tissue temperature induced by RF energy deposited or absorbed in biological systems through local, partial-body or whole-body exposures.

As mentioned previously, the bulk RF properties of biological materials are characterized by complex permittivity and electrical conductivity. These bulk properties cause the electric fields and currents induced to be absorbed and dissipated in cells and tissues of the human body with thermal consequences. The extent of tissue temperature rise depends on the various pathways through which heat is transferred and removed from the tissue inside the body, heat exchange between the body surface (namely, the skin) and the external environment, and the thermoregulatory process, besides RF energy.

The temperature distribution, T = T(r,t), as a function of location and time inside the body may be modeled using the so-called Bioheat equation for RF exposures, where Qv(r) is the SAR distribution in W·m^{-3} or SAR divided by the volume density, ρ(r).

$$\nabla \cdot \left(K(\mathbf{r})\,\nabla T\right) + A(\mathbf{r},T) + Q_v(\mathbf{r}) - RL(\mathbf{r}) - B(\mathbf{r},T)\left(T - T_B\right) = C(\mathbf{r})\,\rho\,(\mathbf{r})\,\frac{\partial T}{\partial t} \quad [W/m^3] \quad \text{Eqn. 5.4.13}$$

The other terms on the left side of equation (5.4.13) represent heat transfer through passive conduction, where K [W/(m°C)] is the tissue thermal conductivity; A (W·m^{-3}) is metabolic heat production; RL (W·m^{-3}) is respiratory heat losses from the lungs; and the last term is heat exchange due to capillary blood perfusion, which is proportional to blood flow, and is represented by the parameter B [W/(°C m^{-3})], and the difference between blood and tissue temperature (T_B ~ T). Note that T_B is a function of time [i.e., $T_B = T_B(t)$]. The right side of equation (13) denotes the temperature increase (or decrease) per unit time. The thermal capacitance per unit volume is given by the product between the tissue specific heat, C [J/(kg °C)] and density, ρ (kg m^{-3}) (Lin and Bernardi 2007c).

For pulsed or brief applications of RF energy, the exposure duration is not long enough for significant conductive or convective heat transfer to contribute to tissue temperature rise. In this case, the time rate of initial rise in temperature (slope of transient temperature response curve) is related to SAR through,

$$SAR = \frac{c\,\Delta T}{\Delta t} \qquad \textit{Eqn. 5.4.14}$$

where ΔT is the temperature increment (°C), c is the specific heat capacity of tissue (J/kg°C), and Δt is the pulse width or duration of RF exposure. Thus, the rise in tissue temperature during the initial transient period of RF energy absorption is linearly proportional to SAR and inversely proportional to the specific heat capacity of tissue. As mentioned before, only a minuscule (~10^{-6} °C), physiologically insignificant temperature rise would result from the absorption of brief (~10 μs) but high peak pulse of RF energy, as in the case of microwave auditory effect.

For longer durations and especially at sufficiently high intensities, RF energy can produce temperature rises that can result in thermal effects and adversely impact functioning of the human body. As suggested by equation (5.4.13), the nature of temperature rise depends on the animal or tissue target and their thermal regulatory behavior and active compensation process. For local or partial body exposures, if the amount of RF energy absorbed is excessive, rapid temperature rise and local tissue damage can occur. Under moderate conditions, a temperature rise on the order of 1°C in humans and laboratory animals can result from an SAR input of 4 $W \cdot kg^{-1}$. However, this value falls within the normal range of human thermoregulatory capacity. Above this temperature or SAR value, disruption of work in trained rodents and primates has been reported for normal environmental conditions (ICNIRP 1998).

A major consideration of existing guidelines is the prevention of adverse biological effects resulting from either partial-body or whole-body exposures that could bring about temperature rises on the order of 1°C in humans and laboratory animals. Under ambient environmental conditions where the temperature and humidity are already elevated, the same SAR could produce body temperatures that reach well beyond normal levels permitted by the 1°C increment, and it could precipitate undesired heat-stress-related responses. The central premise of the exposure guidelines to protect exposed subjects against temperature increases could be eclipsed, breaching the temperature threshold for induction of adverse thermal effects. Thus, attention to temperature as a basic restriction may be a necessity in developing RF exposure guidelines. It should be noted that an increasing number of investigations are beginning to address the problem of human exposure to RF fields with a thermal analysis to estimate the temperature increment induced inside an exposed subject (Lin and Bernardi 2007c). It is emphasized that tissue heating during RF exposure is strongly influenced not only by the power dissipated in the local tissue mass, but also by how the absorption is distributed in the surrounding volume, by the thermal characteristics of the tissue and its unexposed neighboring tissues and, finally, by the heat exchange with the external environment.

I.6. DOSIMETRY

I.6.1. Introduction

Dosimetry is a term to represent “evaluation of dose”. It is therefore necessary to identify the dose metric or the quantity that is closely related to the effect of concern. Internal field in tissue is the primary cause for biological effect of RF fields regardless of the mechanism (Lin 2007a). Thus the induced electric field or the derived dosimetric quantities of specific absorption rate (SAR) and current density must be evaluated and correlated with the observed phenomenon. This is the role of dosimetry.

The thermal effect is the dominant established mechanism of biological and health effects of RF exposures. The current guidelines of human exposure are based on thermal effects. Elevation of deep-body temperature is closely related to the energy absorption rate in the whole body, or whole-body average SAR, when the exposure duration is more than the thermal time constant of the body (> 6 minutes) (ICNIRP 1998). Thus dosimetry of RF exposure is generally equivalent to the determination of

SAR in the body exposed to RF fields. It is noted that nerve and muscle stimulation are dominant at lower frequencies and are relevant for health effects up to approximately 10 MHz. This effect is related to electric currents inside the human body. Studies on contact and induced currents are summarized in Chapter I.6.3.

In the case of extremely localized exposures on some body part, significant temperature rise could occur around the exposed part resulting in thermal injury of the tissue regardless of the deep-body temperature elevation. Local SAR in the part of the body should be considered in this case. Temperature elevation in the body part, however, is not necessarily proportional to the local SAR because of the heat conduction. Thus dosimetry of RF exposure sometimes includes measurement or estimation of temperature as an adjunctive dose metric since it is more directly related to thermal injury.

The RF exposure guidelines are derived from the threshold of thermal effects in terms of SAR. A set of basic restrictions have been recommended in terms of SAR (ICNIRP 1998, IEEE Std. C95.1-2005). Local SAR limits are defined up to 10 GHz for ICNIRP guidelines while different applicable frequency region for local SAR limits are defined for different guidelines, e.g., up to 6 GHz for IEEE Std. C95.1-2005. The SAR is a quantity that is not directly measurable. Reference levels are provided in order to be used in practical assessment of compliance with the basic restrictions in actual exposure situations. Dosimetry plays an important role in the implementation of guidelines especially in the derivation of the reference levels. Dosimetry is also important when the exposure exceeds the reference levels. In this case it is necessary to examine whether the exposure actually exceeds the basic restriction or not by means of dosimetry.

Dosimetry is also important in scientific researches. The well-defined exposure conditions for biological experiments are required for adequate interpretation and reproducibility of the result. Since the International EMF project of WHO started, the importance of dosimetry has become much more recognized than before (Repacholi 1998). Minimum requirements for exposure systems were proposed for biological experiments addressing health concern of RF exposure due to wireless communications systems (Kuster and Schönborn 2000).

Various procedures are available for dosimetry. Those are classified into theoretical manner and experimental one. Each method has advantages and disadvantages. For example, theoretical dosimetry using realistic biological models can provide very fine spatial distribution of SAR, induced current density, and so on. Actual exposure conditions can be assumed in experimental dosimetry. Details are described in the following subchapters. It is highly recommended to select relevant dosimetry techniques for each purpose and to validate the evaluated dose by comparing between theoretical dosimetry and experimental dosimetry.

I.6.2. Biological models and materials

I.6.2.1. Physical phantom

For health risk assessment, it is necessary to evaluate SAR or induced current density in a human body exposed to high-frequency electromagnetic fields. It is very difficult to measure the internal E-field strength or temperature elevation in the actual human body using non-invasive methods (See Chapter I.6.4.2.). Therefore a surrogate of the human body, a so called “phantom” is used.

Phantoms for RF dosimetry are required to simulate the electrical properties equivalent to those of the human body. Various types of materials have been developed for phantoms and their references may be found in international standards on RF dosimetry (IEEE Std. C95.3-2002; IEC 62209-1-2005). In this subchapter, important characteristics of the phantoms are summarized.

Liquid, gel or jelly phantoms have widely been used for RF dosimetry because it is easy to prepare these materials and to adjust their electrical properties. Another advantage is easy to scan E-field sensors in these phantoms. One of the disadvantages of these materials is poor stability of the electrical properties due to water evaporation. Although dry phantoms with fine stability have also been developed, they require complex and skilled procedures and high cost (Kobayashi 1993; Nikawa 1996).

Because electrical properties depend on the type of tissues and organs and on frequency, the phantom must be fabricated for each condition. High-water-content tissues such as muscle are easily simulated with wet material although low-water-content tissues such as fat and bone are usually realized with dry material. A phantom recipe, optimized for each tissue or organ, can generally simulate the electrical properties from several hundred MHz to several GHz (Hartsgrove 1987; Okano 2000). It is however difficult to adjust the electrical properties of the phantom within small deviation, e.g., 5 %, from those of the actual biological tissues over broad frequency region. Different recipes optimized to the target electrical properties at each frequency are therefore used for strict measurements such as compliance tests (Chou et al 1984b; IEC 62209-1 2005). Nevertheless many studies to develop broad-band phantoms are now undertaken (Youngs 2002; Lazebnik 2005; Gabriel 2007a).

There are some difficulties for preparation and maintenance of the phantom:

- It is not easy to adjust both real part and imaginary part of the complex permittivity of the phantom to the target values simultaneously.
- The uncertainty of the electrical properties measured by commercially available systems is sometime considerable.
- Temperature change and water evaporation also affect the electrical properties of the phantom materials.

In order to overcome above difficulties, detailed investigations are necessary although it requires tedious work (Fukunaga 2004).

Generally homogeneous tissue is used for physical phantom, e.g., a standard head phantom for compliance tests of cellular phones (IEC 62209-1-2005, IEEE Std 1528-2003), or full-size models of the human body (Olsen 1979; Olsen and Giner 1989) because it is difficult to develop heterogeneous structure with liquid or jelly materials. However some heterogeneous phantoms were developed. For example, Stuchly et al (1987a) developed a whole-body phantom which simulates heterogeneous structure with solid material for bone within liquid phantom for high-water content tissues such as muscle. They measured E-field distributions by scanning with an E-field probe in the heterogeneous phantom. Several heterogeneous head phantoms have also been developed for SAR evaluation during use of a mobile wireless handset (Cleveland 1989, Okano 2000). Actual bones have been used in some heterogeneous phantoms.

I.6.2.2. Numerical model

Basic characteristics of the RF energy absorption in a human body have been established by simple models such as a sphere and a spheroid. Those have been systematically summarized (Durney 1986) and used for the rationale of RF safety guidelines.

One of the most important recent dosimetric techniques is the development of voxel based anatomical human-body models. A voxel is a small volume element or cube with a few millimeters on each side and identified with corresponding tissues and organs. A whole-body human voxel model can consist of several million voxels.

Various whole-body human models and laboratory animal models have been developed (Dimbylow 1997, Dimbylow 2005a, Dimbylow 2005b, Mason 2000b, Nagaoka 2004, Gandhi 1995, Dawson 1997, Lee 2006)). The voxel model developed by Brooks AFB Laboratory based on the database of the Visible Human Project (VHP), has been most used in RF dosimetry. Various dosimetric characteristics have been investigated with the VHP Man (Mason 2000a). However the disadvantage of VHP Man, i.e., significant deviation of the size and weight from the averaged values, has promoted development of other whole-body human voxel models with average height and weight which are specified in ICRP and other standards (Dimbylow 1997, Dimbylow 2005a, Nagaoka 2004). Recent investigation suggests that the calculated SAR values of those whole-body human voxel models are generally within the variation of the calculation results of the simple human models.

Other whole-body human voxel models such as various postured ones, children, fetuses and embryos, have also been developed and described in Chapter I.6.7.2. Most of those models have been developed by deforming the up-right standing adult human models (Dimbylow 2006a, Findlay 2005, Wang 2006c,

Cech 2007, Nagaoaka 2007, Kainz 2003). On the other hand, recently, whole-body child models have been developed based on MRI or CT database of children (Lee 2006, Kainz 2007, Christ et al 2008).

An anatomically based human voxel model is essential for FDTD calculation (See Chapter I.6.4.1.). Such a numerical model is developed commonly from MRI or CT scans. MRI or CT provides gray-scale image data as many transverse slices, at a designated spacing, from the head to feet of the human body; the resolution in each slice is on the order of several millimeters. MRI data are generally superior to CT data in identifying interior tissues because of high contrast images of soft tissues. Consequently MRI data are used more often in the development of numerical models. In order to develop a voxel model for FDTD calculation, original gray-scale data are interpreted into tissue types, referred to as segmentation. Since the gray scales in MR images do not correspond to tissue types directly, the tissue- and organ-identification processing has to be performed manually to a large extent. Even if software for automatic identification is applied, manual verification or correction is required. The highest complexity used in contemporary models of the whole human body is about 50 tissue types, and the finest resolution is about 1 mm.

Furthermore CAD-based human models have also been developed. The CAD models can easily move and rotate in any direction with 3-D CAD software and no limitation of their spatial resolution (Kainz 2007). The surfaces of the model can be readily deformed but care must be taken for the joints of the body to be correctly articulated. CAD models are usually segmented with voxels when applied to FDTD calculations.

Numerical dosimetry using these novel numerical models is now underway. Although it is important to pay attention to the results of the numerical dosimetry using the novel numerical models, it is also noted that most results of the realistic voxel models have generally agreed with those of the simple anatomical models of the whole body.

I.6.3. Dosimetry of contact and induced currents

In many industrial operations, RF current is induced in the body of operators, for example, plastic sealers. The magnitude of induced currents dependents on many factors, such as the electric and magnetic field strength, the polarization of the field, and the grounding conditions. In deed, operators of RF plastic sealers represent an occupational category that is highly exposed to RF electromagnetic fields (Wilen et al 2004).

The induced current flowing from the feet to the ground may reach values up to 600 mA. Table I.3.4 shows some representative values measured for typical polyvinylchloride (PVC) welders. Often, operators report of a heating sensation in their arms during the heating period, which clearly indicates that currents of several hundred milliamperes are induced per arm.

In general, the coupling of the body to the electric field is stronger than the coupling to the magnetic field. High electric fields around the electrode induce RF-currents flowing along the legs and torso. Absorption is at its maximum in the limbs where the current density increases considerably due to a small cross-section and high amount of low conductivity bone. Recent estimates strongly suggest that the local 10 g average SAR is about 10 $W \cdot kg^{-1}$ for the arm and 5-8 $W \cdot kg^{-1}$ for the foot with 100 mA current through that limb (Dimbylow 2001; Findlay and Dimbylow 2005). More than 70 percent of the power absorbed in the body is absorbed in the limbs. Good galvanic and capacitive contact to the ground increases considerably the current in the lower legs, and the current maximum shifts to the ankles. Additionally, the whole body average (WBA) SAR may increase by a factor of 2 as Chen and Gandhi (1991) have reported. Extending the hands over the electrode increased SAR by an additional factor of 2. Screening the room and adopting a sitting posture further increases SAR (Gandhi et al 1997). Therefore, it is important to isolate the feet, to keep the hands far away from the electrode and to avoid metallic structures near the HF heater. The effective blocking of the ankle current requires at least 10 cm of insulation between the feet and ground.

Measurements with a big body current transformer (Jokela et al 1999) indicate that the induced current is less sensitive to the variation in the electric field as a function of the distance. This is to be expected because the whole body integrates capacitive displacement current. For increasing distance the electric

field becomes more uniform which partly compensates for the decrease of the peak field at the electrode plane. Induced current is a very useful indicator of the exposure at frequencies below 30 MHz. Recent study (Kännälä et al 2008) indicates that the induced current even in the torso is mainly longitudinal when the distance from the electrode is greater than 30 cm. This suggests there exists a simple relationship between the current and both local and whole body average SAR and consequently non-invasive SAR assessment on the site may be possible by measuring the current induced by the electric field in the operator. There exists a local hot spot in the surface of the torso region closest to the applicator but this hot spot becomes critical only when the distance is less than 30 cm.

Any assessment of the exposure from dielectric heaters must take into account the effect of the off-time on the exposure. To determine the rms (root-mean-square) value of the electric field strength and current, the short-time peak value must be multiplied by the square root of the duty factor. Additionally, the electric field strength should be averaged spatially over the whole body (see Table I.3.4.).

Table I.3.4.: Electric and magnetic field strengths and currents induced in operators seated at PVC welding machines (AGNIR 2003).

Frequency (MHz)	Power (kW)	Ground	*E*max (V m^{-1})	*H*max (A m^{-1})	*Duty factor (DF)*	Operating ankle current (mA) DF corrected		Non-operating ankle current (mA) DF corrected	
27	-	Rubber/concrete	280	-	0,3	84	46	79	43
27	3		280	0,45	0,3	270	148	280	153
27	3	Concrete	212	-	0,3	85	47	55	30
27	1	Concrete	40	-	0,5	18	13	17	12
27	1,6	Concrete	150	0,16	0,3	100	55	-	-
28	15	Concrete	316	-	0,5	70	49	70	49
29	3	Concrete	37	-	0,2	27	12	19	8
36	1,6	Wood/concrete	30	-	0,5	17	12	13	9
45	7		100	-	0,16	60	24	60	24
50	3	Rubber/concrete	113	-	0,13	190	69	120	43
51	7	Concrete Concrete Wood	100	-	0,2	37	17	37	17

[1]Rubber; [2]Concrete

In the frequency range of 100 kHz-110 MHz, shocks and burns can result either from an individual touching an ungrounded metal object that has accumulated electric charges or from contact between a charged individual and a grounded metal object. Human body impedance is essential to estimate induced current in human. Kanai et al (1984) measured the contact body impedance and then developed a human-equivalent circuit model in the frequency range between 10 kHz and 3 MHz for limited number of human subjects. In the study of Chatterjee et al (1986), measurement has been conducted for 367 adult volunteers. Two contact areas are considered in the same frequency band, and the body impedance of a human is found to be inversely proportional to the body dimensions, i.e., the height for the case of finger-touching to the metal object by a human standing on the ground plane. They also showed that the threshold current is proportional to the cross section of the body. Kamimura et al (2005) proposed a simple equivalent circuit model in the frequency from 75 kHz to 15 MHz based on measured data for Japanese adults. Unlike the human-equivalent circuits proposed by Kanai et al (1984) and Chatterjee et al (1986), this model does not need to consider the circuit time constant for muscle.

Gandhi et al (1985) investigated current induced in the human body for plane wave exposure in the range between 3 and 30 MHz. They then found that vertically polarized electromagnetic waves induced high SAR around the ankle of a barefoot human standing on the ground plane. Foot currents were proportional to the frequency of incident wave in the frequency rage of 0.63-27.4 MHz, suggesting that the quasi-static approximation is roughly applicable up to this frequency region (Lin et al 1973), as also indicated by a

simple analytical model derived by Jokela et al (1994). Gandhi et al (1986) extended their investigation to the frequency region up to 50 MHz. Measured foot current was found to become maximal at 40 MHz, and the value was 780 mA for the incident power density of 10 W m^{-2}. This phenomenon is known as the whole-body resonance. Further studies have been conducted in the whole-body resonance frequency region (See Chapter I.6.4.1.).

From the studies by Gandhi et al (1986) and Chen and Gandhi (1989), quasi-static approximation is reasonably applicable to a few tens megahertz despite lack of detailed discussion. This implies that the human body is considered as good conductor in such frequency region, and thus computational techniques developed in the extremely low frequency (ELF) region is applicable in this frequency region. Several computational techniques were proposed and successfully applied to human body – ELF interactions. A review of computational methods based on quasi-static approximation can be found in the study by Stuchly and Dawson (2000).

In the 1980s, the impedance method was used for calculating induced current due to magnetic field (Gandhi et al 1984). In this method, the human is represented as impedance mesh. For each face of voxel, Kirchhoff voltages are equated to the electromotive force produced by the rate of change of magnetic field flux normal to the loop surface. The system of equations for loop currents is solved with the successive over-relaxation method. This method was applied for the calculation of induced current for 450-kHz induction heaters (Gandhi and Deford 1988) and electronic article surveillance (Li and Gandhi 2005) and for the calculation of body-equivalent impedance (Kamimura et al 2005).

In the 1990s, the scalar potential finite difference method (SPFD) was developed by Dawson et al, (1996). In this method, the equations for the electric field components in each voxel are derived from Maxwell's equation. The set of equations is solved using the conjugate gradient method. A main difference between the impedance method and the SPFD method is that the first is based on vectors and the second on scalars. Then, the computational cost for the latter is more reasonable than the former.

The Finite-Difference Time-Domain (FDTD) method was proposed by Yee in 1966 and well reviewed in the book by Taflove and Hagness (2005). Although this method has mainly been used for higher frequencies such as VHF and UHF bands, it is often applied for the analysis in the ELF and the intermediate frequency (IF) regions (300 Hz to 10 MHz) using frequency scaling techniques (Furse and Gandhi 1998; Gustrau et al 1999). Namely, actual simulation is performed at a frequency of several megahertz, and then the results are scaled down linearly by the ratio of the target frequency to the frequency assumed at the FDTD calculation. A quasi-static FDTD method has been proposed for proper evaluation of induced current due to electric or magnetic field separately. In this method, two plane waves propagating in the opposite directions are excited for cancellation of electric or magnetic field (Dawson et al 1996).

As mentioned above, there are various calculation methods for dosimetry in IF region. The best method would be different for specific applications. It is also noted that the post processing of the calculation, i.e., the spatial average of the induced current density, significantly affects the results. The detailed description of the procedure is necessary to hold repeatability of the numerical calculation (Dimbylow 2005a; Hirata and Fujiwara 2007).

I.6.4. Specific absorption rates (SAR)

I.6.4.1. Numerical calculation

Numerical methods

Early dosimetry calculations were mainly focused on dielectric spheres, circular cylinders and prolate spheroid bodies (Durney 1980, Durney et al 1986; Lin 1986), which were considered as a highly simplified human head or human body model. For a homogeneous or stratiform structure, an analytical solution is possible to these models under the condition of plane-wave exposure. Although the analytical

solutions do not provide detailed dosimetry information for actual human bodies, they contribute to qualitative analyzes, especially for the resonance in the whole body.

From the end of 1970s, numerical calculation methods have attracted more attentions due to their advantage in modeling the anatomy of a human body. The most notable one is the method of moments (MoM) in which the human body was divided into many blocks and the corresponding dielectric properties were assigned in each block to model the anatomical structure (Liversy and Chen 1974; Chen and Guru 1977; Hagmann et al 1979, Gandhi 1980). The MoM is based on solving linear simultaneous equations for unknown electric fields in the blocks so that its computational scale is proportional to the number of blocks squared. Furthermore, the block number is inversely proportional to the size of the blocks. This limits its application at frequencies higher than several hundred MHz because smaller size blocks are required for higher frequencies, i.e., shorter wavelength. The MoM was mainly used in 1980s for numerical calculations of the whole-body average SAR. In fact, the reference levels of incident electric field or power density in various guidelines were derived mainly from the MoM calculations of the whole-body average SAR. It was demonstrated that an incident electric field or power density under the reference levels never yield a whole-body average SAR larger than the basic restrictions.

Since 1990s, the finite-difference time-domain (FDTD) method (Taflove and Hagness 2005) becomes the most widely accepted means for SAR calculation. The FDTD method is based on Maxwell's time-domain equations. The discretization of the Maxwell's equations is based on a Yee cell approach (Yee 1966). A special feature of the Yee cell is that the electric field (E) and magnetic field (H) components are staggered one half space-cell apart. That is , the E field is assigned at the edges of the Yee cell and the H field is assigned on the faces of the Yee cell to facilitate the differencing scheme. The computational scale of FDTD method is proportional to the number of cells, which enables to apply to millimeter-resolution human models with several millions of cells (See Chapter I.6.2.2.). The fine block models, that is, voxel models, can be used for electromagnetic simulations over 1 GHz.

In applying the FDTD method for numerical dosimetry calculation, the Yee cells correspond completely to the voxels in biological models. By assigning the corresponding permittivity and conductivity to each voxel, one can easily model the anatomical tissues and organs, and calculate the internal electric and magnetic fields. As for the permittivity and conductivity values of each tissue, the parametric models using 4-Cole-Cole equations based on measured data from 10 Hz to 20 GHz by Gabriel constitute the most widely accepted database (Gabriel 1996).

Since the FDTD method requires discretization of the entire domain over which the solution is to be calculated, it is impossible to discretize an infinite space because of the finite memory capability of computers. The calculation domain, therefore, must be truncated to a finite size. Once the infinite space is truncated to a finite size, absorbing boundary conditions must be applied to the outside boundaries of the calculation domain in order to simulate the non-reflective nature of open space. One of the most popular and effective absorbing boundary conditions is known as the perfectly matched layers (PML) (Berenger 1994). The basic concept of PML is based on impedance matching to minimize reflections. Theoretically speaking, semi-infinite PML provides a perfect absorption for traveling waves with any angle of incidence. However, in practice, the PML must be terminated, because of finite computer memory. Typically termination is accomplished using a perfect electric conductor, which introduces a reflection back into the calculation domain. The performance of PML therefore is characterized by three parameters: (1) thickness, (2) conductivity profile, and (3) the reflection coefficient at normal incidence.

In addition to the FDTD method, some hybrid methods have also been developed for SAR calculation. A typical one is the combination of the ray-tracing method and the FDTD method (Bernardi et al 2000b), in which the ray-tracing method is used to calculate the incident electric field, e.g., base station environment, and the FDTD method is used to calculate the SAR. Such an approach avoids the huge calculation burden in modeling the actual electromagnetic environment with the Yee cells. Another typical one is the combination of the MoM and the FDTD method (Mangoud et al 2000, Mochizuki et al 2004). Such an approach is commonly used in the SAR calculation of a helical antenna next to a human head because the FDTD method is not suited for modeling a curved wire.

Whole-body average SAR

In the 2000's Dimbylow (2002; 2005), Mason et al (2000a), Nagaoka et al (2004), and (Wang 2006c) conducted whole-body SAR calculation by the FDTD method together with anatomically based high-resolution models of the human body human. The whole-body average SAR for adult voxel models exposed to plane wave at 1 W/m^{-2} are equal or less than 0.04 W kg^{-1} at the whole-body resonance frequency, e.g., about 70 MHz for adult male in free space, and 0.008 W kg^{-1} at 2 GHz. However, for children, nearly 40-% increases in the whole-body SAR have been reported at the body resonance frequency and around 2 GHz (See Chapter I.6.7.2.).

Spatial peak SAR

The numerical calculation in the human head for various wireless communication devices has become an area of active research since the 1990s (Dimbylow and Mann 1994; Gandhi et al 1996; Watanabe et al 1996; Schönborn et al 1998; Lazzi and Gandhi 1998; Bernardi et al 2000; Wang and Fujiwara 2002a; Wang et al 2004a). The main efforts were focused on calculation of the spatial peak SAR as averaged over one-gram or ten-grams of tissue.

In order to investigate causes of the differences in the evaluated spatial peak SARs among different FDTD calculations with different head models, it is essential to use a common procedure to derive such a spatial-averaged SAR. Otherwise unnecessary confusion will occur especially in the case of complex tissue structure. ICNIRP guidelines define the spatial peak SAR as a contiguous 10-g tissue (ICNIRP 1998) while IEEE defines 10-g cubic tissue (IEEE Std. C95.1-2005). IEEE has also defined procedures to evaluate spatial average SAR for voxel human models (IEEE Std. C95.3-2002).

Recent inter-laboratory comparison using the same human head models and the mobile phone models reported that the maximum 10-g SARs for an adult head model with a mobile phone model at the cheek position for 1-W output power are 3.92 W kg^{-1} (+/- 0.35 W kg^{-1} STD) and 5.12 W kg^{-1} (+/- 1.78 W kg^{-1} STD) at 835 MHz and 1900 MHz, respectively (Beard et al 2006). For mobile antennas, the maximum electromagnetic absorption is found at the superficial tissues and the SAR decreases with depth into the head. No maximum local SAR occurs in the deep region of the head below 6 GHz (Dimbylow and Mann 1994; Gandhi et al 1996; Watanabe et al 1996; Schönborn et al 1998; Lazzi and Gandhi 1998; Bernardi et al 2000a; Wang and Fujiwara 2002a; Wang et al 2004a) while it occurs in a homogeneous sphere model exposed to plane wave (Kritikos 1975).

For a cellular telephone, the spatial peak SAR is strongly dependent on the antenna types. Previous studies suggest that the maximum one-gram or ten-gram averaged spatial peak SAR is induced by a helical antenna with a metal box, and this is followed by the 1/4-wavelength monopole antenna, the 3/8- or 5/8-wavelength monopole antenna, the 1/2-wavelength dipole, and the back-mounted planar-inverted-F antenna. It is noted that the actual cellular phones generally cause lower SAR than the half-wavelength dipole antenna (Ali 2007). These findings can be explained by the current distribution along the antenna and box, and the distance between the antenna and box and the head, because the current on the antenna and box, or the incident magnetic field, is directly related to the spatial peak SAR (Kuster 1992).

The ankle SAR is also an important index for the whole-body resonance region, especially for the case where a human stands on the ground plane. Limb current can be measured easily and linked to the ankle SAR. Dimbylow conducted some numerical simulations with voxel human models for investigating the relationship between the ankle SAR and limb current (Dimbylow 1988; Dimbylow 1991; Dimbylow 2001; Dimbylow 2006b).

I.6.4.2. Measurement

In order to evaluate the SAR and induced current density inside of the human body exposed to EMF, various measurement methods have been developed (IEEE C95.3-2002). For the measurements, human-body phantoms are frequently used (See Chapter I.6.2.1.) while in other cases, volunteers or cadavers have been used (Conover et al 1992; Hill 1984; Swicord et al 1999). In order to keep the repeatability of the measurement, human-body phantoms are preferable although the human-body phantoms are usually

homogeneous or very simple heterogeneous structure such as bones and high-water content material which has similar permittivity to those of muscle and brain.

For local SAR measurement, there are two methods. One is E-field measurement and another is temperature measurement. E-field measurement is used for compliance tests of mobile phones because the sensitivity is relatively high and 3-D measurement is available if liquid-type phantom is used. The procedures of the compliance tests of wireless terminals such as mobile phones based on E-field measurement have been standardized internationally between 300 MHz and 3 GHz (IEC 62209-1-2005; IEEE Std. 1528-2003).

It is also noted that E-field probes must be calibrated at each frequency and in phantom materials with the electrical properties adjusted to those of the biological tissues at the frequency of interest. Thus an E-field probe which is only calibrated in free space cannot be used to measure E-field strength in phantoms that have electrical properties different from free space. Various calibration systems for E-field probes have been developed (Hill 1982; Meier 1996; Jokela 1998) and summarized in the international standards (IEC 62209-1-2005; IEEE Std. 1528-2003; IEEE Std C95.3-2002).

For the temperature method, SAR is derived from the following equation (see Chapter I.5.2.6.).

$$SAR = c\left.\frac{dT}{dt}\right|_{t \to 0} \qquad \textit{Eqn. 6.4.1}$$

where c is specific heat, T is temperature, and t is duration of exposure. This equation means that temperature elevation is proportional to SAR if conduction and other thermal diffusion mechanism can be ignored during a brief RF exposure. The advantage of the temperature method is non/low-invasiveness because infra-red cameras or very-small temperature probes such as fiber-optic probes or small thermistor probes are available (Guy and Chou 1986; Okano 2000). Liquid-crystal has also been used for non-invasive temperature measurement in a phantom (Suzuki 2006).

The temperature-measurement methods are very effective for dosimetry in small laboratory animals such as rats and mice for *in vivo* studies (Lin et al 1977; Swicord et al 1999; Wake et al 2007a) and of *in vitro* studies (Pickard 2000; Schuderer 2004c). Whole-body averaged SAR can also be evaluated with calorimeters (Padilla and Bixby 1986; Olsen and Griner 1989).

I.6.5. Temperature elevation

Temperature elevation is one of the dominant factors to induce adverse health effects. The temperature elevation inside the human body, however, cannot be measured directly. In order to overcome this difficulty, computational schemes for calculating temperature variations have become very useful . A well-known bioheat equation was proposed by Pennes (1948) for following the time variation of temperatures in a human body (see Eqn. 5.4.13). When discretized, this formula has the capability of handling inhomogeneous media, and takes into account the heat conduction, basal metabolism, blood flow, heat production due to RF heating, and heat transfer between body and air. Increased blood flow and perspiration rate with the temperature elevations were also incorporated into the equation (Spiegel 1984; Hoque and Gandhi 1988). The effectiveness of the bioheat equation is discussed by Wissler (1998). The bioheat equation did not account for thermoregulatory response until later. Thermal responses were first modeled by Stolwijk and Hardy (1977) with highly-simplified human bodies . The effectiveness of this thermal response model was verified by Foster and Adair (2004) on the basis of experimental data with human volunteers (Adair et al 2003). This thermoregulatory model was incorporated into the bioheat equation by Bernardi et al (2003). This combined formula enables computation of the temperature elevation in an anatomically-based human body model in the time domain. As a drawback, the computational cost of this scheme was large. Recently, alternating direction implicit (ADI) finite-difference formulation was successfully applied to the bioheat equation for reducing the computational cost (Pisa et al 2003; Ibrahiem et al 2005).

For localized exposure, the thermal time constants in human tissues are mainly determined by the balance of the rate of RF power deposition and a time constant for heat convection by blood flow and for heat conduction. Due to tissue inhomogeneity and the frequency-dependent penetration depth of EM waves, thermal time constants cannot be estimated in a straightforward manner. A 1-D model analysis is discussed by Johnson and Guy (1972) and Foster et al (1998). For whole-body and intense-localized exposure, the absorbed EM energy compared with basal metabolism leads to body-core temperature elevation (Guy et al 1975; Adair et al 2003). Body-core temperature elevation is caused by EM energy absorbed in different body parts and then transferred to body core via blood flow. Due to body-core temperature elevation, some thermoregulatory responses activate to maintain body temperature (Adair and Black 2003). Due to these factors, the thermal time constant of the body core would be somewhat larger than that of temperature elevation in a body part due to localized exposure. It is also noted that some tissues can increase their blood flow even when body core temperature is not significantly increased, and this mechanism can play an important role in limiting temperature rises for intense localized exposure (Wainwright 2003).

When considering the temperature elevation due to whole-body exposure, the temporal variation of blood temperature should be taken into account. This factor was ignored in the original bioheat equation; Bernardi et al (2003) incorporated the blood temperature into the bioheat equation and found that for plane-wave exposures at 40 MHz with a power density of 2 W m^{-2}, the maximum steady-state temperature elevation at the ankle reached 0.7 ^{o}C where whole-body resonance occurs in a man on the perfect ground. An additional finding was that the presence of the thermoregulatory response reduces temperature elevations especially in the body core. Hirata et al (2007b) investigated elevation in body-core temperature for far-field exposures at a whole-body resonance frequency (65 MHz) and 2 GHz. In particular, they discuss the effect of perspiration on body-core temperature elevation. The variability of temperature elevation caused by sweating was found to be 30%. A whole-body average SAR of 4.5 W kg^{-1} was required for a body-core temperature elevation of 1 ^{o}C after 60-min exposure in the model of human with the lower sweating coefficients. The thermal time constant in the body core was 20 min, which was shown to be almost the same at frequencies of 65 MHz and 2 GHz. In these studies, however, the effect of clothing on temperature elevation was not taken into account. Nelson et al (2005) proposed a scheme for determining heat transfer coefficient of garments suitable for high-resolution computations. Further research would be required to quantify the effect of clothing on temperature elevation.

The temperature elevation in the eye is often singled out since intense localized exposure on the eye was shown to induce a variety of effects, including cataract formation. One of the key studies was conducted by Guy et al (1975), in which microwave-induced cataract formation was reported in rabbit eyes. To computationally predict temperature elevation, Emery et al (1975) developed a heat transfer model for the rabbit eye. In this early model, the eye was assumed to be an object thermally isolated from the rest of head on the basis of l high blood flow rates in the choroids and tissues surrounding the eyeball. Lagendijk (1982) employed improved heat transfer coefficients between the eye and air and that between the eye and the rest of the head in anatomically-based human models to quantify the temperature elevation in the eye.

Bernardi et al (1998) investigated the temperature elevation at millimeter frequency bands used in WLAN applications. For frequencies above 6 GHz, the maximum temperature elevation (0.04 ^{o}C for the incident power density of 10 W m^{-2}) appears near the surface of the eye due to small penetration depth of EM waves. For the same reason, the maximum temperature elevation in the lens decreases with increasing frequency. Hirata et al (2000) obtained a maximum temperature elevation of 0.06 ^{o}C in the lens for the same incident power density at 0.6 – 6 GHz. The temperature elevations estimated by Bernardi et al (1998) and Hirata et al (2000) were comparable at 6 GHz.

The results obtained using improved heat transfer models that take into account blood flow in the choroidal and retinal tissues and heat transfer in the whole head showed a maximum temperature elevation of 0.3^{o}C for an eye-average SAR of 2 W kg^{-1} (Hirata 2005; Buccella et al 2007; Wainwright 2007). As expected, a correlation was observed between the average eye SAR and the maximum temperature elevation in the lens. However, a lower temperature elevation was reported in Flyckt et al (2007) using a heat transfer model involving discrete vasculatures (DIVA). (This is more of a side issue)With the rapid progress of wireless communications system, considerable attention has been

devoted to the temperature elevation due to handset antennas. Several studies with anatomically-based head models have been published on this issue (Wang and Fujiwara 1999; Van Leeuwen et al 1999; Bernardi et al 2000a, 2001; Wainwright 2000; Gandhi et al 2001; Hirata and Shiozawa 2003; Hirata et al 2003, 2006c; Ibrahiem et al 2005). In these studies, the blood temperature in humans is assumed to be constant, since the output power of handset antenna is on the order of a few hundred mW which is much lower than the basal metabolic rate of an adult male of 100 W or more. However, using DIVA modeling, Van Leeuwen et al (1999) t showed that the local temperature elevation around the blood vessel could be lower due to the cooling effect of blood flow in the vessel. Wainwright (2000) applied the finite-element method which can better simulate surface curvatures of the human head to calculate SAR and temperature elevation. The results obtained were comparable to those reported in the above mentioned works. The issue of overestimating surface areas in the FDTD voxel models, which could potentially result in excessive heat transfer from human head to air was investigated by Samaras et al (2006) to help improve the accuracy for FDTD modeling of the bioheat equation (Neufeld et al 2007). Nevertheless, the thermal time constant of temperature elevation of 6-8 min was consistent with other studies (Wang and Fujiwara 1999; Bernardi et al 2000a). Note that it takes 30 min or more to reach a thermal steady state in human head models.

A direct comparison of the maximum temperature elevations reported in different papers is difficult since different handset antennas and head models were used. In addition, different average schemes, masses, and algorithms are used for the computation of peak spatial-average SAR and temperature. An analysis of the correlation between peak spatial-average SAR and maximum temperature elevations in the head was conducted by Hirata and Shiozawa (2003) for different frequencies, polarizations, feeding positions, and antennas. They showed fairly good correlations between peak spatial-average SAR and maximum temperature elevation in the head excluding the pinna. In addition, Hirata et al (2006c) investigated the correlation of maximum temperature elevation in the head with peak SAR calculated by different average schemes and masses. Under steady state conditions for exposure times of 60 min or longer, or, the maximum temperature elevation in the head reached 2.4 or 1.4 ^{o}C, respectively depending on whether the pinna is included or excluded from the head, at a peak SAR of 10 W kg^{-1} for 10g of contiguous tissue. At a peak SAR of 10 W kg-1 for averages over a 10g cubic volume, the maximum temperature elevation in a head without the pinna was 2 °C, which is higher than that for contiguous tissues (Bernardi et al 2000a; Wainwright 2000; Hirata and Shiozawa 2003; Hirata et al 2008b; Razmadze et al 2009).

It should be noted that a high degree of spatial correlation between peak SAR and maximum steady state temperature elevation for durations of 60 min or longer is not expected, especially for exposures of large biological bodies with efficient thermal transfer characteristics (Hirata 2006b,c). Heat transfer by passive diffusion and active blood flow convection in biological tissues have the averaging effect of flattening the temperature elevations even though RF heat deposition from SAR is local and instantaneous. It is also worth noting that the proximity of mobile phone handset and battery to the head allows them to behave as heat sources to cause temperature elevation (Bernardi et al 2001; Gandhi et al 2001; Ibrahim et al 2005). The temperature elevations have been shown to rise by 1^{o}C or more, comparable to that caused by RF energy deposition.

I.6.6. Uncertainties of RF dosimetry

t is important for risk assessment to investigate the uncertainty associated with dosimetry. Uncertainty is defined as the amount by which the estimated value may depart from the true value. The expanded uncertainty with a coverage factor of k=2 means the confidence interval is nearly 95 %. The general concept and evaluation procedure are described in ISO/IEC Guide to the Expression of Uncertainty in Measurement (ISO/IEC 1995).

The expanded uncertainty (k=2) of the SAR measurement for compliance tests of mobile phones has been reported to be within 30 % (IEC 62209-1-2005; IEEE Std. 1528-2003). The dominant factors are probe calibration, boundary effect, test sample positioning and device holder (IEEE Std. 1528-2003).

However, the uncertainty of SAR calculations has not been established. One of the important factors to cause uncertainty in FDTD calculations is “staircase modeling” (Holland 1993). It has been reported that

the numerical calculation of temperature elevation with voxel models is also significantly affected by the staircase modeling (Samaras 2006). Boundary conditions that require truncating the region used for FDTD calculations is also a source of uncertainty although reported significance of this effect (PML boundaries) on the whole-body SAR has not been consistent among related studies (Wang et al 2006c; Findlay et al 2006; Laakso et al 2007). Some standard organizations have undertaken inter-laboratory comparison for evaluating the uncertainty of SAR calculations. A recent study reported that a standard deviation of 30 % was found in 12 separate SAR calculations of heads exposed to the near field of a mobile phone with the same voxel models and exposure conditions (Beard et al 2006).

For risk assessment, additional uncertainty factors, i.e., the generality and/or worst-case situation of human models and exposure conditions should be taken into consideration. This is especially important when realistic voxel human models are used. Simple models have long been considered as typical worst-case models, some reports of comparison of various realistic voxel models have been published (Kainz et al 2005a). It is however noted that the simple models frequently may provide considerably higher doses (SAR or induced current density) or artificial phenomena such as the appearance of maximum local SAR in the deep region of the model (Lin 2002b). It has been reported that the standard deviation of whole-body average SARs from 20 MHz to 2.4 GHz for six adult voxel models can reach up to 40% (Conil et al 2008). An inter-laboratory comparison of whole-body SAR calculations and the uncertainty of the calculations are given in Dimbylow et al (2008).

For *in vivo* animal studies, there have been several investigations on dosimetric uncertainties (Wang et al 2004b; Wang et al 2006b; Kuster et al 2006; Wake et al 2007). Specifically, the estimated uncertainty of SAR was within 15 %, i.e., 0.6 dB, for a large-scale *in vivo* study involving 300 rats during the 2-year exposure period (Wake 2007a). However, in another systematic uncertainty evaluation the expanded uncertainty (k=2) was found to be greater than 2 dB (Kuster et al 2006). Thus, the expanded uncertainty (k=2) dosimetry in animal studies is between 1 to 2 dB if the models and exposure conditions are strictly defined although careful consideration of additional uncertainty factors of the models and exposure conditions are necessary for risk assessment.

I.6.7. Other topics

I.6.7.1. Dosimetry for biological and epidemiological studies

In vivo studies

There are two general types of exposure situations in experiments designed to investigate effects of RF exposures *in vivo*: near-field and far field exposures. The near-field or local body exposure is used to simulate exposures by a mobile phone handset held near the head of a user. The far-field or whole-body exposure is used to simulate exposures to the RF fields radiated from broadcasting stations or mobile phone base stations.

In the near-field exposure situation, the exposure is localized so that the local SAR is significantly higher than the whole body average SAR. The ratio of the maximum local SAR to the whole body average SAR can exceed 100 in the actual human exposure to mobile phones. It is therefore required that the exposure system should provide such the localized exposure condition in animals. This condition is not easy to achieve in animals as the body size of animals is much smaller than humans while the antenna size is determined by the wavelength. In recent studies localization of exposure has been provided by sophisticated exposure system design with appropriate dosimetry. The dosimetry has made use of anatomically realistic numerical animal models with different body sizes which takes into account animal growth during long term exposure studies.

One example of near field systems is carousel-type exposure systems for rats or mice (Swicord et al 1999; Schönborn et al 2004; Wake et al 2007a). A dipole or monopole antenna is located in the center of circularly arranged animal holders, like in a carousel, with the animal's head toward the antenna. The reported ratio of brain average to whole-body average SAR is 5 – 9 in these systems when operating at

900 MHz and 1.5 GHz, respectively. The ratio is much less than that of actual mobile phone exposures in humans and is also less than the ratio of maximum local SAR to whole-body SAR in the basic restrictions of the current exposure guidelines (ICNIRP 1998; IEEE C95.1-2005). Another example of near-field exposure concerns effects on the eyes of rabbits (Guy et al 1975; Kramar et al 1975; Wake et al 2007b) and primates (Kues et al 1985; Kamimura et al 1994). In these studies waveguide antennas or applicators for microwave hyperthermia treatment were used. Dosimetry on these studies was based on temperature measurements made with a probe inserted in the animal eye (Guy et al 1975; Kramar et al 1975; Kues et al 1985). More recently numerical calculations have provided more detailed data on SAR and temperature elevation in and around the eye (Hirata 2007b) (See Chapter I.6.5.).

It should be noted that near-field, localized exposure systems usually require constrain of animals to keep constant the relative position of body to the radiating structure. This allows better-defined exposure conditions for more precise dosimetry. On the other hand it causes restriction in the experimental design. In some cases, animals can move even in the holder for constraint, resulting in variation of exposure. Thus, many factors could affect the actual exposure during the experiment (Kuster 2000).

The far-field or whole-body exposure systems allow movement of animals without or with minimal restraint. A whole-body exposure apparatus used in an experiment involving long term exposure of transgenic mice reported an elevated risk of lymphoma at a whole body SAR ranged from 0.008 – 4.2 W kg^{-1}(Repacholi et al 1997). The large exposure uncertainty was mainly attributed to unconstrained condition of exposure of the animals . The exposure was improved in the subsequent replication studies using Ferris wheel type exposure systems with animal holders located on the perimeter of the wheel excited by a loop antenna in the center (Utteridge et al 2002; Oberto et al 2007).

The Ferris wheel exposure system consists of a radial electromagnetic cavity formed by parallel circular plates mounted on a polycarbonate frame. A tunable transition from a 50-Ω coaxial feed line excites a cylindrical TEM wave that propagates in a carousel of symmetrically arranged mice, equidistant from the excitation. The mice, restrained in plastic tubes inserted through circular holes in the plates, are held co-polarized with the incident electric field to maximize the absorption of RF energy (Balzano et al 2000). While the Ferris wheel system allows more accurate dosimetry, constraining the animals causes stress on the animals during long term exposure experiment and limits the exposure duration per day and constraint. In addition the design makes impedance matching more sensitive to the cavity load, e.g., the size of mice.

Reverberation chamber exposure systems have been developed to overcome some of the identified limitations restrictions, principally with more extensive computer simulation of exposure scenarios (Kainz 2006). However, the dosimetry provides SAR values characterized by stochastic properties as the SAR varies in a random manner. A wide variability of exposure is expected for individual animals, akin to those associated with the Repacholi et al (1997) experiment.

In vitro studies

In vitro biological experiments usually involve cells contained within flasks or Petri dishes and are exposed to a well-defined EM field. Several types of exposure systems have been developed for *in vitro* studies. Many of them are closed systems based on a waveguide or a TEM cell (Schönborn 2001; De Prisco et al 2008). The coupling with field depends on the polarization, or direction of electric field relative to the surface of the medium. The E-polarization has a weak coupling, i.e. low efficiency to provide RF energy to the medium with cells and the perturbation of the field in the presence of the culture dish is small. An efficiency of 0.04 W kg^{-1} per 1 W input power was reported for TEM cell system with E-polarization (Schönborn et al 2001). Standing waves are sometimes utilized to improve the efficiency. It should be noted that standing waves have minimum H-field at the location of maximum E field, and vice versa. The exposure condition *in vitro* could be different from that in the free space because of this fact.

A far field or anechoic chamber exposure system has also been used in some experiments (Iyama et al 2004). The signal of IMT-2000 (2.14 GHz) is radiated from a horn antenna and led through a dielectric

lens to be focused on a plate on which culture dishes are arranged. This system aims to expose many dishes at a time with fairly homogeneous and efficient conditions.

Dosimetry for RF *in vitro* experiments characterizes the SAR distribution in the medium containing the cell specimen. The energy absorbed by cells is taken to be the same as absorption in the medium. Thus the meaning of dosimetry is different from the dosimetry for *in vivo* or human volunteer experiments in this case.

In general, exposure should be uniform for the entire cell population to achieve a well-defined condition of exposure. It is difficult, however, to realize uniform exposures throughout the whole culture dishes especially for high frequency RF fields with the wavelengths in the medium comparable to the dimension of the dishes (Kuster et al 2000). Moreover, the SAR distribution is sensitive to the presence of meniscus at the perimeter of the culture dish. Numerical simulation of SAR in a Petri dish with meniscus revealed that it not only affects the distributions but also the average values of SAR in the dish (Schuderer et al 2003).

Non-uniform SAR can cause significant temperature gradients in the medium. Temperature gradient can then cause convective transfer in the medium, resulting in changes in temperature of the cell. The movement of fluid can also cause shear stress on the cells. These phenomena make the experimental results difficult to interpret. Toroidal convection has been observed when a culture dish was exposed to millimeter waves resulting in periodical fluctuations of temperature in the medium (Khizhnyak 1996).

Human studies

Human volunteer studies can provide important data for risk assessment as they directly assess the effects on humans. Exposure levels are low in these experiments due to ethical reasons and subtle effects on neurological functions are of principal interest. Thus, the target organ is the central nervous systems (CNS). A particular hypothesis in human studies is that the site of interaction is localized. Results of detailed dosimetry have been reported recently for several exposure systems used in human studies (Boutry et al 2008). The exposure systems examined include those used in Turku (Haarala et al 2007, Krause et al 2007), Swinburne (Loughran et al 2005), and Zurich (Regel et al 2006). The Turku and Swinburne studies employed modified commercial mobile phones. The dosimetric analysis is based on numerical approach which has been validated by comparing with measurements in phantoms. SAR distributions in the brain are estimated for each exposure system in detail. The results show that the highly exposed part is limited and the location is different from phone to phone. In fact, the peak spatial SAR within the human cortex can vary by more than a factor of 20 from phone to phone (Kuster et al 2004).

In some studies a base station antenna located at a distance of 3 m from the subject sitting in an anechoic room was employed to simulate far-field, whole-body exposure humans. Numerical calculations by FDTD method revealed that the whole-body average SAR is 6.2 μ W kg^{-1}, and that the average and peak (1 g average) SAR in brain is 11 and 73μ W kg^{-1}, respectively for 1 V m^{-1} incident power density of UMTS signal at 2.1 GHz (Regel et al 2006).

Epidemiological studies

Assessment of exposure plays a crucial role in epidemiology investigations. However, the exposure metric for this assessment is not easily defined. A plausible hypothesis is that the tissue which experiences the stronger and the more prolonged exposure could have more risk of diseases. Thus an exposure metric could be defined as the energy absorbed at a point due to the exposure as

$$Dose = \sum_{i=1}^{N} SAR(t_i) \cdot t_i$$

where t_i is the time interval of the exposure with dose rate SAR (t_i) (Balzano 1999). This metric is a function of tissue or the location exposed to RF fields. It is necessary for dosimetry in this case to identify and quantify SAR distribution in tissue, radiation source, duration of exposure, characteristics of the field, output power, incident field distribution, etc. for individuals. It is important to take into account a priori the exposure contributions from all relevant sources and not to restrict the evaluation to one source so

long as it is not demonstrated that this source is dominant with respect to others (Neubauer et al 2007). However, data accuracy and reliability are difficult to ascertain because data acquisition in many of the RF epidemiology research are from self reporting. In addition the radiated power varies significantly, as it is controlled by the wireless systems which depend on the status of the communication signal (Wiart 2000). Thus there are many uncertainties in the dosimetry for epidemiological study of RF exposure.

I.6.7.2. Dosimetry for children, fetuses, and embryos

The dosimetry for children and the unborn from RF exposure has gained considerable attention given their special status during human development and growth. Aside from the physical size, the variation of tissue electromagnetic properties as a function of age may have significant influence on RF energy absorption and distribution.

Dielectric properties

The most widely accepted database of dielectric property for biological tissues lacks data for children (Gabriel 1996). The dielectric properties, i.e., permittivity and conductivity, are considered to decrease with age due to the changes of water content and organic composition of tissues. This consideration has been demonstrated in Peyman et al (2001) i.e., compared to adult rats, at 900 MHz, 16% and 43% higher conductivity were found for the brain and skull of new-born rats, respectively, which suggests a possibility of SAR increase due to the higher tissue conductivity. They also reported relatively lower increase of permittivity, i.e., 9.9% and 33%. Recently, they have reported a significant dependence of the dielectric properties of the white matter and spinal cord on age while no age-related variation was found for the gray matter (Peyman et al 2007). The establishment of a database for children's dielectric properties should be an essential and urgent task.

Spatial peak SAR for cellular telephones

In 1996, Gandhi et al reported a deeper penetration and considerable increase in the spatial peak SAR in children's heads for cellular telephones by using linearly scaled child head models with adult dielectric properties (Gandhi et al 1996; Gandhi and Kang 2002). An increase up to 50% in the one-gram averaged spatial peak SAR was found in a child head model. On the other hand, Kuster et al developed two child head models from magnetic resonance imaging (MRI) data and used them to conduct similar calculations (Schönborn et al 1998). Their results revealed no significant differences in the peak SARs between adults and children, and also for children approximated as scaled adults. To clarify, Wang and Fujiwara (2003) repeated Gandhi's and Kuster's calculations using a scaled Japanese head model. The scaling was conducted based on a statistical database of child heads in order to get a better approximation. They were able to reproduce both Gandhi's and Kuster's calculation results, suggesting that the contradictory conclusions drawn are due to differences in their calculation conditions, specifically, whether the results were normalized with the output power or with the antenna current. Moreover, the authors pointed out the need of further studies on standardization of the averaging procedure used for spatial peak SAR calculations. The same conclusion was reached based on a statistical approach from another study (Bit-Babik et al 2005).

A multi-laboratory collaboration (Beard et al 2006) for computational comparison of spatial peak SAR was conducted by an international task force comprising 14 groups from government, academic and industrial research institutions. The study protocol specified the use of the Specific Anthropomorphic Mannequin (SAM) head (without a pinna) model designed for mobile phone compliance measurement (IEC 62209-1-2005; IEEE Std 1528-2003), an anatomically correct adult head model and a scaled 7-year-old head model. Each institution used a different FDTD code and independently positioned the cellular telephone and head models following the protocol. Each participant ran twelve simulations to fill a data sheet comprising the three head models, two frequencies (835 and 1900 MHz), and two phone positions (cheek and tilt). The spatial peak SARs for one- and ten-grams averages were required according to the IEEE C95.3-2002 averaging procedure, and tissues considered in the SAR averaging volume included all tissues, head only tissues, and pinna only tissues. In addition, the SAR values were normalized to both the antenna input power and feed-point current. The results were very different for the two frequencies and

phone positions. For 1900 MHz cell phones, the peak 1 and 10 g SAR values in the head, pinna and average tissue of the adult model were consistently higher than those for the child model, either normalized to the antenna current or power for the cheek and tilt positions. However, a majority of the SARs were higher in the child than the adult model, especially for the 835 MHz phone in tilt position when normalized to antenna current.

A study by Hadjem et al (2005) using child-sized (CS) and child-like (CL) head models showed that since the brain is closer to the mobile phone in the case of the CS or CL heads, the SAR in the child brain models is slightly higher than that of the adult. The difference between the heads of 5 and 10 year olds and between the CS head and the CL head are very small, except for brain tissues at 900 MHz. More recently Wiart et al (2008) reported that exposure of the cerebral cortex of children is higher than in adults.

It should be note that Wang et al (2006) derived an empirical formula for dielectric properties in children according to Lichtenecker's exponential law for the complex permittivity of various tissues as a function of the total body water (TBW). Following validation by comparing with the measured data for rats (Peyman et al 2001), they showed that the adjusted dielectric properties of children do not affect significantly the spatial peak SAR or the penetration depth. The finding can be qualitatively explained as cancellation of the increased conductivity and decreased electric field penetrating into tissue because of the same degree of increase between the conductivity and permittivity in children compared to the adults.

Whole-body average SAR

Some published studies (Wang et al 2006, Dimbylow and Bolch 2007, Conil et al 2008, Nagaoka et al 2008, Kuehn et al 2009) showed that in the frequency ranges of body resonance (~100 MHz) and from 1 to 4 GHz for bodies shorter than 1.3 m in height (corresponding approximately to a child of 8 years or younger) at the recommended ICNIRP reference level the induced SARs could be up to 40% higher than the current basic restriction under worst case conditions. Since the shape and tissue properties of child models can influence whole body SAR in children, there have been several efforts in developing more realistic child models based on actual anatomy (Lee 2006; Kainz 2007, Christ et al 2008, Nagaoka 2008).

Dosimetry of fetuses

Numerical dosimetry of fetuses was mainly conducted for metal detectors at several MHz (Kainz et al 2003) and MRI equipment at several 10's of MHz (Wu 2006; Hand 2006). By modeling only the abdomen region and using nine different pregnancy stages, Wu et al showed significant increase of SAR and temperature elevation in patients at late pregnancy stage. Recently, whole-body pregnant female models have been developed. Dimbylow (2007) developed a pregnant female model by combining a non-pregnant female model and a mathematical fetus model. Nagaoka et al (2007) reported a more realistic whole-body pregnant female model by embedding a MRI-based voxel fetal model inside a non-pregnant female model. The induced current and SAR of fetuses are shown to be generally similar or lower than those of the mother. At microwave frequencies, the electromagnetic fields attenuate more rapid in the pregnant body so that the energy reaching the fetus is insignificant. Using Nagaoka's pregnant female model, SAR calculations for the fetus in a mother holding a cellular phone around her lower abdomen showed that the averaged SAR in the fetus is lower than that in the mother (Togashi et al 2008).

More detailed calculations, it would require more knowledge of dosimetric parameters of pregnant females and fetuses. Note that Kawai et al (2006) reported that the conductivity of rabbit fetuses is 1.3 times of that of muscle at 150 MHz. The same situation is true for temperature simulation because the thresholds of thermal effects in fetuses and embryos have not been established in terms of SAR.

I.6.7.3. Dosimetry for implant issues

ICNIRP and other exposure guidelines (ICNIRP 1998; IEEE Std C95.1-2005) do not address human bodies with implanted metal objects. The guidelines do not consider the enhancement of SAR around the objects and the malfunction of medical implant equipments such as cardiac pacemakers. The number of the persons who have such implant objects within their bodies however rapidly increasing. Therefore the

dosimetry of a human body with implant objects is an important topic. The enhancement of SAR due to metal implant objects are described in this chapter while the malfunction, i.e., electromagnetic interference, of medical implant devices and equipments may be found elsewhere (Hayes et al 1997; Irnich 2002; Kainz et al 2005b; Silny 2007).

Implant objects with metal parts generally cause enhancement of SAR in a human body exposed to RF fields. The enhancement depends on various factors. One of the important factors is geometrical resonance. That is, excessive enhancement of local SAR may occur when the size of a metal object in the body is comparable to wavelength in tissues. McIntosh et al (2005) also reported on resonance like multi-reflection between the metal object embedded in the cranial bone and the skin surface. The size, shape, location and orientation of a metal object implanted in a human body can affect the enhancement of the SAR and SAR distribution. These dependencies are summarized in a recent review paper (Virtanen et al 2006).

Although significant enhancement of local SAR can occur around an implanted metal object, the impact on SAR values is limited if the local SAR is averaged over 1 g and 10 g of tissues. A study with realistic heterogeneous head model exposed to a dipole antenna at 900, 1800, and 2450 MHz showed that the factor of enhancement of the maximum SAR is 3 and 2 for 1-g SAR and 10-g SAR, respectively (Virtanen 2007). Temperature simulation demonstrated that the temperature elevation due to enhanced SAR around a metal object is not higher than 1 ^{o}C for exposure of RF safety guidelines (McIntosh et al 2005).

It has been suggested that temperature elevation would be a more appropriate measure to evaluate the safety of the thermal effects due to implant metal objects in a human body exposed to RF fields instead of 1-g SAR or 10-g SAR (Virtanen et al 2006). Experimental investigation is also highly recommended, although there may be many difficulties. Detailed knowledge of thermal and physiological parameters should be considered in the thermal simulation. A very high-resolution and low-perturbation sensor is required to experimentally evaluate the effect of the implanted metal object.

I.6.7.4. Dosimetry for millimeter and THz wave exposure

Above 30 GHz, the power absorption of EMF waves becomes increasingly superficial, where the penetration depth, i.e., the distance from the boundary of a medium to the point at which the field strength or induced current density have been reduced to 1/e of their values at the boundary, is 1 mm or less. Absorption of these high-frequency EMF waves takes place in a very shallow region and depends strongly on the incident power density, thus the basic restriction of the safety guidelines are more appropriately set in term of the incident power density instead of SAR.

An important issue in millimeter and THz-wave exposures is the paucity of available data on dielectrical properties. Recent measurements of the dielectrical properties of the human and mouse skin in the 37-100 GHz frequency range showed good agreement among various reports (Alekseev and Ziskin 2007; Alekseev et al 2008a; Gabriel et al 2007b).

Dosimetric calculations of power density, penetration depth, and SAR using a single layer and multilayer models of skin showed that. Alekseev et al 2008b) the thin stratum corneum (SC), has little influence on the interaction of mm waves with skin. In contrast, the thick SC in the palm played the role of a matching layer and significantly increased power deposition. In addition, the palmar skin manifested a broad peak in reflection within the 83-277 GHz range. The viable epidermis plus dermis, containing a large amount of free water, greatly attenuated mm wave energy. Therefore, the deeper fat layer had little effect on the power density and SAR profiles. The appearance of a moderate SAR peak in the 42-62 GHz frequency range within the skin at a depth of 0.3-0.4 mm. Millimeter waves penetrate into the human skin deep enough (0.65 mm at 42 GHz) to affect most skin structures located in the epidermis and dermis (Alekseev et al 2008b). Moreover, in murine models, mm waves penetrate deep enough into tissue to reach muscle. However, in human skin, mm waves are mostly absorbed within the skin. Therefore, when extrapolating the effects of mm waves found in animals to humans, it is important to take into account the possible involvement of muscle in animal effects (Alekseev et al 2008a).

A recent report showed that the Pennes bioheat equation is not adequate to quantify mm wave heating of the skin at high blood flow rates (Alekseev and Ziskin 2009). It was necessary to incorporate an "effective" thermal conductivity to obtain a hybrid bioheat equation. The presence of the fat layer (non-specific tissue) resulted in the appearance of a significant temperature gradient (up to a few ^{o}C) between the dermis and muscle layer which increased with the fat layer thickness.

It should be noted that other models have been used for millimeter wave dosimetry (Riu et al 1997; Walters 2000). However, because the voxel size of realistic human models is comparable to the wavelength in tissue, significant errors can occur in numerical calculation of EMF power absorption. Nevertheless, simple models have been used to predict a threshold of the thermal sensation due to temperature elevation (about 0.06 ^{o}C) at the skin surface. Foster and Glaser (2007) calculated the corresponding threshold in terms of incident power density from 10 to 94 GHz. They found that the threshold of incident power density decreases as frequency increases, i.e., 200 W m^{-2} at 10 GHz to 50 W m^{-2} at 94 GHz, and that the threshold at 94 GHz is the same level of the IR (50 THz) radiation. The above calculations generally assumed short-term and large-area exposures. Short-term exposures can ignore the effects due to thermo-physiological response of the human body. For large-area exposure conditions, very simple 1-D human surface models can be used because the thermal diffusion at the tangential direction can be ignored. Further dosimetric investigations considering actual complex conditions are therefore necessary. Gustrau and Bahr have reported detailed dosimetry of human skin and eye exposed to 77 GHz millimeter-wave which is used for radar systems for adaptive cruise control (Gustrau and Bahr 2002).

I.6.7.5. Microdosimetry

Microdosimetry refers to the determination of the microscopic distribution of absorbed energy. It deals with the quantitative study of the distributions of EM fields imparted in cellular and subcellular biological structures and their relationship to biological effects.

Recently a growing attention has been devoted to RF-microdosimetry. WHO has considered such item in the research priorities agenda (http://www.who.int/peh-emf/research/children/en/index1.html) and it has been argument of discussion in many workshops, one of them specifically devoted to this topic (Physical Effects of Pulsed RF Fields at Microscopic and Molecular Dimensions – Microdosimetry, Dresden 2001).

Supposing that RF bioeffects could manifest under exposure conditions that do not present detectable levels of heating of the body, then the search for biophysical mechanisms involving energy transfer over molecular dimensions and the field strength knowledge at this level is needed to establish a quantification of the effect (Schwan 1999; Apollonio et al 2000; Valberg et al 2007). In order to achieve this result it is crucial to relate the average field absorbed by the whole system, organ or tissue, obtained through macroscopic dosimetry, to the local field induced inside cells and their compartments. While the EM field distribution inside the exposed biological system can be determined via macroscopic dosimetry (Chapter I.6.7.) the problem must at the single cell level be solved considering $\lambda >> d$, where λ is the EM field wavelength and d the maximum dimension of the cell. It can be worthwhile to recall that, in the frequency range of interest, λ is around tens of centimeters and d is of the order of μm. This assumption implies quasi-static conditions, where the EM wave has a constant phase in all the points of the cell (Liu and Cleary 1995; Postow and Swicord 1996; Kotnik and Miklavcic 2000; Simeonova and Gimsa 2006).

Induced field at the microscopic level may in part be instantaneous (due to electronic and atomic polarizability) and also may have proper time delays (due to the polarization phenomena involved in the specific structures). A fist example has been suggested (Liu and Cleary 1995; Kotnik and Miklavcic 2000) in a dielectric model of a cell. The different microscopic structures imply differentiation in the polarization phenomena involved. As a consequence there will be different time (frequency) responses from some parts of the dielectric model in local EM field absorption (Kotnik and Miklavcic 2000).

Some attention has been devoted to quantifying the differences in absorption at microscopic and/or molecular levels and determining if these differences, or associated temperature gradients and energy transfer, could influence biological functions. However, there seems to be a general consensus (Schwan 1999; Foster 2000; Pickard and Moros 2001) that microthermal heating has to be considered negligible (Schäfer and Schwan 1943). The cell, cell membrane, and structures of molecular size, that may absorb

more energy than surrounding matter, nonetheless are likely to remain essentially at the same temperature as the surrounding matter.

Moreover, marked field discontinuities at microscopic level of cell membrane (up to 20 fold (Kotnik and Miklavcic 2000; Munoz et al 2003)) are plausible due to differences in dielectric properties (e.g., between protein and lipid regions in the cell membrane and cytoplasm and extracellular medium) (Liu and Cleary 1995; Kotnik and Miklavcic 2000; Apollonio et al 2000). For such reason, in approaching microdosimetric studies, membrane dielectric models, valid through a wide frequency range, seem to be particularly appropriate (Kotnik and Miklavcic 2000; Simeonova and Gimsa 2006). The molecular structure and dynamics of lipid membranes and of protein domains in membranes have been extensively explored, both theoretically (Klosgen et al 1996; Simeonova and Gimsa 2006; Hu et al 2006) and experimentally (Bordi 1993; Chan et al 1997; Asami 2002; Feldman et al 2003; Bonincontro and Cametti 2004), although much more work remains to be accomplished. Membranes exhibit a complex anisotropic, frequency-dependent structure and proteins (in both membranes and cytoplasm) can have markedly different dielectric permittivity and conductivity with respect to those of the surrounding media (Bordi et al 1993; Klosgen et al 1996; Simeonova and Gimsa 2006).

The EM field solution can be held by two principal approaches: the first considers analytical methods applied to simplified cell shapes: spherical and spheroidal multi-shell models (Liu and Cleary1995; Apollonio et al 2000; Kotnik and Miklavcic 2000, 2006; Gimsa and Wachner 2001; Simeonova and Gimsa 2006). The main advantages of this approach are the simplicity of the technique (Stratton 1941) and the possibility to furnish simplified formula to evaluate influence of different parameters (Postow and Swicord 1996; Wachner et al 2002; Maswiwat et al 2007). The second way is through numerical techniques that allow irregular shape of the cells and the inhomogeneous spatial distribution of the fields due to realistic shapes (Sebastian et al 2001; 2004, Munoz 2003, 2004; Stewart et al 2005; Smith et al 2006; Pucihar et al 2006). With such techniques some authors have also approached the mesoscopic problem of cells assemblies (Pavlin et al 2002).

Further research on microdosimetry applying dielectric theory to cells and subcellular entities is needed to achieve a better understanding of the possibility that in the absence of overall temperature change, RF radiation might influence biochemical processes over microscopic dimensions and sub-microsecond times.

Microdosimetry is of interest also in all cases where the interaction of fields with biological materials at the microscopic level is studied for biomedical reasons. This is the case in the rapidly evolving field related to electric field manipulation of cells, electroporation, and a variety of possible laboratory diagnostic techniques based on dielectric spectroscopy (Pucihar et al 2001, 2007; Stewart et al 2004; Hu et al 2005; Frey et al 2006; Vasilkoski et al 2006; Gowrishankar et al 2006; Munoz et al 2006).

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II. Review of Experimental Studies of RF Biological Effects (100 kHz – 300 GHz)

ICNIRP Standing Committee II – Biology

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II.1. INTRODUCTION

This report reviews the results of biological studies of the effects of exposure to radiofrequency (RF) radiation published after the World Health Organization (WHO) Environmental Health Criteria monograph on electromagnetic fields in the range 300 Hz – 300 GHz (WHO 1993). Biological studies are taken here to include laboratory experiments using volunteers, as well as those using various animal species such as rats or mice and those using cultured cells. The report focuses on individual volunteer, animal and in vitro experimental studies published after 1993, but it also takes account of the numerous national and international reviews of RF studies published since that date. Of particular note are those published by the International Commission on Non-Ionizing Radiation Protection (ICNIRP 1997, 2001), the Health Council of the Netherlands (HCN 2004-2009), the UK Independent Expert Group on Mobile Phones (IEGMP 2000), the Royal Society of Canada Expert Panel on RF (Krewski et al 2001a, b; 2007), the UK independent Advisory Group on Non-Ionizing Radiation Protection (AGNIR 2001, 2003), the US National Council for Radiation Protection (NCRP 2003), the French Agency for Environmental Health Safety (AFSSE 2003, 2005) and the Swedish Radiation Protection Authority (SSI 2004-2008). Papers written in languages other than English have been included in the present review.

WHO (1993) reviewed a large number of biological studies of the effects of RF radiation. The studies often used levels sufficient to induce considerable heating at frequencies commonly used for industrial, scientific and medical purposes, most commonly 915 and 2450 MHz. The RF radiation was usually continuous wave (CW), sometimes pulse-modulated and occasionally amplitude-modulated at extremely low frequencies (ELF). In subsequent years, the rapid increase in wireless telecommunications, particularly those used in mobile telephony resulted in public health concerns regarding the increasingly ubiquitous exposure to the complex but generally low-level RF signals emitted by such devices. A number of large, well-coordinated programs of biological research have been undertaken, often at frequencies of around 900 and 1800 MHz which are typical of GSM (Global System for Mobile Telecommunications) signals. More recently frequencies at around 2100 MHz, typical of the third generation systems (e.g. UTMS, Universal Mobile Telecommunication System) have been investigated. Much of this effort has been centered in Europe. Past and ongoing multi-laboratory European biological research programs have included: CEMFEC, GUARD, PERFORM, RAMP 2001, and REFLEX. These projects comprise volunteer studies of physiological effects; animal studies of cancer, reproduction and other end-points; and *in vitro* studies of genotoxicity, gene expression, etc. In addition, many countries support their own biological research programs, e.g. Australia, China, Denmark, Finland, France, Germany, Italy, Japan, the Netherlands, South Korea, Sweden, Switzerland and the UK.

These research programs have to varying degrees been coordinated through regular meetings, workshops and conferences; an approach strongly supported by WHO through the publication of its Research Agenda (www.who.int/emf/research/en/). Such an approach favors the coordinated replication of notable study outcomes that are of some concern. These studies include, for example, the report of lower levels of well-being in volunteers following UTMS exposure (Zwamborn et al 2003), reports of increased permeability of the blood-brain barrier and the number of dark-staining neurons, thought to indicate degenerating neurons, in rat brains following exposure to GSM-type signals (Salford et al 2003), and reports by several groups of increased levels of heat shock proteins (hsps) and of increased DNA strand breaks in cultured human fibroblasts following exposure to low level GSM-type radiation (e.g. Leszczynski et al 2002; Kwee et al 2001; Diem et al 2005). In addition, an increase in the number of single- and double-strand DNA breaks in rats exposed to pulsed and CW 2.45 GHz fields was reported by Lai and Singh (1995, 1996a,b), and a two-fold increase in the incidence of lymphoblastic lymphomas in transgenic mice was reported by Repacholi et al (1997). Replications of some of these and other studies are published and discussed in this review; other replication studies are currently in progress.

Different types of laboratory study contribute to the evaluation of possible risks to human health in different ways. Studies using volunteers can give valuable insight into the transient, physiological effects of acute exposure of human populations. Animal studies provide the opportunity to investigate possible effects of prolonged exposure on reproductive outcome for example, or on the incidence of cancer, that cannot be conducted using volunteers. They play an essential role in evaluating the integrated responses of the systems of the body, particularly the nervous, endocrine and immune systems. However, the direct

extrapolation of the outcome of such studies to human populations may be limited because of species differences such as lifespan or tumor susceptibility. Finally, experimental observations on cultured cells, tissue samples and biological molecules can, in principle, give insight into the basic mechanisms by which effects might be induced in more complex organisms. Again however, anomalous cellular behavior generated by the culture conditions and other factors may limit the extrapolation of such data to humans.

Criteria for assessing the strength of these experimental data include the adequacy of experimental design, the statistical analysis of the data, and the avoidance of possible confounding that might otherwise result in a misleading or erroneous conclusion (Repacholi and Cardis 1997). In this respect, it is a fundamental principle of scientific investigation that effects described in one laboratory can be repeated in the same and in other laboratories, providing the appropriate procedures and protocols are followed. Thus replication of an effect by an independent laboratory considerably strengthens the view that any effect represents a true response. In addition, the identification of a dose-response relationship would clearly strengthen the view that an agent such as RF interacts in a systematic way with a biological process. Finally, a lack of conflict with current scientific understanding further strengthens the plausibility of any effect. However, these criteria can, of course, only serve as a guide to judgment.

This review specifically examines the biological evidence for different proposed RF interaction mechanisms (Chapter II.2.), the evidence for genotoxic and non-genotoxic effects in cultured cells (Chapter II.3.) and the evidence for genotoxicity and effects on cancer, reproduction and development, the nervous, endocrine, cardiovascular, immune and hematological systems in animals (Chapter II.4.). A review of human laboratory studies (Chapter II.5.), which covers effects on the nervous system and behavior, and the endocrine and cardiovascular systems is followed by a summary and conclusions (Chapter II.6.).

II.2. BIOLOGICAL EVIDENCE FOR INTERACTION MECHANISMS

In this chapter the biological evidence for interaction mechanisms is reviewed. Physical aspects are addressed in Chapter I.5. In addition, this chapter focuses on non-thermal interactions; RF-induced heating and thermal dosimetry are also addressed in Chapter I.5. and I.6. The distinction between thermal and non-thermal interactions is rather important, particularly in the interpretation of biological studies, and has been discussed recently by Glaser (2005) and Foster and Glaser (2007). RF-induced heating is well understood, resulting from the dielectric relaxation of water and other molecules and the translational motion of ions. From a biophysical point of view, Glaser (2005) notes that a mechanism is non-thermal if the interaction of the electric or magnetic vector of the RF field leads to specific effects other than heating. Pragmatically, however, experimental effects are often termed non-thermal when they are not accompanied by a predictable or measurable temperature increase. In practice it is difficult to ensure that small localized temperature increases, in a cell culture for example, have not occurred during RF exposure. In addition, Foster and Glaser (2007) note that cells possess various temperature sensitive molecules that can activate cellular responses to small changes in temperature, sometimes of less than $0.1^{o}C$. One class of thermally sensitive molecules are the temperature-dependent 'riboswitches', RNA sensors that direct gene expression through changes in RNA conformation (e.g. Serganov and Patel 2007) and are, for example, involved in the heat-shock response and the expression of hsps (e.g. Chowdhury et al 2003; Shamovsky et al 2006). Another class are the transient receptor potential (TRP) family of membrane ion channel proteins which respond to a variety of changes in their local environment including temperature (e.g. Moran et al 2004; Bandell et al 2007); both warm and cold sensitive TRP ion channels have been described (Benham et al 2003; Patapoutian et al 2003). As noted by Glaser (2005), subtle temperature effects may occur following low level RF exposure that would be part of the normal repertoire of cellular responses to the small temperature changes encountered in everyday life and are therefore unlikely to be of any health significance.

Another important consideration is the plausibility of any proposed non-thermal mechanism of interaction. In terms of energy per RF photon for example, the available energy of $4x10^{-5}$ eV at 1 GHz is much lower than the average thermal energy of molecules at room temperature ($3x10^{-2}$ eV), and very much lower than the strength of a weak chemical bond (around 1 eV) or a threshold for ionization

(around 10 eV), suggesting that RF will be unable to cause direct damage to biological molecules through chemical bond disruption or ionization.

However, because of the ubiquity of RF exposure and the remaining uncertainties regarding possible low-level effects, it is crucial to perform theoretical analyzes and biophysical investigations in order to yield information on plausible interaction mechanisms and suggest further research.

II.2.1. Biophysical studies

Several processes have been considered that could lead to biological effects. They have been reviewed (Adair 2003; Challis 2005; Foster and Glaser 2007) and are summarized below.

II.2.1.1. Dielectric properties

All living matter contains electrical charges (ions, electrically polarized molecules such as water, etc) and insulating materials such as lipids; it is therefore a weakly conducting medium (called a dielectric). The dielectric properties of tissues determine the net electromagnetic energy absorbed (specific absorption or SA; J kg^{-1}) which is ultimately converted into heat due to an increase in molecular translational and rotational kinetic energy (see Chapter I.5.). Above about 500 MHz, macroscopic loss mechanisms shift from predominantly ionic conduction to more equal contributions from ionic conduction and dielectric relaxation (Pickard and Moros 2001). Increased knowledge of the dielectric properties of biological tissues has enabled a more accurate derivation of the dosimetric relationship between exposure, specific energy absorption rate (SAR; W kg^{-1}) and the elevation of tissue temperature (see Chapter I.6.).

II.2.1.2. Magnetite

Magnetite (Fe_3O_4), found in magnetosomes that are present in the human body, including brain tissue, is a strong absorber of RF radiation between 500 MHz and 10 GHz (Kirschvink 1996). However, it is present at very low concentrations (5-100 ppb) in human tissues and the resultant heating should be biologically unimportant at localized SARs below guideline levels (Adair 1994; Kirschvink 1996; Pickard and Moros 2001).

A preliminary study by Cranfield and co-workers of the effect of exposure of the magnetite-containing bacterium *Magnetospirillum magnetotacticum* using a GSM 900 MHz handset reported that exposure increased the proportion of cell deaths (Cranfield et al 2003a). However, the experimental protocol was only briefly described and dosimetry was inadequate, although the exposure was presumably below guideline levels so the results were potentially of interest. In later work in which cells were exposed inside a waveguide with proper dosimetry (GSM-1800 MHz, with an SAR of up to 2 W kg^{-1}), there was no effect on cell viability (Cranfield et al 2003b). The ELF magnetic fields produced by the handset were not present in the later study and the authors speculated that this might have accounted for the difference in study outcome. However, there is no clear evidence that low-level ELF magnetic fields are associated with increased cell death (ICNIRP 2003; WHO 2007).

The presence of magnetite in human tissues is not associated with any known function as it is in birds and other species and is unlikely to result in increased heating under RF exposure or in non-thermal biological effects.

II.2.1.3. Demodulation

The possibility that biological tissue can demodulate an RF signal through the non-linear conversion of RF energy, generating a signal within the tissue at the modulation frequency, is of considerable interest (Foster and Repacholi 2004). Generally, RF signals are modulated at low frequencies to which neurons and neuronal networks such as those in the CNS are particularly sensitive, and so even weak demodulation could be significant. Ionic conduction through membrane ion channels results in demodulation but only at frequencies below about 10-20 MHz (Pickard and Barsoum 1981; Pickard and Moros 2001). So demodulation at higher frequencies would need to involve other biological components

and an investigation to look for these is currently underway in the UK using a setup composed of a doubly resonant cavity (Balzano 2002, 2003; Balzano and Sheppard 2003; Balzano et al 2008). In this study, the CW RF test signal at 900 MHz irradiates a sample of cultured cells held in the resonant cavity tuned to this frequency; any non-linear processes will generate a second harmonic at 1800 MHz, to which the cavity is also tuned. The sensitivity of the doubly resonant cavity system is such that it should allow the detection of one or two non-linear oscillators per 1000 cells (Balzano et al 2008).

II.2.1.4. Radical pair mechanism

The "radical pair mechanism" is one of the most plausible hypotheses for explaining the biological effects of low-level (< 1 mT) static and ELF magnetic fields (see Brocklehurst and McLauchlan 1996; Timmel and Henbest 2004). Scission of a covalent bond in biological molecules results in the formation of a radical pair, usually as an intermediate stage in some metabolic reaction. If the radical pair lives long enough, a magnetic field can affect the probability of radical recombination and thereby change the reaction yield. There is ample experimental evidence for this mechanism in biochemical systems but less so for biological processes, although some support has been given by the evidence from studies on animal navigation mechanisms (Ritz et al 2004).

For RF fields, transitions between non-degenerate states should be induced when the transition frequency equals the RF frequency. For most biomolecules, these frequencies are below 100 MHz, although molecules containing transition metal ions can have hyperfine splitting of 1000 MHz or more (Challis 2005). Generally, this phenomenon is unlikely to take place in normal solvents in living tissues such as water because of the very short lifetime of the radical pair, typically tens of nanoseconds. However, conditions are more favorable in membranes and/or bound to an enzyme where the radical pair may be held in close proximity for longer periods, possibly microseconds, increasing the possibility of singlet-triplet mixing (Brocklehurst and McLauchlan 1996; Eveson et al 2000). Ritz and colleagues have reported that an RF field of 1.315 MHz can disorient the magnetic compass orientation of the migratory bird species *Erithicus rubecula* in agreement with theoretical predictions based on a radical pair mechanism (Thalau et al 2005). Further experimental work should explore possible biological effects in mammalian cells and animal models.

II.2.2. Biochemical studies

Biochemical studies are carried out using cell free systems such as proteins, membranes, liposomes, etc. Such investigations can yield useful information concerning the validity of hypotheses made at the physical or biophysical level and about the way RF exposure might trigger biological effects, possibly leading to health effects.

II.2.2.1. Biological macromolecules

A few studies have addressed the effects of RF exposure on the structure and function of biological macromolecules such as proteins or DNA. The hypothesis being investigated is that absorption of RF energy by these molecules could modify their structure and/or perhaps their behavior, as first noted by Frölich (1968).

Bohr and Bohr have performed a series of experiments on globular proteins, particularly β-lactoglobulin. RF was applied for 5 s in a microwave oven at 2.45 GHz and 800 W, causing a ~0.3°C temperature increase in the protein solution. In the first publication (Bohr and Bohr 2000a) using optical rotational dispersion, the authors showed that exposure accelerated conformational changes of the protein and in a second paper (Bohr and Bohr 2000b) they reported an enhancement of folding and denaturation of the protein. These observations were interpreted as evidence of coherent RF excitation of vibrational or torsional modes leading to altered conformation of the protein molecules. However, their discussion did not consider the difficulty of direct excitation of vibrational modes by RF nor the effects of damping (Adair 2002; Challis 2005).

The hypothesis of an alteration of the conformation of proteins through transient heating resulting from pulsed RF exposure was suggested by Laurence et al (2000). However, the maximum temperature rise produced by the RF heating depends on the heat capacity of the heated volume and the rate at which it diffuses away. The authors had used an incorrect value of the heat capacity and the temperature rises became extremely small when the correct figure was used (Laurence et al 2003).

D'Ambrosio and colleagues have investigated the effects of RF exposure on protein model systems for several years. In earlier work (La Cara et al 1999), they had compared the effects of RF and conventional heating on the activity of a thermophilic beta-galactosidase. This thermostable enzyme was exposed at 70°C at SAR levels of 1100 and 1700 W kg^{-1} for 15, 30, 45, or 60 min and its activity compared to that of a sample heated in a water bath at the same temperature. Enzyme activity was reduced to 10% at the highest SAR level while water-bath heating did not affect activity. In further work by the same group (Bismuto et al 2003) solutions of the myoglobin protein were exposed at 1.95 GHz in a thermostatically-controlled waveguide for 2.5 h at 30°C (rising from 25°C at the start of exposure at an SAR of 51 W kg^{-1}). Absorption spectroscopy, circular dichroism, and fluorescence emission decay in the frequency domain were used to assess the influence of RF exposure on the native structural state of the protein. Under those exposure conditions, the structural organization of myoglobin molecule, its internal dynamics and CO binding affinity were not affected. As a follow-up to the work on myoglobin, Mancinelli et al (2004), under identical exposure conditions, used an acidic solution at pH 3 to look at the kinetics of protein refolding. The kinetics of the exposed samples was slower than that of the sham-exposed one. This was interpreted as an effect of RF on the propensity of myoglobin to populate specific conformational substates among which it fluctuates at acidic pH, possibly leading to protein misfolding. However, the observations of these small-amplitude effects are prone to artefacts caused by small variations in the temperature control of the samples and the results await confirmation.

More evidence that RF exposure can alter protein conformation without bulk heating comes from the work of de Pomerai et al (2003). The exposure of solutions of bovine serum albumin at 1 GHz (15-20 mW kg^{-1}, exposure lasting from 3 to 48 h and temperature from 25 to 45°C) enhanced the aggregation of the protein in a time- and temperature-dependent manner.

More recently, Copty et al reported on some specific effects on a solution of green fluorescent protein exposed at 8.5 GHz (Copty et al 2006). Samples were either exposed to RF or heated using resistive heating. [At maximum RF power, the calculated SAR was 4 kW kg^{-1} and ΔT was 3°C.] In both cases, heating produced a decrease in the protein fluorescence intensity and the spectrum became red-shifted. It was noted though that, for a similar temperature rise, the alteration of fluorescence was larger in the RF-exposed samples, which was interpreted as evidence of a specific nonthermal effect of RF exposure. However, the theoretical and experimental determination of ΔT under RF exposure is very uncertain but critical for any conclusion regarding nonthermal effects.

There have been a few investigations on isolated DNA in solution, all dating back to the 1980s. Initially there were some reports showing a frequency-specific absorption in DNA from plasmids or DNA breakage due to RF exposure in solution (Swicord and Davis 1982; Edwards et al 1984, 1985, and Sagripanti and Swicord 1986). However, follow-up studies showed that this was incorrect (Foster et al 1987; Gabriel et al 1987). DNA breakage was most likely to have been the result of free radical formation due to the use of copper electrodes and hence the presence of copper ions in solution, but not the result of a direct action of RF absorption (Sagripanti et al 1987). Further theoretical calculations by Foster and Baish (2000), Adair (2002) and Prohofsky (2004) support the view that viscous damping would be sufficient to make any 'resonant' behavior of DNA molecules in solution very unlikely.

Vanderstraeten and Vander Vorst (2004) have evaluated the dielectric properties of DNA in the nucleus and estimated that the local SAR in the layers of condensed counterions and bound water molecules is one and two orders of magnitude above that in muscle tissue. However, the authors conclude that the increased local RF absorption will not generate appreciable rises in temperature in those regions because of the high levels of thermal conductivity of the surrounding fluid medium.

In summary, the search for nonthermal effects of RF on biological macromolecules such as proteins and DNA has not been very active in recent years and to date there is no good evidence to suggest that such effects exist.

II.2.2.2. Liposomes and membranes

Liposomes are artificial phospholipid vesicles, constructed in the laboratory, which have often been used as models for studies of membrane properties. Early work by Liburdy & Magin (1985) reported an enhanced release of drugs trapped in the liposomes under exposure at 2.45 GHz, 60 W kg^{-1}. The effect occurred at temperatures below the membrane phase transition temperature of 41°C.

Following initial work on liposomes exposed to RF (Ramundo-Orlando et al 1993), Ramundo-Orlando and colleagues have more recently used liposomes entrapping glycoenzyme ascorbate oxidase (Ramundo-Orlando et al 2004). Exposure was performed at 2.45 GHz, at SAR levels up to 5.6 W kg^{-1}. Exposure at the maximum SAR level reduced enzyme activity, although the conformation of the enzyme was not affected. The authors suggested that RF interactions with the oligosaccharide chains of the enzyme were critical in eliciting this effect. Further work by the same group at 130 GHz using the carbonic anhydrase enzyme led to increased liposome permeability under pulsed exposure, but only when modulation was at 7 Hz and not 5 or 10 Hz (Ramundo-Orlando et al 2007).

Overall, there is limited evidence to date that nonthermal RF effects occur in model liposomes although the biological significance of such effects is not clear.

II.2.3. Summary on mechanisms

There are several theoretical hypotheses describing potential nonthermal mechanisms for low-level RF biological effects. Some have been tested experimentally, but so far there has been no compelling evidence that they might plausibly account for any such effects. From a biophysical point of view, the most plausible include the possibility that RF can affect metabolic reactions involving a radical-pair mechanism, and that biological tissue can somehow demodulate an RF signal through the non-linear conversion of RF energy. Both are of interest with regard to potential health effects but there is as yet no convincing evidence that such interactions occur in mammalian systems.

Whilst biological effects resulting from low level RF exposure are usually taken to indicate evidence for a non-thermal interaction, it is important to note that cells possess various thermally sensitive molecules such as the TRP family of ion channels and RNA 'riboswitches' that are able to initiate cellular responses to temperature changes possibly as small as 0.1°C. The implication is that low level RF exposure might result in subtle thermal effects that would be part of the normal physiological cellular response and are therefore unlikely to be of any health significance.

In conclusion, whilst it is in principle impossible to disprove the possible existence of nonthermal interaction, the plausibility of the nonthermal mechanisms discussed above is very low.

II.3. CELLULAR STUDIES

II.3.1. Introduction

Cell-based assays are used extensively in toxicological investigations. This is because they can provide essential information about the potential effects of chemicals and other agents such as radiation on specific cell properties, and provide a more rapid and cost-effective approach to molecular and mechanistic studies than can conventional laboratory animal models. A wide variety of cell types, ranging from stem cells via undifferentiated fibroblast-like or epithelial-like cells to highly differentiated tissue-specific cells, can be isolated from many tissues in various species and cultured over extended periods of time and/or cryopreserved for future use. They are therefore interesting tools in toxicity studies and preferred above the many organotypic preparations that have limited *in vitro* longevities. One important cell type is the human lymphocyte, precisely because of its human origin and the ease with which they may be obtained (e.g., by venipuncture). Human blood lymphocytes can easily be cultured for at least 72

hours which is, for example, sufficient for cytogenetic investigations after *in vitro* or *in vivo* exposures to pollutants or radiation. However, these white blood cells do not necessarily respond to chemicals or radiations in the same way as other cells; the choice of the cell system may greatly influence the results of an experiment. Lymphocytes obtained by venipuncture and stimulated by phytohaemagglutinin are a diploid, partially synchronized cell population but many cell lines may be transformed or have an abnormal chromosome count (= aneuploïdy) or have other genetic lesions and may therefore show an abnormal behavior. The cell cycle and DNA repair capacities may be different from one cell type to another, as may the presence or absence of particular membrane receptors and xenobiotic activator systems. Also the choice of the culture medium can play an important role in modulating the effects of environmental factors. It was for example shown that the concentration of folic acid in the medium greatly influences the baseline frequency of micronucleated cells in the culture (Fenech 2000; Wang and Fenech 2003).

Studies *in vitro* have proved to be useful in elucidating mechanisms of action and are predictive for some health hazards and illnesses. Increased frequencies of structural chromosome aberrations and micronuclei in human peripheral blood lymphocytes from a given population were, for example, indicative of an increased cancer risk, not at the individual level, but at least at the level of the study population (Bonassi et al 1995, 2007; Hagmar et al 1994). However, when using simplistic cell-based systems to assess toxicity, it is important to recognize that cells are finely-balanced homeostatic machines that respond to external stimuli through complex pathways. As toxicity can be the result of a multitude of cellular events, and because cell culture systems often lack essential systemic contributors to overall absorption, distribution, metabolism and excretion, as well as to the complex interactions and effects of the immune, endocrine and nervous system, it is clear that no *in vitro* assays can completely mimic the *in situ* condition in animals and humans of complex interactions between stem cells, proliferating progenitor cells and terminally differentiated cells within a tissue and between tissues (Bhogal et al 2005). *In vitro* investigations therefore only contribute to toxicity testing and risk assessment but, standing alone, they are insufficient predictors of toxicity and hazard. This is certainly also true with respect to investigations of cellular effects from RF radiation and this should be kept in mind when evaluating these data.

The possibility that exposure to RF radiation affects DNA has, particularly since the introduction of wireless communication systems, been the subject of much debate. If it were shown that low-level exposure to RF electromagnetic fields induces genetic damage, this would certainly be indicative of a potentially serious public health risk. Yet, the assumption that genetic effects are exclusively and in all cases predictive for cancer is certainly an overstatement. It is now apparent that many chemicals can contribute to the carcinogenic process without inducing mutations. Such chemicals can induce intracellular signaling, alter gap junctional intercellular communication and alter patterns of gene expression, for example by modifications of methylation and acetylation of DNA and histones. They may contribute to cancer by an ‘epigenetic’ mechanism rather than by mutation (Trosko and Upham 2005).

Non-genotoxic studies reviewed here focus on the effects of RF exposure on intracellular and intercellular signaling, gene and/or protein expression, cellular metabolism, cell cycle progression, proliferation, differentiation, apoptosis, and transformation. Cell signaling controls cellular metabolism, gene transcription, protein expression and modification, and finally cell behavior. Therefore, an effect of RF exposure on these pathways could be expected to produce biological effects in cells. If RF radiation does act as an external signal, however, the mechanism by which the external physical signal is transduced into a biological signal remains elusive. Studies on gene transcription, protein expression and modification, and cellular metabolism are thought to provide data for understanding the mechanism of RF radiation interaction, and may provide new biomarkers for further animal or even epidemiological studies. Exploring the effects of RF radiation on cell behaviors, including cell cycle progression, cell proliferation, cell differentiation, apoptosis, and cell transformation, could provide information regarding possible impacts of RF exposure on development, tumorigenesis, and other physiological or pathological processes.

With respect to *in vitro* investigations of RF radiation it should also be emphasized that the way RF exposure is done and hence proper dosimetry are crucial. Major improvements have been made in the quality of the exposure systems and their dosimetry. The various designs (waveguides, wire-patch cells, radial transmission lines, transverse electromagnetic cells, horns, etc.) with their respective advantages

are described elsewhere (see Chapter I.3.4.). The average SAR value is a weak substitute for the real and rather complex exposure distribution in the Petri dishes or tissue culture vessels used. For a given exposure setup, cells can be exposed to SAR values that vary by several fold within a Petri dish. In addition, it is difficult to specify temperature distribution accurately within the cell culture.

II.3.2. Genotoxicity

There have been a number of reviews on genotoxicity of RF radiation, all of them reaching the conclusion that the existing data suggest that RF radiation is not directly mutagenic and that it probably does not enhance the genotoxicity of physical or chemical genotoxic agents (e.g., Brusick et al 1998; Verschaeve and Maes 1998; Meltz 2003; Vijayalaxmi and Obe 2004; Verschaeve 2005; McNamee and Bellier 2007). Positive results have been reported but these were usually attributed to hyperthermia, to possible methodological errors or to misinterpretation of the data. However, following low-level (non-thermal) exposure conditions, there may be some subtle indirect effects on, for example, the replication and/or transcription of genes under relative restricted exposure conditions, and some new studies (e.g., REFLEX 2004) have re-opened this discussion. Hence, a final consensus among investigators has not yet been obtained.

Although studies in this area have been performed at a variety of levels of biological complexity, the majority of them were cytogenetic investigations in which the frequencies of chromosomal aberrations, sister chromatid exchanges and micronuclei were investigated. This is due to ‘historical’ reasons and because it is known that increased levels of chromosomal aberrations in human peripheral blood lymphocytes are predictive for cancer risk (Hagmar et al 1994; Bonassi et al 1995). Recently, evidence was presented indicating that this is most probably also true for increased levels of micronuclei in cytokinesis-blocked lymphocytes (Mateuca et al 2006; Bonassi et al 2007). Thus, these markers can be used to identify potential cancer risk well before the clinical onset of disease. However, cytogenetic methods essentially reveal severe genetic damage and are not able to detect most of the subtle indirect effects that may be induced. Improved methods or new technologies that may be more sensitive are therefore of great importance.

The (alkaline) comet assay was introduced some twenty years ago (Singh et al 1988). For this technique cells are mixed with agarose gel and spread onto a microscope slide. The cells are lysed with high salt concentrations and detergents and the remaining nuclear DNA is then denatured and electrophoresed in a buffer solution. DNA fragments and ‘loops’ migrate out of the nucleus towards the positive pole. Hence a ‘comet like’ figure is formed that can be visualized after staining with a fluorochrome. An image analysis system can be used to measure several damage parameters, for example ‘comet tail length’ and ‘tail DNA content’. The major advantages of this assay are that the test can indeed be considered more sensitive than the cytogenetic methods and that it can be performed on virtually all cells containing DNA (including non dividing cells). Furthermore, individual cells can be analyzed and this is an advantage in terms of identifying subpopulations that respond differently to cytotoxic treatment or exposures. The comet assay is usually performed in one of two variations. The alkaline comet assay can be used to detect the combination of DNA single-strand breaks (SSBs), double-strand breaks (DSBs) and alkali-labile sites in the DNA. The second procedure is performed under neutral conditions and detects predominantly DNA double-strand breaks (Olive and Banáth 2006). The comet assay has, despite several advantages over other technologies, also a number of limitations that may hamper the interpretation of the results. The method is for example not yet completely validated. Also, a sample size of only 50 analyzed “comets” was initially recommended and therefore no more cells were investigated in most studies; however, this may not be adequate if there is significant heterogeneity in DNA damage within a population. Furthermore, if samples contain predominantly necrotic or apoptotic cells, accurate information on the presence of specific lesions like strand breaks or base damage cannot be obtained. Also, tissue disaggregation methods need to be developed to minimize any DNA damage produced by the preparation procedures. The possibility that there may be preferential loss of heavily damaged cells during single cell preparation should also be considered (Olive and Banáth 2006). As indicated by Olive and Banáth (2006) the interpretation of comet test results is complicated by the fact that there is no simple relationship between the amount of DNA damage caused by a specific agent and the biological impact of that damage.

Each agent can differ in terms of the number of DNA breaks that are associated with a given biological effect. Comparing the results of the comet assay with other measures of DNA damage is necessary to interpret the biological relevance of the damage. In other words, the comet assay has become an important tool to assess DNA damage but the interpretation of the results is not always easy, and standing alone, the results can be misleading.

For this reason further new technologies might gain importance in the coming years. One such technology may be the detection of γ-H2AX phosphorylated histone (Huang X et al 2005). One of the earliest marks of a DNA double-strand break in eukaryotes is phosphorylation of the histone variant H2AX to create γ-H2AX–containing nucleosomes (Rogakou et al 1998). γ-H2AX is essential for the efficient recognition and/or repair of DNA double-strand breaks and many molecules, often thousands, of H2AX become rapidly phosphorylated at the site of each nascent double-strand break. The phosphorylated γ-H2AX can be visualized as discrete foci with the use of specific antibodies with fluorescent tags and directly counted using a fluorescent microscope. Detection of the number of DNA double-strand breaks is made possible via polyclonal antibodies to γ-H2AX. The γ-H2AX assay was found capable of detecting DNA damage at levels 100-fold below the detection limit of the alkaline comet assay. It was shown that this simple method was suitable to monitor response to radiation or other DNA-damaging agents (e.g., Nazarov et al 2003; Ismail et al 2007) and to measure cellular radiosensitivity that is potentially applicable in the clinic (Klokov et al 2006). However, it also detects intermediates in repair, or double-strand breaks induced by replication and so there is always a residual background level seen in untreated cells which can vary depending on a number of factors including cell type and, in proliferating cells, the stage of the cell cycle.

So far, this technique has been used by Markova, Belyaev and colleagues (e.g. Markova et al 2005; Belyaev et al 2005, see Table II.3.2.). Markova et al (2005), for example, carried out an analysis of the γ-H2AX protein together with an analysis of 53BP1 protein that binds with γ-H2AX to form a DNA repair complex and also examined an index of chromatin condensation termed anomalous viscosity time dependence (AVTD), developed by the authors, in lymphocytes from 'normal' and self reported electromagnetic hypersensitive subjects exposed *in vitro* to GSM-type RF radiation at 905 and 915 MHz at a mean SAR of 37 mW kg^{-1}. The authors reported that exposure to 915 MHz resulted in a distinct reduction in the number of 53BP1/γ-H2AX DNA repair foci from both normal and hypersensitive subjects, whereas the response to 905 MHz was not consistent amongst subjects, with both increases, decreases or no effect seen, whereas exposure to 3 Gy γ rays increased the number of foci. In addition, a significant effect on chromatin condensation was reported. However, as no replication studies were performed and as it is known that many confounding factors may influence the results, it is at present difficult to assess the significance of this study in the evaluation of possible RF-induced genetic effects.

Another group (Zhang et al 2006) explored the effect of GSM 1800 on DNA damage in Chinese hamster lung (CHL) cells using γ-H2AX focus formation. The cells were intermittently exposed or sham-exposed to GSM 1800 RF (5 min on/10 min off) at an SAR of 3.0 W kg^{-1} for 1 or 24 h. A cell was classified as positive when more than five foci were detected in it. The data revealed that exposure to 1800 MHz RF at 3.0 W kg^{-1} for 24 h caused more γ-H2AX focus formation, but 1 h did not.

II.3.2.1. Studies of RF-effects alone *in vitro*

Genotoxicity in prokaryotes

In an experiment on *Escherichia coli* bacteria carrying the plasmid puc9, Daşdağ et al (1999a) found that the number of plasmid copies per cell was not changed by exposure to 9450 and 2450 MHz RF radiation for up to one hour and up to thermal exposure conditions. In an experiment on the safety of magnetic resonance imaging (MRI) using *Salmonella* tester strains TA98 and TA7001-7006, Mineta et al (1999) did not find any increase of point and frameshift mutations following RF exposure under a 6.3 teslas magnetic field. Belloni et al (2005) used a transmission line to investigate the effects of 900 MHz RF fields on DNA mutability and repair in *E. coli*. They did not find induced DNA damage following RF exposure (up to 66 V m^{-1} and 260 nT) but, on the contrary, they observed a protective effect that they ascribed to an improved efficiency of the mismatch repair system. Chang et al (2005) concluded from

their experiments on *Salmonella typhimurium* and *E. coli* bacteria that 835 MHz CDMA RF exposure at 4 W kg^{-1} during 48 h neither affected reverse mutation frequencies nor accelerated DNA degradation *in vitro.* However, Belyaev et al (1997) did find effects from millimeter waves (51.64-51.84 GHz) on the chromatin conformation in *E. coli* cells using the method of anomalous viscosity time dependencies (AVTD, see above).

Genotoxicity in mammalian cells

A series of reports from Lai and Singh have caused a lot of controversy and discussion within the scientific community and the general public. These studies were conducted on rats and will therefore be discussed later on (see Chapter II.4.1.). However, they also generated a lot of *in vitro* investigations using the single-cell-gel-electrophoresis assay (comet test) on rodent or human cells (see Table II.3.1.). Most investigations have shown negative results. In one investigation on human Molt-4T-lymphoblastoïd cells, Phillips et al (1998) found that TDMA signals caused a significant decrease in SSBs as was also observed for the iDEN signal (frequency/modulation form specially designed for use in vehicles) at the lowest exposure. However, a 2 hour exposure at the higher exposure level increased the DNA damage as measured by the alkaline comet assay. These results may point to different biological effects of the two signals and a possible activation of DNA repair mechanisms and hence a protective effect at low exposure in contrast to the Lai and Singh papers on DNA SSBs and DSBs following *in vivo* exposure of rats.

Other positive findings were reported by Diem et al (2005) and Schwarz et al (2008). Both studies were from the same research group. In the first study cultured human skin fibroblasts and SV-40 transformed rat granulosa cells were examined following exposure to 1800 MHz radiofrequency radiation. Exposure was either continuous or intermittent. The authors concluded that both continuous and intermittent exposures induced SSBs and DSBs with the greatest effect found with intermittent exposure. In the second study of the effects of UMTS, 1950 MHz electromagnetic fields, exposure was found to increase DNA damage assessed using the alkaline comet assay and frequency of centromere-negative micronuclei in human cultured fibroblasts, and this occurred in a dose- and time-dependent way. However, the qualitative technique used to evaluate the "DNA comets", especially the method by which the comets were transformed into an 'objective' tail factor, has been highly criticized. In a 'letter to the editor' Vijayalaxmi et al (2006) listed a number of arguments that they consider refute or at least question the conclusions of the first study and recommend waiting for data from confirmation/replication investigations before drawing any conclusions. Such a repeat study, on 1800 MHz continuous or intermittent exposure, has already been performed in part by Speit et al (2007). These investigators used the same (ES1 human fibroblast) cells, the same equipment and the same exposure conditions and found no effects. They also performed the same experiments with V79 cells, a sensitive Chinese hamster cell line, and did not observe any genotoxic effect in the comet assay or micronucleus test.

The criticisms mentioned above also hold true for the second study. Further objections have been formulated by Lerchl (2008) who performed a critical analysis of the data and reported unusually low levels of variability in critical data. Although one of the authors replied to the points raised (Rüdiger 2008), it is clear that these results at least need further confirmation before they can be seriously taken into consideration.

Table II.3.1.: Study of RF-induced DNA damage using the single cell electrophoresis assay

Assay endpoint	Exposure Conditions	Response	Comment	References
SSB in human white blood cells, sampled immediately after exposure	GSM signal, 935.2 MHz; SAR: 0.3-0.4 W kg-1; 2 h exposure	No effect		Maes et al 1997
SSB in human glioblastoma cells, sampled immediately and up to 4 h after exposure	2450 MHz CW; SAR: 0.7 and 1.9 W kg^{-1}; 2, 4 and 24 h exposure	No effect		Malyapa et al 1997a

Assay endpoint	Exposure Conditions	Response	Comment	References
SSB in mouse fibroblast cells, sampled immediately and up to 4 h after exposure	2450 MHz CW; SAR: 0.7 and 1.9 W kg^{-1}; 2 and 24 h exposure	No effect		Malyapa et al 1997b
SSB in human glioblastoma cells, sampled immediately after exposure	835.62 MHz FMCW and 847.7 MHz, CDMA CW, SAR: 0.6 W kg^{-1}; 2, 4 and 24 h exposure	No effect		Malyapa et al 1997b
SSB in human lymphoblastoid cells, sampled immediately after exposure	iDEN, 813.5 MHz and TDMA, 835.5 MHz; SAR: 0.0024 and 0.024 W kg^{-1}, resp. 0.0026 and 0.026 W kg^{-1}; 2, 3 and 21 h exposure	"Protective" effect for TDMA and iDEN (lowest exposure) and increased damage at highest iDEN exposure		Phillips et al 1998
SSB in human white blood cells, sampled immediately and 4 h after exposure	2450 MHz PW; average SAR: 2.14 W kg^{-1}; 2 h exposure	No effect		Vijayalaxmi et al 2000
SSB in mouse fibroblast cells, sampled immediately and 4 h after exposure	835.6 MHz FDMA and 847.7 MHz FDMA ; SAR: 3.2 and 5.1 W kg^{-1}; 2, 4 and 24 h exposure	No effect		Li et al 2001
SSB in human brain tumor-derived MO54 cells, sampled immediately after exposure	2.45 GHz; SAR: 13~100 W kg^{-1}, 2 h; SAR: 100 W kg^{-1}, 2 h	No significant difference in the tail moments of cells exposed to the RF field and sham control.		Miyakoshi et al 2002
SSB in human white blood cells, sampled immediately after exposure	837 MHz analog, CDMA, TDMA and 1909.8 MHz GSM and PCS signal ; 3 and 24 h exposure; average SAR: 1 to 10 W kg^{-1}	No effect		Tice et al 2002
SSB in human white blood cells, sampled immediately after exposure	1900 MHz CW; SAR: 0.1 to 10 W kg^{-1}; 2 h exposure	No effect		McNamee et al 2002a
SSB in human white blood cells, sampled immediately after exposure	1900 MHz PW; SAR: 0.1 to 10 W kg^{-1}; 2 h exposure	No effect		McNamee et al 2002b
SSB in human white blood cells, sampled immediately after exposure	1900 MHz CW and PW; SAR: 0.1 to 10 W kg^{-1}; 24 h exposure	No effect		McNamee et al 2003
SSB in Xenopus laevis erythrocytes, sampled immediately after exposure	HPMP 8.8 GHz (180 ns pulse width, peak power 65 kW, repetition rate 50 Hz); SAR: 1.6 W kg^{-1} (peak SAR 300 MW kg^{-1}); 40 min exposure.	DNA damage induced by temperature rise	No indication of non-thermal effects	Chemeris et al 2004

Assay endpoint	Exposure Conditions	Response	Comment	References
SSB in MOLT-4T lymphoblastoid cells, sampled immediately after exposure	847.74 MHz CDMA, 835.6 MHz FDMA, 813.6 MHz iDEN, 836.6 MHz TDMA; SAR: 3.2 W kg^{-1} (CDMA, FDMA), 0.0024 or 0.024 W kg^{-1} (iDEN), 0.0026 or 0.026 W kg^{-1} (TDMA); exposure for up to 24 h.	No effect		Hook et al 2004a
SSB and DSB in human diploid fibroblasts and rat granulosa cells, sampled immediately after exposure	1800 MHz; CW or modulated; continuous and intermittent (5 min on, 10 min off) exposure during 4, 16 and 24 h; SAR: 2 W kg^{-1}	Induction of DNA single- and double strand breaks after 16h intermittent exposure in both cell types and at different mobile phone modulations	Some objections have been raised concerning the analysis of the data – see text above.	Diem et al 2005
SSB in human glioblastoma A 172 cells and normal human fibroblasts, sampled immediately after exposure	A172 cells: W-CDMA at 80, 250, 800 mW kg^{-1} and CW at 80 mW kg^{-1} for 2 and 24 h. IMR-90 cells: W-CDMA and CW at SAR: 80 mW kg^{-1} for 2 and 24 h.	No effect		Sakuma et al 2006
SSB in human white blood cells, sampled immediately after exposure	8.8 GHz, HPMP (180 ns pulse width); Average SAR 1.6 W kg^{-1} (peak SAR 300 MW kg^{-1}); for 40 min	No significant change to the percentage of DNA content in the comet tail compared to the respective negative and temperature controls.		Chemeris et al 2006
SSB in human white blood cells	1950 MHz UMTS signal at SAR: 0.5 and 2.0 W kg^{-1}; 24 h exposure	No genotoxicity and cytotoxicity at both SAR levels	Cytotoxicity assessed by the trypan blue exclusion test	Sannino et al 2006
SSB in human lens epithelial cells, sampled at 0, 30, 60, 120 and 240 min after exposure	1.8 GHz (217 Hz AM); 2 h exposure at SAR: 1, 2 and 3 W kg^{-1}	DNA damage at 3 W kg^{-1} at 0 and 30 min following exposure	Exposure for 2h at 2 and 3 W kg^{-1} also exhibited significantly increased hsp 70 protein expression.	Sun et al 2006
SSB in human diploid fibroblasts and Chinese hamster V79 cells, sampled immediately after exposure	1800 MHz; CW or modulated; continuous and intermittent (5 min on, 10 min off) exposure varied between 1 and 24h; SAR: 2W kg^{-1}	No effect	This study was aimed at replicating earlier findings (REFLEX 2004; Diem et al 2005) – Results were not in accordance with these.	Speit et al 2007

Assay endpoint	Exposure Conditions	Response	Comment	References
Alkaline comet assay in human leukocytes	1950 MHz UMTS (intermittent exposure for 24 – 6 min on, 2 h off) in TEM cell; SAR: 2.2 W kg^{-1}.	No effect	Also no effect on micronucleus frequency.	Zeni et al 2008
SSB in human cultured fibroblasts and white blood cells	1950 MHz UMTS; 24 h exposure and (intermittent exposure for 16h – 5min on, 10 min off and 10 min on, 20 min off); SAR <2 W kg^{-1}	Increased "Comet Tail Factor" in a dose and time-dependent way	Some objections have been raised concerning the analysis of the data – see text above.	Schwarz et al 2008

HPMP: high power microwave pulses; FMCW: Frequency Modulated Continuous Wave; AM: amplitude modulated; CDMA = code-division multiple-access; FDMA = frequency-division multiple-access; TDMA = time-division multiple-access ; iDEN = iDEN(R) frequency/modulation form specially designed for use in vehicles.

Besides studies on DNA damage as assessed by the comet assay, many other cytogenetic studies have investigated possible RF-genetic effects in mammalian cells. Most have been performed on human lymphocytes but other cells were also studied. An overview of studies carried out after 1990 is given in Table II.3.2. (some also include data on the comet assay that were not included in Table II.3.1.). It is clear from the table that the results are more mixed than the comet assay results. However, positive findings were most often found when the exposure level was high (e.g., Garaj-Vrhovac et al 1992; Maes et al 1993; Tice et al 2002) resulting in an overall or localized thermal effect. In many other (mainly positive) investigations (e.g., Garaj-Vrhovac et al 1996) insufficient data were provided to judge the validity of the findings or interpretation of the results (see discussion in Vijayalaxmi and Obe 2004).

In recent years the observation by Tice et al (2002) of an RF-induced increase in micronucleus frequency in resting lymphocytes following a 24 h exposure attracted a lot of attention, essentially because no effects were found in the same (or related) investigations with regard to other genetic endpoints (e.g., absence of induced SSBs) and because increased micronucleus frequencies may, in the absence of chromosome aberrations, point towards an aneugenic effect (an abnormal chromosome number). It should be remembered, apart from structural chromosome aberrations, micronuclei may also originate from abnormal chromosome segregation during cell division (e.g., Fenech 2000) which may give rise to aneuploïd daughter cells. Only a few studies have so far investigated aneuploïdy with regard to RF. Mashevich et al (2003) reported increased aneuploïdy of chromosome 17, which may be seen as a corroboration of the possible aneuploïdy-inducing potency of RF radiation. These authors applied a fluorescence *in situ* hybridization technique to determine the incidence of aneuploidy of chromosome 17. The same group later confirmed these findings in a further investigation on aneuploïdy of the chromosomes 1, 10, 11 and 17 (Mazor et al 2008). As discussed elsewhere (Vijayalaxmi and Obe 2004) these data are intriguing and certainly need to be replicated and confirmed by others before any firm conclusion can be reached. It should furthermore be stated that there are also many investigations that do not show any micronucleus-inducing potency of RF. However, the experimental protocols used varied (e.g., 24 h exposure time vs. 2 h exposure) and these differences may eventually account for the contradictory results.

Other recent studies have also failed to reach a consensus view. Stronati et al (2006) did not find cytogenetic effects in RF exposed cells, and no co-operative effect was found with X-rays. However, Diem et al (2005) reported that RF possesses genotoxic properties, provided exposures are intermittent according to a particular protocol (5 min on/10 min off), depending on cell type. RF exposure at a SAR level below 2 W kg^{-1}, for example, induced SSBs and DSBs and micronuclei and chromosomal aberrations in human fibroblast cells, HL-60 cells and/or rat granulosa cells. Blood lymphocytes were apparently unresponsive (REFLEX 2004). According to the authors the effects were possibly caused by an RF-induced increase in free oxygen radicals. As human lymphocytes were not responding it was believed that this cell type is not sensitive to RF and that previous negative findings that were obtained in lymphocytes (cf. Table II.3.2.) were simply due to the wrong choice of cells in these experiments. However, as mentioned above, the methodology and analysis employed in these investigations has been

criticized (e.g., Vijayalaxmi et al 2006; Lerchl 2008) and at least partially failed to replicate in an independent repeat investigation (Speit et al 2007).

Table II.3.2: Cytogenetic investigations of RF genotoxicity

Assay endpoint	Exposure Conditions	Response	Comment	References
Chromosome aberrations test in V79 Chinese hamster cells	7700 MHz CW; power density 300 W m^{-2} for 15, 30 and 60 min	Increased chromosome aberration frequency	SAR not given	Garaj-Vrhovac et al 1990
Chromosome aberrations and micronucleus test V79 Chinese hamster cells	7700 MHz CW; power density 5 W m^{-2} for 15, 30 and 60 min	Increased chromosome aberration and micronucleus frequency	SAR not given	Garaj-Vrhovac et al 1991
Chromosome aberration test in human white blood cells	7700 MHz CW ; 5, 100 and 300 W m^{-2} power density for 10, 30 and 60 min	Time dependent increase in chromosomal aberrations (e.g., dicentric, acentric fragments)	Thermal effect probable; SAR not given	Garaj-Vrhovac et al 1992
Chromosome aberration, sister chromatid exchange and micronucleus test in human white blood cells	2450 MHz PW; SAR: 75 W kg^{-1} for 30 and 120 min.	Increased frequency of chromosomal aberrations and micronuclei but not of SCEs or effect on cell proliferation	Thermal effect probable	Maes et al 1993
Chromosome aberration test in human white blood cells	954 MHz SAR: 1.5 W kg^{-1} for 2 hr	Slight increase in chromosome aberration frequency.	No increase in SSBs according to the alkaline comet assay (unpublished results)	Maes et al 1995
Micronucleus test in human white blood cells	9600 MHz; SAR: 100 W kg^{-1} for 10 min	Increased micronucleus frequency	Thermal effect (increase of 5°C)	D'Ambrosio et al 1995
Micronucleus test in human white blood cells	415 MHz; standard NMT; Exposure for 10, 20 and 30 min with output power of 15 W.	Time dependent increase in micronucleus frequency	SAR not given	Garaj-Vrhovac et al 1996
Chromosome aberration, sister chromatid exchange, micronucleus and HGPRT-test in human white blood cells	440, 900, and 1800 MHz; exposure for 30 – 70 h with an output power of 2 W (440 MHz).	No increased frequency in chromosome aberrations, sister chromatid exchanges, micronuclei and HGPRT-mutations	SAR not given	Eberle et al 1997

Assay endpoint	Exposure Conditions	Response	Comment	References
Chromosome aberration and sister chromatid exchange test in human white blood cells	380 MHz PW; TETRA; SAR: 0.08 W kg^{-1} Waveguide, 900 MHz DCS; SAR: 0.2 W kg^{-1} Wave guide; 1800 MHz PW (GSM); exposure for up to 68 h	No increased frequency in chromosomal aberrations and sister chromatid exchanges		Antonopoulos et al 1997
Chromosome aberration and micronucleus test in human white blood cells	2450 MHz CW; SAR: 12.5 W kg^{-1}; continuous or intermittent exposure for a total of 90 min	No increased frequency in chromosomal aberrations and micronuclei following continuous or intermittent RF exposures		Vijayalaxmi et al 1997a
Micronucleus test in human white blood cells	2450 and 7700 MHz and power density of 100, 200 and 300 W m^{-2}; exposure for 15, 30 or 60 min	Increased frequency of micronuclei at a power density of 30mW/cm^2 and after an exposure time of 30 and 60min (not at 10min)	SAR not given	Zotti-Martelli et al 2000
Micronucleus test in human white blood cells	1800 MHz, CW at power densities of 5, 10 and 200 W m^{-2} for 60, 120 and 180 min.	Microwaves were shown to be able to induce micronuclei in short-term exposure to medium power density fields	SAR not given	Zotti-Martelli et al 2005
Chromosome aberration and micronucleus test in human white blood cells	847.7 MHz CW; CDMA; SAR: 4.9 and 5.5 W kg^{-1}; 24 h exposure	No increased frequency in chromosomal aberrations and micronuclei		Vijayalaxmi et al 2001b
Chromosome aberration and micronucleus test in human white blood cells	835.6 MHz CW; FDMA; SAR: 4.4 and 5.0 W kg^{-1}; exposure for 24 h.	No increased frequency in chromosomal aberrations and micronuclei		Vijayalaxmi et al 2001c
Micronucleus test in C3H $10T^{1/2}$ mouse fibroblast cells	835.6 MHz CW, FDMA and 847.7 MHz CW, CDMA; SAR: 3.2 and 5.1 W kg^{-1}; exposure for 3, 8, 16 or 24 h	No increased frequency of micronuclei		Bisht et al 2002

Assay endpoint	Exposure Conditions	Response	Comment	References
Micronucleus test in human white blood cells	CW or GMSK 1748 MHz; SAR: 5 W kg^{-1}; 15min. exposure	The micronucleus frequency was not affected by CW exposure; but a statistically significant micronucleus effect was found following exposure to phase modulated field	No changes were found in cell proliferation kinetics after exposure to either CW or GMSK fields.	D'Ambrosio et al 2002
Micronucleus test and alkaline comet assay in human white blood cells	837 MHz, analog, CDMA, TDMA; SAR: 1, 2.5, 5 and 10 W kg^{-1} and 1909.8 MHz, GSM; SAR=1.6, 2.9, 5 and 10 W kg^{-1}; exposure for 3 or 24 h.	No DNA damage as assessed by the alkaline comet assay Reproducible increase in the frequency of micronucleated cells for each of the RF signals at an average SAR of 5.0 or 10.0 W kg^{-1} and an exposure time of 24 h		Tice et al 2002
Micronucleus test and alkaline comet assay in human white blood cells	1900 MHz CW; SAR: 0.1, 0.26, 0.92, 2.4, 10 W kg^{-1}; exposure for 2 h	No increased frequency of micronuclei or DNA damage as assessed by the alkaline comet assay		McNamee et al 2002a
Micronucleus test and alkaline comet assay in human white blood cells	1900 MHz PW; SAR: 0.1, 0.26, 0.92, 2.4, 10 W kg^{-1}; exposure for 2 h	No increased frequency of micronuclei or DNA damage as assessed by the alkaline comet assay		McNamee et al 2002b
Micronucleus test and alkaline comet assay in human white blood cells	1900 MHz CW and PW; SAR: 0.1, 0.26, 0.92, 2.4, 10 W kg^{-1}; exposure for 2 h	No increased frequency of micronuclei or DNA damage as assessed by the alkaline comet assay		McNamee et al 2003
Aneuploidy detection in human white blood cells	830 MHz CW; SAR :2, 2.9, 4.3, 8.2 W kg^{-1}; exposure for 72 h	SAR dependent increase in aneuploidy of chromosome 17		Mashevich et al 2003

Assay endpoint	Exposure Conditions	Response	Comment	References
Micronucleus test in human white blood cells	900 MHz CW, GSM; SAR: 0.2 and 1.6 W kg^{-1}; exposure for 14 sessions of 6 min over ~2 days or 1 h per day for 3 days	No increased frequency of micronuclei		Zeni et al 2003
Chromatin conformation in human white blood cells	895 and 915 MHz PW SAR: 5.4 mW kg^{-1} for 30 min and 1 h	Microwaves from GSM mobile phone affect chromatin conformation similar to stress response	Because of the very low SAR value the microwave effect was not attributed to heating	Sarimov et al 2004
Alkaline comet assay, structural chromosome aberrations and sister chromatid exchange in human white blood cells	GSM 900 MHz; SAR :0.3 and 1.0 W kg^{-1}; exposure for 2 h	No increased frequency in DNA damage (alkaline comet assay), chromosome aberrations or sister chromatid exchanges		Zeni et al 2005
SSBs and DSBs (comet assay), structural chromosome aberrations and sister chromatid exchange in mouse embryonic stem cell derived neural progenitor cells.	1.71 GHZ (GSM) signal at a time-average SAR of 1.5 W kg^{-1} for 6 and 48 h with intermittency cycles of 5 min on/30min off.	Low and transient increase of DSBs after 6 h exposure (no effect following 48 h exposure). No effect on chromosomal aberrations or SCEs.	No effects on nuclear apoptosis or proliferation.	Nicolova et al 2005
Chromosome aberration test in mouse m5S cells	1800 MHz, CW at power densities of 5, 10 and 200 W m^{-2} for 60, 120 and 180 min.	Microwaves induced micronuclei in short-term exposure to medium power density fields		Zotti-Martelli et al 2005
Chromosome aberration test in mouse m5S cells	2450 MHz CW and PW at a SAR: 5, 10, 20, 50, 100 W kg^{-1}; exposure for 2 h	No induced chromosome aberrations by CW or PW fields		Komatsubara et al 2005
Chromatin conformation in human lymphocytes and 53BP1//γ-H2AX in murine cells	GSM 900 mobile phone with standard GSM modulation (905 and 915 MHz) SAR: 37 mW kg^{-1}; exposure for 1 h	Effect on chromatin conformation, as measured by AVTD, and 53BP1/γ-H2AX foci similar to heat shock.	AVTD is a technique used only by this group	Markova et al 2005

Assay endpoint	Exposure Conditions	Response	Comment	References
Chromatin conformation in human lymphocytes	915 MHz; SAR: 37 mW kg^{-1}	Significant condensation of chromatin found as measured by AVTD. No induction of apoptosis.	AVTD is a technique used only by this group	Belyaev et al 2005
Micronucleus test in human lymphocytes	900 MHz GSM signal, SAR: 1, 5 and 10 W kg^{-1}, 24 h exposure	No evidence for genotoxic (micronucleus test) or cytotoxic effects		Scarfi et al 2006
Chromosome aberrations and micronuclei in human lymphocytes	2.45 GHz, 8.2 GHz, 21W, 60W; 50 W/m^2 or 100 W/m^2 ; SAR: 2.13 W kg^{-1} or 20.71 W kg^{-1} 2 h	No adverse effects on the kinetics of cell proliferation or on the amount of chromosomal damage.		Vijayalaxmi 2006
Human diploid fibroblasts and Chinese hamster V79 cells, sampled immediately after exposure	GSM 1800 MHz; SAR: 3 W kg^{-1}; intermittent exposure (5min on, 10min off) for 1 and 24 h	No difference with sham exposed cells after 1h; however increased DNA damage after 24h exposure		Zhang et al 2006
Alkaline comet assay and micronucleus test in human ES1 diploid fibroblast cells and in Chinese hamster V79 cells	1800 MHz; CW or modulated; continuous and intermittent (5min on, 10 min off) exposure varied between 1 and 24 h; SAR: 2W kg^{-1}	No induction of micronucleated cells in independently repeated experiments	This study was aimed at replicating earlier findings (REFLEX 2004 and Diem et al 2005) – Results were not in accordance with these (see also comet assay results)	Speit et al 2007
Aneuploidy studies in human lymphocytes	800 MHz, CW, SAR: 2.9 and 4.1 W kg^{-1} for 72 h	Induced aneuploidy as determined by interphase FISH using semi-automated image analysis.	Findings were attributed to an athermal RF-effect	Mazor et al 2008
Micronucleus test and alkaline comet assay in human white blood cells	1950 MHz UMTS signal; intermittent exposure (6 min on, 2 h off) for 14 and 68 h; SAR: 2.2 W kg^{-1}	No increased frequency of micronuclei	Also no effect on (alkaline) comet assay	Zeni et al 2008

GMSK = Gaussian minimum shift-keying; AVTD = Anomalous Viscosity Time Dependence

Cytogenetic effects in plants

Only a few cytogenetic studies on RF bioeffects have been performed in plants since 1993 and are briefly summarized here for completeness (see discussion in Chapter II.3.3.2.). Haider et al (1994) used the Tradescantia-micronucleus test in an *in situ* experiment to find out whether short-wave electromagnetic fields (10-21 MHz) are genotoxic. Plant cuttings bearing young flower buds were exposed during 30 h on both sides of a slewable curtain antenna (40-170 and 90 V m^{-1}), a vertical cage antenna (70 V m^{-1}) and at

200 m from a broadcasting station (1-3 V m^{-1}). Higher micronucleus frequencies were found for all exposure sites except one. The authors concluded that their findings clearly underline the clastogenic (chromosome breaking) nature of short-wave electromagnetic fields. Pavel et al (1998) showed increased levels of micronuclei and chromosome aberrations in wheat when their seeds were exposed to non thermal levels of 9.75 GHz microwaves.

These two studies provide insufficient data on which to base any conclusions regarding cytogenetic effects in plants.

II.3.2.2. RF radiation combined with chemical or physical mutagens

The possibility that the genotoxic potential of certain chemical mutagens or ionizing radiation may be affected by co-exposure to electromagnetic fields has been raised. Theoretically radiofrequency radiation can be directly or indirectly genotoxic by affecting DNA repair mechanisms or by "co-operating" with known chemical or physical mutagens. Such indirect effects have been investigated in a number of studies summarized in Table II.3.3. The possibility that RF radiation inhibits DNA repair was investigated by Meltz & Walker (1987) in a study on MRC-5 normal human diploid fibroblast cells that were exposed to a very high dose of UVC (21 J m^{-2}) and 350, 850 or 1200 MHz pulsed wave signals at SARs ranging from 0.39 to 4.5 W kg^{-1}. In this study, RF irradiation followed the UV-exposure. No impairment of DNA repair synthesis was found or interference with different enzymatic steps of the DNA repair synthesis process was found.

Most of the co-exposure studies were not directed towards DNA repair but were aimed at investigating potential synergistic or co-operative effects. According to these studies, simultaneous exposure of cells to RF and a mutagen did not result in an increased frequency of genetic damage compared to treatment with the mutagen alone. These studies were performed in different cell lines (e.g., CHO cells, L5178Y cells) and used different assays (e.g., SCEs, forward mutation assay, chromosome aberrations). When RF-exposure was prior to mutagen exposure the genetic damage was sometimes higher than when cells were treated with the mutagen alone (Maes et al 1996; Scarfi et al 1996; Zhang et al 2002). A thermal effect could be assumed in some studies (e.g. Koyama et al 2003) but not in all cases. So far, it is by no means established that the order of exposure determines the presence or absence of a co-operative effect. It should, for example, be stressed that different results were found in different comparable experiments even when performed in the same laboratory and by the same investigators (e.g., compare Maes et al 1996, 1997 and 2000). Although the exposure conditions were never exactly the same, this suggests that other factors, unaccounted for in the experimental protocols, might explain the differences in response.

Table II.3.3: Combined exposures to RF radiation and chemical/physical mutagens

Assay endpoint	Exposure Conditions	Response	Comment	References
SCEs in CHO cells	2450 MHz pulsed, 490 W m^{-2}; SAR: 34 W kg^{-1}; exposure for 2 h.; Simultaneous irradiation and mitomycin C (MMC) exposure.	No increased SCE frequency in cells exposed to RF alone or with MMC compared to MMC alone		Ciaravino et al 1987
DNA repair in human fibroblasts	350 and 850 MHz and 1.2 GHz, pulsed 10 to 100 W/m^{-2}; SAR: 0.39 - 4.5 W kg^{-1}; exposure for 1 to 3 h; RF irradiation followed UV irradiation	No effects		Meltz & Walker 1987

Assay endpoint	Exposure Conditions	Response	Comment	References
Forwards mutation assay (thymidine kinase (TK) locus) in L5178Y mouse leukemia cells	2450 MHz, pulsed, 488 W m^{-2}; SAR: 30 W kg^{-1}; exposure for 4 h; Simultaneous irradiation and MMC exposure	RF exposure alone is not mutagenic	RF does not affect either the inhibition of cell growth or the extent of mutagenesis resulting from treatment with MMC alone	Meltz et al 1989
Forward mutation at TK-locus in L5178Y mouse leukemia cells	2.45 GHz, pulsed; SAR ~ 40 W kg^{-1}; exposure for 4 h: Cells are simultaneously exposed to proflavin	RF alone is not mutagenic. No increased mutation frequency for the combined treatment compared to proflavin alone. No difference in colony size distribution of the mutant colonies		Meltz et al 1990
Chromosome aberrations in CHO cells	2450 MHz, pulsed 490 W m^{-2}; SAR: 33.8 W kg^{-1}; exposure for 2 h; Cells are simultaneously irradiated and exposed to MMC and adriamycin.	RF alone does not enhance the chromosome aberration frequency	No alteration in the extent of chromosome aberrations for the combined treatment compared to the chemicals alone	Kerbacher et al 1990
Cell cycle progression and SCEs in CHO cells.	2450 MHz pulsed, 490 W m^{-2}; SAR: 33.8 W kg^{-1}; exposure for 2 h; Cells are simultaneously irradiated and adriamycin exposed	RF does not affect changes in cell progression caused by adriamycin, nor change the number of SCEs that were induced by adriamycin.		Ciaravino et al 1991
SCE and cell proliferation in human lymphocytes	954 MHz (GSM), SAR ~ 1.5 W kg^{-1}; cells exposed to MMC following a 2 h RF exposure	RF alone is not mutagenic. Increased incidence of SCEs in cells exposed to RF + MMC compared to MMC alone.	No influence on the cell proliferation for RF alone and for RF+MMC compared to MMC alone	Maes et al 1996
Micronucleus induction in bovine lymphocytes	9 GHz, SAR: 70 W kg^{-1} (CW); cells exposed to MMC following a 10 min RF exposure	Cooperative effect of microwaves and MMC		Scarfi et al 1996
SCE and cell proliferation, chromosome aberrations and DNA damage (comet assay) in human lymphocytes	935.2 MHz (CW and GSM), SAR ~ 0.3-0.4 W kg^{-1} for 2 h (TEM cell); cells exposed to MMC following a 2 h RF exposure	No effect of RF alone on DNA and chromosomes. Weak synergy with MMC (SCEs)		Maes et al 1997

Assay endpoint	Exposure Conditions	Response	Comment	References
Forward mutation, petite formation and recombination in Saccharomyces cerevisiae	900 MHz pulse modulated at SAR 0.13 and 1.3 W kg^{-1} + methyl methane sulfonate (MMS); exposure for 1 h and 36 h respectively	No effect on any of the genetic endpoints for the RF alone or combined with MMS		Gos et al 2000
Chromosome aberrations, SCEs in human lymphocytes	455.7 MHz, SAR: 6.5 W kg^{-1}; 2 h exposure. Cells exposed to MMC and X-rays following RF-exposure	No consistent results (MMC); no synergy with X-rays		Maes et al 2000
Chromosome aberrations, SCEs and DNA damage (comet assay) in human lymphocytes	900 MHz (GSM; CW, dummy burst and pseudo-random); SAR: 0 - 10 W kg^{-1}; MMC following a 2 h RF exposure, or RF exposure immediately followed by a 1 Gy X-ray exposure	No effect of the RF field alone. No evidence of a synergistic effect with MMC or X-rays		Maes et al 2001
DNA damage (comet assay); micronucleus test in human lymphocytes	2450 MHz, 50 W m^{-2}; exposure for 2 h; followed by MMC	Cooperative effect of RF with MMC	SAR not given	Zhang et al 2002
Micronucleus test in CHO-K1 cells	2450 MHz; SAR: 13, 39, 50, 78 and 100 W kg^{-1}; RF + bleomycin; 18 h exposure	Increased micronucleus frequency at SARs of 78 W kg^{-1} and higher. Potentiation of MN formation induced by bleomycin was found at SARs of 78 W kg^{-1} and higher.		Koyama et al 2003
Micronucleus test in CHO-K1 cells	2450 MHz; SAR: 5, 10, 20, 50, 100, 200 W kg^{-1}; exposure for 2 h; RF + bleomycin combined treatment	Only increased micronucleus frequency at SARs of 100 and 200 W kg^{-1}. No combined effect of RF and bleomycin		Koyama et al 2004

Assay endpoint	Exposure Conditions	Response	Comment	References
Comet assay in C3H 10T$^{1/2}$ cells	2450 MHz CW at SAR: 1.9 W kg^{-1}, 2h exposure followed by 4 Gy γ-irradiation	No DNA damage induced by 2450 MHz RF alone. 2450 MHz microwaves did not impede the DNA migration induced by γ-irradiation. No evidence for induction of DNA-protein crosslinks or changes in amount of protein associated with DNA by 2450 MHz CW microwave exposure		Lagroye et al 2004a$_a$
Chromosome aberrations in human lymphocytes	2.5 GHz and 10.5 GHz + γ-radiation exposure for 40 sec. at 3 W for 2.5 MHz and 5 min at 15 mW for 10.5 GHz). SAR estimated at 626 W kg^{-1} and 0.25 W kg^{-1} resp.	No induction of chromosome aberrations from the microwaves alone.	No combined effects in cells pre-treated with the RF fields followed by gamma-irradiation, but increased cell mortality was observed.	Figueiredo et al 2004
Comet assay performed at 0 and 21h following exposure in human lymphocytes	1.8 GHz, SAR: 3 W kg^{-1} + MMS, 4-NQO, MMC and bleomycin. Exposure for 2 and 3 h in three exposure combinations	No effect of RF alone but combined treatments with MMC and 4-NQO were significantly different from the chemical exposures alone.		Wang et al 2005
Chromosomal aberrations, SCEs, micronuclei, DNA damage (comet assay), nuclear division index in human lymphocytes	935 MHz GSM signal, SAR: 1 and 2 W kg^{-1}, 24 h continuous exposure + 1 min. 1 Gy 250 kVp X-rays	In all instances no effect from the RF signal alone or in combination with X-rays was observed		Stronati et al 2006
DNA damage (comet assay) in human white blood cells	1.8 GHz at SAR 3 W kg^{-1} for 0, 1.5 and 4 h following UVC exposure at 0.25, 0.5, 0.75, 1.0, 1.5 and 2.0 J m^{-2}.	The microwaves were shown to reduce UV induced DNA damage after 1.5h of exposure and increased UV induced DNA damage at 4 h.	No effect of 1.8 GHz exposure alone.	Wang et al 2007

II.3.2.3. Summary on genotoxicity

To date many studies have investigated RF-induced genetic effects in human and other cell types. Most, but not all, studies have found no evidence of *in vitro* RF-induced genetic damage at non-thermal exposure regimes and indicate that RF has no marked synergistic or additive effect together with other environmental agents (mutagens/carcinogens). However, many studies were devoted to genetic endpoints

that correspond to gross structural chromosome anomalies and hence possible subtle indirect effects on, for example the replication of genes under relatively restricted exposure conditions, could not be seen. Therefore more sensitive methods are probably necessary to determine whether such effects might exist. The comet assay was introduced as a method that might meet this demand. Our present-day knowledge of the comet assay shows that this is only partially true. Novel methods, for example the determination of γ-H2AX protein intranuclear foci and mini/microsatellite mutation analysis, may prove more valuable in RF genetic risk assessment.

These conclusions are supported by a recent meta-analysis of RF-genetic toxicity (Vijayalaxmi and Prihoda 2008). The authors quantitatively analyzed the results from 63 papers published between 1990 and 2005, deriving indices and 95% confidence intervals for various genetic endpoints in relation to frequency, SAR and CW or pulsed exposure. The overall genotoxicity indices obtained in from the RF exposed and control groups were similar. Also, the mean indices for chromosome aberrations and micronuclei in RF-radiation exposed and sham or unexposed controls were within spontaneous levels reported in the historical database.

II.3.3. Studies on non genotoxic cellular effects

In addition to evaluating RF radiation effects on the integrity of DNA, numerous studies have addressed other effects on cellular functions that could potentially influence development of disease in humans.

One major class of non-genotoxic cellular studies comprises studies of cell signaling, the means by which cells respond to extracellular signals such as cytokines, neurotransmitters and hormones via receptors located on the cell surface and to intracellular signals, which may be generated for example by the activation of an intracellular signaling cascade. A number of RF studies have been carried out on both intercellular and extracellular signaling processes.

Cellular responses depend on production of proteins (enzymes), key regulators of cell metabolic activity and behavior. Protein structures are encoded in DNA (genes) and are produced by the transcription of genes into mRNA and the translation of the mRNA into protein. This activity is called gene expression and RF effects on gene expression are, more precisely, classified as either effects on mRNA at the transcriptional level or on protein production. A large body of RF research has been conducted on gene and protein expression in mammalian and other cell types.

Other non-genotoxic studies address the ability of RF to affect the production of reactive oxygen species and on cell behaviors such as proliferation, differentiation, and apoptosis. In addition, studies on cell transformation, which examine the 'transforming' or carcinogenetic potential of RF radiation are also described.

II.3.3.1. Intracellular and intercellular signaling

The transduction of signals or 'signal transduction' is the integration of intra- and/or extracellular messages to or within a cell. The transduction of extracellular signals for example is common to the endocrine, immune, and nervous systems of mammals. Cells produce mediators such as ligands and hormones that can be detected by other cells via specific receptors located at the periphery of the cell on the plasma membrane and which induce responses in these cells. The signal can be transmitted inside the cell via a change of conformation of the receptor. This transconformation may initiate various intracellular pathways via secondary messengers: either the ligand penetrates into the cell to bind to a cytoplasmic receptor, which will then generally act at the nuclear level as a transcription factor, or it binds to the extracellular part of a transmembrane receptor, thus modifying its conformation including the intracellular part of the receptor. Then the receptor will have an increased enzymatic activity or bind to other proteins (e.g., kinases or G proteins). These reactions induce intracellular signaling such as protein phosphorylation cascades, which trigger specific cellular responses of the cell: expression of certain genes, exocytosis, etc. Signal transduction has thus three stages: communication, transduction, and signaling. The last two have been extensively studied in cell models exposed to RF radiation.

Among the several pathways of signaling, a few have been investigated in cellular models under RF exposure: processes involving calcium ion concentrations, intercellular gap junctions, and the clustering of receptors at the cell surface. However, other important signaling pathways, such as those activated by G Protein-Coupled Receptors (GPCRs), a large family of transmembrane receptors, have received little attention.

Calcium signaling

The calcium ion Ca^{2+} is one of the most important species in intra- and inter-cellular signaling as it plays a major role as a second messenger. Intracellular calcium is a crucial and ubiquitous intracellular messenger, regulating many cellular proteins involved in intracellular signaling cascades and in cellular homeostasis (Berridge et al 2000). Calcium is known to regulate processes such as cell division, differentiation, exocytosis, and differential gene transcription. Stimulation by external signals such as hormones or neurotransmitters results in intracellular Ca^{2+} oscillations in a large number of cell types. These can propagate through intercellular gap junctions to co-ordinate the activity of groups of cells (Berridge et al 2000). Transient increases in intracellular calcium ion concentration, referred to as calcium spikes, are more pronounced in nerve and muscle cells and trigger cellular responses such as contraction.

Early studies, comprehensively reviewed by AGNIR (2001), reported that very low levels of ELF-amplitude-modulated RF exposures, too low to involve heating, increased calcium efflux from isolated brain hemispheres. This efflux was assessed by measuring the movement of radiolabelled calcium ions out of brain tissue both *in vitro* (Bawin et al 1975; Sheppard et al 1979; Blackman et al 1979 1980a 1980b, 1985; Lin-Liu and Adey 1982; Dutta et al 1984; 1989) and *in vivo* (Adey et al 1982). Briefly, a modulation-frequency dependent 'window' was reported, increasing the efflux of calcium ions from brain tissue with a peak effect around 16 Hz, and a number of studies reported several 'windows' of effective power density. However, it must be noted that in many of the *in vitro* studies, the brain tissue was dead and that the outcome of the experiment was highly sensitive to temperature (Green et al 2005). Two attempted corroborations of these effects by other authors failed to support these previous findings. Shelton and Merritt (1981) found no effect on calcium efflux in rat brains exposed *in vitro* to 1 GHz pulsed at 16 or 32 Hz. An attempted replication of the effects of amplitude-modulated RF on calcium efflux from chick brain tissue exposed *in vitro* by Albert et al (1987) in which tissue slices were bathed in oxygenated saline and hence viable also failed to find any positive effect. The interpretation of these efflux data is therefore difficult and the experimental deficiencies *a fortiori* restrict any extrapolation concerning human health.

The relatively crude measures of calcium ion exchange between tissue compartments, as described above, reveal very little information of direct physiological consequence. The source of calcium in these experiments is unclear but is likely to include calcium bound to cell membranes and/or located in the extracellular spaces between cells and may also have included calcium ion exchange across the cell membrane. More sophisticated investigation of the possible effect of modulated RF on calcium metabolism has been carried out using ion-sensitive fluorescent dyes for the real-time measurement of intracellular calcium ion concentrations $[Ca^{2+}]_i$. Changes in the amount of calcium bound to the external surface of the cell membrane will influence the behavior of membrane ion channels and receptors and would affect $[Ca^{2+}]_i$ indirectly, whereas changes in calcium ion exchange between the cytoplasm and extracellular solution would directly affect $[Ca^{2+}]_i$ (Green et al 2005). Wolke et al (1996) used ion-sensitive fluorescent dyes to measure the intracellular calcium ion concentration in cultured guinea pig ventricular cardiac myocytes exposed to 900 MHz, 1.3 GHz or 1.8 GHz pulse-modulated at 217 Hz or to CW or amplitude-modulated 900 MHz (modulation at 16 and 50 Hz) at SARs of approximately 1 mW kg^{-1}. No effects on intracellular calcium levels were found in the exposed myocytes compared to those sham-exposed except for a small but statistically significant difference in cells exposed to 900 MHz modulated at 50 Hz. Similar techniques were used by Cranfield et al (2001) in their investigation of the CW and pulse-modulated 915 MHz RF at an SAR of 1.5 W kg^{-1} on intracellular calcium concentration in human leukemic T-cells. No effects were seen on mean calcium concentration, or on spontaneous intracellular calcium transient (spiking) activity.

Green et al (2005) measured the intracellular calcium ion concentration in cultured rat cerebellar granule cells and cardiac myocytes exposed to TETRA (Terrestrial Trunked Radio) signals. The cells were exposed *in vitro* to 380 MHz RF pulse-modulated at 17.6 Hz at SARs of between 5 and 400 mW kg^{-1}; changes in intracellular calcium concentrations were measured during exposure. In the granule cells, exposure had no effect on resting intracellular calcium levels, however, differences between the potassium-induced increases in intracellular calcium levels between the sham and exposed cells were attributed to initial differences between the two cell populations. In the cardiac myocytes, no effect of exposure was seen on the spontaneous intracellular calcium transients. Overall, the authors concluded that there was no consistent or biologically significant effect of TETRA fields on intracellular calcium levels.

Several research groups have examined RF effects on calcium ion channel dependent neuronal spiking (nerve impulse or action potential) behavior. A joint study by two research groups in Rome of the effects of 50 Hz magnetic fields or unmodulated 900 MHz RF exposure on single channel ionic currents and firing frequency in isolated rat dorsal root ganglion neurons found no effect of RF exposure (Marchionni et al 2006). The experimental 'targets' were the high-threshold voltage-gated calcium channels, which are responsible for the modulation of interspike interval during action potential bursts, and the calcium-activated potassium channels. The authors interpreted their data as an absence of alteration of the membrane potential under RF exposure.

In another Italian study, no effects of CW and GSM 900 MHz exposure were seen on Ba^{2+} currents through voltage-gated calcium channels in rat primary cortical neurons using patch-clamp techniques (Platano et al 2007). The authors noted that influx through voltage gated calcium ion channels is one of the main determinants of $[Ca^{2+}]_i$, the other being the release of calcium from intracellular stores. This is in agreement with results published earlier on the absence of effects of CW and GSM RF exposure on voltage-gated calcium channel permeability in rat and guinea pig ventricular myocytes (Linz et al 1999).

In contrast to these studies, a recent publication by Rao et al (2008) gave some positive evidence of a nonthermal effect of exposure on calcium dynamics in stem cell-derived neuronal cells. Exposure of the cells for 60 min to between 70 and 1100 MHz at 0.5 to 5 W kg^{-1} significantly increased the number of $[Ca^{2+}]_i$ spikes/per cell, showing a peak effect at 800 MHz. However, the authors did not exclude the possibility that these effects are due to an experimental artifact.

Table II.3.4: Calcium ion metabolism and ion channel dependent activity

Assay endpoint	Exposure conditions	Response	Comment	References
$[Ca^{2+}]_i$ in cultured guinea pig ventricular cardiac myocytes using fluorescent dyes.	900, 1300 and 1800 MHz pulse-modulated at 217 Hz; 900 MHz CW, 16 or 50 Hz modulation; at 1 mW kg^{-1} for 500 s.	No change in $[Ca^{2+}]_i$	Small statistically significant difference in cells exposed to 900 MHz modulated at 50 Hz	Wolke et al 1996
L-type calcium ion currents and potassium ion currents in cultured rat and guinea-pig myocytes using patch-clamp techniques.	GSM 900 MHz and 1800 MHz at 15 or 250 mW kg^{-1} and 80 – 880 mW kg^{-1} respectively	No effect on cell membrane potential or ion channel currents		Linz et al 1999
$[Ca^{2+}]_i$ in human leukemic T-cells using fluorescent dyes.	915 MHz CW or pulsed (GSM) at 1.5 W kg^{-1} for 10 min	No change in $[Ca^{2+}]_i$; no effect on spontaneous transients		Cranfield et al 2001

Assay endpoint	Exposure conditions	Response	Comment	References
$[Ca^{2+}]_i$ in rat cerebellar granule cells and cardiac myocytes using fluorescent dyes.	TETRA: 380 MHz RF pulse-modulated at 17.6 Hz at 5 - 400 mW kg^{-1} for consecutive 10 min periods	No change in $[Ca^{2+}]_i$; no effect on spontaneous transients		Green et al 2005
Isolated rat dorsal root ganglion neurons; single channel ionic currents and firing frequency using patch-clamp techniques.	900 MHz CW at 1 W kg^{-1}	No effect on calcium ion channels or on calcium activated potassium ion channels		Marchionni et al 2006
Primary rat cortical neurons; Ba^{2+} currents through voltage-gated calcium channels using patch-clamp techniques.	900 MHz CW and GSM; at 2 W kg^{-1} exposed 1-3 times for 90 s.	No effect on Ba^{2+} currents in voltage-gated calcium channels		Platano et al 2007
$[Ca^{2+}]_i$ spikes in neuronal cells derived from mouse embryonal P19 carcinoma cells using fluorescent dyes.	700 to 1100 MHz MHz at 0.5-50 W kg^{-1} for 60 min	Increase in number of $[Ca^{2+}]_i$ spikes per cell showing clear frequency response; maximum effect at 800 MHz.		Rao et al 2008

Nitric oxide signaling

Nitric oxide (NO) is an important intra- and intercellular signaling molecule that acts in many tissues to regulate a diverse range of physiological and cellular processes including immune system and cardiovascular system functions.

The effects of RF exposure on processes involving NO have been investigated by two Japanese groups. Vasodilatation of arterioles in the webbing of the feet of anaesthetized South African clawed toads was found to be increased under exposure to pulse modulated 10 MHz RF (Miura and Okada 1991). This effect was abolished by the addition of an NO synthase inhibitor (Miura et al 1993). These authors also reported that the exposure of a supernatant fraction of homogenized rat cerebellum to a similar pulsed 10 MHz RF field resulted in an increase in concentration of NO and cyclic guanosine monophosphate (cyclic GMP). More recently, Morimoto et al (2005) used a similar exposure setup (10 MHz, 50% duty factor, up to 8 mW kg^{-1}) and found that exposure caused a decrease in the thrombin-induced production of endothelin-1 (ET-1), a potent vasoconstrictor, and ET-1 mRNA by cultured endothelial cells. The effect on ET-1 production was abolished by addition of a nitric oxide synthase inhibitor, which was interpreted by the authors as evidence that the inhibitory effect of RF exposure is mediated, at least partly, via an NO related pathway.

Table II.3.5.: Nitric oxide signaling

Assay endpoint	Exposure conditions	Response	Comment	References
Vasodilatation of arterioles in the skin of anaesthetized *Xenopus laevis*	10 MHz pulsed at 10 kHz burst rate, 50% duty cycle; 219 V m^{-1} for up to 3 h.	RF-induced vasodilatation of arterioles preconstricted with noradrenalin.	Maximum effect after 1 h. Dosimetry rather poor.	Miura and Okada 1991
NO and cyclic GMP production by the particulate fraction from homogenized rat cerebellum	10 MHz pulsed at 10 kHz burst rate, 50% duty cycle; 790 V m^{-1} for up to 30 min.	Increased NO and cyclic GMP production	Dosimetry rather poor.	Miura et al 1993

Assay endpoint	Exposure conditions	Response	Comment	References
Thrombin-induced ET-1 and ET-1 mRNA production in bovine aortic endothelial cells, and human umbilical, aortic and microvascular endothelial cells	10 MHz pulsed at 10 kHz burst rate, 50% duty cycle; SAR: 1-8 mW kg^{-1} for up to 24 hr.	Decrease in thrombin-induced ET-1 and ET-1 mRNA production in RF-exposed cells.	Effect abolished by NG-monomethyl-L-arginine. Dosimetry rather poor.	Morimoto et al 2005

Gap junctions

Gap junctions are clusters of channels formed by proteins known as connexins, and permit the transfer of ions and small molecules between contiguous cells. Gap junction intercellular communication (GJIC), one component of the cell signaling system, is very important for cell homeostasis. A defect in GJIC is considered to be an important step during the multistage process of carcinogenesis, and disruption of GJIC has been recognized as one of the non-genotoxic mechanisms of carcinogenesis. Therefore, GJIC could be used as a biomarker to evaluate the possible health impact of RF exposure.

There are few reports about RF effects on GJIC. Ye et al (2002) exposed the heads of rabbits to 2450 MHz at 50 and 100 W m^{-2} for 3 h. One open eye was continuously exposed; the other eye of the same rabbit was covered tightly by copper grid cloth and served as control. Anterior lens capsules were obtained for a study of GJIC function using fluorescence recovery analysis after photobleaching (FRAP) technique and the localization analysis of connexin 43 using indirect immunofluorescence histochemical analysis. The results showed that the GJICs of rabbit lens epithelial cells were inhibited in a dose-dependent manner in response to RF exposure. This also caused a reduction in the amount of membrane-located gap-junction protein connexin 43. Unfortunately, the authors did not record the temperature changes during the exposure nor provided any estimate of the SAR.

Chen et al (2004a,b) studied the effect of 30.16 GHz millimeter wave RF exposure at 10 and 35 W m^{-2} on GJIC in cultured HaCaT keratinocytes using the FRAP approach. The results showed that RF exposure alone for 1 h at either 10 or 35 W m^{-2} did not affect GJIC. However, RF exposure in combination with treatment with the chemical promoter phorbol-12-myristate-13-acetate (PMA) reversed the PMA-induced suppression of GJIC. Exposure at 10 W m^{-2} resulted in a partial reversal, and exposure at 35 W m^{-2} resulted in a full reversal of the PMA-induced suppression. Temperatures during exposure were measured in this study, but no estimate of SAR was given.

Table II.3.6.: Gap junctions

Assay endpoint	Exposure Conditions	Response	Comment	References
GJIC and connexin 43 in lens epithelial cells of rabbits	2450 MHz RF at 50 W m^{-2} and 100 W m^{-2} for 3 h	RF- induced inhibition of GJIC and damage to connexin 43.	No measure of temperature or SAR.	Ye et al 2002
GJIC of human (HaCaTs) keratinocytes	30.16 GHz RF exposure at 10 and 35 W m^{-2} for 1 h	GJIC suppression induced by PMA could be eliminated or diminished by exposure to RF.		Chen et al 2004a, b

Receptor clustering

Receptors are cellular membrane proteins that can bind specific signal molecules (ligands) and initiate a response in the cell. The response often starts with the clustering of receptors after binding its ligand, then activating certain signal pathway(s), changing cellular biological and/or biochemical processes, and resulting in the alteration of cell behaviors such as proliferation, apoptosis, invasion, or metastasis. Such receptors may be present on the outer cell membrane, the nuclear envelope or other membrane structures.

In recent years, the effects of EMFs on the clustering of cellular membrane surface receptors, such as epidermal growth factor receptor (EGFR) or tumor necrosis factor receptor (TNFR) have been investigated. While there are many reports focusing on 50 Hz magnetic fields, only two studies have specifically addressed RF. Xie et al (2006) exposed Chinese hamster lung (CHL) cells to 1800 MHz fields at SARs of 0.1, 0.5, 1.0, 2.0 and 4.0 W kg^{-1}. The results showed that clustering of EGFR was induced by exposure to 217 Hz modulated RF for 15 min at the lowest SAR of 0.5 W kg^{-1}. Unmodulated RF did not induce this phenomenon, and superposition of 2 μT 50 Hz noise inhibited EGF receptor-clustering induced by RF. However, functional significance of this effect could only be determined by the investigation of RF effects on the activation of the normal EGFR signal pathway and resulting changes in cellular physiology.

Xu et al (2006) investigated glutamate receptor clustering and synaptic activity in rat brain cells. Glutamate receptor channels play key roles in excitatory synaptic transmission and are involved in many physiological and pathological processes. The authors exposed cultured hippocampal neurons of rats for 15 min per day for 8 days to GSM-1800 MHz signals at an average SAR of 2.4 W kg^{-1}. Whole-cell patch clamp techniques were used to assess the miniature excitatory postsynaptic currents (mEPSCs) in NMDA (*N*-methyl-D-aspartate) and AMPA (α-amino-3-hydroxy-5-methyl-4-soxazole propionic acid) glutamate receptor channels. Synaptic density on the distal neuronal dendrites of the hippocampal cells was assessed using immunohistochemical staining. The authors reported that RF exposure reduced excitatory synaptic activity in AMPA receptors. Neither AMPA nor NMDA receptor clustering affected; however, the expression of post-synaptic density 95 (PSD95), which is involved in orchestrating excitatory synapse maturation and synaptic plasticity, was decreased after RF exposure. These observations require experimental confirmation.

Table II.3.7.: Receptor clustering

Assay endpoint	Exposure Conditions	Response	Comment	References
Epidermal growth factor (EGF) receptor clustering in Chinese hamster lung fibroblasts.	1800 MHz GSM RF or CW; SAR: 0.1, 0.5, 1, 2, and 4 W kg^{-1} for 15 min.	Clustering induced after exposure to GSM 1800 MHz RF modulated by 217 Hz at SAR ≤ 4 W kg^{-1}.	Superposition of a 2 μT 50 Hz MF inhibited RF-induced EGF receptor clustering	Xie et al 2006
Synaptic activity and receptor density in cultured rat neurons using patch clamp and immunohistochemical staining.	1800 MHz GSM RF at SAR of 2.4 W kg^{-1} for 15 min per day for 8 days	Decreased the amplitude of AMPA mEPSCs; no change in NMDA mEPSCs amplitude.	The expression of postsynaptic density 95 (PSD95) in neuronal dendrites was decreased.	Xu et al 2006

Summary on signaling, gap junctions and receptor clustering

Overall, the evidence of effects on calcium signaling from recent, well conducted studies, particularly those using functionally significant measures of calcium ion concentration, do not support the earlier reports suggesting that low-level amplitude modulated RF can modify intracellular calcium ion metabolism, particularly with regard to its role as an intracellular messenger. In addition, no compelling effects have been seen on the spiking activity of neurons dependent on calcium ion channel properties.

The evidence for any effects of RF exposure on nitric oxide signaling processes is rather weak and insubstantial. No definite conclusion can be drawn based on the few data on effects of RF exposure on cellular gap junctions or receptor clustering.

II.3.3.2. Gene and protein expression

Older studies focused on the response of small numbers of genes and/or proteins, so greatly restricting the ability of any individual study to determine the potential cellular responsiveness to RF. More recently, technological advances have facilitated the ability to screen for RF responsive gene(s) or protein(s) on a large scale using for example DNA microarray and proteomic technologies. Such automation provides the means for greatly increasing the amount of information that may be derived from a single experiment but at a cost, namely the increased difficulty in identifying biologically significant responses from experimental 'noise'. Interpretation of the results relies heavily on complex statistical analysis that is very sensitive to the applied level of stringency with which meaningful responses are identified (Mayo, et al 2006). The various strengths and pitfalls of some of these high throughput technologies for screening for RF-induced 'epigenetic' changes have been discussed in detail by Leszczynski and Meltz (2006) who concluded that the techniques are at present useful primarily as experimental research tools. However, they may eventually be used to identify endpoints suitable for screening for animal, volunteer and epidemiological investigation, leading to a better understanding of the potential health effects, if any, of environmental levels of EMF exposure.

There are a number of different conventions for distinguishing the mRNAs that result from gene expression and the proteins which they encode. In this report, the following convention is used: genes are italicized and proteins are not. The prefix c- (as in *c-fos*) is sometimes used to indicate a gene of cellular origin, as opposed to viral (*v*) origin.

Gene expression

The conventional method for analysis of gene expression is Northern blotting. In this method total cellular RNA or mRNAs are separated by alkaline agarose gel electrophoresis and transferred (blotted) to membranes. Specific RNA transcripts are identified by hybridization of gene-specific probes, usually radioactively labeled, to membranes. Transcript levels are assessed by the relative strength of the signal of the radioactively labeled probe to a specific sized gene fragment. The method is at best semi-quantitative.

More recently, reverse transcriptase polymerase chain reaction (RT-PCR) methods have been introduced. In this method RNA is converted to complementary DNA (cDNA) sequences by the enzyme reverse transcriptase and gene-specific fragments are amplified by successive rounds of DNA synthesis using thermostable DNA polymerase enzymes. Gene specificity is achieved by using specifically synthesized primers that are unique to the gene being analyzed. In its simplest form RT-PCR is not highly quantitative. However, several systems such as real-time RT-PCR have been developed that allow highly precise quantification through the use of fluorescence measurements of specific gene products. This method is generally referred to as quantitative RT-PCR (QRT-PCR or Q-PCR). The Q-PCR methods are the most sensitive methods available for quantification of transcript levels and can detect differences in transcript levels over several orders of magnitude.

Transcriptomics describes the study of global gene expression; the genome in human and other mammalian cells comprises typically 20,000 – 30,000 genes. The transcriptome comprises the RNAs produced from the genome of a cell or tissue. Techniques using oligonucleotide chips, cDNA glass microarrays or microbead array systems rely on the binding of fluorescence labeled cDNA from the cells of interest to a set of complementary sequences on the chip or array and measuring the fluorescence intensity at each site. In this way the quantitative measures of gene expression within the entire genome in cells from two populations can be compared. However, it is widely acknowledged that there is a need to verify any ensuing changes in gene expression through other techniques such as Q-PCR. In particular, the sensitivity of array systems and their 'dynamic' range are considerably less than those of Q-PCR. Alternative sensitive techniques such as HICEP (high coverage gene expression profile) in which all RNA transcripts are amplified and separated by capillary electrophoresis for subsequent sequencing are also becoming available.

It is now becoming clear that there are additional levels of regulation of gene expression; these include the expression of regulatory micro RNAs (miRNAs), DNA methylation and a variety of modifications of chromatin-associated proteins. Methods to analyze such changes have not yet been applied to investigate

the possible impact of RF, but EMF studies have, however, utilized gene-specific approaches such as Northern blotting, PCR and array-based genomic approaches to examine RF-induced changes in gene expression. Studies on mammalian cells are summarized and reviewed in the following two chapters; a third chapter addresses studies looking specifically at gene expression in plants.

Gene-specific approaches in mammalian cells

RF studies of gene expression have focused typically on early response genes, otherwise known as proto-oncogenes, such as *c-fos*, *c-myc* and *c-jun*, tumor suppressor genes such as *p53*, and stress responsive genes such as the family of heat shock protein (*hsp*) genes.

Czyz et al (2004) exposed pluripotent wild-type and *p53*-deficient mouse embryonic stem cells to pulse-modulated 1.71 GHz GSM RF field at SARs of up to 2 W kg^{-1}. The authors reported that *hsp70* m-RNA was significantly upregulated and transient slight increases were also found in *c-jun*, *c-myc* and *p21* expression. However, such changes were not found in wild-type cells which have the normal *p53* gene, suggesting that cellular responses to RF were determined by the genetic background of the cells, including the expression of *p53*.

Exponentially growing human lymphoblastoma cells were exposed to 1900 MHz pulse-modulated RF fields at average SARs of 1 and 10 W kg^{-1} (Chauhan et al 2006). The authors reported that the expression of the proto-oncogenes *c-fos*, *c-jun*, *c-myc* and the stress protein genes *hsp27* and inducible *hsp70* in RF-exposed cells, assessed through the use of Q-PCR, were similar to those in sham-exposed cells. Thus, the study found no evidence that the 1900 MHz RF-field exposure caused a general stress response in these cells, while a heat shock (43^{o}C for 1 h) positive control increased the transcript levels of *hsp27*, *hsp70*, *c-fos* and *c-jun*.

Rat pheochromocytoma (PC-12) cells treated with nerve growth factor (NGF) were exposed to 836.55 MHz (TDMA) for 20 to 60 min at 0.09 to 9 W m^{-2}, and expression levels of *c-jun* and *c-fos* were determined using Northern blot analysis (Ivaschuk et al 1997). The mRNA level for *c-fos* was not changed. However, expression of *c-jun* in cells that were exposed for 20 min at 90 W m^{-2} was lower than that of the sham group. Additionally, in cells that were exposed for 40 to 60 min, the expression of *c-jun* did not differ from sham-exposure, perhaps implying a rather variable response.

In the logarithmic growth phase, the phase transiting to the plateau phase, and the plateau phase, mouse-derived C3H10T1/2 cells were exposed to two kinds of RF fields (835.62 MHz (FMCW)) or 847.74 MHz (FDMA) for 4 days at SAR of 0.6 W kg^{-1} (Goswami et al 1999). In all the RNA that was isolated from the cells, mRNAs of *c-fos*, *c-jun* and *c-myc* were synthesized using the RT-PCR method and verified using gel electrophoresis. No differences from the sham-exposed group were found. In addition, there was no difference in DNA binding capacity of the AP1, AP2, and NF-κB transcription factors. However, in the FMCW-exposed group in both the phase transiting to plateau level and the plateau phase, mRNA of *c-fos* was increased about 2-fold. A similar increase (approximately 1.4 fold) mRNA of *c-fos* also was observed following CDMA RF exposure.

The RF effect on expression of genes other than hsps and oncogenes has been examined in several studies. The effect on *egr-1* gene expression of a modulated RF field of 900 MHz generated by a wire patch cell antenna exposure system was studied as a function of time in human SH-SY5Y neuroblastoma cells. Short-term exposure induced a transient increase in the *egr-1* mRNA level paralleled with activation of the MAPK subtypes ERK1/2 and SAPK/JNK (Buttiglione et al 2007). The results suggest that exposure to 900 MHz modulated RF field affects both *egr-1* gene expression and cell regulatory functions involving apoptosis inhibitors such as *bcl-2* and *survivin*.

Intermittent exposure of human Mono Mac 6 (MM6) cells to ultra-wideband (UWB) pulses for a total of 90 min, (Natarajan et al 2006), revealed no difference in NF-κB-dependent gene expression profiles at 8 or 24 h post-exposure, indicating that activated NF-κB does not lead to differential expression of κB-dependent target genes.

Table II.3.8.: Gene specific approaches

Assay endpoint	Exposure Conditions	Response	Comment	References
c-jun and *c-fos* expression in PC12 cells using Northern blot analysis	836.55 MHz, TDMA, at 0.09, 0.9, and 90 W m^{-2} for 20, 40 or 60 min	No change in c-*fos* transcript levels. Transcript levels for *c-jun* were decreased only after 20 min exposure to 90 W m^{-2}.	Data are shown in only tables. No figures of northern blot in the results chapter. No SAR given.	Ivaschuk et al 1997
c-fos, c-jun and *c-myc* mRNA levels in C3H 10T½ mouse embryo fibroblasts using gel mobility shift assay for DNA-binding, RT-PCR.	FMCW, 835.62 MHz, FDMA, 847.74 MHz, SAR: 0.6 W kg^{-1} for 4 days	Significant increases in *c-fos* mRNA levels were detected in exponentially growing cells.		Goswami et al 1999
c-jun, *c-fos*, *c-myc*, *p53, hsp27* and *hsp70* in pluripotent embryonic stem (ES) cells, (wild-type and *p53*-deficient), using RT-PCR (mRNA)	1.71 GHz (GSM-217, GSM-Talk, GSM-DTX), intermittent (5 min on/30 min off) at an SAR: 0.11-2 W kg^{-1} for 6-48 hr.	Upregulation of *hsp70* mRNA levels in *p53*-deficient ES cells, but not in wild-type cells.	Only *p53*-deficient ES cells were up-regulating *hsp70* mRNA.	Czyz et al 2004
c-jun, *c-fos*, *c-myc*, *hsp27* and *hsp70* in human TK6 lymphoblastoma cells, using RT-PCR (total RNA).	1.9 GHz pulse-modulated RF fields at SAR: 1 and 10 W kg^{-1}, for 6 hr.	No effects on transcript levels of these genes in RF-field-exposed cells.		Chauhan et al 2006
NF-κB-dependent gene expression profiles in human Mono Mac 6 (MM6) cells.	UWB, 100 kV m^{-1}, pulse width = 0.79±0.01 ns, at 250 pps. for 8 or 24 h	No effect on the NF-κB-dependent gene expression profiles	However, the NF-κB DNA binding activity increased at 24 h incubation after EMF exposure.	Natarajan et al 2006
Apoptosis-related gene expression: *Egr-1*, *p53*, *Bcl-2*, *survivin*, etc) using RT-PCR in human SH-SY5Y neuroblastoma cells.	900 MHz, GSM modulated at 1 W kg^{-1}, for up to 24 hr.	No effect on *p53* expression but significant changes in *Egr-1*, *Bcl-2* and *survivin* expression.	Significant 2.3% increase in apoptotic cell population and G2/M cell cycle arrest	Buttiglione et al 2007

Transcriptomics in mammalian cells

In an initial study utilizing a membrane-based cDNA microarray, Harvey and French (1999) studied the effects 864.3 MHz (CW) on HMC-1 human monocytes. Exposure was carefully controlled and averaged at an SAR of 7 W kg^{-1}. Three exposure runs each of 20 min were performed at 4-h intervals daily for 7 days. cDNA microarray revealed consistent alterations in steady-state mRNA levels of 3 of the 558 genes represented on the membranes including one proto-oncogene *c-kit* (increased), one apoptosis-associated gene *dad-1* (decreased) and one potential tumor suppressor gene *ndpk* (decreased). However, there was considerable variability between the two separate experiments reported. The exposure did not result in a broad effect on gene expression and the relative change of each differentially expressed gene was small (< 1.5 fold). The authors did not use other quantitative methods to confirm their finding, which is generally accepted as necessary when determining the significance of such small changes.

Pacini et al (2002) investigated the effect of gene expression in human skin fibroblasts by using cDNA arrays including 82 genes, and reported that exposure to GSM 902.4 MHz RF at an average SAR of 0.6

W kg^{-1} for 1 h increased the expression of 14 genes which function in mitogenic signal transduction, cell growth and apoptosis. The authors further demonstrated a significant increase in DNA synthesis and intracellular mitogenic second messenger formation which matched with the high expression of MAP kinase family genes. The authors suggested that the RF exposure has significant biological effects on human skin fibroblasts. However, only one experiment was performed with array analysis and no further experiment was made by the authors to confirm the array data.

Using a cDNA microarray to examine the expression of 3600 genes, Leszczynski et al (2004) reported that exposure to GSM 900 MHz RF at an average SAR of 2.4 W kg^{-1} for 1 h changed expression of a number of genes, including down-regulated genes involved in forming the Fas/TNFa apoptotic pathway in human endothelial cell line EA.hy926. The authors performed three separate experiments by array analysis, but no confirmation experiments were conducted to validate the array result. More recently, Nylund and Leszczynski (2006) compared the global gene response of two human endothelial cells, EA.hy926 and its variant EA.hy926v1, to RF and reported that the same genes were differently affected by the exposure to GSM 900 MHz RF at an average SAR of 2.8 W kg^{-1} for 1 h in each of the cell lines. However, the differentially expressed genes in this study were not confirmed using other methods.

Lee et al (2005) used the Serial Analysis of Gene Expression (SAGE) method to measure the RF effect on genome scale gene expression in HL 60 cells. The cells were exposed to 2.45 GHz RF at an average SAR of 10 W kg^{-1} for 2 h and 6 h. The authors observed that, after 2 h and 6 h exposure, 221 and 759 genes altered their expression, respectively. Functional classification of the affected genes revealed that apoptosis-related genes were among the upregulated ones and the cell cycle genes among the downregulated ones, but no significant increases in the expression of heat shock genes were found. However, the SAGE experiment was repeated only once and only one control with a 2 h sham exposure was used and no confirmation experiment was reported to validate these results.

Huang et al (2006) investigated the effect of 1763 MHz RF on gene expression in Jurkat cells using Applied Biosystems 1700 full genome expression microarray. The authors found that 68 genes were differentially expressed in the cells after exposure to RF at SAR of 10 W kg^{-1} for 1 h and harvested immediately or after 5 h. The authors repeated the sets of runs five times to collect biological triplicates in every sample. However, the results were not confirmed by other methods.

Whitehead et al (2006a; 2006b) have performed *in vitro* experiments with C3H 10T(1/2) mouse cells to determine whether FDMA or CDMA modulated RF radiations can induce changes in gene expression using the Affymetrix U74Av2 GeneChip. The data showed the number of probe sets with an expression change greater than 1.3-fold was less than or equal to the expected number of false positives in C3H 10T(1/2) mouse cells after 835.62 MHz FDMA or 847.74 MHz CDMA modulated RF exposure at SAR of 5 W kg^{-1} for 24 h. The authors concluded that the exposures had no statistically significant effect on gene expression. Leszczynski (2007) raised the criticism that false positives had not been validated as 'false' using non-transcriptomic methods, but this view was challenged by Whitehead et al (2007).

In the study by Gurisik et al (2006), human neuroblastoma cells (SK-N-SH) were exposed to GSM 900 signals at SAR of 0.2 W kg^{-1} for 2 h and recovered without field for 2 h post-exposure. Gene expression were examined by Affymetrix Human Focus Gene Arrays including 8400 genes and followed by real-time RT-PCR of the genes of interest. Only six genes were found to be slightly down-regulated in response to RF exposure comparing with sham-exposed cells, but this response could not be confirmed by real-time RT-PCR analysis. Thus, the authors concluded that the RF exposure applied in this study did not change gene expression in SK-N-SH cells. However, the array experiment was repeated only once and only one array was used for each exposure or sham exposure group.

Qutob et al (2006) have assessed the ability of exposure to a 1.9 GHz pulse-modulated RF field to affect global gene expression in U87MG glioblastoma cells by application of Agilent Human 1A (v1) oligonucleotide 22K microarray slides. The cells were exposed to pulse-modulated (50 Hz, 1/3 duty cycle) RF fields at an SAR of 0.1, 1.0 and 10.0 W kg^{-1} for 4 hours, and incubated for an additional 6 hours. The authors found no evidence that exposure to RF fields under different exposure conditions can affect gene expression in the cells. In this study, the authors performed five experiments, each containing a single replicate and some of genes were confirmed as real "unaffected genes".

As a follow-up to this study, Chauhan et al, (2007b) examined the effect of RF field exposure on the possible expression of late onset genes in U87MG cells after a 24 h RF exposure period and found no changes of gene expression. They also tested immediately and 18 h post-exposure the gene expression of a human monocyte-derived cell-line (Mono-Mac-6, MM6) in response to intermittent exposure (5 min on/10 min off) for 6 h, and found again a negative effect.

Zeng et al (2006) have investigated gene expression profile in MCF-7 cells after exposing to GSM 1800 RF using Affymetrix Genechip U133A. The results showed that gene expression did not change consistently following intermittent exposure (5 min on/10 min off) at an average SAR of 2.0 W kg^{-1} for 24 h but the expression of five genes was changed consistently after exposure at SAR of 3.5 W kg^{-1}. However, this result could not be further confirmed by real-time RT-PCR assay.

Remondini et al (2006) investigated the effect of RF on gene expression profile in six different cell lines or primary cells, and found that various types of cell reacted differently in RF exposure. Gene expression changed in 900 MHz-exposed EA.hy926 endothelial cells (22 up-regulated, 10 down-regulated), 900 MHz-exposed U937 lymphoblastoma cells (32 up-regulated, two down-regulated), and 1800 MHz-exposed HL-60 leukemia cells (11 up-regulated, one down-regulated), while NB69 neuroblastoma cells, T-lymphocytes, and CHME5 microglial cells did not show significant changes in gene expression. The authors concluded that there were alterations in gene expression in some human cells types exposed to RF but these changes depended on the type of cells and RF signal. However, these RF-responsive candidate genes in different types of cells were not confirmed by other methods. In addition, the RF exposures were different for the different types of cells, so a simple comparison of the effects of RF exposure on gene expression in these cells was not possible.

Zhao R et al (2007) investigated the effects of RF EMF on gene expression of cultured rat neuron with Affymetrix Rat Neurobiology U34 array. Among 1200 candidate genes, 24 up-regulated genes and 10 down-regulated genes associated with multiple cellular functions were identified after 24-h intermittent exposure (5 min on/10 min off) at an average SAR of 2.0 W kg^{-1}. The changes of most of the genes were successfully validated by real-time RT-PCR; these included genes involved in cytoskeleton, signal transduction pathway, and metabolism.

Adopting a similar research strategy, Zhao TY et al (2007) investigated whether expression of genes related to cell death pathways are dysregulated in primary cultured neurons and astrocytes by exposure to a working GSM cell phone rated at a frequency of 1900 MHz for 2 h. Array analysis and real-time RT-PCR showed up-regulation of *caspase-2*, *caspase-6* and *Asc* (apoptosis associated speck-like protein containing a caspase recruitment domain or 'card') gene expression occurred in both "on and "stand-by" modes in neurons, but only in "on" mode in astrocytes. Additionally, astrocytes showed up-regulation of the *bax* gene. The authors concluded that even relatively short-term exposure to cell phone radiofrequency emissions can up-regulate elements of apoptotic pathways in cells derived from the brain, and that neurons appear to be more sensitive to this effect than astrocytes. However, the authors used a working mobile phone as the source of RF signal, and thus the exposures were not well defined or controlled.

Table II.3.9.: Transcriptomics

Assay endpoint	Exposure Conditions	Response	Comment	References
Gene expression in human mast cell line	864.3 MHz CW; SAR: 7.0 W kg^{-1}, three 20 min exposures at 4-h intervals daily for 7 days	Changes in the expression of *c-kit*, n*ucleoside diphosphate kinase B*, and *DAD-1* genes by less than 1.5 fold.	Only two separate experiments, and no confirmation experiments for differentially expressed genes	Harvey and French 1999

Assay endpoint	Exposure Conditions	Response	Comment	References
Gene expression in human skin fibroblasts	GSM 902.4 MHz, at an SAR: of 0.6 W kg^{-1} for 1 hr	14 genes were up-regulated by more than 1.5 fold.	No replicate experiment in array analysis, and no confirmation experiments for the differentially expressed genes	Pacini et al 2002
Gene expression in human endothelial cell line EA.hy926	GSM 900 MHz; SAR: 2.4 W kg^{-1} for 1 hour	3600 differentially expressed genes, including down-regulated genes involved the Fas/TNFa apoptotic pathway	Three separate experiments, but no confirmation experiment	Leszczynski et al 2004
Gene expression in HL60 cells	2.45 GHz, SAR: 10 W kg^{-1}, for 2 h and 6 h	Apoptosis-related genes up-regulated and the cell cycle genes down-regulated	The experiment was repeated only once and only one control with 2 h sham exposure, no confirmation experiment	Lee et al 2005
Gene expression in C3H 10T(1/2) mouse cells	835.62 MHz FDMA or 847.74 MHz CDMA modulated RF radiation; SAR: 5 W kg^{-1} for 24 hours	No effects.	The number of probe sets with an expression change greater than 1.3-fold was less than or equal to the expected number of false positives.	Whitehead et al 2006a; Whitehead et al 2006b
Gene expression in human neuroblastoma cells (SK-N-SH)	GSM 900 MHz RF SAR: 0.2 W kg^{-1} for 2 hours and recovered without field for 2 h post-exposure.	No effect. Only six genes were found to be slightly down-regulated, but these genes could not be confirmed by real-time RT-PCR analysis	The array experiment was repeated only once and only one array for exposure or sham exposure group.	Gurisik et al 2006
Gene expression in U87MG glioblastoma cells	1.9 GHz pulse-modulated (50 Hz, 1/3 duty cycle) RF, SAR: 0.1, 1.0 and 10.0 W kg^{-1} for 4 hours, and incubated for an additional 6 hours	No effect. No differentially expressed genes were found by different statistical analysis.	Five experiments were performed, each containing a single replicate.	Qutob et al 2006

Assay endpoint	Exposure Conditions	Response	Comment	References
Gene expression in Jurkat cell	1763 MHz, SAR: 10 W kg^{-1} for 1 hour and harvested immediately or after five hours	68 genes were differentially expressed after exposure	The authors repeated sets of experiment five times to collect biological triplicates in every sample. But the differentially expressed genes were not confirmed by other methods.	Huang et al 2006
Gene expression in EA.hy926 and EA.hy926v1	GSM 900 MHz, SAR: 2.8 W kg^{-1} for 1 hour	Four up-regulated genes and 89 down-regulated genes were found in EA.hy926 cell line while 61 up-regulated genes and one down-regulated gene were found in EA.hy926v1 cell line.	Each array experiment was repeated three times (n=3) for each cell line using three different cultures of cells. But no attempt was made to confirm the differentially expressed genes by other methods.	Nylund and Leszczynski 2006
Gene expression in MCF-7 cells	GSM 1800 MHz at an SAR: 2 W kg^{-1}, 3.5 W kg^{-1}, intermittent exposure (5 min on/ 10 min off) for 24 hours	No consistently changed genes at 2 W kg^{-1}. 3.5 W kg^{-1} exposure produced five consistently changed genes, but these genes could not be confirmed by real-time RT-PCR.	Duplicate arrays were Applied to two independent exposure or sham exposure groups.	Zeng et al 2006
Gene expression in six types of cells, including NB69 neuroblastoma cells, T lymphocytes, CHME5 microglial cells, EA.hy926 endothelial cells, U937 lymphoblastoma cells, and HL-60 leukemia cells	900 and 1800 MHz RF EMF with different exposure patterns, SAR: 1-2.5 W kg^{-1} for up to 44 h,	22 up-regulated and 10 down-regulated genes in GSM 900-RF exposed EA.hy926 cells. 32 up-regulated, 2 down-regulated genes in U937 cells. 11 upregulated, 1 downregulated genes in HL-60 cells.	RF-responsive candidate genes in different types of cells were not confirmed by other methods.	Remondini et al 2006

Assay endpoint	Exposure Conditions	Response	Comment	References
Gene expression in cultured rat neurons	GSM 1800 MHz, SAR: 2.0 W kg^{-1}, intermittent exposure (5 min on/ 10 min off) for 24 hours	24 up-regulated genes and 10 down-regulated genes, most of these changes were successfully validated by real-time RT-PCR	The array experiment was repeated only once and only one array for exposure or sham exposure group.	Zhao R et al 2007
Gene expression in human glioblastoma-derived cell-line (U87MG) and human monocyte-derived cell-line (Mono-Mac-6, MM6)	1.9 GHz pulse-modulated RF intermittent (5 min on/ 10 min off) exposure: U87MG cells for 24 h; SAR: 0.1, 1.0 and 10.0 W kg^{-1}; MM6 cells for 6 h; SAR: 1.0 and 10.0 W kg^{-1}.	RF field exposure did not alter gene expression in either cultured U87MG or MM6 cells	5 biological replicates per exposure condition.	Chauhan et al 2007b
Gene expression in primary cultures of neurons and astrocytes	GSM 1900 exposure using a mobile phone for 2 hours	RF exposure up-regulated apoptosis related genes in neurons under both stand-by and on mode, but in astrocytes only under on mode	SAR not measured; two arrays in each group; differentially regulated genes were confirmed by real-time RT-PCR	Zhao TY et al 2007

Gene expression in plants

As many *in vivo* studies on EMF have led to highly conflicting results and investigations on intact organisms are to be preferred to cultured cells, Vian et al have performed a series of experiments on tomato plants (*Lycopersicon esculentum*) as these constitute a model system for studying plant responses to environmental stresses. For their investigations they used a reverberation chamber that allows RF exposure as found in urban environments but protecting the experiment from unwanted external RF. They were particularly interested in the very rapid molecular responses following RF exposure in order to minimize side effects and the possible influence of other factors. To do this, they monitored the levels of several wound-induced transcripts within minutes after short-term RF-exposure. Their findings have been reported in several publications (Vian et al 2006, 2007; Roux et al 2006, 2008; Beaubois et al 2007). Two findings can be highlighted. The first is that all transcripts that were shown to be upregulated had been previously found to be wound-induced. This implies that tomato plants perceive and respond to low-level RF as if it were injurious. Furthermore, the response observed at 4.2 W m^{-2} was comparable to that evoked at 66 mW m^{-2}. This "all-or-none" response, along with the fact that responses were shown to be systemic (Beaubois et al 2007), suggests that the RF-evoked "wound signal" is an electrical signal within the plant.

These investigations on plants are certainly interesting from a scientific and mechanistic point of view but are unlikely to be directly transferable to man. The results should therefore not be overestimated in terms of their relevance to human health. This is especially true as there might be methodological shortcomings; for example, the absence of any SAR estimation and dosimetry are limiting factors in evaluating the significance of the findings.

Protein expression

Conventional methods of protein analysis depend upon methods such as Western blotting and traditional biochemistry. In Western blotting, proteins are separated using acrylamide gels and transferred to membranes. The membranes are subsequently stained with antibodies to specific proteins of interest. The presence or absence of specific proteins and crude indications of relative abundance can be determined. Proteins can also be visualized in histological or cellular preparations using immunocytochemistry.

Proteomics is the term applied to the global analysis of the protein complement of a cell. The 'proteome' is complex consisting of tens of thousands of proteins each of which may be subject to post-translational modification. Such modifications can be important for determining the enzymatic activity half-life and location of a protein or its propensity to interact with other molecules, following phosphorylation for example. Typically, analysis is by 2 dimensional (2D) gel electrophoresis, separating individual proteins on the basis of size and electric charge. These methods have been greatly improved in recent years by the development of standardized protocols and sophisticated image analysis software. These 2D gel systems may also be able to detect different post-translationally modified forms of individual proteins. Various mass spectrometry techniques can be used to identify individual proteins. In addition, protein microarrays and chips, often based on monoclonal antibodies, are being developed that will provide quantitative information regarding the expression of a series of functionally linked proteins. These techniques can also be applied to measure the functional state of proteins by examining their phosphorylation status.

EMF studies have generally taken advantage of protein specific approaches such as Western blotting and 2D gel approaches for studying exposure-induced effects on the proteome; few groups have examined such effects using array-based proteomic approaches. However, the proteome of higher eukaryotes is far from being completely understood and it must be recognized that the techniques currently available are unable to describe all effects of toxins on the proteome. The various studies are summarized and reviewed in the following two chapters.

Protein-specific approaches

Many recent studies of RF effects at the cellular level have investigated possible effects on heat shock proteins (hsps), the expression of which is induced by various environmental stresses and forms part of a general cellular stress response, increasing stress tolerance and cytoprotection against stress-induced molecular damage. However, it is not always clear in these studies whether hsp expression has been induced by RF heating or results from a non-thermal RF field-specific stress. Such a distinction requires studies to be conducted under rigorously controlled conditions.

A few biological experiments have been designed and performed to test the hypothesis of nonthermal induction of hsp as a mechanism for RF bioeffects. One such investigation was carried out by De Pomerai et al (2000) who reported increased expression of hsp16 in the nematode *Caenorhabditis elegans*. These nematodes were exposed in a TEM cell at 750 MHz (CW, SAR estimated as 1 mW kg^{-1}). However, the same group reported that a small temperature rise may have contributed to the elicitation of the effect, as losses in the TEM cell induced temperature elevation in the exposed samples of ~0.2°C (Dawe et al 2006); these authors also revised the previous estimate of SAR up to 4-40 mW kg^{-1}. This implies that, in the initial report, at least part of the cause was thermal. More recently, Dawe et al (2006, 2007) have reported that exposure to CW or GSM 1800 RF at an SAR of 1.8 W kg^{-1} did not induce hsp16 in this experimental model.

Kwee et al (2001) had reported that the expression of hsp70, but not hsp27, was induced when transformed human epithelial amnion cells were exposed to a GSM 900 signal at a SAR of 2.1 mW kg^{-1} for 20 min.

Miyakoshi and colleagues have also investigated hsp expression. Using an exposure dish with 3 sections, human brain tumor derived MO54 cells were exposed to 2450 MHz RF fields (SAR: 5, 20, 50, and 100 W kg^{-1}) and cell survival rates and hsp70 expression were determined. At SAR below 20 W kg^{-1}, no effect on hsp70 expression was observed using Western blotting, but, at 20 W kg^{-1} and higher, hsp70 expression was increased in an SAR and exposure-duration dependent manner (Tian, et al 2002). They also examined the effects 2450 MHz exposure on hsp expression in A172 cells, using a wide range of SARs.

There was no significant change in hsp27 expression caused by RF at 5-20 W kg^{-1} or by comparable heating for 1-3 h. However, hsp27 phosphorylation increased transiently at 100 and 200 W kg^{-1} to a greater extent than at 40-44°C (Wang et al 2006). In another experiment, MO54 cells were exposed to 1950 MHz RF fields at SARs of 1 to 10 W kg^{-1} and the expression levels of hsp27, hsp70 and phosphorylated hsp27 (serine 78) were determined. No differences in expression volumes of hsp27 and hsp70 were found compared with the sham group, but expression of phosphorylated hsp27 was significantly decreased after 1- and 2-hour exposure at 10 W kg^{-1} (Miyakoshi et al 2005).

No significant differences in the expression levels of phosphorylated hsp27 at serine 82 were observed between the test groups exposed to W-CDMA or CW signal (80 and 800 mW kg^{-1} for 2-48 h) and the sham-exposed negative controls, evaluated immediately after the exposure periods by bead-based multiplex assays on human A172 and IMR 90 cells. Moreover, no noticeable differences in the gene expression of hsps were observed between the test groups and the controls by DNA chip analysis and indirect immunofluorescence methods (Hirose et al 2007).

RF radiation (27 MHz or 2450 MHz, CW signal for 2 h) at much higher SARs (25 and 100 W kg^{-1}) failed to induce the heat shock response in HeLa and CHO cells (Cleary et al 1997). Lim et al (2005) reported that heat caused an increase in the number of cells expressing stress proteins (hsp70, hsp27), measured using flow cytometry, and this increase was dependent on time. However, no statistically significant difference was detected in the number of cells expressing stress proteins after RF-field exposure of 900 MHz at three average SARs (0.4, 2.0 and 3.6 W kg^{-1}).

The expression of three heat-shock proteins (hsp70, hsc70, hsp27) using immunohistochemistry after exposure to RF fields was investigated on human primary keratinocytes and fibroblasts (Sanchez et al 2007). The results showed no effect of a 48-h GSM 1800 exposure at 2 W kg^{-1} on either keratinocytes or fibroblasts, in contrast to ultraviolet B (UVB)-radiation or heat-shock positive control treatments.

Caraglia et al (2005) reported that RF at 1.95 GHz (3.6 W kg^{-1}) induces apoptosis in human epidermal cells through the inactivation of the ras $\rightarrow$ erk survival signaling due to enhanced degradation of ras and raf-1 determined by decreased expression of hsp90 and the consequent increase of proteasome-dependent degradation.

Friedman et al (2007) also found that exposure to 875 MHz RF for 5 to 30 minutes (0.05 to 3.1 W m^{-2}) activated erk signaling pathways. Erk phosphorylation was observed in Rat1 and Hela cells. The cell response was observed already at 0.05 W m^{-2} (1.4 and 2 fold in Rat1 and Hela cells, respectively) and reached the maximum level at 1.1 W m^{-2}. Other stress signaling pathways under investigation (p38 mapk and jnk signaling) were found unaffected. In this study however, SAR level was not measured or calculated and the uniformity of SAR was not determined.

Hirose et al (2006) tested the hypothesis that RF exposure could activate the p53-dependent signaling pathways in human A172 and IMR 90 cells. They found no significant differences in the expression levels of total P53 and phosphorylated p53 at serine 15 were observed between cells exposed to 215 MHz W-CDMA or CW signal (80, 250 or 800 mW kg^{-1} for 24-48 h) and the sham-exposed negative cells, as evaluated by bead-based multiplex assays. Moreover, no noticeable differences in expression of a number of p53-dependent genes mainly involved in apoptosis were observed between exposed and control cells by real-time RT-PCR and DNA chip analysis in contrast to positive controls (Doxorubicin or heat-shock).

Table II.3.10.: Protein-specific approaches

Assay endpoint	Exposure Conditions	Response	Comment	Reference
Electrophoresis of whole cell extract with [^{35}S] methionine protein labeling in HeLa and CHO cells.	27 and 2450 MHz, SAR: 25W kg^{-1}, for 2 h (HeLa cells); 27 MHz, SAR: 100 W kg^{-1}, for 2 h (CHO cells)	No detectable effect on 'stress protein' induction.	Only molecular weight was used to determine if the proteins examined were 'stress proteins'; no other evidence such as Western blotting was given.	Cleary et al 1997
Immunofluorescence staining of AMA (transformed human epithelial amnion) cells.	960 MHz (GDM), SAR: 2.1 mW kg^{-1}, for 20 min	Higher amounts of hsp70 were present in the cells exposed RF-field at 35 and 37^{o}C than in sham-exposed cells.	The induction of hsp70 by RF was not confirmed by other methods.	Kwee et al 2001
Western blotting of human malignant glioma (MO54) cells.	2450 MHz; SAR: ≤ 100 W kg^{-1} for up to 24 hr.	Increased expression of hsp70 was observed at 20 W kg^{-1} and higher SARs.	Annular culture plate was used for RF exposure. The difference in SAR distribution is relatively high even in the same ring.	Tian et al 2002
Flow cytometry analysis for detection of hsp70 in human blood mononuclear cells.	1.8 GHz (GSM, GSM-DTX, GSM-Talk); SAR: 2 W kg^{-1} (GSM, GSM-Talk) or 1.4 W kg^{-1} (GSM-DTX) intermittently (10 min on/20 min off) for 44 hr	RF exposure did not induce apoptosis, or affect mitochondrial function or hsp70 expression.	Detection of hsp70 was done only by flow cytometry.	Capri et al 2004b
Immunocytochemistry and Western blotting in human malignant glioma (MO54) cells.	1950 MHz; SAR: ≤ 10 W kg^{-1} for up to 2 hr.	No effect on hsp27 and hsp70 expression. However, phosphorylated hsp27 level decreased after RF exposure at 10W kg^{-1}.	A slight decrease in p-hsp27 (Ser^{78}) expression. Other phosphorylation sites at Ser^{15} and Ser^{82} were not examined.	Miyakoshi et al 2005
Flow cytometry analysis for detection of hsp70 and hsp27 in human leukocytes (lymphocytes, monocytes) from healthy volunteers.	900 MHz (CW and GSM), SAR ≤ 3.6 W kg^{-1} for up to 4 hr.	No statistically significant differences were detected in the number of cells expressing hsp70 and hsp27 after RF-field exposure.	Expression of hsp70 and hsp27 was observed only by flow cytometry analysis.	Lim et al 2005

Assay endpoint	Exposure Conditions	Response	Comment	Reference
Western blotting for hsp90 in human epidermal cancer cells (KB cells).	1.95 GHz, SAR: 3.6±0.2 W kg^{-1}, for 1~3 hr	An increase of jnk-1 activity and hsp70 and hsp27 expression with a reduction of P38 kinase activity and hsp90 expression.		Caraglia et al 2005
Flow cytometric measurement for hsp70 in Human Mono Mac6 or K562 cells.	1800MHz, (CW, GSM-nonDTX, GSM-DTX and GSM-Talk), SAR ≤ 2.0 W kg^{-1}, for 45 min	No significant effects on hsp70 expression were detected.	Expression of hsp70 was examined by only flow cytometric measurement.	Lantow et al 2006a
Flow cytometric measurement for hsp70 in human umbilical cord blood-derived monocytes and lymphocytes	1800 MHz (CW, GSM-DTX and GSM-Talk) SAR: 2 W kg^{-1}, for 30 or 45 min (continuous or intermittent exposure, 5 min on/5min off)	No effect on hsp70 expression level after exposure to GSM-DTX signal	Expression of hsp70 was examined only by flow cytometric.	Lantow et al 2006b
Western blotting in human malignant glioma (A172) cells.	2450 MHz; SAR: 5~200 W kg^{-1}, for 1 h~3 h	No significant change in hsp27 expression was observed at up to 20 W kg^{-1} or by comparable heating. hsp27 phosphorylation increased transiently at 100 and 200 W kg^{-1} of RF.	No significant induction of hsp70 and hsp27 was observed even at the highest SAR level.	Wang et al 2006
p53[pS15], total p53 using indirect immunofluorescence method, bead-based multiplex assay in human malignant glioma cells A172 cells and human IMR-90 fibroblasts from fetal lungs.	2.1425 GHz (CW and W-CDMA: A172 cells: SAR: 80, 250 and 800 mW kg^{-1} for 24 or 48 h; IMR-90 cells: 80 mW kg^{-1} for 28 h	No significant differences in the expression levels of total p53 and phosphorylated p53 at serine 15 (p53[pS15]) were observed between RF exposed and sham samples.	Phosphorylation site examined was only at Ser 15 of p53.	Hirose et al 2006
hsp27[pS82], total hsp27, indirect immunofluorescence method, bead-based multiplex assay in A172 cells and IMP-90 fibroblasts.	W-CDMA, SARs of 80 and 800 mW kg^{-1} for 2 h, W-CDMA radiation at SARs of 80, 250, and 800 mW kg^{-1}, and to CW radiation at 80 mW kg^{-1} for 24 or 48h	No significant differences in the expression levels of phosphorylated hsp27 at serine 82 (hsp27[pS82]) were observed.	Phosphorylation site examined was at only Ser^{82} of hsp27. No experiments at Ser^{15} and Ser^{78}.	Hirose et al 2007

Assay endpoint	Exposure Conditions	Response	Comment	Reference
Total ERK expression and phosphorylation, p38-MAPK and JNK phosphorylation, release of Hb-EGF, NADH oxidation using the western-blotting method, NADH oxidase enzyme activity in human Hela carcinoma cells and rat Rat-1 fibroblasts.	875 MHz (800 and 950 MHz also tested); 0.05 to 3.44 W m^{-2} for 5 to 30 min.	RF exposure increased ERK phosphorylation but not total ERK expression. P38-MAPK and JNK were not found activated after exposure.	The use of multiple exposure conditions, lack of statistical analysis, lack of evidence that the study was blind, and lack of state-of-the-art dosimetry weaken the study.	Friedman et al 2007
hsp70, hsc70, hsp2 using fluorescence level in human normal epidermal (NHEK) keratinocytes and dermal (NHDF) fibroblasts	GSM-1800, 1800 MHz, with a 217 Hz modulation, 48 h; SAR: 2 W kg^{-1},	The GSM-1800 signal does not act as a stress factor on human primary skin cells	No Western blot.	Sanchez et al 2007

Proteomics

Leszczynski and co-workers (Leszczynski et al 2002; Nylund and Leszczynski 2004, 2006) have conducted several *in vitro* studies on the effects of GSM 900 RF exposure. In their first study (Leszczynski et al 2002), human endothelial (EA.hy926) cells were exposed to RF at an SAR of 2.0 W kg^{-1} for one-hour and RF exposure changed the overall pattern of protein phosphorylation, upregulated the level of the hsp27 protein and induced its hyper-phosphorylation. This induction was revealed by a variety of independent protein analytical techniques including Western blotting and indirect immunofluorescence methods. The data also revealed that activation of p38 mitogen-activated kinase (MAPK) was partially responsible for the phosphorylation of hsp27. Nylund and Leszczynski (2004) reported that 38 proteins had statistically significant alteration in expression levels in the same cell line after exposure to GSM 900 at SAR of 2.4 W kg^{-1} for 1 h. Western blotting and indirect immunofluorescence methods were used to confirm that one isoform of vimentin was expressed in the cells after exposure. The authors also suggested that the cytoskeleton might be one of the mobile phone radiation-responding cytoplasmic structures. Nylund and Leszczynski (2006) compared *in vitro* response to GSM 900 RF in EA.hy926 with its variant EA.hy926v1 by examination of protein expression using 2-D gel electrophoresis. The results showed that protein expression profiles were altered in both cell lines after RF exposure: 38 and 45 differentially expressed proteins were found in EA.hy926 and EA.hy926v1, respectively. However, the affected proteins were altered differently in each of the cell lines. Several differentially expressed proteins in EA.hy926 cells were confirmed by other methods, but differentially expressed protein in EA.hy926v1 cells was not confirmed by Western blotting (Nylund and Leszczynski 2006). Based on the proteome analysis data, the authors concluded that the response might be genome- and proteome-dependent.

Zeng et al (2006) systematically explored the effects of 1800 MHz RF on protein expression in MCF-7 cells by 2-D gel electrophoresis, and found that a few proteins were differentially expressed under continuous or intermittent RF exposure at 3.5 W kg^{-1} for 24 h or less, implying that the observed effects might have occurred by chance. This study combined proteomics and transcriptomics data, and did not provide convincing evidence that RF exposure could produce distinct effects on gene and protein expression in the MCF-7 cells. The authors suggested that the MCF-7 cells may be insensitive to RF exposure.

Table II.3.11.: Proteomics

Assay endpoint	Exposure Conditions	Response	Comment	References
Protein expression in human endothelial (EA.hy926) cell line	GSM 900 MHz, SAR: 2.0 W kg^{-1} for 1 hour	RF exposure changed protein phosphorylation pattern, up-regulated the levels of hsp27 protein and induced its hyper-phosphorylation.	Confirmed by independent protein analytical techniques including Western blotting and indirect immunofluorescence method	Leszczynski et al 2002
Protein expression in human endothelial (EA.hy926) cell line	GSM 900 MHz, SAR: 2.4 W kg^{-1} for 1 hour	Up to 38 various proteins have statistically significantly altered their expression levels after RF exposure. Increased expression of vimentin in RF exposed cells.	10 replicates in 2-DE analysis were performed, Western blotting and indirect immunofluorescence method were conducted as confirmation.	Nylund and Leszczynski 2004
Protein expression in EA.hy926 and EA.hy926v1 cells	GSM 900 MHz, SAR: 2.8 W kg^{-1} for 1 hour	38 and 45 differentially expressed proteins were found in EA.hy926 and EA.hy926v1 respectively. The changes observed in the two cell lines were different	10 replicates in 2-DE analysis were performed, no differentially expressed protein was confirmed by Western blotting.	Nylund and Leszczynski 2006
Protein expression in MCF-7 cells	GSM 1800 MHz, SAR: 3.5 W kg^{-1}, continuous or intermittent exposure (5 min on/ 10 min off) for 1-24 hours	No effects. A small number of different proteins were differentially expressed under different exposure conditions, possibly by chance.	Triplicate gels were performed in each exposure condition.	Zeng et al 2006

Summary on gene and protein expression

The effects of RF exposure on the expression of cancer-related genes (proto-oncogenes and tumor suppressor gene) are considered to be very weak or absent. Some studies, however, reported proto-oncogene expression in *p53*-deficient cells and a transient effect on the increase in *egr-1* gene expression. Although negative reports predominate in this gene-specific approach in mammalian cells, the few positive effects cannot be ignored and further studies should be carried out before reaching a final conclusion.

High-throughput studies of gene expression in various cell types have yielded a variety of results, including a lack of effect, and the up-regulation and down-regulation of various genes. Many studies however are technically incomplete in that they lack sufficient experimental repetition and replication and further validation through the use of more precise quantitative measures of gene expression. In addition, the magnitude of the changes is small, and may be of limited functional significance. However, to date, insufficient research has been carried out to allow definitive conclusions to be drawn.

Many studies have examined the effect of RF exposure on stress proteins, especially hsps. However, the results of most of these studies are inconsistent, although mostly negative outcomes have been reported *in vitro*. Some experiments suggest that some of the positive findings might result from heating alone. Among the few signaling pathways that have been investigated, the ERK pathway was found altered but again the studies gave inconsistent data. Further studies should be conducted to evaluate the influence of RF exposure on major stress signaling pathways (MAPK, ERK, etc.). Protein-specific approaches may provide more information in studies of these pathways, which are driven mainly through phosphorylation cascades, than monitoring protein expression itself.

High-throughput studies of protein expression by one group have reported changes in protein expression and phosphorylation in two cell lines, whereas another group attributed the small changes observed in another cell type to chance. No clear patterns of response emerged. At present, the available data don't allow valid conclusions to be drawn.

II.3.3.3. Reactive oxygen species and oxidative stress

Ageing, exercise, UV and many other forms of stress are known to increase the production of reactive oxygen species (ROS). These are generally very small and highly reactive species and include $O_2^{\cdot-}$, free radicals and both inorganic and organic peroxides. The harmful cellular effects of ROS include (i) damage to DNA, (ii) oxidation of polyunsaturated fatty acids in lipids, and (iii) oxidation of amino acid residues in proteins. Therefore, cellular damage is increased by elevated ROS levels. In addition, oxidative stress has been implicated in the initiation and promotion of carcinogenesis. Only a few studies have examined the effects of RF fields on spontaneous or induced ROS production, mostly in cells of the immune system that generate ROS as part of their function. In addition some studies have monitored more general assessment of oxidative stress, including intracellular oxidant and antioxidant levels, antioxidant defense, and heat-shock protein levels, the latter function being as molecular chaperones to protect cells from various types of stresses. [Heat shock protein expression is further discussed in the previous chapter (II.3.3.2.).]

Zmyślony et al (2004) examined the effects of 930 MHz CW RF on ROS levels in rat lymphocytes. Some of the lymphocyte samples were treated with Fe^{2+} ions to induce oxidative processes. The results showed that acute (up to 15 min) exposure at around 1.5 W kg^{-1} did not affect ROS production. However, the addition of $FeCl_2$ to the lymphocyte suspensions significantly increased the magnitude of fluorescence, used to measure intracellular ROS levels, by ~ 15% in the exposed lymphocytes.

Hook et al (2004b) investigated the effects of FMCW-modulated 835 MHz and CDMA-modulated 847 MHz RF on the production of oxygen radicals, the enhancement of radicals produced by oxidative stress, the resulting oxidative damage and the induction of an oxidative stress response, in a mouse J774.16 macrophage cell line. Oxidative stress was induced prior to exposure using γ-interferon (IFN) and bacterial lipopolysaccharide (LPS), both of which activate cellular oxidases producing reactive nitrogen and oxygen species. No effects of RF exposure were seen on any of the endpoints, in unstimulated or in IFN/LPS stimulated macrophages.

Simko and colleagues (Lantow et al 2006a; Lantow et al 2006b) have examined the effect of 1800 MHz RF CW or various GSM modes (DTX and Talk) at up to 2 W kg^{-1} for 45 min on hsp70 and ROS production in human Mono Mac 6 cells (a monocyte leukemia cell line) and K562 cells (an erythroid leukemia cell line). No significant difference in free radical production was detected after RF exposure compared with their respective controls, and no additional effects on the production of superoxide radical anions was detected in cells after co-exposure to RF plus the phorbol ester PMA (phorbol-12-myristate-13-acetate) or RF plus LPS treatment (Lantow et al 2006a), both of which known to increase ROS production in monocytes and other cells of the immune system. In addition, no significant effects of RF exposure on hsp70 expression were found. The same group (Lantow et al 2006b) also used human umbilical cord blood-derived monocytes and lymphocytes to examine ROS release after continuous or intermittent (5 min on/5 min off) exposure to CW or the various GSM 1800 modes listed above at 2 W kg^{-1} for 30 or 45 min. No effects of RF exposure on ROS production in PMA-stimulated human monocytes or lymphocytes were seen once a correction had been made for the reduced production of ROS in the sham-exposed cells compared to incubator controls. In addition, no significant effects of RF exposure on hsp70 expression were found.

As part of the CEMFEC program, Scarfi et al investigated the induction of ROS in murine L929 fibrosarcoma cells exposed to a GSM 900 RF field, with or without co-exposure to 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX), a potent environmental carcinogen produced during the chlorination of drinking water (Zeni et al 2007). Treatment with MX was found to significantly increase ROS production, with a concomitant decrease in levels of the antioxidant glutathione; however, RF exposure, either alone or in combination with MX, did not induce formation of ROS under any of the experimental conditions investigated.

Overall, the data are consistent and suggest that RF exposure has no effect on ROS production in several different cell lines.

Table II.3.12.: Reactive oxygen species and oxidative stress

Assay endpoint	Exposure Conditions	Response	Comment	Reference
ROS (fluorescent probe assay) in rat lymphocytes. Positive control: $FeC\ell_2$	930 MHz, CW, SAR: 1.5 W kg^{-1} for 5 or 15 min.	No ROS induction by RF alone, but RF exposure enhanced ROS production induced by the addition of $FeC\ell_2$.		Zmyślony et al 2004
Cell viabiliy (typan blue exclusion), oxidant and antioxidant levels, oxidative damage and nitric oxide production in mouse J774.16 macrophage cells stimulated with IFN and LPS before exposure.	835.62 MHz, FMCW modulation and 847.74 MHz CDMA modulation; at 0.8 W kg^{-1} for 20-22 hr	No effects of RF on cell viability, intracellular oxidants, oxidative damage or antioxidant defenses in IFN or LPS stimulated cells.		Hook et al 2004b
Measurement of superoxide radical anions, ROS and hsp70 in human Mono Mac6 or K562 cells. Positive controls: PMA, LPS and heat.	1800 MHz, (CW, GSM-nonDTX, GSM-DTX and GSM-Talk) SAR $\leq$ 2.0 W kg^{-1} for 45 min	No RF effects on free radical production were detected, and no RF effects on superoxide radical anion production were detected after co-exposure with PMA or LPS.		Lantow et al 2006a
ROS measurement by flow cytometry in human umbilical cord blood-derived monocytes and lymphocytes. Positive control: PMA	1800 MHz (CW, GSM-DTX and GSM-Talk) continuous or intermittent, (5 min on/5min off) SAR: 2 W kg^{-1} for 30 or 45 min.	No effect on ROS production of RF alone or in combination with PMA in either cell type.	ROS production was significantly different in RF exposed human monocytes compared to sham-exposed controls, possibly due to lowered value in the sham-exposed cells.	Lantow et al 2006b
ROS production (fluorescent intensity of 2'7'-dichlorofluorescein) in mouse L929 fibrosarcoma cells. Positive control: MX	900 MHz, SAR: 0.3 or 1 W kg^{-1} for 10 or 30 min; with or without co-exposure to the carcinogen MX.	No effect on ROS production of RF alone or in combination with MX.		Zeni et al 2007

II.3.3.4. Cell proliferation, differentiation and cell cycle control

Cancer develops when cells acquire specific growth advantages through the stepwise accumulation of heritable changes in gene function. Basically, this process is directed by changes in two different classes of genes: tumor suppressor genes that inhibit cell growth and survival, and oncogenes that promote cell growth and survival. At the cellular level, the development of cancer is associated with sustained proliferation, dedifferentiation, angiogenesis, invasion and resistance to apoptosis. This chapter reviews *in vitro* studies related to these phenomena under RF exposure.

Studies published since 1993 on cell proliferation after RF exposure are reviewed which show a mixture of responses including either no effect, or increases, or decreases in these various end-points. The difficulty comes, as often, from the variety of exposure conditions, exposure setups and cell types. Adequate temperature control and dosimetry in particular are critical to the evaluation of any non-thermal effects. Taken together however, some common features arise from these studies.

A number of studies showed no effects on cellular proliferation as determined by cell count, DNA synthesis and cell cycle distribution in cells exposed to RF. In primary cells, proliferation is usually unaffected by RF exposure (Stagg et al 1997; Capri et al 2004a; Nikolova et al 2005; Sanchez et al 2006a; Sun et al 2006). Stagg et al (1997) showed no effects of 836.55 MHz, TDMA RF on rat primary glial cells exposed at very low level SARs ranging from 0.15 to 59 mW kg^{-1} for up to 24 hr. Sanchez et al (2006a) exposed human reconstructed epidermis using keratinocytes to GSM 900, 2 W kg^{-1} for 48 hr and found no increase in the number of Ki67 positive cells, a marker for cell proliferation. Sun et al (2006) found no effect of a 2 h GSM-1800 exposure at 1, 2, and 3 W kg^{-1} on the proliferation of human lens epithelial cells. Yao et al (2004) however found a decrease in cell proliferation correlated to a repressed $P27^{KIP1}$ protein expression in rabbit lens epithelial cells exposed to 2450 MHz, CW signal from 5 to 20 W m^{-2} for 8 hr, although the exposure conditions were not well described. By contrast, Pacini et al (2002) showed an increased proliferation in human normal fibroblasts, but the use of a genuine mobile phone for RF exposure does not enable reliable exposure measurement and dosimetry and this study is thus difficult to evaluate. Interestingly, studies using the cytokinesis-blocked micronucleus assay, mostly in human peripheral blood mononucleated cells, usually failed to detect cytotoxicity and changes in cell proliferation as determined by the mitotic index or the frequency of binucleates (see Chapter II.3.2.), even, in some cases, when increased micronucleated binucleated cells were detected (Maes et al 1993; Zotti-Martelli 2000; 2005; D'Ambrosio et al 2002; Tice et al 2002).

Proliferation and cell cycle distribution were unaffected in a number of cancer or transformed cell lines (Higashikubo et al 2001; Merola et al 2006; Gurisik et al 2006; Lantow et al 2006c; Takashima et al 2006; Chauhan et al 2007a). In fact, Takashima et al (2006) showed that the threshold for an effect on proliferation (decrease) was 200 W kg^{-1} CW when Chinese hamster ovary CHO-K1 cells and human glioma MO54cells were exposed for 2 hr at 2450 MHz.

Earlier however, Cao et al (1995) showed that 27-MHz RF (5 and 25 W kg^{-1}) altered the cell cycle of Chinese hamster ovary CHO cells in an SAR-dependent way. The same group (Cleary et al 1996) found that the effect of 2450-MHz RF (5 to 50 W kg^{-1}) was highly dependent on the concentration of the mitogen IL2 in CTLL2 mouse cytolytic T lymphocytes, and hypothesized that the effect was dependent on the presence of high affinity-IL2 receptors, suggesting that the effect is cell-type dependent. Donnellan et al (1997) found an increased proliferation of rat mast cells repeatedly exposed at 850 MHz (81 W m^{-2}, 3 times a day for 7 days) suggesting that cells lost their contact inhibition. Unfortunately, these studies have not been independently confirmed.

There have been several studies of ornithine decarboxylase (ODC) activity after RF exposure. ODC is an enzyme involved in cell growth and ODC overexpression has been consistently reported to lead to neoplastic cellular transformation (Kubota et al 1997; Dhalluin et al 1998; Tabib and Bachrach 1999), and may thus be involved in cancer cell invasiveness. An increased ODC enzyme activity was consistently reported in murine L929 fibroblasts after an 8-hour *in vitro* exposure to modulated 835-840 and 915 MHz RF at 2.5 W kg^{-1} (Litovitz et al 1993, 1997; Penafiel et al 1997). Results from two independent groups did not confirm such effect in the same cell type exposed at a similar SAR (Desta et al 2003; Höytö et al 2006, 2007a). Both groups also reported that a temperature increase resulting from either RF exposure or

conventional heating of about 1°C decreased ODC enzyme activity. When cells were isothermally exposed at higher SARs (up to 15 W kg^{-1}) however, different outcomes in ODC activity were found according to the type of exposure system and temperature control method used. No clear explanation could be given, which suggests that temperature control is critical in the interpretation of possible non-thermal effects of RF exposure, at least in ODC experiments. Höytö et al (2007b) also exposed L929 murine fibroblasts and other cell lines (rat C6 glioblastoma cells, human SH-SY5Y neuroblastoma cells) and rat primary astrocytes to 815 MHz, CW and GSM-modulated. They found essentially no effect in secondary cell lines but a consistent significant decrease in ODC activity in primary astrocytes. While increased ODC activity has been considered as an indication of potentially harmful health effects, the health relevance of decreased ODC activity is not known.

It is known that the malignancy of a cancer is directly related to the degree of de-differentiation of tumor cells, related to their rate of growth. Differentiation under *in vitro* RF exposure has been sparsely studied. Nikolova et al (2005) found an effect of intermittent GSM 1800 signal (1.5 W kg^{-1} for up to 48 hours) in mouse pluripotent embryonic stem cells; while neural-specific *Nurr-1* mRNA expression was decreased, no change in neural-specific proteins could be detected. Merola et al (2006) showed that exposure to GSM-900 MHz at 1 W kg^{-1} for up to 72 hours did not affect spontaneous or retinoic acid–induced differentiation of LAN-5 human neuroblastoma cells.

Finally, in yeast (*Saccharomyces cerevisiae*) cells, Gos et al (1997) investigated possible non-thermal effects on cell division rate in exponentially growing cells that were exposed to RF in the millimeter frequency range around 41.7 GHz at low power densities (5 and 500 mW m^{-2}). No significant differences were seen between exposed and unexposed cells for value of S-phase and G1-phase at two different power levels. Pakhomov et al (2002) investigated the effects on the density of yeast cells, achieved after a 6 h growth period in a solid nutrient medium (agarose gel) during EHPP (extremely high power pulses) or CW exposure. They reported that CW and EHPP exposures produced highly non-uniform but identical heating patterns in exposed samples. Cell density was strongly affected by irradiation, and the changes correlated well with the local temperature rise. However, the data revealed no statistically significant difference between CW and EHPP samples across the entire studied range of SAR levels (over six orders of magnitude). A trend ($p < 0.1$) for such a difference was observed in gel slices that were exposed at a time averaged SAR of 100 W kg^{-1} and higher, which corresponded to a peak SAR above 20 MW kg^{-1} for the EHPP condition.

In summary, many studies have been published that suggest there are no effects of RF exposure on cell proliferation and cell cycle control. A few early studies have been published that suggest that there are effects of RF exposure below 100 W kg^{-1}, but these should be confirmed using improved exposure equipment, temperature control and dosimetry. The very few studies on the effects of RF exposure on differentiation *in vitro* do not suggest any effect.

Table II.3.13.: Proliferation, differentiation and cell cycle control

Assay endpoint	Exposure Conditions	Response	Comment	References
ODC activity ($^{14}CO_2$ generation) in L929 murine fibroblasts	915 MHz; CW, 55, 60 & 65 Hz AM 915 MHz, switched between AM frequencies at different intervals; SAR: 2.5 W kg^{-1} for 2 - 24 hr	No effect of CW RF. Doubling of ODC activity at 8 hr of modulated-RF exposure applied for periods exceeding 10 s.	SARs averaged over exposure flask; variable ODC activities in controls	Litovitz et al 1993
Cell cycle distribution (flow cytometry) in Chinese hamster ovary (CHO) cells exposed in different phases of the cell cycle	27 MHz, CW, SAR: 5 or 25 W kg^{-1} for 2 hr	SAR-dependent alterations in cell cycle progression with a maximum effect 3 days after exposure at 25 W kg^{-1}.	Data showed considerable interexperimental variability. Cells exposed in phases G0/G1 and S phase were most sensitive.	Cao et al 1995

Assay endpoint	Exposure Conditions	Response	Comment	References
Interleukin-2 (IL2)-dependent cell proliferation (incorporation of [H^3]-thymidine) in CTLL-2 mouse cytoloytic T lymphocytes.	2450 MHz CW, SAR: 25, 50 W kg^{-1} or pulsed 2450 MHz at 5 W kg^{-1} for 2 hr.	RF-induced increase in proliferation at 5 and 25 W kg^{-1} and decrease in induced proliferation at 50 W kg^{-1}.	Effect highly dependent on IL2 concentration. Temperature controls revealed increased proliferation at 39°C and decreased proliferation at 40 or 41°C.	Cleary et al 1996
Cell proliferation (incorporation of [H^3]-thymidine, cell count) in rat RBL-2H3 mast cell line	835 MHz, at an estimated maximum of 81 W m^{-2} for 20 min, 3 times per day for 7 days; SAR not given.	Increased thymidine uptake and cell counts at day 6 and day 7.	Power density variable across exposure chamber; exposed cultures on average 0.8°C above controls.	Donnellan et al 1997
Cell proliferation (growth curve, doubling time, incorporation of [H^3]-thymidine) in rat, primary glial cells and C6 glioma cells	836.55 MHz, TDMA, average SAR: 0.15-59 mW kg^{-1} for 4 or 24 hr	No effects of RF on primary glial cell proliferation.	Small effect on thymidine uptake in C6 glioma cells at 5.9 mW kg^{-1} but no effect on cell growth.	Stagg et al 1997
ODC activity ($^{14}CO_2$ generation) in L929 murine fibroblasts	835 MHz, CW or amplitude modulated at 16, 60 Hz, 6-600 Hz; 835 MHz with TDMA, speech, AMPS or DAMPS modulation; SAR: 2.5 W kg^{-1} for 2 - 24 hr.	Transient increases in ODC activity following 835 MHz, amplitude-modulated at 16 – 65 Hz, TDMA or DAMPS modulation, after exposure for between 6 to 16 hr, depending on signal modulation, and returning to control values after 24 hr.	No effect of CW, speech modulation or AMPs modulation. Experimental data variable; multiple t-tests.	Penafiel et al 1997 ; Litovitz et al 1997
Cell proliferation (formazan test) in human transformed epithelial amnion AMA cells	960 MHz, GSM modulation; SAR: 0.021, 0.21 and 2.1 mW kg^{-1} for 20 - 40 min	RF exposure at 37°C decreased cell proliferation in time dependent manner	Brief description of experimental protocol; multiple t-tests.	Kwee and Rasmark 1998
Cell proliferation (formazan test) in human transformed epithelial amnion AMA cells	960 MHz, GSM modulation; SAR: 2.1 mW kg^{-1} for 30 min	RF exposure at 35°C and 39°C altered cell proliferation compared to controls.	Data presented only as differences between exposed or sham exposed and controls; multiple t-tests.	Velizarov et al 1999

Assay endpoint	Exposure Conditions	Response	Comment	References
Cell cycle progression (BrDU pulse-chase assay) in mouse fibroblasts C3H 10T1⁄2 and human U87MG glioblastoma cells	847.74 MHz CDMA, 835.62 MHz FDMA, SAR: 0.6 W kg^{-1} for 13 - 100 hr	No effects of either RF signal on progression through G_1, G_2 and S phase in either cell line.	Positive temperature effects only at 38, 39 and 40°C.	Higashikubo et al 2001
Cell proliferation (incorporation of [H^3]-thymidine) in human Detroit 550 skin fibroblasts	902.4 MHz, GSM at 1 W m^{-2} (estimated SAR: 0.6 W kg^{-1}) for 1 hr	Increase in thymidine uptake reported (no statistical analysis).	Exposed samples placed above mobile telephone; limited dosimetry and temperature control.	Pacini et al 2002
ODC activity ($^{14}CO_2$ generation) in L929 murine fibroblasts	TDMA 835 MHz; SAR: 1 to 15 W kg^{-1} for 8 hr	No difference as compared to controls at non-thermal SAR levels. Linear fall in ODC activity with RF or conventional heating above 1.0°C (SARs > 5 W kg^{-1}).	Attempted replication of Penafiel et al (1997), above.	Desta et al 2003
PHA- or αCD3-induced cell proliferation and cell cycle analysis in human peripheral blood mononucleated cells	900 MHz, GSM or CW, SAR: 70 - 76 mW kg^{-1} for 1 hr per day for 2 or 3 days	900 MHz GSM exposure over 3 days significantly decreased (by 9%) PHA- but not αCD3-induced cell proliferation.	No effects of 900 MHz GSM on cell cycle. No effects of CW 900 MHz on any parameter investigated.	Capri et al 2004a
Cell proliferation (MTT formazan assay), and cell cycle distribution (flow cytometry) in rabbit lens epithelial cells.	2450 MHz, CW at 1 - 20 W m^{-2} for 8 hr	RF decreased cell viability and proliferation above 5 W m^{-2}, with G0/G1 arrest and a decreased cell number in S-phase.	Inadequate description of exposure conditions	Yao et al 2004
Cell cycle distribution (flow cytometry) and cell growth (MTT formazan assay), in human CCRF-CEM T-lymphoblastoid leukemia cells	900 MHz CW, SAR < 1 mW kg^{-1} for 2 - 48 hr	Drop in cell growth at 24 and 48 hr compared to controls. Cell cycle arrest in S-phase at 48 hr; decreased cell count in G0/G1.	Single FACS analysis. Very low SAR	Marinelli et al 2004a,b
Cell proliferation (BrdU incorporation) in pluripotent mouse embryonic stem (ES) cells	1710 MHz, GSM modulation, SAR: 1.5 W kg^{-1}, intermittent (5 min on/off 30 min), for 6 or 48 hr.	No effects on cell proliferation	Cells derived from nestin positive neural crest cells	Nikolova et al 2005

Assay endpoint	Exposure Conditions	Response	Comment	References
Cell proliferation (Ki67 positive nuclei) in reconstructed epidermis using human primary keratinocytes	900 MHz, GSM modulation; SAR: 2 W kg^{-1} for 48 hr	No effect on proliferation		Sanchez et al 2006a
Cell proliferation (formazan test) and retinoic acid induced differentiation in human LAN-5 neuroblastoma cells	900 MHz, GSM modulation, SAR: 1 W kg^{-1} for 24, 48 and 72 hr	No effects on spontaneous or serum-induced cell proliferation and differentiation.	Student's t test for n=3	Merola et al 2006
Cell viability (trypan blue exclusion), cell cycle distribution (flow cytometry) in human neuroblastoma SK-N-SH and monocytoid U937 cells	900 MHz, GSM modulation, SAR: 0.2 W kg^{-1} for 2 hr	No effects on cell viability or on cell cycle distribution		Gurisik et al 2006
Cell cycle distribution (flow cytometry), DNA synthesis (BrdU incorporation) in human macrophagic Mono Mac 6 cells	1800 MHz pulsed-modulated (GSM-DTX) ± Gliotoxin or PHA, SAR: 2 W kg^{-1} for 12 hr	No effects on cell cycle distribution or cell proliferation	Effects seen in PMA positive controls. Student's t test for n=3	Lantow et al 2006c
Cell Growth, cell survival (colony-forming efficiency), cell cycle distribution in Chinese hamster ovary cells CHO-K1 and human glioma cells MO54	2450 MHz CW; SAR: 0.05 to 200 W kg^{-1} for 2 hr; 2450 MHz Intermittent at peak SARs of 300 to 1500 W kg^{-1} (mean SARs of 50 or 100 W kg^{-1}) for 2 hr	No effects of CW or intermittent RF at a mean SAR of up to 100 W kg^{-1}. CW RF at 200 W kg^{-1} or incubation at 42°C decreased cell growth and survival; no effect on cell cycle distribution.	The effect on cell growth and survival is thermal and depends on the mean SAR. Most data seem to come from a single experiment. No % data for sham control for cell cycle.	Takashima et al 2006
Cell proliferation (BrdU incorporation) in human lens epithelial cells	1800 MHz, GSM modulated, SAR: 1 - 3 W kg^{-1} for 2 hr	No effects of RF exposure on cell proliferation up to 4 days after exposure		Sun et al 2006
ODC activity ($^{14}CO_2$ generation) in L929 murine fibroblasts (ATCC)	900 MHz, CW or GSM-modulated (217 Hz), SAR: 0.2 W kg^{-1} and 0.4 W kg^{-1} for up to 24 hr	No effects of CW or GSM RF exposure on ODC activity after correcting for temperature differences.	A 1.1°C temperature increase over 2 hr led to a 43% decreased ODC activity in temperature controls.	Höytö et al 2006

Assay endpoint	Exposure Conditions	Response	Comment	References
Cell viability, cell cycle distribution in human lymphoblastoid TK6, lymphoblastic HL60 and myeloid Mono-Mac-6 cells	1900 MHz pulse-modulated, intermittent exposure (5 min on/10 min off); SAR: 1 and 10 W kg^{-1} for 6 hr,	No effects of RF exposure on cell viability or cell cycle progression	Heat shock (43°C) controls showed decreased viability and G2/M block	Chauhan et al 2007a
ODC activity ($^{14}CO_2$ generation) in L929 murine fibroblasts (ATCC).	835 and 872 MHz, CW or DAMPS-modulated (50 Hz); SAR: 2.5 or 6 W kg^{-1} for up to 24 hr. Two exposure systems - Crawford cell (CC – 835 MHz) and waveguide (WG – 872 MHz) were used with different cooling methods.	No effects of CW or DAMPS on ODC activity at 2.5 W kg^{-1}. Significant decrease of ODC activity after CC exposure for 2 hr at 6 W kg^{-1} but not after 8 or 24 hr. Significant increase in activity after WG exposure for 8 hr at 6 W kg^{-1}.	Unable to replicate the study of Penafiel et al (1997). However, there were discrepancies in the present study outcome at 6 W kg^{-1} when using two different exposure and temperature control systems	Höytö et al 2007a
ODC activity ($^{14}CO_2$ generation) in rat primary astrocytes and in L929 murine fibroblasts (ECACC), rat C6 glioblastoma cells and human SH-SY5Y neuroblastoma cells.	835 MHz, CW or GSM-modulated, SAR: 1.5, 2.5 and 6 W kg^{-1} for up to 24 hr.	Significant decrease in ODC activity in rat primary astrocytes at 1.5 and 6 W kg^{-1} although after different exposure times at different SARs. No overall effects on ODC activity in rat gioblastoma and human neuroblastome cell lines.	No effect of CW or GSM RF at 2.5 W kg^{-1} on ODC activity in L929 murine fibroblasts used by Penafiel et al (1997), but significant reductions at 1.5 and 6.9 W kg^{-1}. However, these results in L929 cells were affected by temperature differences between the two exposure chambers	Höytö et al 2007b

II.3.3.5. Apoptosis

Apoptosis is a "suicide" process of cells in multicellular organisms. It is one of the main types of programmed cell death (PCD), and involves an orchestrated series of biochemical events leading to a characteristic cell morphology and death. The apoptotic process is executed in such a way as to safely dispose of cellular debris. Apoptosis is initiated for various reasons, such as when a cell is no longer needed within the body (i.e. in embryonic development) or when it becomes a threat to the health of the organism (i.e. with high level of DNA damage). Severe pathological consequences, such as autoimmune disorders, neurodegenerative diseases, and cancer can arise from abnormal rates of apoptosis.

There is no single parameter that defines programmed cell death, and therefore a combination of techniques is recommended for the reliable detection of apoptosis. Using timed inductions and comparing relationships between cell populations expressing multiple markers, it is possible to estimate within a given model the relative order in which the different components of an apoptotic process become evident;

these range from the externalization of the phosphatidyl-serines at an early stage to the ladder-type DNA fragmentation and the loss of membrane integrity at late stages of PCD.

PCD is activated by different apoptotic signaling pathways that can be investigated through the expression of apoptosis-related genes and proteins. The "extrinsic" pathway is activated by the binding of death-activator proteins to the cell surface. The "intrinsic" pathway is launched by signals inside the cell, such as damage caused by radiation or toxins, the withdrawal of critical survival factors (growth factors, hormones), or disturbances in the cell cycle. Both pathways converge inside the cell, turning on a central effector family of proteins: caspases. Recently, a caspase-independent pathway has also been described driven through the Apoptosis-inducing Factor (AIF).

A number of studies have been published on the effects of RF exposure, from 800 to 2450 MHz, on cellular apoptosis *in vitro*.

Using normal cells, ranging from yeast to mouse embryonic stem cells, primary rat neurons, and primary human fibroblasts and blood cells, most studies found no evidence that low-level RF exposure could induce apoptosis (Markkanen et al 2004; Capri et al 2004a, 2004b; Nikolova et al 2005; Joubert et al 2006; Sanchez et al 2007). However, Joubert et al (2008) recently reported an increase in AIF-dependent apoptosis in rat primary neurons 24 h after a 24-h exposure to CW-900 MHz RF at 2 W kg^{-1}, while GSM-900 (1 W kg^{-1}, up to 48 h) was ineffective to induce apoptosis in the same cells (Joubert et al 2007).

Contradictory data have been published on RF-induced apoptosis in tumor and mutant cells. Many tumor cell types have been used, showing no apoptotic response after exposure to RF (Peinnequin et al 2000; Hook et al 2004a; Merola et al 2006; Gurisik et al 2006; Lantow et al 2006c; Chauhan et al 2007a; Joubert et al 2007). In these studies, exposure to RF lasted from 1 to 72 hours and SAR ranged from 0.07 to 4 W kg^{-1}. Chauhan et al (2007a) for instance, exposed three human cell lines (lymphoblastoid TK6, lymphoblastic HL60 and myeloid Mono-Mac-6 cells) to intermittent (5 min on/10 min off) PW 1900 MHz at SAR of 1 and 10 W kg^{-1} for 6 h. They observed no pro-apoptotic effect of RF exposure immediately and 18 h after exposure in either cell line. Hirose et al (2006) exposed a transformed (A172) and a non-transformed (IMR90) human cell lines to 2142.5 MHz RF (0.08 to 0.8 W kg^{-1}, up to 48 h) and observed no apoptotic response.

By contrast, some authors reported an effect of RF exposure on tumor cell apoptosis (Marinelli et al 2004a,b; Caraglia et al 2005; Buttiglione et al 2007). In these investigations, exposure to RF lasted from 2 to 48 hours and SARs ranged from 0.001 to ~ 4 W kg^{-1}. Obviously, SAR levels and exposure duration are unlikely to account for the discrepancy. In human SH-5Y-5H neuroblastoma cells, contradictory data have published despite experiments using the same exposure set-up, although slightly different exposure conditions. Joubert et al (2006) showed no apoptosis induction after GSM-900 exposure (0.25 W kg^{-1} for 24 h, where the maximum temperature increase was reported to be 0.3°C) while Buttiglione et al (2007) showed a small 2.3% statistically significant increase in apoptosis 30 h after exposure to GSM-900 (1 W kg^{-1} for 24 h, where the maximum temperature increase was reported to be 1°C); whether the difference is due a temperature increase in the culture medium is unclear at this stage.

Based on these data, the use of low-level RF exposure as a therapeutic tool for inducing apoptosis in tumor cells such as neuroblastoma cells has been suggested. However, the amplitude of the effect was highly variable (i.e. < 2% to 40% apoptotic cell population) and different signaling pathways were reported to be activated, although most indicated an inactivation of cell survival pathways such as the *ras-erk* and *Bcl2* survival pathways.

Interactions of RF exposure with pro-apoptotic agents have been considered (Peinnequin et al 2000; Markkanen et al 2004; Capri et al 2004a,b). Markkanen et al (2004) suggested that differences in genomic background might affect the response to RF. These authors showed that for yeasts mutant for the cell-cycle dependent cyclin 48, but not their normal wild-type counterparts, apoptosis was increased by exposure to UV and RF (872 or 900 MHz GSM, at 3.0 or 0.4 W kg^{-1} respectively for 1 h) in combination with incubation at 37°C. However, whilst incubation at this temperature induced apoptosis in the mutant yeast strain, it did not do so in the wild type, hence an effect on apoptosis in this strain could not be tested. RF potentiation of induced apoptosis has also been shown in mammalian cells at 900 MHz GSM

(Capri et al 2004a) and 2450 MHz CW (Peinnequin et al 2000), but not at 1800 MHz GSM (Capri et al 2004b). In general however, the RF-induced potentiation of apoptosis was of modest amplitude (≤ 3%).

Among genomic studies, some found changes in apoptosis-related genes (Lee et al 2005; Nikolova et al 2005; Zhao TY et al 2007). Lee et al (2005) for example observed in HL-60 cells altered expression of 221 and 759 genes, 2 and 6 h, respectively, after exposure to 2450 MHz RF (10 W kg^{-1}). Apoptosis-related gene expression was found to be up-regulated, while down-regulation was observed for cell-cycle gene expression. It is however noteworthy that although pro-apoptotic gene expression was found to be up-regulated in mouse embryonic stem cells by Nikolova et al (2005), apoptosis induction was not observed at the cellular level. Hirose et al (2006) found no effect of RF exposure on the expression of about 20 *p53*-dependent genes involved in apoptosis.

In summary, results on RF effects on cellular apoptosis do not suggest any deleterious consequences. There is a need for testing other primary cell types and RF exposure schedules to confirm the lack of pro-apoptotic effects of low-level RF exposure in non-tumoral cells as suggested by all but one of the published studies. More investigations on the pro-apoptotic effect of RF in tumoral cells are necessary with regards to possible therapeutic applications. Finally, more investigation on the existence of interactions between low-level RF and physical or chemical agents may be useful for health risk assessment.

Table II.3.14.: Apoptosis

Assay endpoint	Exposure Conditions	Response	Comment	References
Cell proliferation (alarmaBlue assay) using apoptosis inducers: Fas, butyrate, or ceramide for 16 hr after RF exposure of human Jurkat T-lymphocytes.	2450 MHz, CW, at 50 W m^{-2}, SAR evaluated calorimetrically at 4 W kg^{-1}, for 48 h	RF pre-exposure significantly decreased (+ 0.6%) Fas-induced but not butyrate and ceramide-induced cell proliferation	Not a test of apoptosis per se. Questionable use of Student t test for 3 runs (100 points/run)	Peinnequin et al 2000
Apoptosis (Annexin V affinity) measured 12 hr after UV-B ± RF exposure and elevated temperature (+37°C) in yeast *S. Cerevisiae* Cdc-48 wild-type or Cdc-48 mutant. Cdc-48 mutant yeasts undergo apoptosis at +37°C in contrast to the Cdc-48 wild-type.	872 MHz GSM or CW SAR: 3.0 W kg^{-1}; 900 MHz GSM or CW at ca. 0.4 W kg^{-1} ; for 1 h. UVB exposure at 250 J m^{-2}	No effect of GSM or CW RF exposure on the apoptosis rate in either yeast strain. Significant increase in UV-induced apoptosis in mutant yeasts after GSM exposure (about 2.1 fold at 872 MHz and 3 W kg^{-1}	Small numbers of samples (2-4)	Markkanen et al 2004
Apoptosis assay: annexin V affinity in human T lymphoblastic leukemia Molt-4 cells	847.74 MHz CDMA, SAR: 3.2 W kg^{-1}; 835,62 MHz FDMA, SAR: 3.2 W kg^{-1}; 813.56 MHz iDEN®, SAR: 2.4 and 24 mW kg^{-1} ; 836.55 MHz TDMA, SAR: 2.6 and 26 mW kg^{-1}; for up to 24 h	No effect of exposure to any RF signal on apoptosis		Hook et al 2004a

Assay endpoint	Exposure Conditions	Response	Comment	References
Apoptosis assay: flow cytometry - sub-G1 peak of the cell cycle - and DNA fragmentation on gel electrophoresis; pro- and anti-apoptotic protein expression in human CCRF-CEM T-lymphoblastoid leukemia cells	900 MHz CW, SAR < 1 mW kg^{-1} for up to 48 hr.	Time-dependent increase in apoptosis: 15% at 2 hr to 2% at 48 hr. Early pro-apoptotic (bax, p53, p21) proteins over-expressed.	Single FACs analysis and single blots (no quantification) reported.	Marinelli et al 2004a,b
Spontaneous and induced apoptosis - assay: Annexin V affinity and mitochondrial membrane potential - in human peripheral blood mononucleated cells from 8 to 25 healthy donors per condition.	900 MHz, GSM or CW, SAR: 70- 76 mW kg^{-1}, at 1 h per day for 2 or 3 days.	No effects of GSM-900 MHz or CW signal on spontaneous apoptosis and mitochondrial membrane potential. However, 3% increase of dRib-induced Annexin V positive cells after GSM exposure for 3 days.	Annexin V positivity usually taken to be an early marker of apoptosis, but no concomitant increase in late apoptotic cells, or any variation in mitochondrial membrane potential.	Capri et al 2004a
Apoptosis - assay: Annexin V affinity and mitochondrial transmembrane potential in human peripheral blood mononucleated cells from young and elderly healthy donors.	1800 MHz, GSM modulation: GSM-Basic at 2 W kg^{-1}; GSM Talk at 2.0 W kg^{-1} and GSM-DTX at 1.4 W kg^{-1}, intermittent exposure (5 min on/30 min off) for up to 44 hr.	No effect on apoptosis in PBMC of both young (27 ± 5 years) and elderly (88 ± 1 year) donors		Capri et al 2004b
Apoptosis assays: Internucleosomal DNA fragmentation (ladder) and Annexin V affinity plus PI staining in human oropharyngeal epidermoid carcinoma KB cells	1950 MHz, GSM modulation, SAR: 3.6 W kg^{-1} for up to 3 h	Time-dependent significant increase in cell apoptosis: about 20, 32 and 45% after 1, 2 and 3 hours of RF exposure, respectively as compared to 8% in sham-exposed cells.	Means and SEM were not given, but only values from a single experiment.	Caraglia et al 2005
Serial analysis of gene expression (SAGE) method (mRNA) in human HL-60 cells.	2450 MHz, SAR: 10 W kg^{-1}, for 2 or 6 hr	Some apoptosis-related genes were up-regulated and cell cycle genes down-regulated immediately after RF exposure		Lee et al 2005

Assay endpoint	Exposure Conditions	Response	Comment	References
Apoptosis assays: sub-G1 peak of the cell cycle, apoptosis-related gene expression (QRT-PCR) in mouse pluripotent embryonic stem cells.	1710 MHz, GSM, SAR: 1.5 W kg^{-1} intermittent (5 min on/30 min off) for 6 and 48 hr.	No effects on apoptosis. Up-regulation of apoptosis related bax and *gadd45* mRNA levels during the ESC differentiation process.	No effects on nuclear apoptosis or cell proliferation suggesting compensation at the translational or post-translational level.	Nikolova et al 2005
Apoptosis (assay: Annexin V affinity plus PI staining), expression of *p53*-dependent genes in human malignant glioma cells A172 cells and IMR-90 fetal lung fibroblasts.	2142.5 MHz; CW and W-CDMA; SAR ≤ 800 mW kg^{-1} for 24 or 48 h.	No effect on apoptosis or on the expression of *p53*-dependent genes involved in apoptosis.		Hirose et al 2006
Apoptosis (assays: TUNEL, Caspase 3 activation, DAPI staining) in human SH-SY5Y neuroblastoma cells.	900 MHz, CW and GSM at 0.25 W kg^{-1} (GSM), or 2 W kg^{-1} (CW) for 24 h	No effects of 900 MHz CW or GSM-modulated on apoptosis.	$2^{o}C$ rise after 2 h at 2 W kg^{-1} 900 MHz CW necessitated the use of a $39^{o}C$ temperature control.	Joubert et al 2006
Spontaneous and camptothecin-induced apoptosis (assays: Caspase 3 activation, PARP cleavage) in human LAN-5 neuroblastoma cells.	900 MHz, GSM modulation, SAR: 1 W kg^{-1} for up to 72 h	No effects on spontaneous and/or induced cell apoptosis, proliferation, and differentiation	Statistics: validity of the use of the Student t test with n=3.	Merola et al 2006
Apoptosis (assay: YOPRO and/or PI exclusion) in human neuroblastoma SK-N-SH and monocytoid U937 cells.	900 MHz, GSM modulation, SAR: 0.2 W kg^{-1} for 2 h	No effects on cell viability and apoptosis when evaluated 24 hours post-exposure.	No positive control	Gurisik et al 2006
Spontaneour and induced apoptosis (assay: Annexin V affinity and 7-AAD staining) in human macrophagic Mono Mac 6 cells.	1800 MHz pulsed-modulated (GSM-DTX) SAR: 2 W kg^{-1} for 12 h	No effects on spontaneous or chemically induced cell apoptosis evaluated immediately after exposure or up to 72 hr after exposure.		Lantow et al 2006c
Apoptosis (neutral comet assay) in human lymphoblastoid TK6, lymphoblastic HL60 and myeloid Mono-Mac-6 cells.	1900 MHz pulsed-modulated SAR: 1 and 10 W kg^{-1} intermittant (5 min on/10 min off) for 6 h	No effects on cell viability and apoptosis when evaluated immediately after exposure and 18 hr post-exposure.		Chauhan et al 2007a
Apoptosis (assays: TUNEL, caspase-3 activation, DAPI staining) in primary cultured neurons from rat cortices.	900 MHz, GSM modulation; SAR: 0.25 W kg^{-1} for 24 h.	No effects on apoptosis when evaluated immediately after exposure and 24 hr post-exposure.		Joubert et al 2007

Assay endpoint	Exposure Conditions	Response	Comment	References
Apoptosis (assay: Annexin V affinity and PI staining) in human primary skin fibroblasts and keratinocytes.	1800 MHz, GSM modulation SAR: 2 W kg^{-1} for 48 h.	No effects of RF on apoptosis.		Sanchez et al 2007
Apoptosis (assay: cell cycle sub-G1 population, apoptosis-related gene expression: *Egr-1*, *p53*, *Bcl-2*, *survivin*, etc) in human SH-SY5Y neuroblastoma cells	900 MHz, GSM modulated SAR: 1 W kg^{-1}, for up to 24 h.	Significant 2.3% increase in apoptotic cell population and G2/M cell cycle arrest; no effect on *p53* expression but significant changes in *Egr-1*, *Bcl-2* and *survivin* expression.	No positive control	Buttiglione et al 2007
Gene expression (array analysis for apoptosis-related gene expression; real-time RT-PCR for selected genes) in mouse primary neurons and astrocytes.	GSM-1900 phone exposure for 2 h in 'on' mode (exposed) or 'stand-by' mode (sham); no dosimetry, no SAR determination.	RF exposure up-regulation of *caspase-2*, *caspase-6* and *Asc* gene expression in neurons and astrocytes; upregulation of *Bax* gene in astrocytes.	Up-regulation of *caspase-2*, *caspase-6* and *Asc* gene expression also seen in sham-exposed neurons compared to non-exposed controls. No dosimetry	Zhao TY et al 2007
Apoptosis (assays: TUNEL, caspase-3 activation, DAPI staining; apoptosis inducing factor (AIF) expression) in primary cultured neurons from rat cortices.	900 MHz, CW SAR: 2 W kg^{-1} for 24 h	Apoptosis induced 24 hr after RF exposure; AIF-positive but not caspase-3 positive cells significantly increased immediately and 24 hr after exposure.	Exposure induced a $2^{o}C$ rise in culture medium necessitating the use of control experiments carried out at $39^{o}C$. Authors acknowledge possibility of localized thermal effects.	Joubert et al 2008

II.3.3.6. Summary on non-genotoxic effects

With regard to signaling, the evidence from studies using measurement of calcium ion concentration, does not support the earlier reports suggesting that low-level amplitude modulated RF may affect calcium ion physiology. There is insufficient research regarding RF effects on nitric oxide signaling, intercellular gap junction properties and receptor clustering behavior to be conclusive.

Recent studies suggest that the RF exposure has no or very little effect on the expression of cancer-related genes (proto-oncogenes and tumor suppressor genes). However, the results of studies of RF exposure on stress protein expression, particularly on hsps, have so far been inconsistent, although mostly negative outcomes have been reported *in vitro*. Nevertheless, further studies should be conducted to evaluate the influence of RF exposure on major stress signaling pathways.

With regard to the outcome of studies using powerful, high-throughput screening techniques, several authors have suggested that low intensity (less than about 2.0 W kg^{-1}) RF exposure, especially at the mobile phone utilization frequencies (800-2000 MHz), can change gene and/or protein expression in some types of cells. However, the magnitude of these changes is usually small and of doubtful functional significance. In addition, other studies have reported a lack of effects. Because of the inconsistencies and methodological limitations of these studies, final conclusions regarding possible RF effects on the modulation of gene and/or protein expression are not possible at present.

Many studies have been published that suggest there are no effects of RF exposure on ROS production, cell proliferation, cell cycle control or on cellular apoptosis.

Ensuring adequate temperature control has proved difficult in many of these studies and heating may account for some of the positive effects reported.

II.3.4. Cell transformation

The neoplastic cell transformation assay is an integrative assay which is used to test carcinogenic and co-carcinogenic effects of chemical and physical agents. Its main advantage is that it reveals the carcinogenic potential of both genotoxic and non-genotoxic compounds. Several research groups have used this assay to determine whether RF exposure acts as an inducer, a promoter, or a co-carcinogen; most have used the chromosomally highly abnormal mouse fibroblast C3H/10T½ cell line.

In a series of experiments, Balcer-Kubiczek & Harrison (1985, 1989, 1991) exposed C3H10T½ cells to 2450 MHz RF (24 h), alone or in combination with known tumor initiators (X-rays or benzo(a)pyrene (B(a)P)), or the chemical promoter phorbol-12-myristate-13-acetate (PMA). No neoplastic transformation occurred with RF treatment alone at an SAR of up to 4.4 W kg^{-1} but Balcer-Kubiczek and Harrison (1991) reported that RF interacted with the promoter PMA in an SAR-dependent manner by increasing the transformation efficiency. However, unusually for *in vitro* RF studies, the authors exposed the cells in culture flasks situated in a waterbath situated in the far field of an anechoic chamber - dosimetry and temperature control may well be questionable. The data regarding effects on plating efficiency and the effect of RF exposure on neoplastic transformation induced by X-rays in presence of PMA were different in different experiments

Cain et al (1997) used the model of UV-TDT10e mutant cells in co-culture with parental C3H/10T½ murine fibroblasts to determine whether intermittent RF exposure (TDMA, 836.55 MHz) could influence the PMA dose-dependent promotion of focus formation. Cells were intermittently exposed (20 min on/20 min off) at SARs of 0.15, 1.5, and 15 mW kg^{-1}, 24 h per day for 28 days. No influence of RF exposure at any SAR level was seen on PMA-induced focus formation.

Roti Roti et al (2001) investigated the neoplastic transformation potential of mobile phone signals (CDMA, 847.74 MHz; FDMA, 835.62 MHz) at an SAR of 0.6 W kg^{-1} in mouse C3H10T½ cells. Exposure to RF lasted 7 days and combination treatments included X-rays and PMA. RF exposure did not affect neoplastic transformation whatever treatment combination.

Wang et al (2005) exposed C3H10T½ cells to 2450 MHz CW RF at SAR levels of 5 to 200 W kg^{-1} for 2 hours, sufficient to raise culture medium temperatures to ~ 40 and ~ 44°C at 100 and 200 W kg^{-1}, respectively. Cells were exposed to RF, either alone or in combination with 3-methylcholanthrene (MCA), PMA or MCA+PMA. RF alone and in combination with PMA did not affect the background neoplastic transformation. No significant differences were observed in the malignant transformation frequency in other combined treatments at SARs of up to 50 W kg^{-1}. However, RF at 100 and 200 W kg^{-1} increased the transformation frequency induced by MCA or MCA plus PMA. The authors reported that the transformation assay was negative when cells were exposed at corresponding temperatures (up to 44°C), although the heating profiles may have differed.

Hirose et al (2008) used the mouse BALB/3T3 cell transformation model to evaluate the effect of a continuous 6-week RF exposure in an anechoic chamber to 2140 MHz (W-CDMA) at 80 and 800 mW kg^{-1} on spontaneous and MCA±PMA-driven neoplastic transformation. No significant difference in neoplastic transformation was observed between groups.

All studies detailed above clearly show that RF exposure at SARs of up to 200 W kg^{-1} did not induce cell transformation. RF exposure did not promote the neoplastic transformation potential of either physical (X-rays) or chemical (B(a)P, MCA) inducers at SARs below 100 W kg^{-1}. In one study, a promoter effect of RF was found with MCA alone and combined with PMA, but at SARs sufficient to significantly increase culture medium temperature. An interaction of RF with the promoter PMA was also reported in another study at lower SAR levels, but discrepancies within the same group were reported for RF

interactions with a combination of physical or chemical initiators and PMA. Such effects were not found in other studies from four different laboratories using longer exposure durations.

Overall, the data consistently indicate no effect on neoplastic transformation rate of RF exposure at non-thermal levels, either alone or in combination with physical or chemical inducers.

Table II.3.15.: Cell transformation

Assay endpoint	Exposure Conditions	Response	Comment	References
Transformation (RF combined with B(a)P or X-rays ± PMA treatment) in mouse C3H10T½ cells	2450 MHz, 120 Hz pulse modulation, SAR: 4.4 W kg^{-1} for 24 hr.	Significant increase in transformation frequency in cells exposed to RF and X-rays followed by PMA	Questionable dosimetry and temperature control. RF significantly reduced cell plating efficiency by about 2-fold but had no effect on transformation.	Balcer-Kubiczek & Harrison 1985
Transformation (RF and/or X-rays ± PMA treatment) in mouse C3H10T½ cells	2450 MHz, 120 Hz pulse modulation SAR: 4.4 W kg^{-1} for 24 hr.	Significantly increased transformation frequency in cells exposed to RF and PMA. No effect in cells exposed to RF and X-rays followed by PMA.	Questionable dosimetry and temperature control. Different effects on transformation and plating efficiency (no effect) compared to previous paper.	Balcer-Kubiczek and Harrison 1989
Transformation (i. RF ± PMA; ii. RF preceded or followed by X-rays ± PMA) in mouse C3H10T½ cells.	2450 MHz, 120 Hz pulse modulation SAR: 0.1, 1, or 4.4 W kg^{-1}; ii. 4.4 W kg^{-1}; for 24 hr.	In the presence of PMA, RF increased neoplastic transformation in an SAR-dependent way. RF exposure slightly enhances effect of X-rays and PMA.	Questionable dosimetry and temperature control. No effect on plating efficiency.	Balcer-Kubiczek and Harrison 1991
PMA-induced focus formation in mutant UV-TDT10e cells in co-culture with parental mouse C3H10T½ cells.	836.55 MHz TDMA intermittently (20 min on/ 20 min off) SAR: 0.15, 1.5 or mW kg^{-1} for 24 hr per day for 28 days.	No significant effect of RF exposure up to 15 mW kg^{-1} on PMA–driven transformation	Variability in the transformation assay in response to PMA	Cain et al 1997
Transformation (i. RF alone; ii. X-rays followed by RF; iii. RF + PMA) in mouse C3H10T½ cells.	847.74 MHz CDMA, or 835.62 MHz FDMA SAR: 0.6 W kg^{-1} for 7 days.	No effect of RF exposure on neoplastic transformation rate with or without PMA, nor any effect on X-ray-induced transformation.		Roti Roti et al 2001
Transformation (i. RF alone; ii. MCA + RF; iii. RF + PMA; iv. MCA+ RF + PMA) in mouse C3H10T½ cells	2450 MHz, CW SAR ≤ 200 W kg^{-1} for 2 h.	No effect of RF exposure alone and in presence of PMA on transformation. Increased level of MCA ± PMA-induced transformed foci by RF exposure at 100 and 200 W kg^{-1}.	Significant RF heating. However, a lack of effect of heat treatment up to 44°C suggested that the increased levels of MCA ± PMA-induced transformed foci are not linked to raise temperatures.	Wang et al 2005

Assay endpoint	Exposure Conditions	Response	Comment	References
Transformation (i. RF alone; ii. MCA+ RF; iii. MCA+PMA+RF) in mouse BALB/3T3 cells assay.	2142.5 MHz, W-CDMA modulation SAR: 80 or 800 mW kg^{-1} for 6 weeks.	No induction, promotional or co-carcinogenic effect of RF exposure on transformation.		Hirose et al 2008

II.3.5. Summary on cellular studies

Over the last 30 years there have been many *in vitro* studies on potential cellular effects of RF. These studies gave insight into the basic mechanisms by which effects might be induced in more complex animal or human organisms. Interpretation is, however, limited by anomalous cell behavior generated by the culture conditions and other factors which limit the extrapolation to humans. The studies conducted so far have not provided consistent evidence of biological effects under non-thermal RF exposure conditions. In the case of genetic effects, for example, most results were negative and some of the few positive findings may be attributable to a thermal insult rather than to the RF-exposure as such. The same holds true for other endpoints. With regard to signaling, studies done using measurements of calcium ion concentration related to cellular function do not support earlier positive reports on calcium ion physiology. There is insufficient research regarding RF effects on nitric oxide signaling, gap junctions and receptor clustering to be conclusive, but the results of studies on cell proliferation and differentiation, apoptosis and cell transformation are mostly negative.

Changes in cell physiology and function imply changes in gene and protein expression. An early publication on heat shock gene expression in the nematode *C. elegans* initiated further investigation of various genes known to be stress-responsive. However, this positive finding was later shown to have resulted from inadvertent heating, due to lack of rigorous dosimetry. Recent studies suggest that RF exposure has no or very little effect on the expression of cancer-related genes (e.g., proto-oncogenes and tumor suppressor genes). However, the results of studies of the effects of RF exposure on stress protein expression, particularly hsps, have so far been inconsistent, although mostly negative outcomes have been reported *in vitro.* Heating remains a potential confounder, and probably accounts for some of the positive effects reported. Nevertheless, further studies should be conducted to evaluate the influence of RF exposure on major stress signaling pathways

More recently, studies have been carried out using powerful high-throughput screening techniques capable of examining changes in the expression of very large numbers of genes and proteins. Such studies often showed a limited number of alterations where some genes were up- and others down-regulated. Apoptosis-related genes were amongst the up-regulated ones, and cell cycle genes amongst the down-regulated ones, but this was not always the case. High-throughput techniques have thus so far not provided any evidence of an RF 'signature'. Overall, it should be noted that:

- Quantitative methods have not always been used to confirm the initial findings; such a step is generally accepted as necessary for confirmation.
- Repeat experiments in array analysis have not often been conducted, which prevents confirmation of the earlier results.
- Changes have frequently been found in only a few genes out of several hundreds investigated, which might have occurred by chance.
- The changes that have been reported are usually very small compared to those induced from eg known carcinogens such as UVR, and may be of little functional significance.
- Ensuring adequate dosimetry and temperature control has proved difficult in many of these studies and heating may account for some of the positive effects reported.

These advances in molecular studies are promising, but not yet decisive in risk evaluation. The microarray technology, for example, can be very important in confirming results obtained by more conventional scientific methods and helping elucidate mechanisms of action, but, on their own, results

from such studies are not yet sufficiently understood and the methodologies not sufficiently standardized and validated to provide decisive data on RF (and other) health effects. However, if a gene or a protein is identified as an RF-responsive molecule, the possibility that the change has a physiological or pathological consequence should be further explored with both *in vitro* and *in vivo* studies.

II.4. ANIMAL STUDIES

Animal studies are frequently based on experiments using laboratory strains of mice or rats. The advantage of such studies is that they provide information concerning the interaction of RF radiation with living systems which display the full repertoire of body functions, such as immune response, cardiovascular changes, and behavior, in a way that cannot be achieved with cellular studies. Transgenic or gene knockout animal models of certain diseases have further increased the value of animal studies to reveal potential adverse health effects. Animal studies are thus usually a more powerful experimental tool than cellular studies in this context. However, extrapolation to humans is not straightforward since there are obvious differences in physiology and metabolism between species as well as differences in life expectancy and many other variables. Nevertheless, at a molecular level, there are many similarities between processes in animals and humans and such studies have been very useful in helping unravel the sequence of genetic events in the development of a number of human cancers (e.g., Balmain and Harris 2000; Anisimov et al 2005).

Generally, animal studies can be expected to provide qualitative information regarding potential outcomes, but the data cannot be extrapolated quantitatively to give reliable estimates of human risk for the reasons outlined above. In addition, differences in body size, which are particularly marked in laboratory rodents compared to humans, means that dosimetric interaction will be different, small animals showing body resonance to RF radiation at higher frequencies than humans, with a comparatively greater depth of penetration relative to body size. Major improvements in exposure systems for animals have been achieved in the recent years. Several types of setups are being used depending on the type of exposure needed (such as head-only or whole-body). The various systems in common use (such as loop antennas, carousels, Ferris wheels, radial transmission lines and reverberation chambers) are described in Chapter I.3.4. The selection of RF exposure systems used in animal studies is often a compromise between restraint-related stress and the accuracy of RF dosimetry. If animals are allowed to move freely during RF exposure, they change their position and orientation relative to the electromagnetic wave and may also be shielded by other animals, which results in large uncertainties in dosimetry. Therefore, immobilization of animals has been used in many animal studies to achieve well-defined dosimetry. However, immobilization can cause restraint-related stress that might affect the outcome of the experiment unless appropriate steps, such as the habituation of animals to restraint, are taken.

II.4.1. Genotoxicity

Several studies have been conducted over the past 30 years using *Drosophila melanogaster* as the test organism. They all yielded negative results (see Verschaeve 1995; Léonard et al 1983; WHO; 1993).

With regard to laboratory mammals, many studies that have been published so far have not demonstrated convincingly any direct DNA damage after acute or chronic exposure to RF radiation (e.g. Léonard et al 1983; WHO 1993; Verschaeve and Maes 1998; Meltz 2003; Vijayalaxmi and Obe 2004), in particular when temperatures were maintained within normal physiological limits. However, a number of investigations have suggested that RF radiation can affect DNA (Table II.4.1.). Sarkar et al (1994) found evidence of an alteration in the length of a DNA micro satellite sequence in cells from the brain and testis of mice exposed to 2450 MHz fields, whereas Lai and Singh demonstrated in a series of publications (Lai and Singh 1995, 1996a, 1997, 2005; Lai et al 1997) that acute exposure to low-intensity radiofrequency radiation increased DNA strand breaks in the brain cells of rats. A significant increase in DNA strand breaks was found immediately and 4 h after exposure. It was suggested that this could be due either to a direct effect on the DNA or to an effect on DNA repair mechanisms (Lai and Singh 1996a). The authors

furthermore provided data suggesting that free radicals may play a role in the observed SSBs and DSBs as the addition of free radical scavengers reduced the effect (Lai et al 1997).

These observations have been the subject of discussion and criticism in the scientific community. The fact that effects were observed at 4 h post exposure was especially criticized (Williams 1996), but arguments in favor of the findings were subsequently presented by Lai and Singh (1996b). Nevertheless, studies by other authors, including two attempted replications, have not reported RF-induced DNA damage in rat brain cells (Malyapa et al 1998; Lagroye et al 2004b; Verschaeve et al 2006; Belyaev et al 2006). These contrasting results were attributed partly to differences in procedures, especially in the ways the animals were killed and in the time lag between the death of the rats, dissection of the brain, and slide preparation for the comet assay (Malyapa et al 1998). As replication studies were not able to confirm the Lai and Singh data the significance of the findings therefore remain unclear to date but point to an absence of field-dependent effects. The same holds true for other genetic endpoints where both positive and negative findings were reported (e.g., on the incidence of micronuclei, (cf. Table II.4.1.).

Most of the animal studies have been conducted in somatic cells (blood, bone marrow, brain, liver or spleen). Only a few studies have been devoted to germ cells or the reproductive system. Ono et al (2004) did not find any increased mutation frequency in the testes (and other organs) of the offspring of RF exposed pregnant mice. However, Aitken et al (2005) did find a significant genotoxic effect on the epididymal spermatozoa of mice that were exposed for 7 days to 900 MHz low-level RF, whereas no impact on male germ cell development was observed. These studies differed in many aspects (e.g., *in utero* vs. *in vivo* exposure, LacZ gene mutation vs. Q-PCR analysis, etc.*)* which may eventually account for the different results. Aitken et al (2005) note that during epididymal transit spermatozoa have lost all capacity for DNA repair and are therefore vulnerable to factors that might affect DNA integrity. However, the possible genotoxic effect of RF-radiation on epididymal sperm remains unconfirmed at present.

In summary, most studies have failed to convincingly demonstrate any direct genetic effect after exposure of laboratory mammals to RF radiation, in particular when temperatures were maintained within normal physiological limits.

Table II.4.1.: RF-radiation alone or in combination with chemical/physical mutagens

Assay endpoint	Exposure Conditions	Response	Comment	References
DNA analysis with synthetic oligo probes in brain cells and testes of mice	2.45 GHz; CW; SAR: 1.2 W kg^{-1}; 2 h per day for 120, 150 and 200 days.	DNA fragments: altered band patterns of DNA		Sarkar et al 1994
DNA single and double strand breaks (comet assay) assayed in rat brain cells immediately and 4 h after RF-exposure	2450 MHz, pulsed or CW; SAR: 1.2 W kg^{-1}; 2 h exposure	Significant increase in DNA strand breaks immediately and 4h after exposure		Lai and Singh 1995
DNA single and double strand breaks (comet assay) assayed in rat brain cells immediately and 4 h after RF-exposure	2450 MHz, pulsed or CW; SAR: 1.2 W kg^{-1}; 2 h exposure	Significant increase in DNA strand breaks immediately and 4h after exposure		Lai and Singh 1996a
DNA single and double strand breaks (comet assay) assayed in rat brain cells immediately and 4 h after RF-exposure	2450 MHz, 2 h exposure as above; rats were also treated with melatonin or N-tert-butyl-a-phenylnitrone (free radical scavengers)	Treatment of rats with free radical scavengers before and after RF negated the induction of DNA strand breaks		Lai and Singh 1997
Micronuclei in peripheral blood and bone marrow cells in tumor prone mice	2450 MHz; CW; SAR:1 W kg^{-1}; 20 h per day, 7 days per week for 1.5 years	No effects observed		Vijayalaxmi et al 1997b

Assay endpoint	Exposure Conditions	Response	Comment	References
Micronuclei in polychromatic erythrocytes (from peripheral blood and bone marrow) of CF-1 mice	Animals were exposed for 15 minutes to ultra-wide band (UWB) radiation at 37 mW kg^{-1}	No effects observed		Vijayalaxmi et al 1999
DNA single strand breaks (comet assay) in rat brain cells.	2450 MHz; CW; SAR=1.2 W kg^{-1}; 2 h exposure	No observed DNA damage in brain cells of rats euthanized by CO_2 asphyxia or decapitation	Comet assay conduced immediately and 4 h after RF-exposure	Malyapa et al 1998
Micronuclei in peripheral blood and bone marrow cells in rats	2450 MHz; CW; SAR: 12 W kg^{-1}; 24 h exposure	No effects observed		Vijayalaxmi et al 2001a
Somatic intrachromosomal recombination in spleen cells of pKZ1 transgenic mice	900 MHz pulsed-wave; SAR: 4 W kg^{-1}; 30 min per day for 1, 5 and 25 days	No evidence of a genotoxic effect	Significant reduction in inversions below the spontaneous frequency in the 25-day exposure group	Sykes et al 2001
Micronuclei in rat peripheral blood cells	2450 MHz, CW; SAR: 1 and 2 W kg^{-1}; 2 h per day, 7 days per week for up to 30 days	Increased incidence of micronuclei in animals exposed to RF after eight irradiation treatments of 2 h each		Trosic et al 2002
Mutation assay (mutant lacI genes) in brain tissue of Big Blue mice	1.5 GHz at SAR: 2.0, 0.67 and 0 W kg^{-1}. Animals were exposed for 90 min per day, 5 days per week, for 4 weeks	1.5 GHz was not found mutagenic to mouse brain cells		Takahashi et al 2002
Micronuclei in rat bone marrow cells	1600 MHz; iridium signal; SRA: 0.16 and 1.6 W kg^{-1}; 2 h per day, 5 days per week for 2 years	No evidence of a genotoxic effect		Vijayalaxmi et al 2003
Micronuclei in mouse peripheral blood and bone marrow cells	42.2 GHz; SAR: 622 ± 100 W kg^{-1}; 30 min per day for 3 consecutive days; also co-exposure with cyclophosphamide	No evidence of genotoxic effect of RF alone and no influence on cyclophosphamide induced micronuclei		Vijayalaxmi et al 2004
Alkaline comet test (with and without the use of proteinase K in the assay) in rat brain cells.	2450 MHz Pulsed wave; SAR: 1.2 W kg^{-1}; 2 h exposure	No DNA damage found	Comet assay conducted 4 h after RF exposure.	Lagroye et al 2004b
Mutation frequency at the LacZ gene in cells from the spleen liver brain and testes of the offspring of LacZ-transgenic mice.	2450 MHz; SAR: 0.71 W kg^{-1} (intermittent exposure of 10 sec. on with 4.3 W kg^{-1} and 50 sec. off); *in utero* exposure for 16 h per day at gestational age of 0-15 days	No effects observed	Offspring analyzed at 10 weeks of age	Ono et al 2004

Assay endpoint	Exposure Conditions	Response	Comment	References
Micronuclei in rat bone marrow cells	2450 MHz; CW; SAR: 1.25 W kg^{-1}; 2 h per day, 7 days per week and 4, 16, 30 and 60 h	Increased incidence of micronuclei on experimental day 15		Trosic et al 2004b
Micronuclei in rat bone marrow cells	910 MHz; peak SAR: 0.42 W kg^{-1}; 2 h/day for 30 consecutive days	Increased incidence of micronuclei	Observations possibly biased by presence of mast cell granules that cannot be easily discriminated from micronuclei.	Demsia et al 2004
DNA single and double strand breaks (comet assay) assayed in rat brain cells immediately and 4 h after RF-exposure	2450 MHz; SAR: 0.6 W kg^{-1}; 2 h exposure	Brain cells of RF-exposed rats had significantly higher levels of SSBs and DSBs.		Lai and Singh 2005
Micronuclei in blood erythrocytes, bone marrow, keratinocytes and spleen lymphocytes of mice	GSM 900 MHz and DCS 1800 MHz; amplitude modulated; SAR: 0, 3.7, 11 and 33.2 W kg^{-1} (1 week study) and 0, 2.8, 8.3 and 24.9 W kg^{-1} (6 week study); 2 h per day exposure	No DNA damaged observed in brain cells		Görlitz et al 2005
DNA damage assessed by quantitative PCR (Q-PCR) and alkaline- and pulsed field electrophoresis in caudal epididymal spermatozoa of mice	900 MHz; SAR: 0.09 W kg^{-1}; exposure for 7 days at 12 h per day	No impact on male germ cell development but Q-PCR revealed a significant genotoxic effect on the epididymal spermatozoa		Aitken et al 2005
DNA damage (alkaline comet assay) and micronuclei in rat blood, liver and brain cells	900 MHz; amplitude modulated; SAR: 0.3 and 0.9 W kg^{-1}; 2 h per day, 5 days per week for 2 years. Exposure in conjunction with MX exposure in the drinking water.	Co-exposure to MX and RF-radiation did not increase the response of blood (comet and micronucleus assay) or liver and brain cells (comet test)		Verschaeve et al 2006
Changes in chromatin conformation and DNA double strand breaks (pulsed field gel electrophoresis) in rat brain cells	915 MHz (GSM); SAR: 0.4 W kg^{-1}; 2 h exposure	No induction of DNA double strand breaks or chromatin conformation, but changes in gene expression were observed		Belyaev et al 2006
Micronucleus formation in blood from rats being exposed to mobile phone radiation during their embryogenesis	Exposure to cellular phone antenna (834 MHz, 26.8-40 V m^{-1}) from the first day of pregnancy for 8.5 h per day. SAR estimated at 0.55-1.23 W kg^{-1}	Significant increase in erythrocyte MN frequency in newborn pups from exposed pregnant rats.		Ferreira et al 2006

Assay endpoint	Exposure Conditions	Response	Comment	References
DNA damage (alkaline comet assay) in rat brain cells	2.45 GHz and 16.5 GHz at SAR: 1.0 and 2.01 W kg^{-1}. Exposure 2 h per day for 35 days	Statistically significant increase in DNA single strand breaks following RF exposure		Paulraj and Behari 2006
Micronucleus frequency in erythrocytes of mice	902.5 MHz (NMT) signal at a SAR: 1.5 W kg^{-1}; or 902.5 MHz (GSM) signal at SAR=0.35 W kg^{-1} for 78 weeks (1.5 h per day, 5 days per week).	No effect	Animals taken at necropsy from a co-carcinogenicity study by Heikkinen et al 2001. During first weeks also X-irradiation at 4 Gy	Juutilainen et al 2007
Micronucleus frequency in erythrocytes of mice	K2 transgenic and non transgenic mice exposed 52 weeks to digital mobile phone signals, GSM and DAMPS at SAR: 0.5 W kg^{-1}.	No effect	Animals taken at necropsy from a co-carcinogenicity study by Heikkinen et al 2003. Exposure 3 times per week to 1.2 MED UV-radiation	Juutilainen et al 2007
MX=3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone; MED = minimal erythemal dose				

II.4.2. Cancer

Animal studies investigating the carcinogenic potential of RF radiation were reviewed by WHO (1993), while more recent studies have been reviewed by Repacholi (1997), Krewski et al (2001a & b), AGNIR (2003), Elder (2003b), and Krewski et al (2007). This review focuses on papers published after 1993, but some earlier key studies are also described.

Evaluating carcinogenicity in laboratory rodents has remained a cornerstone in identifying agents likely to cause cancer in humans. According to IARC, agents for which there is sufficient evidence of carcinogenicity in experimental animals are considered to pose carcinogenic hazard to humans, unless there is scientific evidence that the agent causes cancer through a species-specific mechanism that does not operate in humans (IARC 2006). However, despite the similarities in many cancer characteristics between humans and laboratory rodents, interspecies differences need to be taken into account when extrapolating data from rodents to humans: many agents that are carcinogenic in rodents (often only at very high doses) are not carcinogenic to humans, and some human carcinogens do not affect rodents (Ames and Gold 1990; Trosko and Upham 2005; Anisimov et al 2005).

The effects of stress resulting from restraint and related daily handling has be seen in many animal cancer studies as a lower body weight among the sham-exposed (restrained) animals than among the cage control (unrestrained) animals (see below: Heikkinen et al 2003; Oberto et al 2007; Shirai et al 2007; Smith et al 2007; Yu et al 2006; Zook and Simmens 2006). In many of these studies, tumor incidence has also been lower and survival higher in the sham-exposed (restrained) group than in the cage control (unrestrained) group, which may be related to the observations that reduced energy intake inhibits the development of tumors (Keenan et al 1996; Sinha et al 1988; Klurfeld et al 1991). Immobilization has not caused experimental bias in studies assessing carcinogenicity of RF radiation, as both the RF exposed and the sham-exposed animals have been restrained, but it can be argued that stress could act as an effect modifier and hide possible RF-induced effects. However, there is no evidence of such modifying effects: many of the studies reviewed above have used freely moving animals, and the majority of studies have produced negative findings independent of the handling (restrained or unrestrained) of the animals.

Classical carcinogenicity bioassays involve exposure of animals over most of their lifetime to the agent being tested. Such studies are potentially capable of revealing whether the tested agent alone could act as a complete carcinogen or serve to increase the incidence of spontaneous tumors. This type of studies are, however, not sensitive in detecting weak carcinogenic effects (because of the low number of tumors induced) and co-carcinogenic effects (interaction with other carcinogens). To overcome these limitations, several studies have used tumor-prone animal strains or combined exposure to RF radiation and known carcinogens. The animal studies are classified here as: i) studies with exposure to RF field alone (Table II.4.2.), including studies using tumor-prone animals strains (Table II.4.3.), ii) studies using exposure to RF radiation combined with a known genotoxic/carcinogenic agent (Table II.4.4.), and iii) studies evaluating effects of RF radiation on the very last steps of carcinogenesis using implanted or injected tumor cells (Table II.4.5.).

II.4.2.1. RF radiation alone

Conventional laboratory animal strains

Long-term rodent bioassays evaluating carcinogenicity of RF radiation alone have been rather consistent and have not found evidence for RF field-related effects on tumor development in conventional rat strains (Chou et al 1992; Zook and Simmens 2001; Adey et al 1999; Adey et al 2000; Anderson et al 2004; La Regina et al 2003) or mouse strains (Utteridge et al 2002). The main emphasis in many of these studies has been the combined effects of RF radiation with known genotoxic agents, but the study design has also involved groups exposed to RF radiation alone. Details of studies evaluating exposure to RF radiation alone are shown in Table II.4.2.

The first carcinogenicity study on RF radiation was published several decades ago (Prausnitz and Susskind 1962). The authors reported some indication of increased testicular degeneration, and increased neoplasias of white blood cells, which they termed “leucosis”, in RF field exposed mice. Both the methods and reporting of this study have been severely criticized (see e.g. Roberts and Michaelson 1983). For example, the daily exposure time was short, but RF dose-rate was high resulting in 2-5ºC increase in body temperature, the methods were not described in detail, a large number of animals were lost due to autolysis, and the conclusions were not based on statistical analysis. Therefore, this report has no real value in cancer risk assessment.

Using a so-called ‘carousel exposure set-up’ for well-defined RF exposure levels in the head, five recent studies failed to find evidence of enhanced brain tumorigenesis in RF field-exposed rats (Adey et al 1999; Adey et al 2000; Anderson et al 2004; La Regina et al 2003; Zook and Simmens 2001) at average SARs in the brain up to about 1.5 W kg^{-1}. In the carousel set-up, the rats are restrained head first in cylindrical tubes arranged in a radial configuration with the RF antenna at the centre of the carousel, where the head is preferentially irradiated. The SAR in other body parts is much lower, and the ratio of brain average SAR to whole body average SAR may be up to 10:1 at mobile phone frequencies (Schönborn et al 2004). The animals in these studies had been exposed for most of their lifetime, and three of the studies also included *in utero* exposures (Adey et al 1999; Adey et al 2000; Anderson et al 2004). There was some indication of decreased CNS glial tumor development in the group exposed to NADC-modulated RF field (Adey et al 1999). The unexpectedly high incidence of spontaneous CNS tumors in the control group, however, suggests that this statistically non-significant difference might be a consequence of chance. The studies that have involved histopathological evaluation of other organs have provided no evidence of enhanced tumorigenesis in other tissues exposed at considerably lower SAR values than the brain (Anderson et al 2004; La Regina et al 2003; Zook and Simmens 2001).

The combined incidence of malignant tumors (all tumor types combined) was statistically significantly increased in male Sprague-Dawley rats exposed to radar-type pulsed 2.45 GHz RF radiation at whole-body average SAR of 0.15-0.4 W kg^{-1} (Chou et al 1992). The organ-specific tumor incidences were low (except those in some endocrine organs). The incidence of any single type of primary malignant or benign neoplasm, the combined incidence of benign neoplasms or survival were not statistically significantly affected, and the authors concluded that, overall, the study did not show any definite biologically significant effects. The incidence of benign pheochromocytoma was reported to be higher in RF-exposed

rats, but the difference did not reach statistical significance. No tumor-enhancing effects of RF field exposure were reported in Sprague-Dawley rats in a later study reporting slightly (but statistically non-significantly) lower incidences of combined adrenal tumors in RF-exposed males (Zook and Simmens 2001). The SAR levels were similar and both studies used relatively long daily exposure times. However, the later study (Zook and Simmens 2001) was concentrated on brain tumorigenesis, and did not include complete histopathology of all other organs. Thus, it did not provide data on combined tumor incidences.

Exposure to RF radiation did not affect the incidence of lymphomas in C57BL/6Ntac mice, the 'wild type' of the Eμ-*Pim1* transgenic mice used in the same study (Utteridge et al 2002). This study was planned as a replication experiment of an earlier study (Repacholi et al 1997) reporting enhanced the development of lymphoma in Eμ-*Pim1* transgenic mice exposed to RF radiation (see below). In addition to transgenic animals, Utteridge and colleagues used also corresponding wildtype C57BL/6Ntac mice exposed at four SAR levels ranging from 0.25 to 4 W kg^{-1}. There were only a few lymphoblastic leukemias in the wild-type animals, and for non-lymphoblastic leukemias there were no statistically significant differences between the sham-RF and RF-exposed animals.

Two studies evaluated carcinogenicity of both a GSM signal at 902 MHz and a DCS signal at 1747 MHz in B6C3F1 mice (Tillmann et al 2007) and in Wistar rats (Smith et al 2007). Three exposure levels ranging from 0.4 to 4 W kg^{-1} (and sham exposure) were used. In the mouse study (Tillmann et al 2007), no significant increase in the incidence of any particular tumor type in the RF exposed groups was observed. Interestingly, in both studies (both RF signals) the incidence of liver adenomas in males decreased with increasing exposure level, with a statistically significant difference between the highest exposure and the sham-exposed group. However, comparison to published tumor rates in untreated mice revealed that the observed tumor rates were within the range of historical control data. In conclusion, the study produced no evidence that exposure at whole body SARs of up to 4.0 W kg^{-1} increased the incidence or severity of neoplastic or non-neoplastic lesions, or resulted in any other adverse health effects. The rat study (Smith et al 2007) was a combined chronic toxicity and carcinogenicity study, and some of the animals (15 males and 15 females per group) were killed at 52 weeks from the start of the study. There were no significant differences in incidence, multiplicity, latency or severity of neoplasms, or any other adverse responses to RF field exposure.

Table II.4.2.: Carcinogenic effects of RF radiation: Exposure to RF radiation alone, normal strains

Assay endpoint	Exposure conditions	Results	Comments	Reference
CNS Tumors				
CNS tumors in F-344 rats 30 females and 30 males/group.	836.55 MHz D-AMPS 1) freely moving pregnant dams, circular polarization, SAR not given, 2 h/d , gestation day 19 until birth 2) freely moving pups, circular polarization, SAR not given, 2 h/d, from birth until weaning 3) restrained (carousel set-up,) from age of 33 d, brain SAR: 0.3–0.5 W kg^{-1} (whole-body SAR: 0.2-0.4 W kg^{-1}), 2 h/d 22 months (intermittent exposure: 7.5 min on/ 7.5 min off)	No effects on CNS/brain tumor incidence. No significant effects on survival	Exposure started *in utero*.	Adey et al 1999

Assay endpoint	Exposure conditions	Results	Comments	Reference
CNS tumors in F-344 rats 45 females and 45 males/group.	836.55 MHz FM 1) freely moving pregnant dams, circular polarization, SAR not given, 2 h/d, gestation day 19 until birth 2) freely moving pups, circular polarization, SAR not given, 2h/d, from birth until weaning 3) restrained (carousel set-up) from age of 33 d, brain SAR: 1.1-1.4 W kg^{-1} (Whole-body SAR: 0.3-0.7 W kg^{-1}), 2 h/d , 4 d/w, for 23 months	No effects on CNS/brain tumor incidence. No effects on survival.	Exposure started *in utero*	Adey et al 2000
CNS tumors in Sprague-Dawley rats 30 females and 30 males/group.	860 MHz CW or 860 MHz MiRS Restrained (carousel set-up) Brain SAR: 0.8-1.2 W kg^{-1} Whole-body SAR: 0.27-0.42 W kg^{-1} 6 h/d, 5d/wk for 22 mo	No effects on CNS/brain tumor incidence. No effect on tumorigenesis in other tissues.	8 non-neural tissues evaluated, but relatively high number of missing tissues in some of them	Zook & Simmens 2001
CNS tumors in F-344 rats 80 females and 80 males/group.	835.62 MHz, FDMA or 847.74 MHz, CDMA. Restrained (carousel set-up). Brain SAR: 1.3±0.5 W kg^{-1} (mean ±SD) Whole-body SAR not given (SAR in other organs less than 1/3 of that in brain). 4 h/d, 5d/wk, for 104 weeks	No increase in CNS tumors. No increase in tumors in other tissues (all major organs evaluated). No increase in total number of tumors. No effects on survival (survival over 90%).	The study hypothesis was whether RF exposure increases tumor incidences, so decreased incidences were not statistically tested	La Regina et al 2003

Assay endpoint	Exposure conditions	Results	Comments	Reference
CNS tumors in F-344 rats 90 females and 90 males/group	1.62 GHz Iridium 1) freely moving pregnant dams (1/cage), brain SAR: (fetuses) 0.1 -0.2 W kg^{-1} (Whole-body SAR ≈ 0.06 W kg^{-1}), 2 h/d, gestation day 19 until birth 2) freely moving pups, brain SAR: 0.1-0.2 W kg^{-1} (Whole-body SAR ≈ 0.06 W kg^{-1}), 2h/d, from birth until weaning 3) restrained (carousel set-up), brain SAR: 0.11-0.18 W kg^{-1} or 1.1-1.8 W kg^{-1} (whole-body SAR ≈ 0.02 W kg^{-1} or 0.2 W kg^{-1}), 2h/d, 5d/w, 2 years	No effects on brain tumor incidence. No effects on incidence of lymphoma. No effects on tumors in other tissues evaluated No effects on survival.	Exposure started *in utero*	Anderson et al 2004
Lymphomas				
Lymphoma in female C57BL/6Ntac mice (wild type of Eμ-*Pim1*) 120 mice /group	898.4 MHz GSM. Restrained ("Ferris wheel") Whole-body SAR: 0.25, 1.0, 2.0 or 4.0 W kg^{-1}, 1 h/d, 5d/wk for 104 wk	No differences in the incidence of non-lymphoblastic lymphomas; incidence of lymphoblastic lymphoma low in all groups No effects on total tumor incidence (12 tissues evaluated) No effects on survival	The study included also transgenic animals, see Table II.4.3.	Utteridge et al 2002
Multiple tumors				
Multiple tumors in male Sprague-Dawley rats 100 rats/group.	2.45 GHz pulsed (10 μs pulses at 800 pps; pulse-modulated also at 8 pps); Freely moving; Whole-body SAR 0.15–0.4 W kg^{-1}; for 21.5 h/day, 7 days/week, for 25 months	No increase in any individual tumor type. Four-fold increase in combined primary malignancies, but no increase in combined primary benign tumors. No effects on survival.		Chou et al 1992
Multiple tumors in male and female B6C3F1 mice 50 males and 50 females/group	902 MHz GSM or 1747 MHz DCS Restrained ("Ferris wheel") Whole-body SAR: 0.4, 1.3 or 4.0 W kg^{-1}, 2 h/d, 5 d/wk for 2 years	No increase in the incidence of any neoplastic or non-neoplastic lesions	Two signals, three exposure levels for each signal	Tillmann et al 2007

Assay endpoint	Exposure conditions	Results	Comments	Reference
Multiple tumors in male and female Wistar rats 65 males and 65 females/group.	902 MHz GSM or 1747 MHz DCS Restrained ("Ferris wheel") Whole-body SAR: 0.44, 1.33 or 4.0 W kg^{-1}, 2 h/d, 5 d/wk for 2 years	No increase in the incidence of any neoplasms; no other adverse effects	Combined chronic toxicity/carcinogenicity study, 15 females and 15 males per group were killed at 1 year	Smith et al 2007

Studies using genetically predisposed animal models

Animal strains developing tumors (in some organs) with particularly high frequency and/or early in life are classified as 'tumor prone strains'. These strains include animals engineered to be more vulnerable via gene manipulation (transgenic animals), as well as other strains with exceptionally high tumor incidence due to their genetic background. The division between "tumor prone" and "other" strains is somewhat arbitrary, because spontaneous tumor frequency varies greatly between different animal strains. Details of studies using genetically tumor-prone animal strains are described in Table II.4.3. The spontaneous incidence of tumors in this kind of experimental models is important: if nearly all animals in the unexposed control group develop tumors, there is not much room for an additional effect from RF field exposure. Therefore, information on tumor incidence in unexposed animals is included in Table II:4.3. [Note, however, that accelerated development of tumors can be detected even if the final incidence is 100%, if the tumors are externally observable during the experiment, as is the case for eg skin tumors and mammary tumors.]

Lymphoma models

Transgenic Eμ-*Pim1* mice overexpressing *Pim1* oncogene in their lymphoid cells are prone to malignant lymphoma. In the first study with this model using RF (Repacholi et al 1997) Eμ-*Pim1* mice were exposed to 900 MHz GSM-type RF radiation at SARs ranging from 0.13 -1.4 W kg^{-1} (if all possible animal orientations are included, the range was 0.008 to 4.2 W kg^{-1}). The RF exposed animals had twofold lymphoma incidence compared to controls. At the time the study was terminated, lymphoma incidence was increasing rapidly in both exposed and sham-exposed animals. The authors emphasize that even if the observed effect were established, the relevance of the animal model for human cancer risk assessment needs to be carefully considered. The findings of this study were not confirmed in a replication study by Utteridge et al (2002), who used the same strain of mouse obtained from the same supplier. The investigators also fed the same food to the mice. The later study had some refinements in experimental design: four SAR levels (0.25, 1.0, 2.0 and 4.0 W kg^{-1}) were used instead of one in the original study; animals were restrained during the exposure for better control of variations in exposure level; animals were exposed once per day instead of two episodes of 30 minutes; and full necropsy was performed on all mice at the end of the study. RF field exposure did not enhance development of lymphoma. The incidence of lymphoblastic leukemia was slightly lower in all RF-exposed groups compared to that of the sham-exposed animals, and the difference was statistically significant at the lowest dose rate. In contrast, the incidence of non-lymphoblastic leukemia was slightly higher in RF exposed groups, but these differences were not statistically significant either in pairwise comparisons or in a trend test. The incidence of lymphomas in the RF-sham-exposed group was surprisingly high, and the publication stirred debate whether some critical features of the original experiment had been changed (Goldstein et al 2003a; Goldstein et al 2003b; Kundi 2003a; Kundi 2003b; Lerchl 2003).

The study reported by Oberto et al (2007) was also a replication and an extension of the Repacholi et al (1997) study. Eμ-*Pim1* transgenic mice were exposed for 1 h/day, 7 days/week to pulsed GSM 900 RF at a whole-body SAR of 0.5, 1.4 or 4.0 W kg^{-1}. 50 animals per sex per group were exposed, sham-exposed or used as cage controls. There were several methodological improvements compared to the original study by Repacholi et al (1997), including use of several exposure levels, well-defined dosimetry and more uniform exposure (achieved through restrain of the animals) and necropsy and extensive histopathology of all animals. Compared to the sham-exposed controls, survival was reduced in the

animals exposed to RF radiation. The intergroup differences were statistically significant in the male animals, but there was no trend with increasing exposure level (lowest survival at 0.5 W kg^{-1}). No increase in lymphoma incidence was observed in the RF exposed groups. Concerning other neoplastic findings, Harderian gland adenomas were increased in male mice, with a significant dose-related trend ($p<0.01$). However, this trend was not supported by the findings on female animals, in which no tumors in the highest exposure groups were observed. The statistical analysis used in this study can be criticized. The cage control and the sham-exposed control groups were combined for statistical comparisons, which is not a valid procedure given the differences in body weight development and tumor incidence between these groups (these differences are most likely related to restraint of the sham-exposed animals). However, based on the data reported in the paper, a different analysis strategy (comparison to the sham-exposed group only) would not essentially change the interpretation that there was no effect of RF exposure on tumor incidence at any site. The reduced survival in RF-field-exposed animals is not thoroughly discussed by the authors; this finding remains unexplained and difficult to interpret without detailed information about the causes of death.

GSM-type RF exposure at nominal SAR of 0.4 W kg^{-1} did not affect development of lymphoma in female AKR/J mice (Sommer et al 2004). This mouse strain is prone to develop lymphoma due to expression of an AKV retrovirus in all of their tissues. About 90% of animals both in the sham-exposed and RF-exposed groups developed lymphoma by the end of the 10-month study. Essentially mortality was reported to be related to the development of lymphoblastic lymphoma, and RF field exposure did not affect survival. No effects of exposure were seen in differential leucocyte count of blood samples collected 5-10 months after the beginning of RF exposure. The nominal SAR was 0.4 W kg^{-1}, but as in other studies using several freely moving animals per cage, the variation in exposure level would undoubtedly have been large.

In another study by the same group (Sommer et al 2007), unrestrained AKR/J mice, 160 animals per group, were chronically sham-exposed or exposed to a generic UMTS test signal for 24 h/day, 7 days/week at a SAR of 0.4 W kg^{-1}. Additionally, 30 animals were kept as cage controls. The animals were checked visually each day and were weighed and palpated weekly to detect swollen lymph nodes. Starting at the age of 6 months, blood samples were taken from the tail every 2 weeks to perform differential leukocyte counts and to measure the hematocrit. Visibly diseased animals or those older than 43 weeks were killed humanely, and tissue slices were examined for metastatic infiltrations and lymphoma type. Cage control animals had a significantly lower growth rate than those kept in the radial waveguides. Incidence of lymphoma, survival time and the severity of the disease indicated that there was no effect from exposure to RF radiation. Cage control animals had significantly lower body weights and higher occurrence of metastatic infiltrations in liver and meninges than the other groups. This difference was most likely related to different housing conditions and stress level.

Models for mammary tumorigenesis

The accelerated tumor development in mammary tumor prone female C3H/HeA mice reported by Szmigielski and co-workers (Szmigielski et al 1982) has not been confirmed by other long-term studies using female C3H/HeJ mice at lower SARs but generally longer daily exposure times (Frei et al 1998a; Frei et al 1998b; Jauchem et al 2001; Toler et al 1997).

In the study of Szmigielski et al (1982) the exposure levels were expressed in W m^{-2}. The SAR values were estimated to be about 2-3 and 6-8 W kg^{-1}, and thermally induced stress may have affected the outcome at least at the higher exposure level. The response to the lower RF level was reported to be similar to that of confinement stress. Similarly to Szmigielski et al, Frei and co-workers used continuous 2.45 GHz RF radiation (Frei et al 1998a; Frei et al 1998b) whereas two other studies used signals consisting of short pulses (Jauchem et al 2001; Toler et al 1997). Although the four later studies were designed specifically to examine mammary tumors, they included histopathological analyzes of other main tissues. Overall, the authors of these studies concluded that RF field exposure did not affect the development of tumors or survival of animals. The only statistically significant differences in tumor incidence reported in these studies were a smaller number (0 vs. 4) of alveolar-bronchiolar adenomas in RF field exposed animals in one study (Frei et al 1998a), and increased incidence of bilateral ovarian

tumors in another study (Toler et al 1997). The latter was, however, not accompanied with increase in the number of mice developing an ovarian tumor.

Multiple tumor models

Saran et al (2007) used *Patched1* heterozygous knockout mice, an animal model of multi-organ tumorigenesis in which exposure of newborn animals to ionizing radiation greatly enhances development of brain tumors (medulloblastoma). Newborn *Patched1* heterozygous mice and their wild-type siblings were exposed to GSM 900 signals at 0.4 W kg^{-1} for 30 min twice a 5 days (starting on postnatal day 2). Brains, any visible tumors and preneoplastic skin lesions were examined histopathologically. No statistically significant differences in survival were found between exposed and sham-exposed animals. Medulloblastomas (in 7 animals) and rhabdomyosarcomas (in 56 animals) were found in the *Patched1* mice but not in the wild-type animals. The incidence of rhabdomyosarcoma was higher (68%, 36 animals) in the exposed group than in the sham-exposed group (51%, 20 animals), but this difference was not statistically significant. The incidences of medulloblastomas, other tumors or preneoplastic skin lesions did not differ between the exposed and sham-exposed groups.

Table II.4.3.: Carcinogenic effects of RF radiation: Exposure to RF radiation alone, tumor-prone animal strains

Assay endpoint	Exposure conditions	Result	Comments	Reference
Lymphoma				
Lymphoma in female Eμ-*Pim1* transgenic mice 100-101 mice/group.	900 MHz GSM. Freely moving (5/cage). Whole-body SAR: 0.13- 1.4 W kg^{-1} (0.008–4.2 W kg^{-1}) 2x 30 min/d, 7 d/wk for 18 months	2-fold increase in lymphoma incidence (mainly non-lymphoblastic follicular lymphoma)	Animals that were clinically healthy at the end of the study were discarded without histopathological analyzes Incidences of lymphoblastic and non-lymphoblastic lymphomas 3% and 19 %, respectively in unexposed animals	Repacholi et al 1997
Lymphoma in female Eμ-*Pim1* transgenic mice 120 mice /group	898.4 MHz GSM Restrained (“Ferris wheel”) Whole-body SAR: 0.25, 1.0, 2.0 or 4.0 W kg^{-1} for 1 h/d, 5 d/wk for 104 wk	No enhancement of lymphoma development Lymphoblastic lymphoma slightly decreased (statistically significant at the lowest SAR) No effects on total tumor incidence (12 tissues evaluated) No effects on survival	The study included also wild-type animals, see Table II.4.2. Incidences of lymphoblastic and non-lymphoblastic lymphomas 12 % and 62 %, respectively in unexposed animals	Utteridge et al 2002

Assay endpoint	Exposure conditions	Result	Comments	Reference
Lymphoma in female AKR/J mice 160 mice/group	900 MHz GSM Freely moving (6-7/cage) Whole-body SAR: 0.4 W kg^{-1} 24 h/d, 7d/wk, for 10 months	No effects on development of lymphoma, differential count of leucocytes or survival. Exposed animals had higher body weights during late stages of the study	Lymphoma incidence 90 % in unexposed animals	Sommer et al 2004
Lymphoma in female and male Eμ-*Pim1* transgenic mice 50 females and 50 males/group	900 MHz GSM Restrained ("Ferris wheel") Whole-body SAR: 0.4, 1.4 or 4.0 W kg^{-1}, 1 h/d, 7 d/wk for 18 months	No effects on the incidence of lymphoma. Harderian gland adenoma increased in male mice, but not in females. Survival was decreased in the exposed animals (significant in males, but no dose-related trend)	Sham-exposed group and cage controls were combined for statistical analysis Incidences of lymphoblastic and non-lymphoblastic lymphomas 4% and 40%, respectively in sham-exposed females and 0% and 18% in sham-exposed males	Oberto et al 2007
Lymphoma in female AKR/J mice, 160 mice/group	1.966 GHz UMTS Freely moving (6-7/cage) Whole-body SAR: 0.4 W kg^{-1} 24 h/d, 7 d/wk for 35 weeks	No effects on incidence or severity of lymphoma. No effects on survival	Lymphoma incidence 96.7 % in unexposed animals	Sommer et al 2007
Mammary Tumors				
Mammary gland tumors in female C3H/HeA mice 40 mice/group	2.45 GHz CW Freely moving (10 /cage) 50 W/m^2 (SAR: 2–3 W kg^{-1}) or 150 W/m^2 (6–8 W kg^{-1}); 2 h/d, 6 d/wk for 10.5 months	Accelerated tumor development Decreased survival due to mammary tumorigenesis	Large uncertainty in estimated SAR Incidence of mammary tumors ≈35% in unexposed animals	Szmigielski et al 1982
Mammary tumors in female C3H/HeJ mice 200 mice/group	435 MHz pulsed (1 μs pulses, 1000 pps) Freely moving (1 /cage) SAR: 0.32 W kg-1 22h/d, 7 d/wk, for 21 months	No effect on mammary gland tumorigenesis Increased number of animals with a bilateral stromal tumors in ovaries (but no effect on incidence of animals with a stromal tumor in ovaries) No effects on other tumors (most organs analyzed). No effects on survival	Incidence of adenocarcinoma ≈ 40 % in unexposed animals	Toler et al 1997

Assay endpoint	Exposure conditions	Result	Comments	Reference
Mammary tumors in female C3H/HeJ mice 100 animals /group	2.45 GHz CW Freely moving (1/cage) SAR: 0.3 W kg^{-1} 20 h/d, 7 d/wk for 78 wk	No effects on mammary gland tumorigenesis Decreased incidence of alveolar-bronchiolar adenomas in lungs No effects on tumors in other organs (most organs evaluated) No effects on survival	Mammary gland tumor incidence 55 % in unexposed animals)	Frei et al 1998a
Mammary tumors in female C3H/HeJ mice 100 animals/group	2.45 GHz CW Freely moving (1/cage) SAR: 1.0 W kg^{-1} 20 h/d, 7 d/wk for 78 wk	No effects on mammary gland tumorigenesis No effects on tumors in other organs (most organs evaluated) No effects on survival	Mammary gland tumor incidence 30% in unexposed animals	Frei et al 1998b
Mammary tumors in female C3H/HeJ mice 100 animals/group	UWB pulsed (1.9 ns pulses, 1000 pps) SAR: 0.01 W kg^{-1} 2 min/d, 1d/wk for 12 wk	No effects on mammary gland tumorigenesis No effects on tumors in other organs (all main tissues evaluated) No effects on survival	Mammary gland tumor incidence 52% in unexposed animals	Jauchem et al 2001
Multiple tumors				
Multiple tumors in newborn *Patched1* heterozygous knock-out mice 50-63 animals (22-36 females and 23-29 males)/group	900 MHz GSM Restrained SAR: 0.4 W kg^{-1} 1 h/d, 5d/wk for 1 wk	No significant effect on medulloblastoma, rhabdomyosarcoma, other visible tumors or preneoplastic skin lesions No effects on survival	Many samples for histopathology were lost because of tissue autolysis (too late detection of death) Incidences in unexposed animals: medulloblastoma 8%, rhabdomyosarcoma 51%	Saran et al 2007

II.4.2.2. Combined RF and known genotoxic/carcinogenic agents

Both theoretical considerations (low photon energy) and experimental evidence (reviewed in Chapter II.2.) indicates that direct DNA-damaging effects of weak RF electromagnetic radiation are not likely. Therefore, there has been considerable interest in testing RF radiation as a non-genotoxic carcinogen or a co-carcinogen that enhances the effects of known carcinogenic agents. Methods for detecting non-genotoxic carcinogens and co-carcinogens are less well developed than those for detecting genotoxic carcinogens. It can be argued that classical animal carcinogenicity bioassays should identify carcinogens independently of the mechanisms. However, because of the very low number of tumors induced, such studies (involving exposure to the agent alone, without co-exposures) may suffer from low statistical

power to detect co-carcinogens. Animal studies on co-carcinogenic effects have usually been designed based on the concepts of "initiation" and "promotion". Such studies involve a short-term exposure to an "initiator" (known DNA-damaging agent), followed by long-term exposure to the putative "cancer promoter". However, it has been questioned whether the initiation-promotion approach is sufficient for describing the complex interaction of genotoxic and non-genotoxic agents (Juutilainen et al 2000). Although most of the studies on co-carcinogenicity of RF radiation have tested RF radiation as a possible "promoter" after a single dose or short-term treatment with a known "initiator", a few studies have used different approaches such as long-term simultaneous exposure to RF radiation and the known carcinogen, or RF field exposure before treatment with the known carcinogen.

Details of studies evaluating combined exposure to RF radiation with known genotoxic/carcinogenic agents are shown in Table II.4.4. As in the case of genetically predisposed models (see Chapter II.4.2.1.2.), the incidence of tumors in the control group (exposed only to the known carcinogen) should be at an appropriate level to allow detection of a possible further increase related to RF field exposure. Therefore, information of tumor incidence in the control (known carcinogen only) group is included in Table II.4.4.

Brain tumors

Several animal studies have evaluated the effects of low-level RF radiation on tumorigenesis initiated by transplacental administration of a known genotoxic agent, n-ethylnitrosourea (ENU) in Fischer 344 rats (Adey et al 1999; Adey et al 2000; Shirai et al 2005) and in Sprague-Dawley rats (Zook and Simmens 2001). Using a carousel exposure set-up to ensure well defined dosimetry of the head, these studies have provided no evidence that RF radiation can promote the development of CNS tumors in this model.

RF exposure (836.55 MHz; pulsed or continuous) did not increase the incidence of brain tumors induced by transplacental administration of ENU in Fischer 344 rats (Adey et al 1999; Adey et al 2000). North American Digital Cellular (NADC)-modulated RF field exposure appeared to decrease the incidence of ENU-induced glial CNS tumors (similar tendency was seen also in spontaneous tumors), but the difference was not statistically significant. The difference was more evident (statistically significant) if the animals surviving to the end of the experiment were excluded from the analyzes. RF exposure did not statistically significantly affect the mortality of ENU-treated animals, although survival was slightly increased in the RF exposed group.

Similarly, a more recent study reported no statistically significant effects of RF exposure on brain tumorigenesis in ENU-treated Fischer 344 rats, although the incidence of brain tumors in females was slightly lower in both RF-exposed animals compared to the sham-exposed group (Shirai et al 2005). The ENU dose was identical to that used earlier by Adey et al (1999; 2000). Considering other tissues, the incidences of pituitary tumors showed a tendency for increase in both sexes treated with ENU compared to the cage-control animals, the effect being more consistent for females. Compared to the sham-RF-exposed group, incidence of pituitary tumors was decreased in males of both RF-exposed groups. At the higher RF-exposure level (2.0 W kg^{-1}) the decrease was statistically significant, and the incidence was slightly decreased also in females. The authors questioned the biological meaning of high pituitary tumor incidence in their study, and stated that the incidences may still be within the wide range of background data of this strain. An earlier study did not report any effect of RF on tumorigenesis in pituitary glands of ENU treated Sprague-Dawley rats (Zook and Simmens 2001), but the proportion of pituitary glands tissues available for histology was only about 80% in this study. Interestingly, the development of brain tumors in ENU-treated female rats was slightly decreased in both RF-exposed groups, like in the earlier study by Adey et al (1999).

A later study from the same group (Shirai et al 2007) had otherwise similar protocol, but a different mobile phone signal was used (1.95 GHz W-CDMA versus 1.439 GHz TDMA used in the first study). In contrast to the previous study, brain tumor incidences of both females and males tended to be higher in the RF exposed groups than in the sham-exposed group, but no statistically significant effects were reported. However, the statistical method used (two-group comparisons with Fisher's exact test) is not sensitive for detecting trends with increasing exposure level. Using combined female and male data from the paper, chi-squared test for trend showed a p-value of 0.0395 for an increasing trend from the sham-

exposed group to the highest exposure group. No differences in pituitary tumors were observed in this study. Given the inconsistent findings and opposite trends observed in these two studies (Shirai 2005; 2007), the differences observed are most likely incidental.

Continuous or pulsed 860 MHz Motorola integrated Radio Services (MiRS) head-mainly RF field exposure at 1 W kg^{-1} did not significantly affect incidence, volume, multiplicity, malignancy or fatality on ENU-induced brain tumors or development of tumors in eight other organs in Sprague-Dawley rats (Zook and Simmens 2001). There was a slight statistically non-significant tendency toward higher incidence of fatal brain tumors in the group treated with higher level of ENU and exposed to the pulsed RF field.

In a follow-up study, Zook and Simmens (2006) investigated further potential promoting effect of the pulsed RF signal. Latency and other characteristics of neurogenic tumors were investigated in the progeny of pregnant Sprague-Dawley rats treated with 6.25 or 10 mg/kg of ENU. The 1080 offspring were randomized equally by number, sex and ENU dose into pulsed RF, sham and cage control groups. The rats were exposed to the RF field (MiRS signal, 860 MHz, 11.1 pulses per second) 6 h/day 5 days/week at a SAR of 1.0 W kg^{-1} averaged over the brain (0.27-0.42 W kg^{-1} averaged over the whole body). The animals were restrained during the exposures. An equal number of rats from each group were killed every 30 days between the ages of 171 and 325 days; 32 rats died and 225 rats were killed when they were moribund. All rats were necropsied and the brain and spinal cord were examined histopathologically. The examinations revealed 38 spinal cord tumors, 191 spinal nerve tumors, 232 cranial nerve tumors, and 823 brain tumors. No evidence was found of RF effects on the incidence, malignancy, volume, multiplicity, latency or fatality associated with any kind of neurogenic tumor. Body weight was higher in the cage control animals than in the other groups, which is most likely related to restraint of the exposed and sham-exposed animals.

Multiple tumors

Heikkinen et al (2006) evaluated possible effects of RF radiation on tumorigenesis induced by the mutagen and multisite carcinogen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) given in drinking water continuously during the experiment. Female Wistar rats were exposed to GSM 900 RF at 0.3 or 0.9 W kg^{-1}. The tumor profile in the MX-exposed animals resembled that reported earlier in MX-exposed female Wistar rats. RF radiation did not statistically significantly affect mortality or organ-specific incidence of any tumor type. The only statistically significant difference was an increase in the combined frequency of vascular tumors of the mesenteric lymph nodes in the high-RF group compared to the sham-RF group. However, comparison to cage-control animals suggested that this difference was due to an unusually low frequency of this type of tumors in the sham-RF group, rather than high frequency in the high-RF group.

Lymphoma

Exposure to continuous (frequency modulated) or pulsed (GSM modulation) 902 MHz RF radiation at 1.5 W kg^{-1} or 0.35 W kg^{-1} for 1.5 h/d on did not affect development of lymphomas, enhance development of other tumors or affect survival in female CBA/S mice irradiated with X-rays (Heikkinen et al 2001). The X-rays were delivered during the first three weeks of the study in three subdoses, and the exposures to RF radiation continued for 1.5 years. The only statistically significant differences in tumor incidences were decreased incidence of glandular polyps in the continuous wave group, and decreased incidence of a benign pheochromocytomas of adrenal glands in both RF field groups.

Mammary tumors

Several studies have investigated effects of RF field effects on mammary gland tumorigenesis induced by 7,12-dimethylbenz(a)anthracene (DMBA) in rodents. Although some indication of enhanced or decreased tumorigenesis was found in some experiments, these findings were not repeatable in other experiments by the same group, or in studies with similar design by different groups.

The study of Bartsch and co-workers (Bartsch et al 2002) differs from most other RF field studies published this far, in that the daily RF exposure time was long (nearly all time exposure). The study

involved exposures of freely moving animals (12 per cage) at low SARs levels (0.1 W kg^{-1} or below). The study consisted of three experiments each started exactly at the same time of the year on three consecutive years. The animals were exposed until "practically all animals had developed a macroscopic mammary tumor" and the last experiment was conducted in blinded fashion. In one of the three experiments median latency for the development of the first malignant tumor was statistically significantly extended in the RF field-exposed group. This finding was not supported by the two other experiments. The overall conclusion was that long-term exposure to RF radiation had no significant effect on the development of DMBA-induced mammary tumors in Spraque-Dawley rats.

The study of Anane et al (2003a) consisted of two separate experiments, one performed in spring-summer and another in autumn. Female Spraque-Dawley rats were administered DMBA ten days before the beginning of the RF field exposures. The group sizes were small (14-16 rats/group in each experiment). Both experiments included one common exposure level, 1.4 W kg^{-1}. In the first experiment, exposure at this SAR led to accelerated development of malignant mammary tumors, whereas an opposite finding was reported in the second experiment. The authors concluded that the study was no overall effect of exposure.

The study by Yu et al, (2006) did not provide evidence for RF field effects on the development of DMBA-initiated development of mammary tumors in rats. Exposure levels up to 4 W kg^{-1} were covered and 100 animals per group were used. The incidence of mammary gland adenocarcinomas was slightly lower in the group exposed to the lowest SAR, but the tumors were slightly larger compared to the animals exposed to DMBA only. A slightly enhanced development of adenocarcinomas was found at the highest SAR level. However, none of these differences were statistically significant. Significant differences were observed between the cage controls and the other experimental groups, with increased body weight and higher number and more rapid development of mammary tumors in the cage control group. These differences are most likely related to restraint of the sham-exposed and RF-exposed animals. The statistical analysis of tumor appearance was apparently done without making a distinction between tumors observed during the study by palpation and tumors detected in histopathological evaluation. While this could in principle mask differences between the groups (also small non-palpable tumors are detected in histopathology), the data shown in the paper suggest that a different statistical analysis would not essentially change the conclusion that RF radiation did not promote mammary tumor development.

Hruby et al (2008) used a study design similar to that used by Yu et al (2006). There were several statistically significant differences between RF field-exposed groups and the sham-exposed group. All RF-exposed groups had significantly more palpable mammary gland tissue masses than the sham-exposed group, but there were no differences between the three RF-exposed groups. The incidence of malignant mammary tissue tumors was lowest in the sham-exposed group, and significantly increased in the high exposure group. However, the incidence of benign tumors was significantly lower in the three RF exposed groups than in the sham-exposed group. The number of animals with benign or malign neoplasms was similar in the sham-exposed group and in the three RF-exposed groups. The cage control group had the highest incidence and malignancy of neoplasms among all groups. Given that the DMBA mammary tumor model is known to be prone to high variations in the results, the authors' interpretation was that the differences between the groups were co-incidental. Comparison to the results of the almost identical study of Yu et al (2006) supports this conclusion: both studies reported similar development of mammary tumors in three groups, but lower rate of development (seen in the appearance of palpable tumors and/or reduced malignancy) in one group. Hruby et al found the lowest rate of development in the sham-exposed group, while Yu et al found it in the 0.44 W kg^{-1} group. Both studies consistently reported highest incidence of tumors in the cage control group, which is most likely related to the different handling of the cage control animals (different stress level, differences in food intake).

Skin tumors

Szudzinski and co-workers reported significant acceleration of the development of benzo(a)pyrene (B(a)P)-induced tumors in mice irradiated with 2.45 GHz (CW) RF at about 6-8 W kg^{-1} (Szudzinski et al 1982). Exposure to both Ba(a)P and RF radiation were long-term (6 months). Enhanced development of

skin tumors was reported also if the RF exposure was for 1-3 months prior to the beginning of B(a)P exposures. Some of these results appear to have been reported in another publication (Szmigielski et al 1982) in the same year. There are some inconsistencies between these two reports (e.g. the group size and the exact handling of the sham-exposed animals) which complicate the interpretation of the results. The authors detected no increase of rectal temperature, but admitted that at the highest exposure level formation of significant "hot-spots" was possible due to non-uniform absorption of RF energy.

Low-level RF field exposures (only a few animals were exposed at 0.27 W kg^{-1}, the others at 0.075 W kg^{-1}) had no effects on tumor appearance or survival in B(a)P-treated female Sprague-Dawley rats (Chagnaud et al 1999). Similarly, the RF field exposures had no effects on the levels of anti-phosphatidylinositol auto-antibodies, a suggested marker of malignant transformation. The animals were exposed to RF radiation for two weeks beginning on day 20, 40 or 75 after B(a)P injection.

RF field exposures have not been observed to induce tumors in skin of DMBA-treated CD-1 mice (Imaida et al 2001) or ICR mice (Huang TQ et al 2005). In both studies mice were subjected to topical application of DMBA on dorsal skin a week before the beginning of RF field exposures. No skin tumors were observed either in sham-RF or RF-field-exposed animals during the 19-week-studies, or not even after a one-year follow-up (Huang TQ et al 2006), whereas a clear tumor response was observed in the positive control animals exposed to repeated topical treatment with the classical tumor promoter phorbol-12-myristate-13-acetate (PMA). RF field exposures did not affect either the epidermal thickness (Imaida et al 2001; Huang TQ et al 2005) or expression of proliferating cell nuclear antigen (Huang TQ et al 2005). No difference was observable in the incidence of lymphoma (Imaida et al 2001; only liver, kidney, adrenal glands and spleen evaluated for lymphomas), and RF field exposures did not affect serum hormonal levels (melatonin, adrenonocorticotrophic hormone (ACTH) or corticosterone) in samples collected at necropsy (Imaida et al 2001).

Heikkinen et al (2003) reported that daily exposure to pulsed 849 or 902 MHz RF with two modulations characteristics (GSM or DAMPS) did not significantly affect the development of skin tumors induced by UV radiation in female ODC-transgenic mice (K2) and in their non-transgenic littermates. Skin tumors were induced by exposure to solar-simulating UV radiation three times a week during the whole two-year study. The development of skin tumors was faster in RF field-exposed animals than in the control group exposed to UV radiation only. This was consistently seen with both RF signals and in both transgenic and non-transgenic animals, but did not reach statistical significance even in a combined analysis.

Colon tumors

Exposure to 2.45 GHz RF, even at relatively high SAR of 10 - 12 W kg^{-1}, did not affect the development of dimethylhydrazine (DMH)-induced colon tumors in Balb/c mice (Wu et al 1994). Although tumor incidence was not increased in animals treated with PMA as a positive control for tumor promotion, this treatment resulted in higher number of tumors per animal and larger tumors.

Medium-term hepatocarcinogenesis models

Exposure to 1.49 GHz (Imaida et al 1998a) or 929.2 MHz (Imaida et al 1998b) pulsed RF at 0.4-0.8 W kg^{-1} (maximum local values in liver 0.9-2.0 W kg^{-1}) did not promote hepatocarconigenesis in a rat medium term bioassay, in which rats were exposed to diethylnitrosamine (DEN), partially hepatectomised a few weeks later, and exposed to RF radiation for six weeks. Interestingly, the development of gluthathione S-transferase (GST-p) positive liver foci, a preneoplastic rat liver lesion used as an end-point marker in this assay, was slightly decreased in the RF field exposed animals in both studies, the difference being statistically significant at 1.49 GHz. Compared to unrestrained DEN-exposed control animals, the level was about the same in RF field exposed animals, but higher in sham RF field exposed animals. Serum levels of ACTH, corticosterone and melatonin were increased in RF-exposed animals at the end of the study.

Table II. 4.4.: Co-carcinogenic effects of RF radiation with known carcinogenic agents

Assay endpoint	Exposure conditions	Results	Comments	Reference
CNS Tumors				
CNS tumors in F-344 rats exposed to a single dose of *n*-ethyl-*N*-nitrosourea (ENU) transplacentally (CNS tumor incidence 17 % without RF exposure)	836.55 MHz D-AMPS 1) freely moving pregnant dams, SAR not given, 2 h/d , gestation day 19 until birth 2) freely moving pups, SAR not given, 2 h/d, from birth until weaning 3) restrained (carousel set-up,) from age of 33 d, brain SAR: 0.3–0.5 W kg^{-1} (whole-body SAR 0.2-0.4 W kg^{-1}), 2 h/d, for 22 months (intermittent exposure: 7.5 min on/ 7.5 min off)	Fewer CNS glial tumors in the exposed group; the difference was statistically significant only in a *post hoc* analysis restricted to preterm animals. No significant effects on survival	Group size 26-30 animals of each gender. Exposure was started *in utero*	Adey et al 1999
CNS tumors in F-344 rats exposed to a single dose of ENU transplacentally (CNS tumor incidence 22% without RF exposure)	836.55 MHz FM 1) freely moving pregnant dams, SAR not given, 2 h/d, gestation day 19 until birth 2) freely moving pups, SAR not given, 2h/d, from birth until weaning 3) restrained (carousel set-up) from age of 33 d, brain SAR: 1.1-1.4 W kg^{-1} (whole-body SAR 0.3-0.7 W kg^{-1}), 2 h/d , 4 d/w, for 23 months	No effects on development of brain/CNS tumors No effects on survival	Group size 38-52 animals of each gender. Exposure was started *in utero*	Adey et al 2000
CNS tumors in Sprague-Dawley rats exposed to a single dose of ENU transplacentally. (brain tumor incidence 10-16 % (low-ENU) and 58 % (high-ENU) without RF exposure)	860 MHz CW or 860 MHz MiRS Restrained (carousel set-up) Brain SAR: 0.8-1.2 W kg^{-1} (whole-body SAR: 0.27-0.42 W kg^{-1}) 6 h/d, 5d/wk for 22 months	No statistically significant effects on CNS/brain incidences No effects on overall tumor rate in other tissues (about 8 non-neural tissues; relatively high number of missing tissues in some of these)	30 females and 30 males/group	Zook and Simmens 2001

Assay endpoint	Exposure conditions	Results	Comments	Reference
CNS tumors in F-344 rats exposed to a single dose of ENU transplacentally. (brain tumor incidences 24 % in males and 30% in females without RF exposure)	1.439 GHz PDC Restrained (carousel set-up) Brain SAR: 0.67 W kg^{-1} or 2.0 W kg^{-1} (whole-body SAR <0.4 W kg^{-1}) 1.5 h/d, 5d/wk for 104 weeks	No statistically significant effects on CNS/brain tumor incidences Incidence of pituitary tumors decreased in males exposed at 2 W kg^{-1}, no other significant effects ("all organs" evaluated, tissues not listed). No effects on survival	50 females and 50 males/group	Shirai et al 2005
CNS tumors in Sprague-Dawley rats exposed to a single dose of ENU transplacentally. (brain tumor incidence 50% without RF exposure)	860 MHz MiRS Restrained (carousel set-up) Brain SAR: 0.8-1.2 W kg^{-1} (whole-body SAR: 0.27-0.42 W kg^{-1}) 6 h/d, 5d/wk for 39 weeks	No effects on incidence, malignancy, volume, multiplicity, latency or fatality of any kind of neurogenic tumor	540 females and 540 males/group Two ENU doses An equal number of rats were killed and examined at 30-d intervals	Zook and Simmens 2006
CNS tumors in F-344 rats exposed to a single dose of ENU transplacentally. (brain tumor incidences10 % in males and 8% in females without RF exposure)	1.95 GHz W-CDMA Restrained (carousel set-up) Brain SAR: 0.67 W kg^{-1} or 2.0 W kg^{-1} (whole-body SAR <0.5 W kg^{-1}) 1.5 h/d, 5d/wk for 104 weeks	No statistically significant effects on CNS/brain tumor incidence, although there was a tendency towards increased incidence in RF exposed groups compared to sham-exposed group	50 males and 50 females/group. The statistical methods used were not sensitive for detecting a trend with exposure level	Shirai et al 2007
Lymphomas				
Lymphoma in female CBA/S mice exposed to an initiating dose (consisting of three subdoses)of X-rays (Incidence of lymphoma 24% without RF exposure)	902.5 MHz NMT (analog) or 902.4 MHz GSM Restrained SAR: 1.5 W kg^{-1} (NMT) or 0.35 W kg^{-1} (GSM) 1.5 h/d, 5 d/wk for 78 weeks	No increase in lymphoma No increase in any primary neoplastic change	50 mice/group	Heikkinen et al 2001
Mammary tumors				
Mammary gland tumors in female Sprague-Dawley rats exposed to a single dose of DMBA (practically all animals developed tumors)	900 MHz GSM Freely moving (12/cage) SAR 0.03-0.13 W kg^{-1} (young) 0.015-0.06 (adult) 24 h/d, 7 d/wk for 9-11 months	Overall no effects on mammary gland tumorigenesis In one experiment, latency for developing first malignant mammary gland tumor was increased in RF- exposed rats, but not in two other experiments	Three experiments; 60 animals/group in each experiment. Response to DMBA varied between the experiments	Bartsch et al 2002

Assay endpoint	Exposure conditions	Results	Comments	Reference
Mammary gland tumors in female Sprague-Dawley rats exposed to a single dose of 7,12-dimethylbenz[*a*]anthracene (DMBA) (Incidence of malignant mammary tumors 60 % without RF exposure)	900 MHz GSM Restrained SAR: 1.4, 2.2 or 3.5 W kg^{-1} (Experiment I) or 0.1, 0.7 or 1.4 W kg^{-1} (Experiment II) 2 h/d, 5d/wk for 9wk	Incidence of malignant tumors increased at 1.4 and 2.2 W kg^{-1} in Exp I, but decreased at 1.4 W kg^{-1} in Exp II. Less tumors/animals in rats exposed at 1.4 W kg^{-1} in Exp II. The authors concluded that, there were no overall effects.	Two experiments;16 animals/group in each experiment	Anane et al 2003a
Mammary gland tumors in female Sprague-Dawley rats exposed to a single dose of DMBA (Mammary tumor incidence 45% without RF exposure)	900 MHz GSM Restrained SAR: 0.44, 1.33 or 4 W kg^{-1} 4 h/d, 5 d/wk for 26 wk	No significant effects on mammary gland tumorigenesis	100 animals/group	Yu et al 2006
Mammary gland tumors in female Sprague-Dawley rats exposed to a single dose of DMBA (Mammary tumor incidence 60% without RF exposure)	902 MHz GSM Restrained SAR: 0.4, 1.3 or 0.4 W kg^{-1} 4 h/d, 5 d/wk for 6 months	All RF-exposed groups had, at different times, significantly more palpable tissue masses compared to the sham-exposed. However, the cage-control group had significantly more palpable masses, benign and malignant tumors compared to the sham-exposed group.	100 animals/group	Hruby et al 2008
Skin tumors				
Skin tumors in male Balb/c mice exposed to repeated doses of Benzo(*a*)pyrene (BaP) after or simultaneously with RF field exposure (Skin tumor incidence 50% without RF exposure)	2.45 GHz CW Freely moving (10/cage) 50 W/m^2 (SAR: 2–3 W kg^{-1}) or 150 W/m^2 (6–8 W kg^{-1}) 2 h/d, 6d/wk, Experiment I: for 1 or 3 months prior to BaP; Experiment II: for 5 months simultaneously with BaP	RF exposures (prior to and simultaneously with BP treatments) accelerated the development of skin tumors and decreased survival	40 mice/group Difficult to interpret: methods are not described in detail. Large uncertainty in estimated SAR	Szmigielski et al 1982

Assay endpoint	Exposure conditions	Results	Comments	Reference
Skin tumors in male Balb/c mice exposed to repeated doses of BaP after or simultaneously with RF field exposure (Skin tumor incidence 95% without RF exposure)	2.45 GHz CW Freely moving or slightly restrained (10/cage) 50 W/m^2 (SAR: 2–3 W kg^{-1}) or 150 W/m^2 (6–8 W kg^{-1}) for 6 months simultaneously with BaP; or 100 W/ m^2 (4-6 W kg^{-1}) for 1, 2 or 3 months prior to BaP 2h/d, 6d/wk	Accelerated development of skin tumors, statistically significant at 150 W/m^2). Also pre-irradiation accelerated tumor development. Increased mortality	100 animals/group The results seem to be partly the same as those reported in Szmigielski et al 1982. However, there are discrepancies in the methods described	Szudzinski et al 1982
Skin tumors in female Sprague-Dawley rats exposed to a single dose of BaP; RF exposure was started 20, 40 or 75 days later. (All animals developed a malignant sarcoma at the site of injection)	900 MHz GSM Restrained SAR: 0.075 ±0.025 W kg^{-1} (a few rats exposed at 0.27 W kg^{-1}) 2 h/d, 5d/wk for 2 wk	No effects on tumor appearance/onset No effects on survival with tumor No effects on anti-phosphatidylinositol auto-antibodies (a suggested marker of malignant transformation)	8 – 18 rats/group	Chagnaud et al 1999
Skin tumors in female ICR-1 mice exposed to a single dose of DMBA (no macroscopic skin tumors without RF exposure)	1.49 GHz PDC Restrained Skin SAR: 0.67 W kg^{-1} (whole-body SAR: 0.028 W kg^{-1}) 1.5 h/d, 5 d/wk for 19 weeks	No effects on skin tumor development (no skin tumors in RF field exposed group). No differences in incidence of lymphoma (only a few tissues evaluated for lymphoma) No effects on serum hormone levels (melatonin, corticosterone, ACTH) No effects on the thickness of epidermis	48 animals /group PMA was used as a positive control for tumor promotion; a clear response to PMA was observed	Imaida et al 2001
Skin tumors in female SENCAR mice exposed to a single dose of DMBA with or without repeated PMA treatment (Incidences of skin tumors 0 % and over 80 % in DMBA and DMBA+PMA treated groups, respectively)	94 GHz CW 1) anesthetized 10 000 W/m^2, for 10 s Temperature increase in skin 13-15 °C 2) restrained 3 330 W/m^2, 10 s/d, 2d/wk, for 12 wk Temperature increase 4 - 5 °C	RF field exposures did not promote or co-promote DMBA-induced skin-tumorigenesis No effects on expression of early biomarkers of tumor-promoting activity (epidermal thickness, 5-bromodeoxyuridine incorporation, ODC activity)	50-55 animals /group	Mason et al 2001

Assay endpoint	Exposure conditions	Results	Comments	Reference
Skin tumors in female transgenic K2 mice and their non-transgenic littermates exposed to repeated doses of UV radiation. (Incidence of skin tumors 22% without RF exposure)	902 MHz GSM or 824 MHz DAMPS Restrained SAR: 0.5 W kg^{-1} 1.5 h/d, 5 d/wk for 52 weeks	No statistically significant increase in skin tumors (although tumor development was faster in both RF field exposed groups compared to the UV only group)	45-49 animals/group K2 mice are more prone to develop skin tumors than their normal counterparts	Heikkinen et al 2003
Skin tumors in male ICR mice exposed to single dose of DMBA (no skin tumors without RF exposure)	849 MHz CDMA or 1.763 GHz CDMA Freely moving SAR: 0.4 W kg^{-1} 2 x 45 min/d, 5 d/wk for 19 wk	No effects on tumor development (no skin tumors in RF-exposed groups). This was confirmed in a replication study with longer follow-up (1 year) No effects on epidermal thickness Staining with anti-proliferating cell nuclear antigen (PCNA) was observed only in the positive control group Cyclin D1 and c-fos proteins were detected only in the skin of the positive controls.	20 animals/group PMA was used as a positive control for tumor promotion; a clear response to PMA was observed	Huang TQ et al 2005
Colon Tumors				
Colon tumors in BALB/c mice exposed to repeated injections of Dimethylhydrazine (DMH) (colon tumor incidence 46 % without RF exposure)	2.45 GHz CW Restrained SAR: 10–12 W kg^{-1} 3 h/d, 6d/wk, for 5 months	No effects on colon tumorigenesis	26-32 animals /group Positive control (PMA) did not increase tumor incidence, but it accelerated tumor development	Wu et al 1994
Multiple Tumors				
Several tumor types in female Wistar rats exposed to 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX) continuously in drinking water (Proportion of animals with malignant tumors 51% without RF exposure)	RF 900 MHz GSM Freely moving (1/cage) SAR: 0.3 or 0.9 W kg^{-1} 2 h/d, 5 d/wk for 104 weeks	No increase in the incidence of any primary neoplasm	72 animals/group	Heikkinen et al 2006

Assay endpoint	Exposure conditions	Results	Comments	Reference
Liver promotion model				
Liver, medium-term promotion model: male F-344 rats exposed to a single dose of DEN and partial hepatectomy 3 wk later	1.439 GHz PDC Restrained Liver SAR 0.9-1.9 W kg^{-1} (peak values), whole-body SAR: 0.45-0.68 W kg^{-1} 1.5 h/d, 5 d/wk for 6 wk	Number of GST-P positive foci decreased Spleen and testis weights decreased; adrenal weight increased Serum levels of ACTH, corticosterone and melatonin increased	45-47 rats/group	Imaida et al 1998a
Liver, medium-term promotion model: male F-344 rats exposed to a single dose of Diethylnitrosamine (DEN) and partial hepatectomy 3 wk later	929.2 MHz PDC Restrained Liver SAR: 1.7-2.0 W kg^{-1} (local peak values), whole-body SAR: 0.6–0.8 W kg^{-1}, 1.5 h/d, 5 d/wk for 6 wk	No significant effects on the development of GST-P positive foci Serum levels of ACTH, corticosterone and melatonin were increased in RF-field-exposed group	47-48 rats/group	Imaida et al 1998b

II.4.2.3. Effects of RF radiation on tumor transplantation

Szmigielski et al, (1982) exposed BALB/c mice injected with sarcoma cells to 2.45 GHz RF for 1, 2 or 3 months at 50 or 150 W m^{-2}. Significantly elevated numbers of neoplastic lung foci nodules after both 1 and 3 months were reported at both RF exposure levels. The interpretation of this study is complicated by the fact that methods were not described in detail and by uncertainties in dosimetry. Thermal effects are possible at least at the higher exposure level.

Three studies using inoculated/implanted rat glioma cell lines (Salford et al 1993; Salford et al 1997) and gliosarcoma cells (Higashikubo et al 1999) in Fischer 344 rats did not find effects on brain tumor growth from exposure to 835 – 915 MHz RF using several different modulations and SAR levels from 0.008 to 1.67 W kg^{-1}.

Santini and co-workers (Santini et al 1988) exposed C57/6J mice to continuous and pulsed 2.45 GHz RF field at 1.2 W kg^{-1}. After 15 days of exposure animals were subcutaneously implanted with B16 melanoma cells. The results did not indicate significant effects of RF either on tumor development or on survival times.

Four daily 20-min RF field exposures of pregnant dams at thermal exposure levels resulted in retarded development of inoculated sarcomas in offspring of CFW mice, but did not affect the final incidences (Preskorn et al 1978). Mice exposed *in utero* lived slightly longer. Postnatal exposures treatments did not affect tumor development in this study. Retarded tumor development was also reported in studies involving short-term exposure of the nasal area of mice to 42-61 GHz RF at very high intensities causing temperature elevation (Radzievsky et al 2004; Logani et al 2004; 2006).

Table II.4.5.: Effects of RF radiation on transplanted/injected tumor cells

Assay endpoint	Exposure conditions	Results	Comments	Reference
Homogenate of sarcomatous tumors (Experiment I) or virus homogenate (Experiment II) subcutaneously in CWF mice (Half of the animals developed tumors)	2.45 GHz, 60-Hz amplitude modulation 35 W kg^{-1}, 20 min/d, *in utero* on gestation days 11-14 and/or postnatally on days 19-54	Exposure *in utero* retarded tumor development in offspring, but did not affect the final incidences. Some indication of increased survival and tumor regression related to fetal exposure (Exp. 2) Postnatal exposure did not affect tumor development	24 males and 24 females/group (Exp. I); 60-84/ group (Exp. II) Hyperthermic exposure; colonic temperature of dams increased by 2 °C	Preskorn et al 1978
L_1 mouse sarcoma cells intravenously in BALB/c mice	2.45 GHz CW Freely moving 50 W m^{-2} or 150 W m^{-2} (SAR: ≈ 2–3 or 6–8 W kg^{-1}) 6 d/wk, for 1, 2 or 3 months	Increased incidence of metastatic tumor colonies on lung surface, more pronounced at the higher exposure level.	Difficult to interpret because methods are not described in detail, group size unknown	Szmigielski et al 1982
B16 melanoma cells Subcutaneously in C57BL/6J mice (average survival with tumor below 4 wk)	2.45 GHz CW or pulsed (10 ms bursts of 10 µs pulses, 25 bursts/s) Freely moving SAR: 1.2 W kg^{-1} 2.5 h/d, 6 d/w	No effects on tumor development No effects on survival	15 animals /group	Santini et al 1988
RG2 rat glioma cells injected into brain in F-344 rats (All animals developed brain tumors)	915 MHz CW or pulsed (4, 8, 16, 50 or 217 pulses/s) Restrained SAR: 0.008–1.67 W kg^{-1} depending on pulse frequency 4+3 h/d, 5 d/wk for 2–3 wk	No effects on tumor growth	37animals/group (4 to 11 animals for each RF signal; pooled for statistical analysis)	Salford et al 1993
RG2 or N32 rat glioma cells injected into brain in F-344 rats (All animals developed brain tumors)	915 MHz CW or pulsed (4, 8, 16, 50 or 217 pulses/s) Restrained SAR: 0.008–1.67 W kg^{-1} depending on pulse frequency 4+3 h/d, 5 d/wk for 2–3 wk	No effects on tumor growth	4 - 29 for each RF signal (total 45 rats with N32; 94 – 109 rats with RG2)	Salford et al 1997

Assay endpoint	Exposure conditions	Results	Comments	Reference
9L gliosarcoma cells injected into brain in male F-344 rats	835.62 MHz FM or 847.74 MHz CDMA Restrained (carousel set-up) Brain SAR: 0.5-1.0 W kg^{-1} 4 h/d, 5 d/wk, for 4 +21 wk	No effects on tumor growth or survival. Brain weight increased in rats that were implanted with the highest number of viable cells and exposed to the CDMA signal. The authors concluded that this was more likely related to irregularities in sham-exposed group than to RF field exposure	96-101 animals/group (3 subgroups according to the number of viable cells injected: 10-67 rats/subgroup)	Higashikubo et al 1999
B16F10 melanoma cells subcutaneously in male Swiss Webster mice	61.22 GHz, Restrained 130 W/m^2, on the nasal area (max. temperature increase at the tip of the nose ≈ 1 ° C) 15 min/d for 5 d	Reduced tumor growth, if RF exposures started on day 5. No significant effects, if RF exposures started on day 1 or on day 10 (slightly enhanced tumor growth) Pre-treatment with Naloxane (a non-specific blocker of opioid receptors) blocked the effect	>10 mice/group	Radzievsky et al 2004
B16F10 melanoma cells subcutaneously in female SKH1 hairless mice CPA treatments on days 4-8 (Experiments I-III) or on days 4 and 11 (Experiment IV)	42.2 GHz (60-Hz amplitude modulation) Restrained 185 W m^{-2} on the nasal area (peak skin SAR: 730 W kg^{-1}, temperature rise 1.5 ºC) 30min/d, for 3-6 d	RF field exposures either concurrently with, prior to or following CPA treatments did not affect tumor development	10 mice/group	Logani et al 2004
B16F10 melanoma cells Intravenously in female C57BL/6 mice, Cyclophospamide (CPA) before RF exposure	42.2 GHz (60-Hz amplitude modulation) Anesthetized 185 W m^{-2} on the nasal area (peak skin SAR: 730 W kg^{-1}, temperature rise 1.5 ºC) One 30 min exposure.	Decreased number of metastatic lung colonies in animals exposed to RF field alone and in combined exposure with CPA Increased activity of natural killer cells in RF+CPA combined exposure	10 mice/group	Logani et al 2006

II.4.2.4. Summary on cancer

The possible carcinogenicity of RF field exposure has been investigated in a number of experimental models including classical rodent bioassays, studies using genetically predisposed animals, co-carcinogenicity studies involving combined exposure to RF and known carcinogens, and studies evaluating effects on the development of tumors after transplantation of tumor cells. With only a few exceptions, these studies have provided no evidence of carcinogenic effects. Positive findings were reported in some early studies, but these studies are of limited value because of shortcomings in methodology and reporting. A notable positive finding was a two-fold increase in lymphoma incidence in

a strain of lymphoma-prone transgenic mice following exposure at 900 MHz with a signal similar to that used in GSM mobile phones. However, this finding has not been supported by two subsequent confirmation and extension studies. Recent studies have generally been of high quality and have consistently reported lack of carcinogenic effects in a variety of animal models. This includes several studies involving *in utero* and postnatal exposures. Overall, the results of these studies are rather consistent and indicate that carcinogenic effects on rodents are not likely at SAR levels up to 4 W kg^{-1}.

II.4.3. Reproduction and development

Reproductive and developmental effects of RF radiation were reviewed by WHO (1993) and more recently by eg, Verschaeve and Maes (1998), O'Connor (1999), IEGMP (2000), Heynick and Merrit (2003), AGNIR (2003), Juutilainen (2005) and Marino et al (2006). The conclusions of these reviews are rather similar, indicating that there is a consensus in the scientific community regarding the interpretation of experimental results on reproductive and developmental effects. Extensive research on a wide range of species has consistently shown effects at exposure levels causing significant temperature increase in tissues, but no effects have been established at non-thermal exposure levels. The present review focuses on studies published after 1992, but some earlier key studies are also included. The review covers classical teratological endpoints such as malformations and fetal loss, postnatal effects of prenatal exposure, and effects on reproduction.

The IEGMP report (IEGMP 2000) focused particularly on possible effects of low level RF radiation on children, particularly in connection with possible effects on the developing nervous system of RF radiation resulting from the use of mobile phone technologies, and recommended further research on this subject. Development after birth largely entails the maturation of existing organ systems (Kheifets et al 2005). With some particular exceptions, most adult neurons are already produced by birth. Two important neurological events that occur postnatally include changes in the number of synapses and increased neuronal myelination, which facilitates the transmission of information within the brain. One recent animal study of the effects of juvenile exposure on subsequent brain histology and the performance of a number of behavioral tests is reviewed.

Studies on avian and other non-mammalian species are also reviewed, although there are fundamental problems in extrapolating such data to mammals, and their relevance to assessment of human health risks is limited. Non-mammalian models are useful for investigating basic mechanisms and as screening tests to detect potential risks that should be studied in mammals or humans.

II.4.3.1. Reproduction

Male fertility has long been recognized as susceptible to heat (AGNIR 2003). Testicular temperature in mammals is normally clearly below that of the rest of the body, and the development of male germ cells can be adversely affected by increased temperatures. Exposure of anaesthetized rodents to RF radiation that elevate testicular or body temperatures can cause depletion of the spermatogenic epithelium and decreased fertility (Gunn et al 1961; Muraca et al 1976; Saunders and Kowalczuk 1981; Kowalczuk et al 1983; Lebovitz et al 1987). In contrast, exposure of conscious animals has resulted in little or no significant effects, except after long exposures at thermally stressful levels (Lebovitz and Johnson 1983, 1987; Johnson et al 1984; Cairnie and Harding 1981; Saunders et al 1988; Berman et al 1980). This difference is most likely explained by the fact that anesthesics impair the regulation of body temperature.

In a small study, Magras and Xenos (1997) reported that exposure to extremely-low-level RF near an antenna park (almost 100 TV and radio antennas), situated at an altitude of ~ 750 m in Northern Greece, produced an apparently dramatic drop in fertility in mice. Twelve male and female mice were caged in various outdoor and indoor locations close to the antenna park. Exposures ranged between 1.7 and 10 mW m^{-2}. After five matings between May and December, the litter size was very small compared to animals living in a microwave-free environment in a laboratory. Unfortunately there was no matched control group, so the result may be due to environmental differences that are unrelated to RF levels.

Akdağ et al (1999) reported that the epididymal sperm count decreased and the percentage of abnormal sperm increased in rats chronically exposed to 9.45 GHz CW RF at a whole-body SAR of about 2 W kg^{-1}. Testis SAR was not given; because of the high frequency, absorption of power is superficial, so temperature increase of the testis may have occurred because of high local SAR. The same group reported that only the seminiferous tubule diameter in rat testes was decreased after one month of 3-min daily exposures at 890-915 MHz at about 0.14 W kg^{-1} from a GSM phone (Daşdağ et al 1999b). However, in a subsequent study carried out to explore these results more thoroughly, longer (20 min) daily exposures to pulsed 800-915 MHz GSM-type signals at 0.5 W kg^{-1} or less had no effect on testicular structure or function (Daşdağ et al 2003). The effects of a 4-week exposure (30 min per day) to 900 MHz CW radiation on testicular morphology were investigated in a small study by Ozguner et al (2005). Significant reductions were observed in the diameter of the seminiferous tubules, in the mean height of the germinal epithelium and in serum testosterone levels. Unfortunately, the dosimetry of the animals was not adequately characterized; SAR values were not given.

II.4.3.2. Development

Effects on non-mammalian species

Several studies have been performed to investigate the effects of 2.45 GHz RF exposure on Japanese quail embryos. The estimated SAR ranged from 3.2 to 25 W kg^{-1}. The results were consistent: no significant effects on hatchability, hatchling weights, viability, or incidence of abnormalities were seen unless the exposure levels were high enough to raise the egg temperatures by a few degrees (see WHO 1993; Heynick and Merritt 2003). The only study that has reported effects on bird embryo development at non-thermal exposure levels was published by Saito et al (1991), who exposed chicken eggs continuously at 428 MHz at low SAR levels (3.1 to 47 mW kg^{-1} in the exposure area). The findings included decreased hatching, increased mortality and functional abnormality in hatched chickens of the exposed group. The interpretation of these results is difficult due to uncertainties in dosimetry and the fact that the exposed and control eggs were not incubated simultaneously.

Weisbrot et al (2003) exposed fruit flies (*Drosophila melanogaster*) to GSM multiband (900 and 1900 MHz) mobile phones for 2 h per day during the 10-day developmental period from egg laying to pupation. An increased number of offspring was reported together with increases in stress protein hsp70 level, serum response element (SRE) DNA binding and phosphorylation of the nuclear transcription factor ELK-1. The results are difficult to interpret because of a lack of RF dosimetry.

Effects on mammals

Effects on prenatal development

Numerous studies have shown that RF radiation can cause increased embryo and fetal losses, increased incidence of fetal malformations and anomalies and reduced fetal weight at term, if the SAR level is high enough to raise the maternal body temperature considerably (for detailed review, see WHO 1993; Heynick and Merritt 2003; Juutilainen 2005). The threshold temperature rise for teratogenic effects varies with timing and duration of exposure. The lowest observed thresholds in maternal temperature increase (in experiments with long-term exposure) have been around 1-2°C, which is consistent with the lowest thresholds for effects from hyperthermia induced by other forms of heating (Edwards et al 2003).

In general, no effects have been found at non-thermal exposure levels, even with exposures that lasted for the whole gestation or continued during the postnatal period. The only exception is the study by Tofani et al (1986) who reported increased post-implantation losses in pregnant rats exposed at 27.12 MHz at a very low exposure level. The interpretation of this study is difficult because the increase of post-implantation loss is completely explained by the high percentage (50%) of total resorptions among the exposed dams. Among dams with viable fetuses, no effects were seen on pre- or post-implantation losses, number of viable fetuses or fetal weight. As there is no obvious reason why RF exposure would increase embryonal death only in some of the dams, it remains possible that the increased total resorptions are explained by environmental conditions other than RF field exposure. The exposed and sham-exposed

animals were not kept in the same room. Other significant findings reported were reduced body weight of dams exposed on days 0-20 of gestation and incomplete ossification of cranial bones of the exposed fetuses.

Exposure to high level RF radiation has also been reported to enhance the effects of chemical teratogens (Marcickiewicz et al 1986; Nelson et al 1991) or ionizing radiation (Roux et al 1986). These effects are most likely due to the rise in fetal temperature. It has been shown that hyperthermia combined with chemical agents such as arsenic, vitamin A, lead and ethanol is more effective in causing developmental effects than when administered alone (Edwards et al 2003).

Klug et al (1997) exposed rat embryos *in vitro* for up to 36 h to 150 MHz RF modulated at 16, 60 or 120 Hz. The electric field strengths were 60 and 600 V m^{-1}, and the magnetic flux densities were 0.2 and 2 μT. Experiments were also carried out using 900 MHz RF modulated at 217 Hz, at SAR levels of 0.2, 1 and 5 W kg^{-1}. No significant effects were observed on the growth and differentiation of the embryos.

Behavioral teratology

Prenatal exposure of animals and subsequent assessment of postnatal neural or behavioral effects can be considered as one of the most sensitive systems for investigating possible toxic effects.

Some of the early studies reviewed by WHO (1993) and Heynick and Merritt (2003) and Juutilainen (2005) included also assessment of postnatal behavioral effects. The results of these studies (Kaplan et al 1982; Jensh et al 1983b, 1984b) indicate that RF exposure does not cause any consistent effects on behavioral endpoints in the absence of hyperthermia.

One early study using primates merits discussion. Kaplan et al (1982) exposed 33 squirrel monkeys during the second trimester of pregnancy for 3 h/day at 2.45 GHz at whole body SAR of 0.034, 0.34, or 3.4 W kg^{-1}. Some of the offspring were additionally exposed for 18 months postnatally. No significant differences were seen in EEG or the behavioral endpoints tested (righting, orienting, climbing down, climbing up, directed locomotion). However, because of the small group sizes, these results have very limited statistical power.

A series of post-natal studies following prenatal exposure carried out by Jensh et al (1982b, 1983b) and Jensh (1984b, 1997), were more equivocal. These reported a number of minor behavioral changes, such as reduced water T-maze performance by females but not males, in the offspring of rats exposed throughout pregnancy to 6.0 GHz at a whole-body SAR estimated to be about 7 W kg^{-1}. No effects were seen in the offspring of rats similarly exposed to 2.45 GHz or 915 MHz at whole body SARs estimated to be about 2-4 W kg^{-1} (Jensh 1997). Although an SAR of 7 W kg^{-1} is usually thermogenic in rats under normal laboratory conditions, the author stated that maternal body temperature was not elevated by exposure to RF radiation in any of these studies.

Bornhausen and Scheingraber (2000) exposed rats to GSM 900 signals continuously during pregnancy. The power density was 1 W m^{-2}, corresponding to a typical level of GSM base station near the antenna. The estimated SAR was 17.5-75 mW kg^{-1}. The offspring were tested at 3 months of age using nine tests of operant behavior performance using tasks with differing levels of complexity. No performance deficits were observed in the exposed animals.

Cobb et al (2000) exposed pregnant rats to ultra-wideband (UWB) pulses (55 kV m^{-1} peak, 1.8 ns pulse width, 300 ps risetime, 1000 pulses s^{-1}, 0.1-1 GHz, SAR 0.45 mW kg^{-1}). The exposure was 2 min per day during gestation days 3-18, and was continued during 10 postnatal days for part of the animals. Lead acetate was used as a positive control. No changes were found in 39 of the 42 endpoints. The authors concluded that there was no unifying physiological or behavioral relationship among the differences observed (more stress vocalization, longer medial-to-lateral length of the hippocampus, less frequent mating in exposed males but no difference in fertility). The positive control, in contrast, caused significant effects in numerous endpoints.

Postnatal exposure

Only a few studies have addressed possible effects of long-term exposure during the development of juvenile animals. In one of the behavioral studies, the RF exposure started during pregnancy was continued also during 10 postnatal days. The results of this study (Cobb et al 2000) are described above.

Exposure to 112 MHz RF amplitude-modulated at 16 Hz was reported to affect calcium-dependent protein kinase C (PKC) activity in developing rat brain (Paulraj and Behari 2004). Thirty-five days old male Wistar rats were exposed 2 h per day for 35 days at a power density of 10 W m^{-2} (estimated SAR 1.5 W kg^{-1}). A significant decrease in PKC level was observed in the exposed group as compared to the sham-exposed group, particularly in the hippocampus. In a later study with similar design (Paulraj and Behari 2006), also 2.45 GHz RF at 3.44 W m^{-2} (estimated SAR 0.1 W kg^{-1}) was reported to affect hippocampal and whole brain PKC activity. Electron microscopic examination also showed an increase in the glial cell population in the exposed group as compared to the sham-exposed group. While these results suggest that long-term exposure to RF radiation might affect brain development, the small study size (6 or 8 animals per group) precludes any firm conclusions.

A few studies have investigated whether exposure to RF fields affects the permeability of blood brain barrier in neonatal and juvenile animals (Salford et al 2003; Kuribayashi et al 2005; Finnie et al 2006c). As described in Chapter II.4.4.2., no evidence of consistent field-dependent effects has been found.

Kumlin et al (2007) investigated the effects of prolonged GSM 900 RF on the developing central nervous system. Young (3 week old) rats were exposed or sham-exposed at average whole-body SARs of 0.3 or 3.0 W kg^{-1} for 2 h per day, 5 days per week for 5 weeks. A variety of behavioral tests were carried out following exposure and brain tissue histology was examined. The immunohistochemical assays did not reveal any significant changes in brain tissue, and the results did not support the observations of Salford et al (2003) of increased dying neurons and leakage of the blood-brain barrier following a single RF exposure. No effects were seen on the performance in the open-field test, the elevated plus maze test or the acoustic startle response test. However, the authors did find an improved task acquisition and retention among the exposed animals in the water maze task, a test of spatial and working memory.

Table II.4.6: Reproductive and developmental effects

Assay endpoint	Exposure Conditions	Response	Comment	References
Testicular function				
Testicular structure and function, sperm count in Sprague-Dawley rats	9.45 GHz CW at a whole body SAR of 1.8 W kg^{-1} for 1 h per day for 13, 26, 39 or 52 days	Reduced epididymal sperm count; increased abnormal sperm	Possible heating of the testis?	Akdağ et al 1999
Testicular structure and function, sperm count in Sprague-Dawley rats	890-915 MHz pulsed (GSM) 0.6 ms pulses at 217 pps for 3 min per day for 1 month at a whole body SAR of 0.14 W kg^{-1}	Seminiferous tubule diameter significantly reduced		Daşdağ et al 1999b
Testicular structure and function, sperm count in Sprague-Dawley rats	890-915 MHz pulsed (GSM) 0.6 ms pulses at 217 pps for 20 min per day for 1 month at a whole body SAR of 0.5 W kg^{-1} or less	No effects		Daşdağ et al 2003

Assay endpoint	Exposure Conditions	Response	Comment	References
Reproductive outcome				
Litter number and size in BALB/c/f mice	RF radiation from an 'antenna park' over a five month period at various outdoor and indoor locations	Reduced number and size of litters compared to laboratory controls	Lack of matched controls	Magras and Xenos 1997
Behavioral teratology following prenatal exposure				
Pregnant rats exposed to RF	915 MHz CW at a whole body SAR estimated to be 3-4 W kg^{-1} of for 6 h per day throughout gestation	No effect on post-natal and adult behavior		Jensh et al 1982b; Jensh 1997
Pregnant rats exposed to RF	2.45 GHz CW at a whole body SAR estimated to be about 2-4 W kg^{-1} of for 6 h per day throughout gestation	No effect on most tests of behavior; exposed offspring more active in an open field test.		Jensh et al 1983b; Jensh 1997
Pregnant rats exposed to RF	6.0 GHz CW at a whole body SAR estimated to be about 7 W kg^{-1} of for 6 h per day throughout gestation	Exposed female offspring showed decreased learning in water T-maze test and decreased activity levels, whereas males showed increased activity levels.		Jensh 1984b, 1997
Exposure of pregnant rats throughout gestation	900 MHz pulsed (GSM) 0.577 pulses at 217 pps; at whole body SAR of between 0.0175 and 0.075 W kg-1 continuously from day 1 to day 20 gestation	No effect on operant task performance.		Bornhausen and Scheingrabe r (2000)
Exposure of pregnant rats and postnatal exposure of the offspring	UWB (dominant frequency range 0.1-1 GHz) pulses of 1.8 ns pulse width at 1000 pps; Two min per day at average whole body SAR of 0.045 W kg^{-1} during days 3-18, or during this period and to postnatal day 10.	No statistically significant effects except more stress vocalization, longer hippocampus and lower mating frequency in exposed offspring.		Cobb et al 2000
Postnatal development				
Exposure of young (~ 5 week old) rats for a further 5 weeks	112 MHz RF amplitude modulated at 16 Hz; whole body SAR estimated as1.5 W kg^{-1}; 2 h per day for 35 days	A significant decrease in PKC level was observed in the exposed group as compared to the sham exposed group, particularly in the hippocampus.		Paulraj and Behari 2004

Assay endpoint	Exposure Conditions	Response	Comment	References
Exposure of young (~ 5 week old) rats for a further 5 weeks	2.45 GHz RF; whole body SAR estimated as 0.1 W kg^{-1}; 2 h per day for 35 days	RF exposed animal showed a significant decreased in PKC activity in the hippocampus compared to the rest of the brain	Small group size (n=6)	Paulraj and Behari 2006
Exposure of young (~ 3 week old) rats for a further 5 weeks.	900 MHz pulsed (GSM) 0.577 pulses at 217 pps; at average whole body SARs of 0.3 W kg^{-1} or 3 W kg^{-1} for 2 h per day, 5 days per week until 8 weeks of age.	RF-exposed animals showed significantly improved performance of a water maze task compared to those sham-exposed.	No effect of RF on the performance of open-field test, plus maze test or acoustic startle response.	Kumlin et al 2007

II.4.3.3. Summary on reproduction and development

Numerous studies have evaluated developmental and reproductive effects of RF exposure on mammals and birds. These studies have clearly shown that RF exposure can cause increased embryo and fetal losses, increased incidence of fetal malformations and anomalies, reduced fetal weight at term and impair male fertility at exposure levels that are sufficiently high to cause significant increase of temperature. There is no consistent evidence of adverse effects at non-thermal exposure levels. Relatively few studies have evaluated possible effects of prenatal exposure on postnatal development; results from such studies have not shown consistent effects on developmental indices or behavior at exposure levels that do not induce hyperthermia. The possibility of effects resulting from long-term RF exposure during the development of neonatal and juvenile animals has been addressed in only a few studies. Some effects on brain development have been reported, but additional experiments would be needed to confirm their reproducibility and to understand their biological significance.

II.4.4. Nervous system

The brain and nervous system have long been considered sensitive targets for the effects of low-level modulated RF exposure following the work of Adey, Blackman and others in the late 1970s and 1980s on the effects of such radiation on the efflux of calcium ions from isolated brain tissue (See Chapter II.3.3.1.1.). The possible effects of exposure on the brain and behavior have been approached in animals using a range of methods and techniques from changes in specific gene expression in cells to investigations of changes in learned behaviors. These studies have been reviewed by WHO (1993), Hermann and Hossman (1997), Pakhomov et al (1998), D'Andrea (1999, 2003a, 2003b), McKinlay et al (2004) and Sienkiewicz et al (2005). Effects on learning and memory have also been considered by Lai (1992, 2001). The focus of this review is on studies published after the WHO (1993) review.

II.4.4.1. Gene expression

A few studies have investigated if the induction of stress-related genes and their proteins increase following exposure to RF radiation. These genes respond to various insults, such as ischemia or hyperthermia, and help to minimize potential damage. As part of a larger behavioral study, Mickley et al (1994) exposed rats at 600 MHz at 9.3 W kg^{-1} and measured increased c-fos protein expression in various areas of the forebrain, especially in cortical and periventricular areas. These changes were blocked by an opioid antagonist and were considered consistent with opioid-mediated stress. In another study, rats exposed to high-peak-power ultrawideband (UWB) pulses (0.25–2.5 GHz) at a peak electric field of 250

kV m^{-1} for 2 minutes did not show any changes in the expression of c-fos protein levels (Walters et al 1995). Body temperatures of the animals in this study rose by less than 0.5°C.

Fritze et al (1997a) exposed the heads of rats to simulated GSM signals (890–915 MHz pulsed at 217 Hz) at 7.5 W kg^{-1} and measured changes in the messenger RNAs of *hsp70*, *c-fos*, *c-jun*, and *gfap* using *in situ* hybridization histochemistry. Only changes consistent with brain hyperthermia or immobilization stress were found either immediately or 24 hours after exposure. Seven days after exposure, no changes were observed in the levels of the relevant proteins. Similarly, Morrissey et al (1999) reported that local exposure of the heads of mice to a 1.6 GHz Iridium satellite phone signal (pulse modulated at 11 Hz with a duty cycle of 4:1 and a pulse duration of 9.2 ms) for 1 hour only significantly increased *c-fos* mRNA expression in the forebrain when the average SAR in the brain exceeded 4.3 W kg^{-1}. The pattern of *c-fos* change was consistent with a thermal stress, thermoregulatory activity and the effects of restraint. There were no differences between continuous and pulsed exposures. Stagg et al (2001) exposed rats for 2 hours to 1.6 GHz Iridium signals using a head-only exposure system that produced local SARs in the brain of up to 5 W kg^{-1}. No significant increases in body temperature were recorded and no field-dependent increases in *c-fos* and *c-jun* mRNA were observed. Finnie (2005) found that no increase in *c-fos* expression was seen in restrained mice given a single far-field exposure to GSM 900 RF at a whole-body SAR of 4 W kg^{-1} for 1 hr; however, both *c-fos* expression in both exposed and sham-exposed groups was elevated compared to free-running control mice, suggesting that the experimental restraint significantly elevated *c-fos* expression. In a subsequent study, Finnie et al (2006a) investigated the expression of c-fos expression in the brain of embryonic and fetal mice exposed or sham-exposed during the whole of gestation (day 1 to day 19) to 900 MHz GSM RF radiation. No effects of GSM RF radiation exposure were seen on c-fos expression in brain tissue compared to the tissue of those sham-exposed, although expression varied between different brain regions.

Belyaev et al (2006) analyzed gene expression profile in RF exposed animals (see also Chapter II.3.3.2.1.2.). Rats were exposed or sham exposed to GSM 900 at whole-body averaged SAR of 0.4 W kg^{-1} for 2 h and total RNA was extracted from the cerebellum. In this study, triplicate arrays were applied for three exposed samples or three sham exposed samples. Gene expression profiles were obtained by Affymetrix U34 GeneChips representing 8800 rat genes and analyzed with the Affymetrix Microarray Suite (MAS) 5.0 software. The results showed that 11 genes were up-regulated by about 1.34 - 2.74 fold and one gene was down-regulated 0.48 fold. The induced genes encode proteins with diverse functions including neurotransmitter regulation, blood-brain barrier, and melatonin production. However, these changes in gene expression were not confirmed by other methods.

In general, the only consistent changes seen in gene expression were associated with hyperthermia or restraint stress.

Table II.4.7: Nervous system effects: Gene expression

Assay endpoint	Exposure Conditions	Response	Comment	References
Gene expression				
Immunocytochemical staining for c-fos protein in rat brain 2 hr after treatment	600 MHz (CW) for 20 min at 9.3 W kg^{-1} only.	Increase in c-fos protein levels following 1°C rise in brain and body temperature.	A small part of a larger behavioral study.	Mickley et al 1994
Immunocytochemical staining for c-fos protein in rat brain 2 hr after treatment	0.25-2.5 GHz; high peak power UWB radiation; 7-8 ns pulses; 60 pps for 2 min; peak E-field of 250 kV m^{-1}.	No effect on c-fos protein levels; body temperatures rose by less than 0.5°C.	High peak power UWB radiation	Walters et al 1995

Assay endpoint	Exposure Conditions	Response	Comment	References
Genomic response in rat brain tissue immediately following *in vivo* exposure viz: *hsp70*, *fos* and *jun* mRNA and their protein products assayed 24 hr after exposure	900 MHz CW or pulsed (GSM); 0. 6 ms pulses at 217 Hz pps for 4 h at brain SARs of 0.3 or 1.5 W kg^{-1} (pulsed) or 7.5 W kg^{-1} (CW).	Slight increase in *hsp 70* expression at the highest SAR (7.5 W kg^{-1}) but no effects on hps70 protein levels or any other exposure-related effects		Fritze et al 1997a
In situ hybridization for *fos* mRNA in the mouse brain immediately following exposure.	1.6 GHz CW or pulsed (Iridium signal); 9.2 ms pulses at 11 pps for 1 h at average brain SARs of ~ 0.3 - 11 W kg^{-1}	Increased *fos* expression in stress responsive and thermoregulatory parts of the brain at average brain SARs ≥ ~ 4 W kg^{-1}	Satellite communication system	Morrissey et al 1999
Body temperature, *fos*, *jun* and *odc* mRNA levels in brain tissue and stress-related plasma hormone levels in Fischer rats	1.6 GHz pulse modulated (Iridium signal); 9.2 ms pulses at 11 pps for 2 h at brain SARs of 0.16, 1.6 and 5 W kg^{-1}	No effect on body temperature, gene expression or plasma hormone levels compared to sham values.	Satellite communication system	Stagg et al 2001
Immunocytochemical staining of c-fos levels in mouse brain	900 MHz pulsed (GSM); 0.6 ms pulses at 217 pps for 1 h at whole-body SAR of 4 W kg^{-1}.	No effect of RF exposure on c-fos expression	Elevated c-fos levels in exposed and sham exposed, but not in free moving controls	Finnie 2005
Immunocytochemical staining of c-fos levels in fetal mouse brain	900 MHz pulsed (GSM); 0.6 ms pulses at 217 pps for 1 h from day 1 gestation to day 19 gestation at whole-body SAR of 4 W kg^{-1}.	No effect of RF exposure on c-fos expression in fetal brain tissue	Elevated c-fos levels in some brain regions in exposed and sham-exposed mice	Finnie et al 2006a

II.4.4.2. Blood-brain barrier

The blood-brain barrier is a dynamic interface that regulates the composition of cerebrospinal and interstitial fluid bathing central nervous system tissue. Physically, the 'barrier' comprises endothelial cells lining the blood capillaries of the brain and spinal cord and epithelial cells lining the choroid plexuses of the ventricles. 'Tight' junctions between these cells restrict the otherwise normal exchange of molecules through extracellular pathways, enabling the endothelial and epithelial cells of blood-brain barrier to regulate the exchange of molecules between the fluid compartments. However, the blood-brain barrier is relatively permeable in some regions of the brain, for example around the ventricles.

About 20 years ago several studies reported that low-level RF exposure may alter the permeability of the blood-brain barrier and cause leakage of molecules from the blood into the cerebrospinal fluid. Such responses could produce severe and lasting consequences. However better conducted studies failed to corroborate these findings and the original observations were ascribed to various confounding factors (see Blackwell and Saunders 1986). Consistent changes in permeability were only found using SARs of about 7 W kg^{-1} or more, which produced significant heating (WHO 1993, IEGMP 2000; Zmirou 2001; Krewski et al 2001a, Lin 2005). Immobilization stress is also associated with changes in the blood-brain barrier; habituation to experimental conditions is therefore essential when animals are restrained during exposure.

However, some recent studies have again suggested that low level RF exposure may affect the blood-brain barrier. Neubauer et al (1990) reported that significant changes occurred with exposures above 2 W

kg^{-1} for 30 minutes or more. Persson et al (1997) and Salford et al (1997) reported that exposure of rats to 915 MHz radiation increased the permeability of the blood-brain barrier to endogenous albumin. Using a TEM cell, animals were exposed in groups of 4 to either a CW field or pulse modulated radiation at pulse repetition rates between 4 and 217 Hz, and exposures lasted from 2 to 960 min at SARs ranging from 0.4-8 mW kg^{-1} to 1.7-8.3 W kg^{-1}. The number of animals showing increased permeability was reported to depend on both SAR and pulse modulation frequency but generally most exposures increased the leakage of albumin. Furthermore, the largest effects were reported using the weakest radiation, and exposure to CW radiation was reported to produce a greater effect than pulsed radiation. Weaknesses of this study include insufficient description of the experimental and exposure protocols used, and the findings are difficult to assess.

A more recent paper from the same laboratory (Salford et al 2003) reported that single, brief exposure of rats to pulsed 915 MHz radiation for 2 h at SARs of between 2 and 200 mW kg^{-1} caused increased blood-brain barrier permeability to albumin and neuronal damage throughout the brain (indicated by darkly staining neurons), especially in the cortex, hippocampus, and basal ganglia. However there are a number of caveats with this study. These include not only the modest size of the study (8 animals per group), a rather wide age range (12-26 weeks) of the rats used but also serious uncertainties about the metrology and dosimetry. The quantification of damaged neurons was also highly subjective, and too few data are presented to justify any conclusions. Overall replication using improved methods and with tighter control of experimental variables is necessary before any extrapolation can be made regarding potential human health effects.

Other studies using rats or mice have failed to corroborate these results, and acute or prolonged exposure to the radiation associated with mobile communication has not produced anything more than negligible effects on albumin permeability. Using a head-only exposure system, Fritze et al (1997b) exposed rats at 900 MHz pulsed at 217 Hz for 4 h at local SARs in the brain of 0.3, 1.5 or 7.5 W kg^{-1}. The leakage of albumin across the blood-brain barrier was examined using immuno-histochemical staining either at the end of exposure or 7 days later. Small increases in permeability were observed in all treatment groups examined immediately after exposure, but these numbers only reached significance in the animals exposed at the highest SAR which represented a thermal challenge. No sustained increases in permeability were reported. Using a similar design of exposure system, Tsurita et al (2000) exposed the heads of rats to a pulsed 1439 MHz TDMA field for 1 h a day, 5 days a week for 2 or 4 weeks. The peak SAR in the brain was 2 W kg^{-1}. Permeability was assessed using immuno-histochemical staining and the Evans blue dye injection method. Neither exposure period caused any discernible effect on the permeability of the blood-brain barrier. In addition, exposure had no apparent effect on body weight or on the Purkinje cells and granular cells in the cerebellum. As positive controls, both local cold injury of the skull or 2 h irradiation at 20 W kg^{-1} produced detectable increases in blood-brain barrier permeability.

Finnie et al (2001) exposed mice to 898 MHz pulsed at 217 Hz for 60 min at 0.4 W kg^{-1} using a well-characterized whole-body exposure system. This system consisted of a cylindrical parallel plate with the animals restrained in clear acrylic tubes arranged radially around a dipole antenna. Exposure had no significant effect on blood-brain barrier permeability as assessed using immunohistochemical staining for albumin. Where leakage had occurred, it was mainly confined to the leptomeningeal blood vessels which have no recognized blood-brain barrier. The same pattern of responses was reported by Finnie et al (2002) using long-term, repeated exposure. In this study, mice were exposed to 900 MHz pulsed at 217 Hz for 60 min a day, 5 days a week for 104 weeks at whole body SARs of 0.25, 1, 2 and 4 W kg^{-1}. Comparable small numbers of extravasations were observed in the brains of exposed, sham-exposed and freely moving control animals, but statistical analysis was not performed.

More recently, Kuribayashi et al (2005) investigated the effects of exposure to pulsed 1.439 GHz TDMA signals on the blood-brain barrier function in immature (4 week old) and young (10 week old) rats. The authors assessed permeability to dextran and the expression of genes involved in the regulation of barrier function, namely those encoding p-glycoprotein, aquaporin-4 and claudin-5, which regulate transmembrane drug transport, water homeostasis and tight junction integrity respectively. Repeated exposure of the head at 2 or 6 W kg^{-1} over a one or two week period had no effect on blood-brain barrier permeability or on the expression of related genes. In addition, no histopathalogical lesions such as gliosis or degenerative lesions were seen.

Cosquer et al (2005a) used the radial arm maze test to investigate whether exposure to RF would increase blood-brain barrier permeability to a drug known to affect radial arm maze performance. The muscarinic antagonist scopolamine hydrobromide readily crosses the blood-brain barrier to alter radial arm maze performance. The authors used a quaternary-ammonium derivative, scopolamine methylbromide, which does not readily cross the blood-brain barrier, to investigate barrier integrity in rats exposed to pulsed 2.45 GHz signals at a whole-body SAR of 2 W kg^{-1} (3 W kg^{-1} in the brain) for 45 min. No effect was seen on behavior nor, in separate groups of rats, on leakage of the dye Evans blue, which binds to albumin. A cold-injury positive control induced blood-brain barrier permeability to Evans blue/albumin.

The *in utero* exposure of embryonic and fetal mice from day 1 to day 19 of gestation for 1 h per day to GSM 900 RF was reported not to increase blood-brain barrier permeability (Finnie et al 2006b), using endogenous albumin as a vascular tracer identified by monoclonal antibody staining. The areas of the brain examined included the cerebral cortex, thalamus, basal ganglia, hippocampus, cerebellum, midbrain and medulla. Positive effects were reported in control animals injected with cadmium chloride. A second experiment (Finnie et al 2006c) examined the effect of a similar exposure for the first seven days following birth, during which time neurogenesis continues. As reported in the previous study, no effects were seen on blood-brain barrier permeability. It is worth noting here that Kumlin et al (2007) did not find any effect on the blood-brain barrier of juvenile (3 week old) rats following a 5 week exposure to 900 MHz GSM mobile phone radiation at average whole-body SARs of 0.3 or 3.0 W kg^{-1} (see above, Chapter II.4.3.2.2.3. and Table II.4.6.).

Overall, earlier reports of increased blood-brain barrier permeability have not been corroborated by later, better conducted studies.

Table II.4.8.: Nervous system effects: blood brain barrier

Assay endpoint	Exposure Conditions	Response	Comment	References
Blood-brain barrier				
Fluorescence assay of a tracer (rhodamine-ferritin) of pinocytic uptake in capillary endothelial cells.	2.45 GHz pulse-modulated, 10 μsec pulses at 100 pps, for 15 -120 min; whole-body SAR: ~ 1 or 2 W kg^{-1}	Increased uptake following exposure at 2 W kg^{-1} for more than 30 min.	Uptake blocked by colchine, which inhibits microtubular formation.	Neubauer et al 1990
Endogenous albumin and fibrinogen immuno-histochemical staining in rat brain tissue	915 MHz CW or pulse-modulated, either 0.57 ms pulses at 4, 8, 16 or 217 pps, or 6.6 ms pulses at 50 pps, for 2-960 min at whole-body SARs: between 0.4-8 mW kg^{-1} to 1.7-8.3 W kg^{-1}	Increase in albumin permeability at different combinations of SAR and modulation; results for fibrinogen not presented.	Weaknesses include insufficient description of experimental and exposure protocols	Persson et al 1997; Salford et al 1997
Albumin immuno-histochemical staining in rat brain tissue immediately or 7 days after *in vivo* exposure	900 MHz CW or pulsed (GSM); 0.6 ms pulses at 217 Hz pps for 4 h at brain SARs of 0.3 or 1.5 kg^{-1} (pulsed) or 7.5 W kg^{-1} (CW).	Increased extravasation of albumin immediately after exposure at 7.5 W kg^{-1} but not 7 days later.	Small but detectable increases in extravasation in rats immobilized for 4 h	Fritze et al 1997b

Assay endpoint	Exposure Conditions	Response	Comment	References
Evans blue injection or immunostaining of albumin, and cerebellar Purkinje cell numbers in rat brain tissue exposed *in vivo*	1439 MHz pulsed (PDC); 6.7 ms pulses at 50 pps for 1 h per day for 10 or 20 days at a brain SAR of 0.2 W kg^{-1}	No effect on blood-brain barrier integrity or Purkinje cell number	Cold injury positive control	Tsurita et al 2000
Albumin immuno-histochemical staining in mouse brain tissue exposed *in vivo*	898.4 MHz pulsed (GSM); 0.6 ms pulses at 217 pps for 1 h at a whole-body SAR of 4 W kg^{-1}	No effect on blood-brain barrier integrity		Finnie et al 2001
Albumin immuno-histochemical staining in mouse brain tissue exposed *in vivo*	900 MHz CW or pulsed (GSM); 0.6 ms pulses at 217 pps for 1 h per day, 5 days per week for 104 weeks at a whole-body SAR of 0.25, 1.0, 2.0, or 4.0 W kg^{-1}	The authors report that results suggest negligible effect on blood-brain barrier integrity	Some increased extravasation in exposed animals, but mainly in areas without effective blood-brain barrier.	Finnie et al 2002
Cresyl violet or albumin immunohistochemical staining in rat brain tissue 'about' 50 days after *in vivo* exposure.	898.4 MHz pulsed (GSM) 0.6 ms pulses at 217 pps for 2 h at a whole body SAR of 2, 20, or 200 mW kg^{-1}	Increased presence of albumin and darkly staining neurons in brain tissue of exposed animals.	Modest study size, wide age range, uncertainties with metrology and dosimetry.	Salford et al 2003
Immunocytochemical staining for vascular permeability to dextran and Evans Blue, and RT-PCR for blood-brain barrier-related gene expression, in immature (4 week) and young (10 week) rats	1439 MHz pulsed (PDC); 6.7 ms pulses at 50 pps at head SARs of 2 or 6 W kg^{-1} for 90 min day^{-1} for 6 days per week for 1 or 2 weeks.	No effect on vascular permeability, neuropathology or blood-brain barrier - related gene expression.	The genes, involved in blood-brain barrier function, showed only weak responses to chemically-induced barrier disruption	Kuribayashi et al 2005
Behavioral (radial arm maze) performance in response to a drug that crosses the blood-brain barrier poorly and Evans blue extravasation	2.45 GHz pulsed; 2 μS pulses at 500 pps at whole body SAR of 2.0 W kg^{-1}; brain SAR of 3 W kg^{-1}, for 45 min	No effect of exposure on blood-brain barrier permeability as revealed by Evans blue extravasation, or by drug-induced behavioral effects	The study assumed that significant changes in blood-brain barrier permeability would permit drug-induced behavioral changes	Cosquer et al 2005a
Monoclonal antibody staining of endogenous albumin in brain tissue of mice exposed *in utero* during gestation.	900 MHz pulsed (GSM) 0.6 ms pulses at 217 Hz pps for 1 hr per day, from day 1 gestation to day 19 gestation at a whole-body SAR of 4 W kg^{-1}	No effect on blood-brain barrier permeability	Blood-brain barrier permeability increased in positive control group.	Finnie et al 2006b

Assay endpoint	Exposure Conditions	Response	Comment	References
Monoclonal antibody staining of endogenous albumin in brain tissue of neonatal mice.	900 MHz pulsed (GSM) 0.6 ms pulses at 217 Hz pps for 1 hr per day, from post-natal day 1 - 7 at a whole-body SAR of 4 W kg^{-1}	No effect on blood-brain barrier permeability	Blood-brain barrier permeability increased in positive control group.	Finnie et al 2006c

II.4.4.3. Electrical activity in brain tissues

Neurons and neuronal networks are believed to be potential targets of RF exposure since they are excitable components that are potentially able to interact with induced electric fields. However, few experiments have been done on neuronal systems.

The hippocampal slice preparation has been much used in neurophysiology to study mechanisms associated with memory. Using a novel parallel-plate exposure system, Tattersall et al (2001) exposed slices of rat hippocampus at 700 MHz (CW) at SARs of between 0.6 and 4.4 mW kg^{-1}. Changes were found in the electrically-evoked field potentials, notably the post-synaptic discharge (population spike) in CA1 that depended on the magnitude of the SAR – low field intensities produced an increase in the amplitude of the population spike by up to 20%, but higher intensity radiation produced either increases of up to 120% or decreases of up to 80%. In addition, it was reported that exposure at about 1.1 mW kg^{-1} reduced or abolished drug-induced epileptiform activity in 36% of slices tested. Any field-induced rises in temperature were too small to be detected even using sensitive measuring equipment. Imposed temperature changes of up to 1°C failed to mimic the effects of RF exposure. However, it was later reported at an international symposium in Dublin (Green et al 2005) that significant heating occurs at the tip of the metallic stimulating electrode at much higher SARs (7-10°C at 400 mW kg^{-1}) which may have influenced these results. Pakhomov et al (2003), using a similar hippocampal slice preparation, found a transient reduction in the electrically-evoked population spike amplitude during exposure to brief, extremely high power (peak SAR of up to 500 MW kg^{-1}) microwave pulses (0.5 – 2.0 μs) at 9.3 GHz that was temperature-dependent; the reported temperature rises ranged up to 6.0°C at time averaged SARs of 3.6 kW kg^{-1}.

The electroencephalogram (EEG) is a description of the spontaneous electrical activity of the brain and can be used to indicate subtle changes in brain function. The interpretation of such studies is often complicated by the possible effects of anesthesia and restraint, and by artifacts resulting from electrical 'pick-up' via the recording electrodes. Exposure to very low levels of amplitude-modulated radiation has been reported to alter the EEG of the brain in cats and rabbits (WHO 1993). Complex changes in electrical activity recorded from the surface of the brain itself from implanted electrodes, particularly in the spectral power of various bands of the EEG, have been reported in recent studies using rats, mice and rabbits. Thuróczy et al (1994) reported that the whole-body exposure of anaesthetized rats to continuous wave 2.45 GHz at thermally significant cortical SARs increased the amplitude of the summed power spectrum of the EEG whereas head-only exposure to amplitude-modulated 4 GHz at similar high cortical SARs increased the amplitude of the beta frequency (14.5-30 Hz) band. More recently, Vorobyov et al (1997, 2004) reported that the intermittent application of amplitude-modulated 915 or 945 MHz RF enhanced the amplitude of certain EEG frequency bands recorded during exposure from conscious rats. However SARs were not reported.

Other studies are difficult to evaluate because little experimental detail has been published. Chizhenkova and Safroshkina (1996) reported slow high-amplitude waves accompanied by an increase in the number of spindle-shaped firings in the rabbit brain EEG in response to 3 GHz RF. Pu et al (1997) found an 800 MHz RF-induced inhibition of total EEG energy recorded from the mouse brain. In both studies, the experimental protocol was very briefly described.

Another difficulty with the interpretation of the EEG in individuals at rest is that the intra-individual variability is very high. Overall, because of these problems, it is not possible to draw any general conclusions regarding mobile phone effects on animal EEGs, although some of the changes appear to

reflect thermal responses. The variability of evoked and event related potentials is much lover, resulting in better reproducibility. Aran et al (2004) found no effect on the electrical activity in the auditory brain stem neural pathways evoked by acoustic stimulation following the chronic exposure of Guinea pigs to GSM 900 RF at local SARs of 4 W kg^{-1} at the cochlea.

In summary, effects seen in hippocampal brain slice activity appear to be temperature dependant. Otherwise, the reports of effects on EEG are rather variable and may be confounded by various uncontrolled experimental factors including 'pick-up' artifacts.

Table II.4.9.: Nervous system effects: brain electrical activity

Assay endpoint	Exposure Conditions	Response	Comment	References
EEG, DC brain impedance and ECG recorded in anaesthetized rats during RF exposure.	2.45 GHz CW whole body exposure at whole body SARs of 0.2, 2 or 7 W kg^{-1} for 10 min; 4 GHz, modulated at 16 Hz, head exposure at local SARs of 8 and 17 W kg^{-1} for 30 min; or 4 GHz CW localized at 42 W kg^{-1} for 30 min.	Increased EEG activity seen following thermally significant whole body and head exposures	Metal electrodes implanted below the skull may have caused localized RF field distortion. No RF 'pick-up' detected.	Thuróczy et al 1994
Electrical activity recorded in awake restrained rabbit brain, following prior electrode implantation, before, during and after RF exposure.	800 MHz at 400 W m^{-2} for 1 min; precise exposure conditions not given	Slow high amplitude 'waves' accompanied by increased in spindle-shaped firing	Experimental details unclear. Metal electrodes may have caused localized RF field distortion. No SARs given	Chizhenkova and Safroshinka 1996
Electrical activity recorded in mouse brain during exposure on 7th day	3 GHz at 50 W m^{-2} for 1 h day^{-1} for 7 days; precise exposure details not given	Exposure reported to produce a decrease in electroencephalic energy (expressed in dB).	Experimental details unclear – were the mice anesthetized on 7th day? Metal electrodes may have caused localized RF field distortion.	Pu et al 1997
EEG frequency spectra in rats with chronically implanted electrodes during RF exposure	945 MHz, amplitude modulated at 4 Hz, at 1 – 2 W m^{-2} applied intermittently (1 min on, 1 min off) for 10 min.	Small but statistically significant differences seen in certain EEG frequency bands during exposure	Implanted carbon electrodes. SARs not given.	Vorobyov et al 1997
Electrically-evoked field potentials recorded in vitro from the CA1 or CA3 region of rat hippocampal slices during RF exposure.	700 MHz CW at SARs (to the tissue slice) estimated between 0.6 an 4.4 mW kg^{-1} for between 5 and 15 min.	SAR-dependent changes in population spike amplitude	In vitro study; Localized temperature increase possible at tips of electrodes	Tattersall et al 2001; Green et al 2005
Electrically-evoked field potentials recorded in vitro from the CA1 region of rat hippocampal slices before, during and after RF exposure.	9.3 GHz pulsed; 0.5 -2 μs pulses at 0.5-2.0 pps at up to time-averaged SARs of 3.6 kW kg^{-1}; peak SARs of up to 500 MW kg^{-1}.	Time-averaged SAR-dependent reduction in population spike amplitude during exposure.	In vitro study; peak SARs very high	Pakhomov et al 2003

Assay endpoint	Exposure Conditions	Response	Comment	References
EEG frequency spectra in rats with chronically implanted electrodes during RF exposure	915 MHz, 20 μs pulses at 4 Hz; 3 W m^{-2} applied intermittently for 30 min day^{-1} for 3 days, followed by treatment with a muscarinic cholinergic antagonist and repeated RF exposure.	RF exposure enhanced EEG amplitudes in the 20-26 Hz frequency band and altered EEG responses to the cholinergic antagonist.	Implanted carbon electrodes. SARs not given.	Vorobyov et al 2004
Auditory brain stem response in Guinea pigs following RF exposure.	900 MHz pulsed (GSM); 0.6 ms pulses at 217 Hz pps at localized SARs of 1, 2 or 4 W kg^{-1}. Exposure for 1 h per day, 5 days per week for 2 months	No effects on auditory brain stem evoked response	No evidence for microwave damage to auditory pathways	Aran et al 2004

II.4.4.4. Neurotransmitters

Changes in various neurotransmitter systems have sometimes been reported in a few studies from different laboratories. Many of these data were reviewed by Hermann and Hossman (1997) who ascribed many of the reported changes to spurious temperature effects. The possibility that confinement or other stresses associated with exposure may produce changes in neurotransmitters levels, particularly in the cholinergic systems, should also be considered.

An extensive series of experiments from one laboratory suggests that exposure to low-level RF radiation may affect cholinergic function in a time-dependent fashion (reviewed by Lai 1992). Both pulsed and continuous 2.45 GHz exposure could elicit decreases in cholinergic activity (Lai et al 1987, 1988, 1989a,b). The threshold with pulsed RF (0.45 W kg^{-1}, specific energy absorption per pulse of 0.9 mJ kg^{-1}) was approximately equal to the rat's auditory perception threshold. It was reported that similar changes in cholinergic function could be induced by stressors such as noise and acute restraint, suggesting that exposure may be associated with mild stress. In addition, exposure was found to increase the concentration of benzodiazepine receptors in the cortex following acute but not repeated exposures (Lai et al 1992a) again suggesting anxiety or stress response. Similar studies provided evidence of the involvement of endogenous opioids (Lai et al 1992b) in the medial septal nucleus (Lai et al 1996).

Testylier et al (2002) reported that RF exposure caused sustained decreases in acetylcholine release from the rat hippocampus. Animals were exposed during the day for 1 h to 2.45 GHz CW or exposed at night to 800 MHz RF modulated at 32 Hz. Acetylcholine release was continuously measured by microdialysis using an implanted membrane in the CA1 region of the hippocampus. No effects were seen using 2.45 GHz at a whole-body SAR of 3.26 W kg^{-1} but exposure at 6.52 W kg^{-1} significantly decreased acetylcholine release for several hours after exposure.

Using semi-quantitative immunochemistry and image analysis to assess neurotransmitter content, Mausset et al (2001) reported that exposure to GSM 900RF at an SAR in the head of 4 W kg^{-1} reduced the cellular GABA neurotransmitter content in the Purkinje cells layer in the rat cerebellum. Similar but more extensive effects were observed following exposure to CW radiation at 32 W kg^{-1}, which suggested that thermal effects may have contributed towards this response. In an extension of this study, Mausset-Bonnefont et al (2004) exposed only the heads of rats to GSM RF for 15 min at a brain-averaged SAR of 6 W kg^{-1}. Using sensitive cellular and molecular techniques, the authors reported significant changes in binding properties of dopamine transporters, GABA receptors, and NMDA receptors in the cortex and striatum, and/or the hippocampus. Exposure was also associated with significant decreases in the expression of NMDA receptor subunits at the postsynaptic membrane, particularly in the striatum. In addition, the amount of glial fibrilliary acidic protein (GFAP), which is considered to be indicative of astrocyte activation, was increased in the cortex, hippocampus and striatum following exposure. The striatum is involved in the control of locomotor activity but a test of this, using an open-field paradigm, did not reveal any change, either immediately or 24 h after exposure. Following a similar exposure protocol, a further study (Brillaud et al 2007), set out to confirm and further evaluate these results on GFAP expression over a 10 day post-exposure period: the authors reported that a significant but transient

increase in GFAP was seen 2-3 days after exposure in the frontal cortex and basal ganglia, which declined thereafter.

Overall, the studies suggest that exposure to RF, including GSM signals, might result in transient changes in cholinergic activity, GABA content and NMDA receptor properties. However, in some cases, auditory perception and/or heating may have contributed to these observations; these possibilities should be clarified by further study.

Table II.4.10.: Nervous system effects: neurotransmitters

Assay endpoint	Exposure Conditions	Response	Comment	References
Choline uptake in rat brain following RF exposure.	2.45 GHz CW or pulsed (2 μs pulses at 500 pps); for 45 min at whole body SARs of 0.6 W kg^{-1}	Decrease in choline uptake in hippocampus (pulsed only) blocked by opioid antagonists	Whole body specific absorption per pulse of 1.2 mJ kg^{-1}, around pulsed RF auditory threshold	Lai et al 1987
Choline uptake in rat brain following RF exposure.	2.45 GHz CW or pulsed (2 μs pulses at 500 pps); for 45 min at whole body SARs of 0.6 W kg^{-1}	Decrease in choline uptake in frontal cortex (CW and pulsed) and in hippocampus (pulsed only).	As above (for pulsed RF only)	Lai et al 1988
Choline uptake activity and muscarinic cholinergic receptor concentration in rat brain following RF exposure	2.45 GHz pulsed; 2 μs pulses at 500 pps for 20 or 45 min at whole body SARs of 0.6 W kg^{-1} once, or in 10 daily sessions	Changes in choline uptake activity and in receptor concentration under some conditions of exposure	As above	Lai et al 1989a
Choline uptake in rat brain following RF exposure.	2.45 GHz pulsed; 2 μs pulses at 500 pps for 45 min at whole body SARs of 0.3 – 1.2 W kg^{-1}	Decrease in choline uptake activity in striatum, frontal cortex and hippocampus but not hypothalamus	Threshold effect of 0.75 W kg^{-1} in the striatum, and of 0.45 W kg^{-1} in the cortex and hippocampus.	Lai et al 1989b
Benzodiazepine receptor concentration in rat brain following RF exposure	2.45 GHz pulsed; 2 μs pulses at 500 pps for 45 min at whole body SARs of 0.6 W kg^{-1} once or in 10 daily sessions	Increase in receptor concentration in cerebral cortex only after acute but not repeated exposure	As above; suggests that low intensity microwave radiation can be a source of stress	Lai et al 1992a
Cholinergic activity in rat brain, pre-treated with antagonists to 3 subtypes of opioid receptors, following RF exposure.	2.45 GHz pulsed; 2 μs pulses at 500 pps for 45 min at whole body SARs of 0.6 W kg^{-1}	All three opioid receptor subtypes involved in RF-induced decrease in cholinergic activity in the hippocampus	As above	Lai et al 1992b
Cholinergic activity in rat hippocampus following RF exposure	2.45 GHz pulsed; 2 μs pulses at 500 pps for 45 min at whole body SARs of 0.6 W kg^{-1}	RF-induced decrease in cholinergic activity blocked by prior injection of μ-opioid antagonist.	As above	Lai et al 1996

Assay endpoint	Exposure Conditions	Response	Comment	References
GABA content of rat cerebellar tissue following *in vivo* exposure	900 MHz pulsed (GSM) 576 μs pulses at 217 pps for 2 h at brain SARs of 4 W kg^{-1} (pulsed) or 32 W kg^{-1} (CW).	Decreased stained area in one cell layer following pulsed RF exposure; reduced optical density in three cell layers following CW exposure.		Mausset et al 2001
ACh release in rat hippocampal tissue during and after exposure *in vivo*	2.45 GHz CW for 1 h during the day at a whole body SAR of ~ 3 or 6.5 W kg^{-1}; or 800 MHz amplitude modulated at 32 Hz for 1 or 14 h overnight at a whole body SAR of 0.3 W kg^{-1}	Exposure to 2.45 GHz at 6.5 W kg^{-1} for 1 h, or to 800 MHz for 14 h at 0.3 W kg^{-1} significantly reduced ACh release.		Testylier et al 2002
Binding properties of neurotransmitter transporters and receptors; number of NMDA receptor subunits and GFAP expression in rat brains	900 MHz pulsed (GSM) 576 μs pulses at 217 pps for 15 min at brain SARs of 6 W kg^{-1}.	Significant changes were seen in receptor and transporter binding properties and in NMD receptor subunit amount. A strong glial reaction in the striatum	No change in striatum-related locomotor activity.	Mausset-Bonnefont et al 2004
GFAP expression in rat brains 2-10 days after GSM-type exposure.	900 MHz pulsed (GSM) 576 μs pulses at 217 pps for 2 h at brain SARs of 4 W kg^{-1}.	Transient increase in GFAP expression in frontal cortex and basal ganglia 2-3 days after exposure.		Brillaud et al 2007

II.4.4.5. Behavior

Exposure to thermally significant levels of RF induces a heat load that elicits the various physiological and behavioral mechanisms animals use to regulate body temperature. These responses have been studied extensively and were reviewed by WHO (1993) and later by Adair and Black (2003). Most of the relevant animal studies were carried out before 1993. Briefly, in cool environments, animals compensate for RF-induced body heating by lowering their rate of metabolic heating and decreasing their food intake (e.g. Adair and Adams 1982). Other thermoregulatory behaviors exhibited by animals include the selection of cooler environments (e.g. Gordon 1983) and a reduction of spontaneous locomotor activity (eg Mitchell et al 1988). Later studies have focused on volunteer responses (Chapter II.5.3.2.).

In addition, a large number of important studies were carried out mostly in the 1970s and 1980s of aversive responses to RF exposure and of RF effects on food-motivated operant (learned) behaviors (reviewed by D'Andrea 1999; D'Andrea et al 2003a, 2007). The authors observed that the performance of operant tasks in which laboratory rodents and primates were trained to press one or more levers on a prescribed schedule in order to receive the food-reward could be disrupted or completely stopped (the 'work stoppage' effect) in a very consistent and repeatable manner by RF exposure sufficient to induce mild, whole-body hyperthermia. In general, behavior was not reliably affected until colonic temperature increased by $1^{o}C$ or more, corresponding to a whole-body SAR of approximately 4 W kg^{-1} (3.2 - 8.4 W kg^{-1}), depending on various factors including the frequency of the applied field, the animal size and species and the ambient temperature and relative humidity. The reduction in task performance seen in these studies has been attributed by Stern (1980) to the effects of competing thermoregulatory behaviors such as cooling off or escape, which the author notes may not necessarily be considered adverse.

More recent studies have focused mostly on effects of RF exposure associated with mobile phone use on learned behaviors. In addition, high-peak-power pulsed RF effects on the startle reflex, and in evoking body movement, have also been studied.

Learned behaviors

This heating effect on learning is illustrated by the results of a study using rats exposed at 600 MHz (Mickley et al 1994). Significant deficits in the performance of a working memory (object recognition) task were observed when exposures caused rises in rectal and brain temperatures of at least 1°C. These changes were correlated with an increase in expression of the *c-fos* gene in the cortex.

However, results of a few studies using pulsed radar-like signals appear to challenge this conclusion. Lai et al (1989a &b, 1994) reported that the behavior of rats performing a test of spatial memory function in a radial arm maze was disrupted by daily exposure for 20 or 45 minutes to pulsed 2.45 GHz RF at 0.6 W kg^{-1}. It should be noted that the pulse sequence used in these studies (2 μs at 500 pps) results in peak SARs of 600 W kg^{-1} and absorbed specific energies (SA) of 1.2 mJ kg^{-1}. Exposure did not cause a measurable rise in colonic temperature but acquisition was retarded and exposed animals consistently made more errors than controls, although Cassel et al (2004) noted that differences in performance between these groups existed at the onset of the task, indicating possible differences in anxiety or motivation. Additional results from the Lai group suggested that exposure had activated the endogenous opioid systems and so caused a decrease in cholinergic activity within the hippocampus. Quock et al (1994) reported that brief (5 min) exposure to 4.7 GHz at relatively high whole-body SARs of 36 W kg^{-1} reversed the sedative and anxiolytic effects of the benzodiazepine drug chlordiazepoxide.

Wang and Lai (2000) placed rats in a Morris water maze immediately after exposure to pulsed 2.45 GHz RF at 1.2 W kg^{-1} for 1 hour. The animals had to learn to escape from the water by locating a submerged (non-visible) platform. Exposed animals took longer to find the platform than control animals throughout the training sessions, and, in contrast to the control animals, spent much time trying to climb the side walls of the maze. In a probe trial without the platform being present, the exposed animals were reported to have spent less time swimming in the quadrant of the maze that should have contained the platform. Therefore, it was concluded that exposure had disrupted spatial reference memory functions and that the exposed animals had to use other, less efficient, learning strategies to locate the platform. However, statistical analysis of the probe trial data by one-way analysis of variance revealed no significant treatment effect, and only post-hoc analysis suggested a statistical difference between the exposed and control animals (see IEGMP 2000).

In contrast, tests of RF effects on spatial memory carried out at mobile phone frequencies found no effect. Sienkiewicz et al (2000) found that that exposure of mice for 45 minutes to pulsed 900 MHz RF at 0.05 W kg^{-1} had no significant effects on performance in a radial arm maze. Animals were tested immediately after exposure or following delays of 15 or 30 min. The animals tested without delay took longer to complete the task, possibly due to some mild stress associated with exposure. Similarly, Dubreuil et al (2002) exposed rats to pulsed 900 MHz radiation for 45 min using a head-only system before daily trials either in a radial arm maze or on a food location task in an open field arena (equivalent to a dry-land version of the Morris water maze). No significant effects on the performance of either task were seen using average SARs in the brain of either 1 or 3.5 W kg^{-1}. In an extension of this study, Dubreuil et al (2003) found no effect of a similar exposure on the performance of more complex radial arm maze tasks, or on the performance of a non-spatial object recognition task. A lack of effect on spatial reversal learning in a T-maze was also reported by Yamaguchi et al (2003) following exposure of rats to pulsed 1439 MHz (PDC) at non-thermal levels for either 4 days or 4 weeks. However, performance was significantly impaired by exposure that increased intraperitoneal temperature by up to 2°C.

Two groups have attempted a direct replication and extension of the radial arm maze study by Lai et al (1994) described above, using the same pulsed 2.45 GHz RF exposure at a whole body average SAR of 0.6 W kg^{-1} for 45 min Cobb et al (2004), using similar experimental procedures to those of Lai et al, including restricted access to distal spatial cues normally used to perform the task, found no effects of exposure on task performance. Similarly, Cassel et al (2004), also using a similar protocol but with distal spatial cues accessible, found that such exposure had no effect on performance. The same group (Cosquer et al 2005b) tested the effect of such exposure, reported by Lai et al (1989a & b, 1992a) to increase the number of benzodiazepine receptors in the cortex, on anxiety responses in rats using the elevated-plus maze test. This maze, which is in the form of a cross, elevated above the floor, has one pair of opposing arms enclosed by high sides, with the adjacent opposing pair were devoid of sides; anxiety increases the

number of entries into the closed arms. Cosquer et al (2005b) found that exposure had no effect on anxiety levels, either in a low-baseline anxiety test (carried out at low levels of illumination) or in a high baseline anxiety test (high levels of illumination). These findings provide no support for the hypothesis that low level RF radiation exposure increases behavioral measures of anxiety.

In summary, the early studies support the conclusion of WHO (1993) that the performance of learned behaviors is reduced following thermally significant RF exposure. Following a study reporting the reduced performance of a spatial memory task after exposure to pulsed RF, several groups have been unable to replicate or extend the initial observations. In addition, one group reported a lack of effect of pulsed RF on anxiety levels. However, the types of behavioral tasks that have been used are by no means exhaustive.

High peak power pulse effects

The auditory perception of pulsed RF radiation by animals is well established (WHO 1993; Lin and Wang 2007). Following RF absorption, a sound wave is generated in the head by the small and rapid thermoelectric expansion of brain tissue which generates a sound wave that stimulates the cochlea. For short pulses (< 30 μs), thresholds are dependent on the energy per pulse and correspond to a specific absorption per pulse of 0.9-1.8 mJ kg^{-1} in rats and 10-16 mJ kg^{-1} in cats (e.g. Guy et al 1975a; Chou et al 1985).

High peak power RF pulses with peak power densities of the order of 10's – 100's MW m^{-2} but of relatively short pulse widths (ns–μs) have been developed for military and other use but their relative biological effectiveness is not well established. Four studies have examined the effects of such pulses on food-reinforced operant behavior.

Using rhesus monkeys D'Andrea et al (1989) examined the effects of such exposure on the performance of a behavioral task that comprised a sequence of three operant schedules: a differential reinforcement of low rate schedule, a time discrimination schedule and a fixed interval schedule. During the performance of these tasks the animals were exposed for 1 h to pulsed 1.3 GHz RF with a pulse width of 3 μs at peak power densities of 1.32 MW m^{-2} and a specific absorption of 280 mJ kg^{-1} per pulse. Whole-body time-averaged SARs were varied by adjusting the pulse repetition rate and ranged between 0.05 W kg^{-1} (at 2 pps) to 0.8 W kg^{-1} (at 32 pps). The authors found no effects of exposure compared to sham-exposed animals.

D'Andrea et al (1994) exposed rhesus monkeys for 20 min to pulsed 5.62 GHz RF at whole body SARs of 2, 4 or 6 W kg^{-1} whilst they carried out a variable-interval, color-discrimination task. The monkeys were exposed to RF pulses with a pulse width of 2.8 μs at 100 pulses per second from a military radar either with or without an additional high peak power pulse with a pulse width of ~50 ns superimposed on the radar signal. Peak power densities were 2.77 MW m^{-2} (radar) and 25.2 MW m^{-2} (radar plus high peak power pulse). Compared to sham-exposed animals, response rates, reaction time and food pellet rewards significantly declined at whole-body SARs of 4 and 6 W kg^{-1} suggesting a heating effect; there was no specific effect of the additional high peak power pulse regime.

Akyel et al (1991) examined the operant performance of rats immediately after exposure to high peak power pulsed 1.25 GHz for 10 min. The rats were exposed to 10 μs pulses each of which produced a whole-body specific absorption of 2.1 J kg^{-1}. By adjusting the pulse repetition frequency, whole-body SARs varied from 0.84 W kg^{-1} to 23 W kg^{-1}. Following exposure, the rats were tested on three successive operant schedules: a fixed-ratio schedule, a variable interval schedule, and a differential reinforcement of low rate schedule. The authors found that the 10 min exposure at 23 W kg^{-1}, which induced a colonic temperature rise of 2.5°C, resulted in the subsequent termination of all operant behavior for about 13 minutes. Afterwards, the animals began to respond, but performance of two of the operant tasks never reached base-line levels, and the performance of the third task was variable. No effects were seen following exposure at the other SARs.

Raslear et al (1993) investigated the effect of exposure of rats to high peak power pulsed RF on their subsequent performance of a time perception and discrimination operant task. In this study, the rats were trained to discriminate between a visual stimulus applied for 0.5 or 5 s for a food reward and were then

also tested at intermediate durations with no reward following exposure for about 27 min to pulsed 3 GHz RF (80 ns pulse width) at a specific absorption of up to 580 mJ kg^{-1} per pulse. Whole-body SARs were small (< 0.1 W kg^{-1}). The authors found that the time taken to complete 300 trials and the number of null responses increased with increasing levels of exposure, suggesting a non-thermal effect on cognitive processes.

Other studies have focused on effects on the startle reflex. Seaman and Beblo (1992) studied the effect of exposure to a single high peak power RF ~1 μs pulse (head specific absorption of 22-43 kJ kg^{-1}, or 59-107 kJ kg^{-1}) on a subsequent 100 dB SPL acoustic noise-induced startle response in rats. They found that the low-energy pulse significantly reduced the amplitude of a subsequent startle response, as did prior exposure to an acoustic noise of 60 dB SPL (sound pressure level), whereas the high energy pulse increased the amplitude, but the variability precluded statistical significance. A later study (Seaman et al 1994) reported that a ~1 μs pulse of 66-142 mJ kg^{-1} and an 8 μs pulse of 525-1056 mJ kg^{-1} would inhibit and increase that latency of a startle response if given >~10 ms before an acoustic or tactile startle stimulus. An acoustic click given in place of the RF pulse had a similar effect. Both studies suggest that this effect of high peak power pulsed RF was mediated through the field-induced thermoelastic expansion of brain tissue.

Finally, Brown et al (1994) investigated the ability of high peak power RF pulses to evoke body movement in restrained mice. These authors exposed mice either to pulsed 1.25 GHz RF at 80 pps for 2 s, or to 'gated' CW 1.25 GHz for a duration of 50-3200 ms and measured induced movement with the aid of piezoelectric sensors. The brain specific energy per pulse varied up to 152 J kg^{-1}, and it was reported that a single pulse could induce body movement. Overall, however, the authors reported that the incidence of evoked body movement increased with the average energy input, and that there was no difference between pulsed RF and the gated CW RF, suggesting a possible heating effect.

In summary, most of the data suggests that high peak power RF pulses have no effect on operant behavior, except at thermogenic levels, when an expected decline in performance ensues. There is however, good evidence that individual high-peak-power pulses reduce and delay the 'startle' response to an acoustic noise and may evoke body movement.

Table II.4.11.: Nervous system effects: Behavior

Assay endpoint	Exposure Conditions	Response	Comment	References
Learned behaviors				
Radial arm maze (12 arm) performance in rats following RF exposure	2.45 GHz pulsed; 2 μs pulses at 500 pps for 20 or 45 min each day for 10 days at whole body SARs of 0.6 W kg^{-1}	Significantly reduced maze performance following exposure for 45 min, but not 20 min	Whole body specific absorption per pulse of 1.2 mJ kg^{-1}, around pulsed RF auditory threshold for short (< 30 μs) pulses	Lai et al 1989a
Radial arm maze (12 arm) performance in rats following RF exposure	2.45 GHz pulsed; 2 μs pulses at 500 pps for 45 min each day for 10 days at whole body SARs of 0.6 W kg^{-1}	Reduced maze performance reversed by pre-treatment with cholinergic agonist or opioid antagonist	As above	Lai et al 1994
Locomotor and rearing in mice, after pre-treatment with chlordiazepoxide, following RF exposure	1.8 or 4.7 GHz CW for 5 min at whole body SARs of 4, 12 or 36 W kg^{-1}	No effect of RF exposure on anxiolytic or sedative effect of drug treatment except at 4.7 GHz, 36 W kg^{-1}	Thermal effect	Quock et al 1994
Object recognition - working memory task in rats following RF exposure.	600 MHz (CW) for 20 min; whole-body SAR of 0.1-10 W kg^{-1}.	Impaired performance at > 9.3 W kg^{-1}; 1°C rise in body and brain temperature.	Thermal effect	Mickley et al 1994

Assay endpoint	Exposure Conditions	Response	Comment	References
Water-maze performance in rats following RF exposure	2.45 GHz pulsed; 2 μs pulses at 500 pps for 1 h twice per day for 3 days at a whole body SAR of 1.2 W kg^{-1}	Reduced performance	Whole body specific absorption per pulse of 2.4 mJ kg^{-1}	Wang and Lai 2000
Radial arm maze (8 arm) performance in mice following RF exposure	900 MHz pulsed; 576 μs pulses at 217 pps for 45 min each day for 10 days at a whole body SAR of 0.05 W kg^{-1}	No effect on performance	Whole body specific absorption per pulse of 0.23 mJ kg^{-1} for long (576 μs) pulses	Sienkiewicz et al 2000
Radial arm maze (8 arm) or a food-location task in an open field arena in rats following RF exposure	900 MHz pulsed; 576 μs pulses at 217 pps for 45 min each day for 10-14 days at a brain SAR of 1 or 3.5 W kg^{-1}	No effect on performance of either task	Whole body specific absorption per pulse of 4.6 or 16 mJ kg^{-1} for long (576 μs) pulses	Dubreuil et al 2002
Radial arm maze (8 arm) with inter-arm confinement or intra-trial delays, or an object recognition task in rats following RF exposure	900 MHz pulsed; 576 μs pulses at 217 pps for 45 min each day for 10-14 days at a brain SAR of 1 or 3.5 W kg^{-1}	No effect on performance of either task	Whole body specific absorption per pulse of 4.6 or 16 mJ kg^{-1} for long (576 μs) pulses	Dubreuil et al 2003
T-maze reversal learning in rats following RF exposure	1439 MHz pulsed (PDC); 6.7 ms pulses at 50 pps for 4 day or 4 weeks at a brain SAR of 7.5 W kg^{-1} and whole body SAR of 1.7 W kg^{-1} or brain SAR of 25 W kg^{-1} and whole body SAR of 5.7 W kg^{-1}	No effect on performance at the lower level of exposure; a reduction at the higher, thermally significant, level	Thermal effect	Yamaguchi et al 2003
Radial arm maze (12 arm) performance in rats following RF exposure	2.45 GHz pulsed; 2 μs pulses at 500 pps for 45 min each day over 10 days at whole body SARs of 0.6 W kg^{-1}	No effect on performance	Fails to replicate Lai et al 1989a, 1994	Cobb et al 2004
Radial arm maze (12 arm) performance in rats following RF exposure	2.45 GHz pulsed; 2 μs pulses at 500 pps for 45 min each day over 10 days at whole body SARs of 0.6 W kg^{-1}	No effect on performance	Fails to replicate Lai et al 1989a, 1994	Cassel et al 2004
Anxiety responses of rats in elevated plus-maze at different ambient light intensities following RF exposure	2.45 GHz pulsed; 2 μs pulses at 500 pps for 45 min at whole body SARs of 0.6 W kg^{-1}; brain SAR estimated as 0.9 W kg^{-1}	RF radiation had neither an anxiolytic nor an anxiogenic effect.	Fails to confirm suggestion of pulsed microwave exposure as a stressor by Lai et al 1994	Cosquer et al 2005b

Assay endpoint	Exposure Conditions	Response	Comment	References
High Peak Power Pulse Effects				
Multiple schedule operant task performance by rhesus monkeys during exposure.	1.3 GHz pulses, pulse width 3 μs, at 2-32 pps at a head specific absorption of 280 mJ kg^{-1} per pulse; Peak whole body SARs 8.3 W kg^{-1}; average whole body SARs 0.05-0.8 W kg^{-1} for 60 min.	No effect on operant task performance.	Head specific absorption per pulse above the auditory stimulus threshold.	D'Andrea et al 1989
Multiple schedule operant task performance by rats after exposure.	1.2 GHz pulses, pulse width 10 μs; average whole body SARs of 0.84, 2.5, 7.6 and 23 W kg^{-1} for 10 min. Whole body specific absorption of 2.1 J kg^{-1} per pulse.	Initial (for 13 min) failure to perform tasks following exposure at 23 W kg^{-1} followed by reduced task performance.	Colonic temperatures increased in the high exposure group by 2.5°C.	Akyel et al 1991
Startle reflex in rats in response to 100 dB SPL acoustic noise	Single 1.25 GHz pulses, pulse width 0.8-1.0 μs; SA to the head of 22-43 mJ kg^{-1} or 59-107 mJ kg^{-1} per pulse. Time-averaged whole-body SARs not given.	The acoustic startle reflex had a lower amplitude following a prior RF pulse at 22-43 kJ kg^{-1}, and a higher but variable amplitude following an RF pulse at 59-107 kJ kg^{-1}.	A prior 60 dB SPL noise increased latency and decreased amplitude of the acoustic startle response.	Seaman and Beblo 1992
Time-discrimination operant behavior in rats after exposure.	3 GHz pulses, pulse width of 80 ns at 0.125 pps for 200 pulses (~27 min) at SA of up to 580 mJ kg^{-1} per pulse. Time-averaged whole-body SAR of up to 0.072 W kg^{-1}.	Dose-response effects observed for session time and null responses; possible effect on time discrimination.	Co-varying sound (~57-89 dBA per pulse) and x-ray exposure did not correlate with effects.	Raslear et al 1993
Color discrimination operant task performance by rhesus monkeys during exposure.	5.62 GHz pulses, radar pulse width 2.8 μs, with or without additional high peak power pulse width 2 ns, at 100 pps for 20 min at average whole body SARs of 2, 4 or 6 W kg^{-1}.	Responses declined significantly at whole body SARs of 4 and 6 W kg^{-1}, as did reaction time and food pellet consumption.	No effect of pulse regime.	D'Andrea et al 1994.
Startle reflex in rats in response to (1) acoustic noise or (2) tactile (air puff) stimulus	Single 1.25 GHz pulses, (1) pulse width ~1 μs, SA of 16-44 mJ kg^{-1} or 66-142 kJ kg^{-1}; (2) pulse width ~ 8μs, SA of 525-1056 kJ kg^{-1} per pulse	A high intensity 1 μs pulse (1) or an 8 μs pulse (2) affected the amplitude and latency of a subsequent startle reflex	The 8 μs pulse (2) had a similar effect on the tactile startle reflex to that of a 94 dB SPL acoustic noise (click).	Seaman et al 1994

Assay endpoint	Exposure Conditions	Response	Comment	References
Evoked body movement in restrained mice.	1.25 GHz pulsed, pulse width 10 μs at 80 pps for up to 2 s; or single gated 1.25 GHz CW, duration 50-3200 ms. Brain SA per pulse up to ~152 J kg^{-1}	Body movement could be induced by a single pulse; generally, incidence increasing with averaged energy input.	No difference between responses to pulsed RF and gated CW suggested a possible skin heating effect.	Brown et al 1994

II.4.4.6. Summary on behavior

Several recent studies support the experimental observations summarized by WHO (1993) that operant behavior in laboratory rodents and primates can be disrupted by thermogenic RF exposure sufficient to raise body temperature by about 1°C. Two studies report the reduced performance of operant tasks during exposure to high peak power RF pulses but attribute these effects to heating. In another study, significant deficits on the performance of a T-maze task were seen only when exposure increased body temperature by 2°C.

Otherwise, studies have continued to investigate the possible effects of RF radiation, often that characteristic of mobile phone use, on the brain and nervous system in animals. Despite sporadic reports of positive effects, most studies have not reported any field-dependent responses either in gene expression or in increased permeability of the blood-brain barrier. One study in particular found a lack of effect on blood-brain barrier permeability following in GSM exposure *in utero* throughout gestation. The evidence from several laboratories indicates that changes may be induced in cholinergic activity in the brain following relatively intense exposure. Such changes might predict effects on spatial learning and memory, but on balance the evidence does not support this view: two studies from one laboratory have reported deficits in performance of spatial memory tasks using pulsed 2.45 GHz microwaves, but were not confirmed in two independent replications of these studies, nor in three other studies using GSM signals.

Studies of the behavioral effects of high peak power RF pulses such as those used in military applications have been rather sporadic and diverse; pulse widths have varied by two orders of magnitude (80 ns – 10 μs) and the specific absorption per pulse by four orders of magnitude (22 mJ kg^{-1} – 152 J kg^{-1}). Two rather elegant studies showed the equivalence of pulsed RF to an acoustic 'click' in affecting the startle reflex.

II.4.5. Auditory system

It has been known for a long time from extensive electrophysiological and behavioral data indicate that animals can perceive pulsed RF radiation (see WHO 1993; Lin and Wang 2007). As described above, the generally accepted explanation is that a sound wave is generated in the head by the short but rapid thermoelastic expansion of the brain resulting from the absorption of the RF pulse. For short pulses (< 30 μs), thresholds are dependent on the energy per pulse and correspond to a specific absorption per pulse of 0.9-1.8 mJ kg^{-1} in rats and 10-16 mJ kg^{-1} in cats. Such effects might be important in the interpretation of behavioral responses to pulsed RF radiation.

Recent studies have focused on possible RF effects on cochlea function *per se* measuring otoacoustic emission. This is an indicator of the normal mechanical contractility of the outer hair cells of the cochlea and is considered to be a reliable method of assessing cochlea functionality *in vivo*. The outer hair cells, which are notoriously susceptible to various endogenous and exogenous stressors, generate an acoustic signal in response to auditory stimuli (measured for example as the distortion product otoacoustic emission or DPOE) which can be monitored in the external ear canal (or auditory meatus).

A lack of effect on otoacoustic emissions was reported in four new-born and two groups of seven adult rats exposed or sham exposed to GSM 900 RF for 1 h per day for 30 days (Kizilay et al 2003); unfortunately field measurements and dosimetric assessments of SAR were not given. DPOEs were recorded in anesthetized adult animals before the first exposure and after 30 days exposure; for the new-born rats, the recordings made after 30 days exposure were compared to the results from adults prior to

exposure. No effects on outer hair cell function were detected. Similarly, Aran et al (2004) found no effect on outer hair cell function in rats following chronic exposure over 2 months to GSM 900 at local cochlea SARs of 1, 2 or 4 W kg^{-1}.

A lack of effect on outer hair cell function by mobile phone RF, as assessed by DPOEs, has also been reported in two papers. Galloni et al (2005a) describe a set of three experiments in which the RF frequency, source, modulation characteristics and period of exposure were varied. In the first experiment, rats were exposed or sham exposed to 936 MHz CW whole-body or to 923 MHz CW head-only for 3 h per day for 5 days; the local SAR to the head was about 1 W kg^{-1}. In the second study, rats were exposed or sham exposed to 960 MHz GSM RF for 3 h per day for 5 days, with a head SAR of 1 W kg^{-1}. In the third study, exposure was to 900 MHz GSM RF for 2 h per day, 5 days per week for 4 weeks, with a head SAR of 2 W kg^{-1}. The authors found no effect of RF exposure on DPOEs in any of these studies. A further study (Galloni et al 2005b) reported that the exposure of rats to GSM signals at 900 MHz or 1800 MHz over a 4 week period at a local SAR of 2 W kg^{-1} had no effect.

In summary, the evidence is rather consistent and suggests that mobile phone type RF exposure has no effect on auditory function in rodents. It is also clear that animals can hear the pulsed RF characteristic of radar above given thresholds, through a thermoelastic expansion mechanism.

Table II.4.12.: Nervous system effects: auditory functions

Assay endpoint	Exposure Conditions	Response	Comment	References
DPOEs in newborn and adult rats before and after RF exposure.	900 MHz pulsed (GSM); 0.6 ms pulses at 217 Hz pps. SARs not given. Exposure for 1 h per day for 30 days	No effects on distortion product otoacoustic emissions	Small numbers of animals and absence of dosimetry.	Kizilay et al 2003
DPOEs in Guinea pigs following RF exposure.	900 MHz pulsed (GSM); 0.6 ms pulses at 217 Hz pps at localized SARs of 1, 2 or 4 W kg^{-1}. Exposure for 1 h per day, 5 days per week for 2 months	No effects on distortion product otoacoustic emissions	No evidence for microwave damage to outer hair cells of the cochlea	Aran et al 2004
DPOEs in rats before and after RF exposure.	936 MHz CW whole body or 923 MHz CW head only, 3 h per day for 5 days; head SAR 1 W kg^{-1}; 960 MHz GSM RF, 3 h per day for 5 days, head SAR 1 W kg^{-1}; 900 MHz GSM RF, 2 h per day, 5 days per week for 4 weeks, head SAR 2 W kg^{-1}.	No effects on distortion product otoacoustic emissions	As above	Galloni et al 2005a
DPOEsin rats before, during and after RF exposure.	900 or 1800 MHz pulsed (GSM); 0.6 ms pulses at 217 Hz pps at localized SAR in the ear of 2 W kg^{-1} for 2 h per day, 5 days per week for 4 weeks.	No effects on distortion product otoacoustic emissions	As above	Galloni et al 2005b

II.4.6. Endocrine system

Most early studies, reviewed for example by WHO (1993) and later by Black and Heynick (2003) described thermally-mediated responses of the endocrine system to RF exposure. Briefly, endocrine responses to acute RF (often CW 2.45 GHz) exposure are generally consistent with the acute responses to

non-specific stressors such as heat. Several papers report that plasma corticosterone or cortisol levels are significantly enhanced in rodents (Lotz and Michaelson 1978; Lu et al 1980, 1981) and primates (Lotz and Podgorski 1982) by exposures resulting in about a 1^{o}C rise in body temperature; corresponding whole-body SARs were of the order of 4 W kg^{-1}. The response seems to be mediated by the release of adrenocorticotrophic hormone by the hypothalamus via the anterior pituitary gland, and is modulated in amplitude by the circadian rhythm of cortisol or corticosterone levels. The hypothalamus also controls the secretion of growth hormone and thyroxin; stressful stimuli such as significantly elevated body temperatures are known to depress circulating plasma levels of both hormones in rodents (Michaelson et al 1975). However, no effects on growth hormone and thyroxin have been seen in primates (Lotz and Podgorski 1982). In addition, no effects on the circulating levels of a number of hormones have been seen in rats chronically exposed for most of their lives at whole-body SARs of up to 0.4 W kg^{-1} (Chou et al 1992), a SAR insufficient to significantly affect body temperature.

II.4.6.1. Pineal-melatonin studies

There have been fewer studies of endocrine effects since 1993; those that have been carried out mostly focused on radiation associated with the use of mobile telephony. Several studies have examined possible effects on circulating melatonin, a hormone produced by the pineal gland in a distinct daily or circadian rhythm which is governed by day length, the disturbance of which has been implicated in breast and other cancers (e.g. Stevens 1987).

Vollrath et al (1997) studied the serum melatonin levels and other markers of melatonin synthesis in two strains of rat and in Djungarian hamsters exposed to GSM 900 or CW RF for up to 6 h. Whole body SARs were estimated as ranging from 0.06 – 0.36 W kg^{-1} in the rats and 0.04 W kg^{-1} in the hamsters. No effects were seen on any of the endpoints examined. However, interpretation is limited by a number of difficulties; the study comprised 26 experiments which were described and assessed individually; the first 12 experiments were dismissed by the authors because the results were affected by differences in sampling times in exposed and sham-exposed animals due to the sequential nature of the sham and exposure treatments. In addition, the sample numbers in all the individual experiments were small (between 4-6 on average), limiting the statistical power to detect differences.

Bakos et al (2003) examined the daily urinary excretion of 6-sulfatoxymelatonin, a waste product of melatonin metabolism, in male rats exposed or sham exposed to either GSM 900 or 1800 RF for a 2 h period between 8.00 am and noon for 14 days. The exposure levels were chosen to correspond to the occupational (1 W kg^{-1}) and public (100 mW m^{-2}) RF exposure levels that apply in Hungary. The authors found no effect of exposure on daily 6-sulfatoxymelatonin excretion.

Hata et al (2005) measured serum and pineal melatonin levels in rats that were on a reversed day/night schedule and were exposed or sham exposed to mobile phone RF radiation from a Japanese Personal Digital Cellular (PDC) system operating at 1.439 GHz. Treatment (exposure or sham exposure) was for 4 h on one day, beginning at the onset of the 12 h dark period; serum and pineal melatonin were assessed 3 and 6 h after the cessation of exposure. No effects of RF exposure on melatonin levels were observed.

Koyu et al (2005a) looked at nocturnal serum melatonin levels in rats exposed or sham-exposed either to GSM 900 or 1800 RF over a 4 week period. Peak SARs in the head were 2 W kg^{-1}. There was no statistically significant effect on melatonin levels recorded in response to 900 MHz or to 1800 MHz GSM RF radiation.

II.4.6.2. Pituitary-thyroid studies

Even fewer studies have examined hormones controlled by the hypothalamus-pituitary axis. Koyu et al (2005b) investigated the effects in rats of exposure to 900 MHz CW RF on circulating levels of thyroid stimulating hormone (TSH), which is released from the hypothalamus via the anterior pituitary gland and regulates thyroid activity, and serum tri-iodothronine (T_3) and thyroxin (T_4) levels. The authors found that exposure for 30 min per day for 5 days a week for 4 weeks at a peak SAR in the head of 2 W kg^{-1} significantly reduced TSH, T_3 and T_4 levels compared to sham exposed animals. Unfortunately, it is not

possible to determine from the brief account of the experimental protocol and dosimetry whether the exposure was sufficient to increase tissue or whole-body temperature.

Table II.4.13.: Endocrine responses

Assay endpoint	Exposure Conditions	Response	Comment	References
Pineal gland				
Pineal seratonin *N*-acetyltransferase (NAT) activity and serum melatonin in rats and Djungarian hamsters; pineal synaptic ribbon profile numbers in rats	900 MHz CW at 1 W m^{-2} or pulsed (GSM) 0.6 ms pulses at 217 pps for between 15 min and 6 h at whole-body SAR: between 0.04 and 0.36 W kg^{-1}.	No effects on any parameter measured	An exploratory study in which experiments were individually described. Early studies confounded by different sampling times for exposed and sham exposed animals.	Vollrath et al 1997
Daily excretion of a major metabolite (6-sulfatoxymelatonin) of melatonin in rats	900 MHz or 1800 MHz pulsed (GSM) 0.6 ms pulses at 217 pps for 2 h per day for 14 days at whole-body SARs of between 0.009-0.012 W kg^{-1} and 0.22-0.045 W kg^{-1} respectively	No effect	Power density levels similar to Hungarian exposure limits for the general public (100 mW m^{-2}) and workers (1 W m^{-2})	Bakos et al 2003
Pineal seratonin and melatonin levels, and serum melatonin levels, in rats.	1439 MHz (PDC); 6.7 ms pulses at 50 pps for 4 h at the onset of the 12 h dark period at whole body SAR ~ 2.0 W kg^{-1}. Head SAR estimated as 7.5 W kg^{-1}	No effect on melatonin or seratonin levels taken 3 and 6 h after exposure.	Light-at-night positive control group showed marked suppression effects.	Hata et al 2005
Nocturnal serum melatonin levels in rats	900 MHz or 1800 MHz pulsed (GSM) 0.6 ms pulses at 217 pps for 30 min per day, 5 days per week, for 4 weeks at a peak (head) SAR of 2 W kg^{-1}	No effect on serum melatonin levels at the end of the 4 week exposure.	Uncertain dosimetry	Koyu et al 2005a
Pituitary-thyroid axis				
Serum TSH, tri-iodothronine (T_3) and thyroxine (T_4) in rats	900 MHz CW for 30 min per day, 5 days per week for 5 day per week for 4 weeks; peak (head) SAR of 2 W kg^{-1}	Significant decreases in serum TSH, T_3 and T_4 levels.	No differences between cage controls and sham exposed levels. Possible RF heating effects.	Koyu et al 2005b

II.4.6.3. Summary on endocrine system

One study reported decreased levels of thyroxin and associated thyroid hormones in rats following exposure to CW RF radiation, similar to reports from earlier studies, although it is not clear in the later study that the exposure was thermal. No effects were seen in circulating serum melatonin levels and other measures of melatonin synthesis and excretion in four studies using mobile phone signals.

II.4.7. Cardiovascular system

Early studies of the effects of RF radiation on the cardiovascular system of animals have been reviewed by Jauchem and Frei (1992) and by WHO (1993). These early studies have also been reviewed more recently by Black and Heynick (2003) and in some detail by Adair and Black (2003). In summary, these

reviews concluded that cardiovascular system responses to RF exposure, such as changes in heart rate and arterial blood pressure, are consistent with those associated with thermoregulatory responses to conventional heating. In general, an increase in body temperature elicits several cardiovascular changes, including increased blood flow to the skin, increasing skin thermal conductance, and increased cardiac output, primarily due to an increase in heart rate, in order to maintain arterial pressure within the normal range. For example, vasodilation of the superficial blood vessels of the skin in primates occurs above a threshold whole-body SAR of about 1 W kg^{-1} when the RF heating is largely superficial (Adair and Adams 1980). Similar responses occur during exposure of primates to 'resonant' frequencies which result in more uniform, less superficial heating (Lotz and Saxton 1987, 1988) but are associated with larger rises in rectal temperature because the less effective stimulation of skin temperature receptors results in reduced thermoregulatory performance. Heart rate was increased in rabbits exposed to 2.45 GHz at whole-body SARs sufficient to raise body temperatures by 0.5^{o}C (Chou et al 1980).

Following the reviews of Jauchem and Frei (1992) and WHO (1993) in the early 90's, most subsequent studies were of thermoregulatory responses of volunteers to RF exposure (see Chapter II.5.) rather than animals. However, one group carried out a study of the effects of RF exposure during fever, which is generally assumed to increase susceptibility to exogenous sources of heat such as RF radiation. In addition, a series of studies was carried out by another group on health effects primarily associated with military applications of RF, including the responses of anesthetized rats to severe RF heating, and the responses of conscious rats to high peak power RF pulses, or to pulsed ultra-wideband RF radiation.

II.4.7.1. Thermoregulatory changes

Adair et al (1997) investigated the effect of exposure to 450 MHz or 2.45 GHz radiation on thermoregulatory responses during experimentally-induced fever in the conscious squirrel monkey, a non-human primate. The authors found that during RF exposure, the magnitude of the fever remained the same but the absorption of RF energy had proportionately reduced the fever-generated increase in endogenous heat production. However, during exposure at 450 MHz, a resonant frequency in the squirrel monkey, energy is deposited deep within the body and the fever was augmented. In addition, the fever was exacerbated when exposure occurs during the period that the fever abates and body temperature begins to fall.

Jauchem and colleagues investigated the cardiovascular and respiratory responses of rats anesthetized with Ketamine to intense RF radiation. Ketamine is reported to have minimal effects on temperature regulation or on the cardiovascular and respiratory system, but the animals would have been unable to thermoregulate behaviorally. Unusually, the experiments were often continued until the animals died. Jauchem and Frei (1997) investigated the effects of exposure to a sub-resonant RF radiation (350 MHz) at a whole-body SAR of about 13 W kg^{-1} on the cardiovascular and respiratory responses, namely heart rate, mean arterial blood pressure, respiratory rate and colonic, tympanic and sub-cutaneous temperatures of anesthetized rats. The authors observed that heart rate increased with rising body temperature; mean arterial pressure and respiratory rate were largely unaffected until body temperatures rose above around 42^{o}C, whereupon they declined.

Jauchem et al (2000) investigated the effects of exposure to 1 GHz, 10 GHz, or combined 1 and 10 GHz RF at whole-body SARs of 12 W kg^{-1} on heart rate, mean arterial blood pressure, respiratory rate and colonic, tympanic and sub-cutaneous temperatures. Colonic temperature was highest in the 1 GHz exposure group, indicating a more uniform heating, whereas subcutaneous temperature on the side facing the antenna was highest in the 10 GHz exposure group, reflecting more superficial heat deposition and greater temperature gradients resulting from exposure to a higher frequency. With regard to the physiological parameters measured, the authors found that the overall pattern of responses was generally similar in all three groups. Heart rate and temperature increased linearly with exposure duration, mean arterial blood pressure increased slightly and then declined, respiration rate initially increased or remained relatively constant and then declined.

These authors also conducted a series of studies of the cardiovascular and respiratory responses of anesthetized rats exposed to 35 GHz RF heating until the death of the animal. Ryan et al (1997a) examined the effect of age and food restriction on the ability of the animal to withstand severe thermal

challenge. In addition, the effect of various pharmacological manipulations, notably nitric oxide administration (Ryan et al 1997b) and histamine receptor blockade (Jauchem et al 2004), on the ability of the animal to withstand the severe thermal challenge induced by 35 GHz heating was studied. These studies of intense heating effects are reviewed by Jauchem (2006) but are of little direct relevance to occupational or public exposures.

II.4.7.2. High peak power pulses

High peak power RF pulses of relatively short pulse widths are a relatively recent technological development, initially intended for military use but now finding a wider range of application, e.g., in radar. Peak power densities may be of the order of 10's – 100's MW m^{-2}, but the short pulse widths (ns–μs) and the low pulse repetition rates result in low average SARs. However, this type of exposure raises a question about the relative biological effectiveness of high peak power SARs compared to low overall average values.

Jauchem and Frei (1995) exposed or sham exposed rats to ten pulses of high peak power density 1.2-1.7 GHz RF. The pulse widths were between 40-70 ns, given at a rate of 1 pulse per second, and the peak power density in each pulse ranged up to a maximum of 520 MW m^{-2}; SARs, however, were not given. The authors reported an initial but transient increase in mean arterial blood pressure and a transient but non-significant decrease in heart rate. These responses disappeared when the acoustic noise associated with the production of each RF pulse was attenuated by Eccosorb® sound attenuator.

II.4.7.3. Ultra-wideband pulses

Ultra-wideband (UWB) RF radiation is a new modality in radar technology that has also been developed initially for military use but which now finds a wide range of application in imaging, sensing and communication systems (ICNIRP 2008). It comprises a RF signal with an ultrashort pulse width (1–10 ns) and a very fast rise-time (10's–100's ps). The spectral power of each pulse is very broad, ranging, for example, from 10's kHz to 10's GHz. The peak electric field can be in excess of the breakdown voltage of air without arcing, and results in a very high energy absorption per pulse, but with a very low average SAR because of the very low pulse repetition rate. The ratio of peak to average SAR is therefore very much higher than has been addressed hitherto and, as with high peak power pulses, raises a question about biological effectiveness.

Jauchem et al (1998) reported a lack of effect on heart rate and mean arterial blood pressure in anesthetized rats exposed to pulsed UWB RF for 2 min. The rats were exposed to 50, 500 or 1000 pulses per second; the average pulse width was ~ 1 ns, the rise-time was 174-218 ps, and the peak E-field was 87-104 kV m^{-1}. Jauchem et al (1999) reported a lack of effect on heart rate and mean arterial blood pressure in anesthetized rats exposed to pulsed UWB RF radiation for up to 5 min. The pulses had a pulse width of 6 ns and an average rise time of ~ 330 ps, a pulse repetition frequency of 2 kHz and a peak electric field of 19-21 kV m^{-1}; power density and SAR were not given.

Lu et al (1999) exposed or sham exposed conscious rats in a GTEM cell for 6 min to pulsed UWB RF radiation at a whole-body SAR of 0.07 W kg^{-1}, or to pulsed UWB RF radiation at a whole body SAR of 0.121 W kg^{-1} and reported that systolic and mean arterial blood pressure, and by implication diastolic blood pressure, were significantly decreased during the monitoring period, from 45 min to up to 4 weeks after treatment. In contrast, no effect was seen on heart rate. The exposure was below thermal levels and the specific energy (SA) per pulse was ~0.12 mJ kg^{-1}, about an order of magnitude below the threshold for the 'microwave hearing' effect. The authors were unable to account for the UWB radiation-induced hypotension but noted that it was a robust and persistent effect.

Table II.4.14.: Cardiovascular responses

Assay endpoint	Exposure Conditions	Response	Comment	References
Thermoregulation				
Metabolic heat production, preoptic, colonic and skin temperature in squirrel monkeys with prostaglandin E_1 (PGE_1)-induced fever	450 MHz (CW) at a whole-body SAR of 2.06 or 3.3 W kg^{-1} or 2.45 GHz (CW) at a whole-body SAR of 3.3 W kg^{-1} for 30 min periods during fever	Metabolic heat production reduced by exposure during early period of the fever; core temperature rose during exposure to 450 MHz.	450 MHz is a resonant frequency in squirrel monkeys.	Adair et al 1997
Colonic and sub-cutaneous temperatures, heart rate, respiratory rate, mean arterial blood pressure in anesthetized rats	350 MHz (sub-resonant) at a whole-body SAR of 13.2 W kg^{-1} until death	Heart rate and mean arterial blood pressure raised during body temperature elevation by 1°C.	Behavioral thermoregulation absent.	Jauchem and Frei 1997
Colonic and sub-cutaneous temperatures, heart rate, respiratory rate, mean arterial blood pressure in anesthetized rats	1 GHz and/or 10 GHz at whole-body SARs of 12 W kg^{-1} until death.	Heart rate and mean arterial blood pressure initially increased in response to different irradiation regimes.	Behavioral thermoregulation absent.	Jauchem et al 2000
High peak power pulses				
Heart rate and mean arterial blood pressure in conscious rats	High peak power pulses: 10 pulses of pulsed 1.7-1.8 GHz; 40-85 ns pulse width at 33-65 MW m^{-2} at 1 pps. Or 10 pulses of pulsed 1.2-1.4 GHz; 40-70 ns pulse width at 146-561 MW m^{-2} at 1 pps.	No significant change in mean arterial blood pressure or heart rate once the acoustic noise associated with each pulse was attenuated.	SAR not given	Jauchem and Frei 1995
Ultra-wideband pulses				
Heart rate and mean arterial blood pressure in anesthetized rats	UWB pulses: 174-218 ps rise time, 0.97-0.99 ns pulse width, 87-104 kV m^{-1} at 50, 500 or 1000 pps for 2 min.	No significant change in heart rate or mean arterial blood pressure.	SAR not given	Jauchem et al 1998
Heart rate and mean arterial blood pressure in anesthetized rats	UWB pulses: 318-337 ps rise time, 6 ns pulse width, 19-21 kV m^{-1} at 1000 pps for 0.5 s or for 2 s alternating with 2 s off for 2 min.	No significant change in heart rate or mean arterial blood pressure.	SAR not given	Jauchem et al 1999

Assay endpoint	Exposure Conditions	Response	Comment	References
Heart rate, systolic and diastolic blood pressure, mean arterial blood pressure in conscious rats at 45 min, 24 h, 72 h, and 1, 2, 3 and 4 weeks after exposure.	UWB pulses: 180 ps rise time, 1.00 ns pulse width, 93 kV m^{-1}, at 500 pps; whole-body SAR of 70 mW kg^{-1}. Or 200 ps rise time, 1.03 ns pulse width, 85 kV m^{-1}, at 1000 pps; whole-body SAR of 121 mW kg^{-1}; for 6 min	No effect on heart rate, but significant delayed decrease in diastolic, systolic and mean arterial blood pressure		Lu et al 1999

II.4.7.4. Summary on cardiovascular system

Cardiovascular system responses to RF radiation, such as changes in heart rate and arterial blood pressure, are consistent with those associated with thermoregulatory responses to conventional heating. In general, an RF induced increase in body temperature elicits several cardiovascular changes, including increased blood flow to the skin, increasing skin thermal conductance, and increased cardiac output, primarily due to an increase in heart rate, in order to maintain arterial pressure within the normal range. Exposure to RF radiation during a fever may increase body temperature above that due to the fever itself if the RF energy is deposited deep within the body, or if the exposure takes places as the fever abates and body temperature begins to fall.

Acute exposure to high peak power pulsed RF or to UWB RF radiation in which the energy per pulse is below the threshold for RF auditory effects does not appear to elicit any changes in the cardiovascular system of anesthetized rats, but one study reported persistent delayed hypotension in conscious rats following brief UWB exposure.

II.4.8. Immunology and hematology

Immune responses serve to protect individuals from infectious disease caused by invading micro-organisms such as viruses, bacteria, and various single-celled or multicellular organisms. They can be grouped into acquired or antigen-specific responses and natural or innate responses, which tend to be less specific. The cells that mediate the acquired responses include B-lymphocytes, which secrete antibodies (humoral immunity) which circulate in body fluids, and the T-lymphocytes, which can function as cytotoxic cells (cell-mediated immunity) or as helper T-cells which assist in B- or T-cell activation. The acquired immune responses also involve the recruitment and amplification of the responses of other, less specific parts of the immune system. These include natural killer cells (large granular lymphocytes), mononuclear phagocytes (monocytes and macrophages), granulocytes (neutrophils, eosinophils, and basophils) and the protein complement system, the latter mediating many of the cytolytic and inflammatory effects of humoral immunity. It is generally accepted that the immune system has considerable redundancy in its various components and regulatory mechanisms such that transient and subtle changes in a few components are unlikely to be of much health significance.

Hematology describes the growth and behavior of the cell populations of the blood. Thus, it encompasses the growth and development of the cell populations of the immune system in addition to the erythrocyte populations. The interpretation of changes to cell population estimates can however be complicated by the migration of some cell groups to different body compartments, such as the lymph system, in response to some physiological changes to the body.

A number of studies of RF effects on immune system responsiveness and on the hematological system were carried out mainly in the 1970s and 1980s. They have been reviewed by WHO (1993) and later by Black and Heynick (2003) and are briefly summarized here along with few more recent studies.

II.4.8.1. Immune system

Studies of immune responses, summarized by WHO (1993), were mostly conducted using 2.45 GHz continuous wave RF. In general, the changes that have been reported with any consistency were usually transient and resulted from acute, thermally-significant exposures. For example, changes in natural killer cell and macrophage activity were reported by several studies after the acute exposure to 2.45 GHz of hamsters at SARs of about 13 W kg^{-1} or of mice at whole-body SARs of around 21 W kg^{-1} (Smialowicz et al 1983; Rama Rao et al 1983; Yang et al 1983). An increase in the primary antibody response of B-lymphocytes has been associated with the exposure of mice to 3.0 GHz at whole-body SARs above 4-5 W kg^{-1} and hamsters to 2.45 GHz at SARs of 8 W kg^{-1} and above (Shao and Chiang 1989; Rama Rao et al 1985). In primates, an enhanced mitogen response was reported in lymphocytes from rhesus monkeys following exposure to 10.5, 19.27 or 26.6 MHz RF radiation between 0.4 and 2 W kg^{-1} (Prince et al 1972). The effects in these studies were associated with transiently increased rectal temperatures (WHO 1993).

With regard to effects on the developing immune system, two studies conducted prior to 1993 by the same group of the pre-natal and postnatal exposure of rats to 2.45 GHz at whole-body SARs of 1-5 W kg^{-1} (Smialowicz et al 1979) or to 425 MHz at 3-7 W kg^{-1} (Smialowicz et al 1982) also reported an increased lymphocyte responsiveness to mitogen stimulation at thermogenic levels. In contrast, a lifetime exposure study in which rats were exposed to pulsed 2.45 GHz at whole-body SARs of up to 0.4 W kg^{-1} between 2 and 27 months of age did not reveal any effects on immunological parameters except for a transient change in the responsiveness of B- and T-lymphocytes to specific mitogens after 13 months exposure (Chou et al 1992).

With regard to studies published after 1993, one group have examined the effect of low-level (whole-body SAR estimated as 2-5 mW kg^{-1}) exposure to 8-18 GHz swept frequency RF on the production of the cytokine tumor necrosis factor (TNF) in the peritoneal macrophages and splenic T-lymphocytes of mice. Fesenko et al (1999) exposed male mice over periods ranging from 0.5 h to 7 days and reported that TNF production was significantly enhanced in both cell types in mice exposed for between 5 h to 3 days compared to sham-exposed mice, and that this persisted over 3 days post-exposure. Following a similar experimental protocol, Novoselova et al (1999) confirmed the increased in TNF production in macrophages and T-lymphocytes following a 5-hour exposure, being maximum about 24 h after exposure in macrophages. T-cell proliferation was also enhanced during this period.

Chagnaud and Veyret (1999) reported a lack of effect on the spleen lymphocyte sub-populations of rats exposed to GSM-modulated RF radiation for 2 h per day for 10 consecutive days at whole-body SARs of 75 mW kg^{-1} or 270 mW kg^{-1}. No effect was seen on the numbers of cells expressing the surface markers CD4+ (helper T-cells), CD8+ (cytotoxic T-cells) or immunoglobulin A (B-cells). In addition, the mitogenic responses of splenic lymphocytes to the mitogen concanavalin-A were unchanged. These authors subsequently investigated the effects of 900 MHz RF exposure at a head SAR of 1.5 or 6 W kg^{-1} over a 21-day period on the onset, duration and termination of experimental allergic encephalomyelitis in rats (Anane et al 2003b). This is a demyelinating auto-immune disease that is often used as model for multiple sclerosis. No statistically significant effects of RF exposure were found compared to the responses of sham-exposed animals.

More recently, two studies by one group evaluated the effects of RF radiation on mouse peripheral lymphocytes and on B cell peripheral differentiation and antibody response in mice (Gatta et al 2003; Nasta et al 2006). Mice were exposed or sham-exposed to GSM 900 RF at whole body SARs of 1 or 2 W kg^{-1} for up to 4 weeks. The first study investigated the effects on T and B lymphocyte frequencies, expression of activation markers (CD28; CD69), cytokine (IL2 and IFNγ) production and T and B cell proliferation (Gatta et al 2003). The second study investigated B cell peripheral differentiation in spleen, and antibody (IgM and IgG) production in response to polyclonal or antigen-specific stimuli (Nasta et al 2006). The authors concluded that T and B lymphocytes were not substantially affected by exposure to RF.

Table II.4.15.: Immune system responses

Assay endpoint	Exposure Conditions	Response	Comment	References
TNF production in macrophages and T-lymphocytes in mice	Swept 8-18 GHz RF SAR ~ 2-5 mW kg^{-1} for 0.5 h to 7 days	Increased TNF production following 5 h to 3 day exposure	Blinded procedures	Fesenko et al 1999
TNF production in macrophages and T-lymphocytes in mice	Swept 8-18 GHz RF SAR ~ 2-5 mW kg^{-1} for 5 h.	Increased TNF production and T-lymphocyte proliferation 24 h after exposure	Blinded procedures	Novoselova et al 1999
Cell surface markers (CD8+, CD4+ and IaAG+) and mitogenic activity of lymphocytes in rats	900 MHz GSM RF at a whole-body SAR of 75 or 270 mW kg^{-1} for 2 h per day for 10 days	No effects on cell surface markers or mitogenic activity	Low-level RF	Chagnaud and Veyret 1999
Experimental allergic encephalomyelitis (EAE) in rats; eight rats per group	900 MHz GSM RF at a brain SAR of 1.5 or 6 W kg^{-1} for 2 h per day for 21 days	No statistically significant effect on onset, duration and termination of EAE crisis.	Cage controls exhibited greatest impairment	Anane et al 2003b
Lymphocyte proliferation, cytokine production and expression of activation markers in mice	900 MHz GSM RF at a whole-body SAR of 1 or 2 W kg^{-1} for 2 h per day for 1, 2 or 4 weeks	No effects on T or B lymphocyte function, except for transient increase seen in IFNγ after 1 week RF exposure.	Blinded procedures	Gatta et al 2003
B-cell peripheral lymphocyte differentiation and antibody production in mice	900 MHz GSM RF at a whole-body SAR of 2 W kg^{-1} for 2 h per day for 4 weeks	No effects on B cell differentiation or on serum antibody levels		Nasta et al 2006

II.4.8.2. Hematology

A large number of studies of effects of RF exposure on hemopoietic tissues and immune function were also carried out prior to 1993 but the results are not always clear; many reports have yielded conflicting data. In addition, some of the older studies suffered from inadequate dosimetry and poor experimental design. As with the immune system, changes that have been reported were usually transient and resulted from acute, thermally-significant exposures (WHO 1993). One response observed by several authors has been the decrease in peripheral lymphocyte count and an increase in the neutrophil count in mice exposed at 26 MHz at whole-body SARs of 5–13 W kg^{-1} (Liburdy 1979) and rats exposed to pulsed 24 GHz RF at whole-body SARs of 1.5–3 W kg^{-1} (Deichmann et al 1959, 1964); sufficient under these particular experimental conditions to raise rectal temperatures by about 1°C. In primates, no field-dependent changes in any of 21 hematological parameters were seen following prolonged (11-day) exposure to 28 MHz at a whole-body SAR of 0.06 W kg^{-1} (Wright et al 1984).

No consistent effect of RF exposure has been seen on peripheral blood cell populations in developing rats (WHO 1993). No consistent changes in erythrocyte, leucocyte or differential leucocyte cell count were found in rats exposed prenatally and postnatally (for up to 41 days) to 2.45 GHz RF at 1–5 W kg^{-1}, at 100 MHz at 2–3 W kg^{-1} or to 425 MHz RF at 3–7 W kg^{-1} (Smialowicz et al 1979, 1981, 1982). In addition, a lifetime exposure study in which rats were exposed to pulsed 2.45 GHz RF at whole-body SARs of up to 0.4 W kg^{-1} between 2 and 27 months of age did not reveal any effects on hematological parameters except for a transient change in the responsiveness of B- and T-lymphocytes to specific mitogens after 13 months exposure (Chou et al 1992).

More recently, the effects of exposure of rats to 2.45 GHz RF at SARs of 1-2 W kg^{-1} for up to 30 days on bone marrow cells and peripheral blood white cells were investigated by Trosic et al (2004a & b). A statistically significant decrease in lymphoblast number in bone marrow cells was observed after 15 and 30 days exposure. Other endpoints, such as the number of lymphocytes and total cells in bone marrow, the number of peripheral blood leukocytes and lymphocytes, were not affected. The same group (Busljeta et al 2004) reported that a similar exposure of rats at 2.45 GHz resulted in increased erythrocyte count, hemocrit and hemoglobin levels after 8 and 15 days of exposure, but not after 30 days exposure. Similarly, the number of erythropoietic precursor cells in bone marrow was decreased after 15 days exposure, but not after other intervals.

Table II.4.16.: Hematological effects

Assay endpoint	Exposure Conditions	Response	Comment	References
White blood cell counts in peripheral blood and in bone marrow in rats.	2.45 GHz at whole body SAR of 1-2 W kg^{-1} for up to 30 days	No effects on cell numbers except for a significant decrease in the number of lymphoblasts in bone marrow		Trosic et al 2004a & b
Erythropoietic precursor cells and erythrocyte count in bone marrow and peripheral blood in rats	2.45 GHz at whole body SAR of 1-2 W kg^{-1} for up to 30 days	Transiently increased erythrocyte counts in peripheral blood; erythropoietic precursor cells in bone marrow transiently decreased		Busljeta et al 2004

II.4.8.3. Summary on immunology and hematology

Few studies of RF-induced effects on the immunological and hematological systems have been carried out since the WHO RF review (WHO 1993), which had concluded that the most consistent changes seen in a relatively large number of studies were transient and were mostly associated with elevated body temperatures and whole-body SARs greater than 4 W kg^{-1}. The more recent studies have been carried out at lower SARs. Two groups reported a lack of RF effects on immune function in mice and rats. In contrast, one group has reported increased expression of the cytokine TNF in mice; another group reported minor changes in the number of bone marrow lymphoblasts and erythropoietic precursor cells in rats. Clearly, these latter studies require some confirmation and corroboration. At present, the conclusion remains that most studies indicate that the most consistently observed RF-induced changes in immune function and hematology are transient and associated with temperature rise of $1^{o}C$ or more.

II.4.9. Skin

The skin lies on the outer surface of the body and is therefore the first tissue to be irradiated by RF radiation emitted by an external source of RF; in addition, the skin has a population of proliferating cells, which might be adversely affected by such exposure. The skin is thus often the organ most exposed to RF radiation, particularly regarding frequencies characteristic of mobile phone signals. Whilst a number of studies have been carried out of the potential of RF radiation to act as tumor promoters (see Chapter II.4.2.), few studies have examined the effects of RF radiation on skin morphology.

In the first of two studies by the same group, Masuda et al (2006) exposed hairless female rats to GSM 900 or 1800 RF radiation for 2 h; the local SARs in the skin near the loop antenna was ~ 6 W kg^{-1} at 900 MHz, and ~ 5 W kg^{-1} at 1800 MHz. In the second study, Sanchez et al (2006b) exposed hairless female rats to the same GSM signals for 2 h per day, 5 days a week for 12 weeks; the local SARs in the skin near the loop antenna were ~ 2.5 W kg^{-1} and ~ 5 W kg^{-1}, respectively. In both studies, no differences were seen in skin thickness, or in filaggrin, collagen and elastin skin content compared to skin taken from an unexposed site on the contralateral side of the body. In addition, the ratio of cells in the epidermis expressing Ki-67, which is a marker for cell proliferation, and basal epidermal cells remained within the normal range. These authors used an exposure to UVB radiation (4 kJ m^{-2}) as a positive control for the

proliferative response. The same authors (Sanchez et al 2008) also assessed the expression of the heat-shock cognate hsc70, and the inducible forms of the heat-shock proteins hsp25 and hsp70 in the skin of rats exposed as above; UVB radiation was used as positive control. There was no difference between sham and exposed groups in hsp expression following either single or repeated exposure.

In contrast to the above data, Ennamany et al (2007) observed a deterioration of stress gene expression in cells from reconstituted skins that had been exposed for 6 h to GSM 900 RF. The SAR level was not given. There were no changes in reconstituted skin morphology, cell apoptosis, or mortality, but from an analysis of the expression of 600 genes, the authors reported that RF exposure induced a modulation of the transcriptomic response in the reconstituted skin, similar to those observed with other stressors. However, a thermal effect cannot be excluded.

Ozguner et al (2004) also reported a number of skin changes following exposure of rats to 900 MHz CW RF radiation at 10 W m^{-2} for 30 min per day for 10 days, including atrophy, an increased thickness of the stratum corneum, impaired collagen distribution and basal cell proliferation. The peak local SAR was cited as 2 W kg^{-1}; unfortunately, so few experimental details were given that it is not clear how this value was derived. In addition, the histological changes were given subjective scores (mild, moderate or severe change) and it was not clear whether the scorer(s) were or not aware of the exposure status of the animals.

Table II.4.17.: Effects on the skin

Assay endpoint	Exposure Conditions	Response	Comment	References
Rats Skin morphology	900 MHz CW 10 W m^{-2} for 30 min per day for 10 days peak SAR: 2 W kg^{-1}	Atrophy, increased thickness of stratum corneum, impaired collagen distribution and basal cell proliferation	Subjective scoring, possibly not blind	Ozguner et al 2004
Hairless female rats and reconstituted skin Skin thickness, filaggrin, collagen and elastin skin content Expression of Ki-67	900 and 1800 MHz GSM (SAR: 6 and 5 W kg^{-1} respectively) 2-h exposure positive control: UVB	No effects		Masuda et al 2006
Same model and assays	Same type of exposure 2 h/day, 5 day/w, 12 weeks	No effects		Sanchez et al 2006b
Same model Expression of Hsc70, hsp25 and 70	Same type of exposure 2 h/day, 5 days/ week, 12 weeks	No effects		Sanchez et al 2008
Reconstituted skin Skin morphology, cell apoptosis and mortality Expression of 600 genes	GSM 900 6 hours	No effects on skin morphology, cell apoptosis and mortality Modulation of transcriptomic response: increased expression of *hsp70* and the *c-myc*, *c-jun*, and *jun-B* proto-oncogenes	No dosimetry	Ennamany et al 2007

II.4.9.1. Summary on skin

The skin lies on the outer surface of the body and is therefore more likely than other tissues to be exposed to external RF fields, including that from mobile phones. Few studies of RF effects on skin have been

carried out. One group has reported an absence of effects of GSM-type mobile phone radiation on a number of different parameters such as skin thickness, elastin and collagen content, proliferative response and the induction of heat-shock proteins. In contrast, two groups reported positive effects, although in both cases there were a number of methodological shortcomings.

II.4.10. Eye

Studies have been carried out on the effects of exposure to RF radiation on the lens of the eye and other tissues including the retina. Many of the early studies carried out in the 1960s and 1970s used rabbits, while later studies tended to use primates because of the greater similarity of their facial and ocular structures to those of humans. These studies have been reviewed by WHO (1993) and more recently by Elder (2003a) and are briefly summarized below, along with a discussion of the evidence from more recent papers.

II.4.10.1. Cataracts

The lens is considered potentially sensitive to RF exposure because it lacks a blood supply and so has a limited ability to dissipate heat. In addition, the fibers which make up the bulk of the lens have only a limited capacity for repair and tend to accumulate the effects of minor insults. Cellular debris resulting from any cytotoxic insult to the lens tends either to be carried to the posterior sub capsular region due to the mechanical forces of epithelial cell proliferation and fiber formation or is trapped *in situ* in the lens matrix.

Briefly, as noted by WHO (1993), cataract is a well-established thermal effect of RF exposure in anesthetized rabbits (e.g., Kramar et al 1975; Guy et al 1975b; Hagan and Carpenter 1976; Kramar et al 1978; Carpenter 1979). High lens temperatures induced by exposure of the head to RF have been shown to induce cataracts in the lenses of anesthetized rabbits (Guy et al 1975b; Kramar et al 1978); threshold temperatures for prolonged (100-200 min) exposure lie between 41-43^{o}C; corresponding local SARs are in the range 100-140 W kg^{-1}. These high local SARs and temperatures resulted from protracted (>140 min) localized exposure of the eye at 1- 10 GHz at power densities greater than 1.5 kW m^{-2}; whole-body exposure at such levels however is limited by thermal stress (Elder 2003a). The few experiments which have investigated the effect of chronic whole-body exposure of conscious rabbits at lower power densities (up to 100 W m^{-2}) reported a lack of effect on the lens. Cataracts were not observed in rabbits after 2.45 GHz RF radiation at 100 W m^{-2} (whole-body SAR of 1.5 W kg^{-1}) for up to 17 weeks (Ferri and Hagan 1976). Nor was any change found by in the eyes of rabbits exposed for ~ 6 months at 2.45 GHz where the maximal SAR in the head was 17 W kg^{-1} (Guy et al 1980). Chou et al (1982, 1983) also reported that low-level pulsed or CW 2.45 GHz RF exposures for 3 months at SARs of 0.55 and 5.5 W kg^{-1} in the head did not cause cataracts.

These early studies also found primates to be less susceptible to cataract induction than rabbits (WHO 1993). Opacities were induced in the eyes of anesthetized rhesus monkeys after acute localized exposures of up to 5 kW m^{-2}, well above threshold levels for anesthetized rabbits (McAfee et al 1979; 1983). In addition, McAfee and colleagues exposed conscious monkeys to 2.45 GHz CW for up to 12 h over a 4 month period or to pulsed 9.3 GHz RF radiation (pulsed or CW) for up to 15 h over a 34 month period at SARs in the head of up to 40 W kg^{-1}. Eye examinations carried out 1-4 years after exposure revealed no effects on the lens, cornea or retina. The lower susceptibility of primates to cataract induction is thought to result from structural differences in the eyes and skull of the two species resulting in lower power absorption and heating of the thinner primate lens.

More recently, Saito et al (1998) exposed the eyes of conscious rabbits for ~ 2.5 – 4 h at 2.45 GHz at an SAR to the head/eye of 26.5 W kg^{-1}, with the contralateral eye serving as a control, and reported transient conjunctival and corneal edema, contraction of the pupil and pupilliary congestion, and fibrinogenesis in the anterior chamber of the lens of the exposed eyes. In contrast to studies with anesthetized rabbits, using higher local SARs, the authors did not observe cataracts. Studies with both conscious and anesthetized rabbits have been carried out. Kojima et al (2004) assessed the effects of localized exposure of rabbit eyes for 20-60 min to 2.45 GHz RF at a local SAR to the eye of 108 W kg^{-1}; the RF-induced changes, which

disappeared within a week, included corneal edema, inflammation of the iris and increased light-scattering from the anterior cortex of the lens. These effects were much more marked in the anaesthetized rabbits than in those not anesthetized; reflecting the greater temperatures (of up to 9^{o}C) measured in the posterior (vitreous) chamber and to a lesser extent in the anterior (aqueous) chamber of the eyes of the anesthetized rabbits. Increased heating of the posterior region of the lens, particularly in anesthetized rabbits due to reductions in blood flow, was confirmed in dosimetric and thermal modeling studies by Hirata et al (2006).

Balci et al (2007) placed 900 MHz GSM phones over cages each housing 10 rats. The phones, on standby, were called intermittently (4 times a day for 10 min) over a 4 week period. There was no RF dosimetry. The authors reported a number of effects. Unfortunately, the absence of proper dosimetry and the poor description of the experimental protocol render the results uninterpretable.

Table II.4.18.: Effects on the eye

Assay endpoint	Exposure Conditions	Response	Comment	References
Cataract formation and other ocular effects in anesthetized rabbits.	2450 MHz (CW) at 150 mW/cm^2 for 100 min at the maximum SAR 138 W kg^{-1} in the eye.	Induce cataract and transient effects (papillary constriction and anterior chamber turbidity).		Guy et al 1975b
Cataract formation in anesthetized rabbits.	2450 MHz CW at 1.8 kW m^{-2} for 140 min at maximum SAR in the eye 100 W kg^{-1}.	Cataract.	This study was performed to determine the cataractogenic threshold.	Kramar et al 1978
Ocular effects in conscious monkeys.	9310 MHz (PW) at 1.5 kW m^{-2} for 30-40 days with 294-665 min totally.	No ocular effects such as cataract after one year observation.		McAfee et al 1979
Ocular effects such as cataract, visual capability loss in conscious monkeys.	9310 MHz (PW) at 1.5 kW m^{-2} for 408-946 min over 34 months; 9310 MHz (PW) at 3 kW m^{-2} for 275-594 min over 34 months; 2450 MHz (PW) at 1.5 kW m^{-2} for 549-750 min over 4 months.	No cataracts; no effects on cornea, aqueous and vitreous humors or retina; and no loss of visual capability 4 years after 9310 MHz exposure and 1 year after 2450 MHz exposure.	These results support that clinically significant ocular effects have not been confirmed in human populations exposed for long period of time to low level RF radiation.	McAfee et al 1983
The effect of CW irradiation on conscious rabbit eye including cornea, anterior chamber, lens, and other ocular tissues.	2450 MHz (CW) on the eyes at SAR 26.5 W kg^{-1} for 160 to 240 min.	No cataracts. Other effects (miosis, keratoleucoma and corneal edema, endothelial cell detachment and floating in aqua oculi, and so on)	The miosis and papillary congestion in all irradiated eyes was first to be detected.	Saito et al 1998
Effects of acute RF exposure on ocular tissue in conscious and anesthetized rabbits	2.45 GHz at localized SAR of 108 W kg^{-1} for 20-20 min.	Corneal edema, inflammation of the iris and increased light-scattering from anterior lens cortex.	Changes more marked in anesthetized animals.	Kojima et al 2004
Oxidative stress in lens tissues in rats.	900 MHz GSM mobile phone signal for 4 weeks; the phone was placed above the cage.	Malondialdehyde level significantly increased in lens and corneal tissue in the mobile phone group compared to controls.	The absence of any RF measurements of any kind renders the experiment uninterpretable.	Balci et al 2007

II.4.10.2. Other ocular tissues

Degenerative changes have been reported in various eye tissues of primates after exposure to pulsed microwaves. A series of studies (summarized by Kues & Monahan 1992) have indicated that localized exposure of the eyes of anaesthetized monkeys to pulsed 2.45 GHz RF (10 μs pulses at 100 pps) at an SAR in the eye of 2.6 W kg^{-1} or more for four hours resulted in transient lesions in the corneal endothelium (Kues et al 1985). These were maximal 16-24 h post-exposed and persisted for several days. Such lesions in the cornea were also induced by exposure to CW 2.45 GHz, but less effectively compared to pulsed radiation. Topical pretreatment with the ophthalmic drug timolol maleate (used in the treatment of glaucoma) appeared to reduce the threshold to a localized SAR of 0.26 W kg^{-1} (Kues et al 1992). In addition, the authors reported a transient increase in the vascular permeability of the iris (blood-aqueous barrier) following similar treatment. In studies by the same group but using conscious monkeys, transient reductions in electroretinogram activity in response to light stimulation have been reported following repeated exposures to pulsed (0.5 μs pulses, 16 pps) 1.25 GHz microwave radiation at a localized SAR of 4.0 W kg^{-1} (Kues & Monahan 1992). Histopathalogical investigation three weeks after exposure revealed photoreceptor degeneration, which, the authors argue, is consistent with the observed decrements in electroretinogramme activity.

In contrast to these studies, Kamimura et al (1994) reported that they were unable to induce corneal, lenticular or retinal lesions in the eyes of conscious monkeys exposed to CW (but not pulsed) 2.45 GHz radiation at levels exceeding the threshold for CW-induced corneal damage described by Kues et al (1985). The technique used for the identification of corneal lesions (specular microscopy) was the same as that used by Kues et al (1985); although the latter authors used histological techniques to confirm damage to both the cornea and retina, in contrast to Kamimura et al (1994). However, Kamimura and colleagues note that the use of anesthesia by Kues et al may have compromised heat dissipation in the eye (see above) increasing susceptibility to RF heating.

Further studies using unanesthetized monkeys by Lu et al (2000) were unable to confirm these earlier observations of Kues and colleagues. Lu et al (2000) exposed or sham-exposed monkeys to pulsed 1.25 GHz over a 3 week period at localized SARs averaged over the retina of 4.3, 8.4 or 20.2 W kg^{-1}. RF-induced changes in the retina were examined using various measures of retinal integrity including fundus photography, fluorescein and indocyanine green angiography, and electroretinography both before and after exposure, and complete retinal histopathology following termination of the experiment. No significant changes were seen in the exposed eyes compared to those pre- or sham-exposed either in the appearance of the fundus or in the angiography examinations. The electroretinogramme response of cone photoreceptors to light flash was enhanced in monkeys exposed at retinal SARs of 8.4 or 20.2 W kg^{-1}, but not in those exposed at 4.3 W kg^{-1}. The authors suggest that this effect is likely to represent a transient physiological change. Histopathologic examination did not reveal any pathological changes. However, an increase in glycogen storage was seen in the photoreceptors in eyes exposed at 8.4 and 20.2 W kg^{-1} and, confusingly, also in sham-exposed animals but not those exposed at 4.3 W kg^{-1}.

Table II.4.19.: Effects on other ocular tissues

Assay endpoint	Exposure Conditions	Response	Comment	References
Corneal endothelium of anesthetized monkeys	CW 2.45 GHz at localized SAR of 5.3-7.8 W kg^{-1} (200-300 W m^{-2}) or pulsed 2.45 GHz (10 μs at 100 pps) at localized SARs of 2.6 W kg^{-1} for 4 h per day, once or repeated over 4 days.	RF-induced transient lesions in the corneal endothelium. Pulsed RF more effective than CW.	Animals used for repeat experiments, separated by 1 week or more.	Kues et al 1985

Assay endpoint	Exposure Conditions	Response	Comment	References
Corneal endothelium and vasculature of the iris in anesthetized monkeys with or without timolol maleate application	Pulsed 2.45 GHz (10 µs at 100 pps) at localized SARs of up to 4 W kg^{-1} for 4 h per day for 3 consecutive days	Timolol pretreatment reduced the threshold for coneal lesions to 0.26 W kg^{-1} and increased vascular leakage from the iris		Kues et al 1992
Scotopic test (rod photoreceptor response) and 30-Hz flicker test (cone-receptor response) of electroretinogram (ERG) in conscious monkey	Pulsed 1.25 GHz (0.5 µs at 16 pps) at a localized SAR of 4 W kg^{-1} for 4 h.	60% reduction in scotopic ERG amplitude and 90% reduction in flicker test ERG correlated with photoreceptor degeneration		Kues and Monohan 1992
Corneal endothelium, lens, vitreous humor and retina in conscious monkeys	CW 2.45 GHz at up to 430 W m^{-2} for 4 h.	No abnormalities of the corneal epithelium or lens; or of vitreous humor or retina	Attempted corroboration of Kues et al 1985 with CW 2.45 GHz using supra-threshold exposures	Kamimura et al 1994
Ocular tissues of anesthetized rabbits and monkeys	60 GHz at 100 W m^{-2} for 8 h, or for 4 h on 5 consecutive days. SAR not given	No histopathalogical effects seen in cornea, iris, or lens		Kues et al 1999
Ocular tissues of conscious monkeys exposed to high peak power RF pulses	1.25 GHz pulsed (5.6 µs at up to 2.8 pps); retinal average SARs of up to 20 W kg^{-1} (peak of 130 MW kg^{-1} per pulse), for 4 h per day for 9 days over a 3 week period.	No histopathological effects seen; transient functional changes in electroretinograms and increased photo-receptor glycogen storage seen above 4.3 W kg^{-1}.	Increased glycogen storage also seen in photoreceptors of sham-exposed group.	Lu et al 2000

II.4.10.3. Summary on eye

The lens of the eye is potentially sensitive to RF because it lacks a blood supply and so has a limited ability to dissipate heat. RF-induced cataract is a well-established thermal effect of RF exposure in anesthetized rabbits; thresholds for prolonged (100-200 min) exposure lie between about 41-43^{o}C, corresponding to localized SARs in the range 100-140 W kg^{-1}. However, recent studies have confirmed that the anesthesia restricted lenticular cooling through a reduction in local blood flow, thereby exacerbating the effects observed. Primates appear less susceptible to cataract induction than rabbits, and opacities have not been observed following either acute or prolonged exposures.

Studies from one laboratory suggesting that the exposure of the eyes of anesthetized primates to pulsed RF could result in corneal lesions and vascular leakage from the iris were not corroborated by later studies by other groups using conscious primates. Transient changes were seen in the electroretinogramme responses following exposure at high localized SARs, but the functional significance of this, if any, was not clear.

II.4.11. Summary on animal studies

Overall, studies published after 1993 provide a further support for the conclusions of WHO (1993) that the most consistent and reproducible responses of animal to acute RF exposure result from RF-induced heating. These studies established that, in general, an increase in body temperature elicits several

cardiovascular changes including increased blood flow to the skin, increasing skin thermal conductance, and increased cardiac output, primarily due to an increase in heart rate, in order to maintain arterial pressure within the normal range. Deficits in learned behaviors, particularly the disruption of ongoing operant behaviors, occur mainly when core temperatures are increased by about 1°C or more. Similar rises in body temperature also result in significantly enhanced plasma corticosterone or cortisol levels in rodents and primates and transient changes in immune function and hematology, generally consistent with the acute responses to non-specific stressors.

In addition, RF radiation can cause increased embryo and fetal losses, increased incidence of fetal malformations and anomalies, reduced fetal weight at term and impair male fertility at exposure levels that are sufficiently high to cause a significant increase in temperature. To date, there is no consistent evidence of effects at non-thermal exposure levels. Relatively few studies have evaluated possible effects of prenatal exposure on postnatal development; results from such studies have not shown consistent effects on developmental indices or behavior at exposure levels that do not induce significant temperature elevation. The few studies that have addressed neonatal exposure or exposure of juvenile animals to low level RF have generally reported a lack of effect on such diverse endpoints such as behavior, blood-brain barrier permeability and tumor induction. However, to date, there remains insufficient evidence to form a firm conclusion regarding neonatal or juvenile sensitivity to RF compared to adults.

RF-induced cataract also remains a well-established thermal effect of RF exposure in anesthetized rabbits. However, recent studies have confirmed that the anesthesia-restricted lenticular cooling through a reduction in local blood flow, thereby exacerbating the effects observed. Primates appear less susceptible to cataract induction than rabbits, and opacities have not been observed in primates following either acute or prolonged exposures. Studies from one laboratory suggesting that the exposure of the eyes of anesthetized primates to pulsed RF could result in corneal lesions and vascular leakage from the iris were not corroborated by later studies by other groups using conscious primates.

Overall, the results of recent carcinogenicity studies are rather consistent and indicate that carcinogenic effects on rodents are not likely at SAR levels up to 4 W kg^{-1} even for long-term exposure. Genotoxicity studies also generally indicate a lack of effect. A notable positive finding was of a two-fold increase in lymphoma incidence in a strain of lymphoma-prone transgenic mice following exposure at 900 MHz with a signal similar to that used in GSM mobile phones. However, this finding was not confirmed in two subsequent replication and extension studies. In addition, studies report an absence of effects of RF radiation characteristic of mobile phone use on melatonin levels.

Studies of the behavioral effects of high peak power RF pulses used in some military applications have been rather sporadic and diverse; pulse widths have varied by two orders of magnitude and the specific absorption per pulse by four orders of magnitude. Two studies have shown the equivalence of pulsed RF to an acoustic ‘click’ in affecting the startle reflex. Otherwise, the effects seen may be attributed either to heating or auditory perception.

II.5. HUMAN STUDIES

Prior to 1993, laboratory studies using volunteer were confined primarily to studies of cutaneous and auditory perception and effects resulting from localized and whole body heating (WHO 1993). Guy et al (1975c), for example, determined a threshold for the auditory perception of pulsed RF as used in radar as 16 mJ kg^{-1} energy absorption per pulse in the head. With regard to the effects of RF absorption by the whole body, this was addressed largely in the context of thermoregulation. It was known that healthy individuals can sustain an increase in body temperature up to an upper safe limit of 39°C, at which level the heart rate is considerably elevated and the sweat rate is about 1 liter per hour (WHO 1993). In addition, early studies on the exposure of patients and volunteers to RF fields in magnetic resonance imaging systems reported that whole-body SARs of up to 4 W kg^{-1} for 20-30 minutes resulted in body temperature increases in the range 0.1-0.5°C (eg Kido et al 1987; Shellock and Crues 1987; Shellock et al 1989).

In subsequent years, the rapid increase in wireless telecommunications, particularly those used in mobile telephony, initiated a number of research programs which included volunteer studies of the possible

physiological effects of the complex but generally low level RF emitted by such devices. In particular, the proximity of mobile phones to the head raised public concern about a potentially toxic effect of electromagnetic radiation on the central nervous system which has prompted a large number of studies to be carried out. These include investigations of mobile-phone type RF radiation on the electrical activity of the brain and regional cerebral blood flow (rCBF), a marker of neural activity in a local brain region, and on various cognitive functions such as memory, attention and concentration. A number of these studies have been reviewed by Cook et al (2006) and Valentini et al (2007), as well as by the major reviews cited in the Introduction. In addition, a variety of subjective symptoms such as headaches, fatigue, etc., have been reported by some users of mobile phones. Finally, some studies have examined possible effects on the endocrine system, particularly in relation to melatonin, and on the cardiovascular system. With regard to the latter, as indicated above, thermoregulatory responses to heat stress and to RF radiation are well understood and are briefly summarized here.

Experiments using volunteers exposed to RF are restricted for ethical reasons to the investigation of transient physiological phenomena which, in the controlled conditions of a laboratory, are at relatively low exposure levels. It is possible, however, that effects judged to be harmless when experienced transiently in the laboratory, may have adverse health consequences if experienced for long periods in an occupational or public context. The advantage of such experiments is that they indicate the likely response of other people exposed under similar conditions, but the disadvantages include the often short duration of investigation and the small number of subjects usually examined. To some extent, shortcoming such heterogeneity in the study population can be addressed through experimental design, in this example by using a crossover experimental design (see below), or retesting of participants to account for possible differences in response. However, due to practical considerations, subjects have tended to be relatively homogeneous and are therefore unlikely to reflect the range of variability encountered within a population. Nevertheless, within this limited context, volunteer studies can give valuable insight into the physiological effects of exposure in normal, healthy people.

Important factors to consider in the evaluation of these studies include the use of double-blind procedures and crossover and counter-balanced protocols. Double-blind procedures apply when both the experimenters and subjects are unaware of the exposure status of the subjects, and so are less likely to be influenced by any expectation of a particular outcome; single-blind procedures, often used in early studies, are where only the subjects are unaware of their exposure status. A crossover design is where subjects are both exposed and sham exposed in different parts of the experiment, so that they act as their own controls (also known as a within-subjects or repeated measures design). This procedure minimizes the effects of intrinsic differences between subject groups, such as might occur between a sham group and an exposed group, which could affect the experimental outcome. A counter-balanced protocol is where all possible orders of exposures are used, with equal numbers of subjects experiencing each order. This counteracts any effect of time-dependency on the subjects' responses, resulting for example from improving in task performance or from loss of attention during the course of a study.

II.5.1. Nervous system

II.5.1.1. Electrical activity of the brain

The electroencephalogram (EEG) is a reflection of synchronous activity in relatively large populations of cortical neurons. The 'spontaneous' EEG of awake subjects is conventionally divided into a number of frequency bands, the relative amounts of activity in which depends upon the psychological state of the subject and the nature of the cognitive function in which he or she is engaged. The designation of the frequency bands is not always applied very strictly, which results in specific frequencies sometimes being assigned to different bands in different studies. Generally, the following division is used: delta (δ) < 4 Hz; theta (θ) 4-8 Hz; alpha (α) 8-12 Hz; beta (β) 12-30 Hz; gamma (γ) > 30 Hz. Slightly different band designations are used by some authors which are also cited in this report. The functional significance of these different components of the normal, waking EEG is poorly understood. Thus, while a demonstration that mobile phone signals influenced these components would be indicative of a biological effect of such

signals, interpretation of the effect in terms of health would be uncertain. In addition, intra-individual variability is very high. However, EEG patterns associated with sleep are well characterized and routinely used as indices of the different sleep stages that a typical healthy individual will move between during the night. There would also be little uncertainty in the interpretation of a change from a normal to a frankly pathological pattern of EEG activity, such as might be observed in epilepsy.

A measure of brain function closely related to the EEG is the 'evoked' or 'event-related' potential (ERP). ERPs are obtained by sampling the EEG time-locked to a reference event such as the presentation of a stimulus or the onset of a motor response, and averaging the samples together so as to obtain an electrical waveform that represents brain activity associated specifically with that class of event. ERPs are commonly used to study the timing and functional integrity of neural systems supporting sensory, cognitive and motor processing. Nevertheless, interpretation is still problematic, since changes in arousal and attention of volunteers can substantially affect the outcome of these studies.

Spontaneous EEG

Laboratory studies investigating the effects of mobile phone signals on the spontaneous EEG have produced somewhat mixed results, although more recent stronger studies point to the existence of effects of exposure primarily to the alpha bands of the EEG.

Reiser et al (1995) reported from a single-blind, sham-controlled crossover experiment that a 15-min exposure to a signal from a GSM mobile phone was associated with an increase some 15 minutes later in the power of EEG frequencies in the 18-35 Hz (defined here as the upper beta) band. The effect is only marginal and the statistical analysis of the data is questionable. Röschke and Mann (1997) were unable to detect any differences in EEG spectra related to exposure to GSM signals. These authors exposed 34 male volunteers in a single blind design to the signal of a GSM mobile phone positioned at 40 cm from the vertex. The power density at the location of the head was 0.5 W m^{-2}. Exposure or sham exposure was for 3.5 min midway during a 10-min EEG recording session. No effect on the EEG was detected and no distinction could be made between sensitive and non-sensitive groups. Hietanen et al (2000) recorded resting EEG from 19 volunteers during sham exposure, and exposure to signals from five different mobile handsets (analogue and GSM at 900 and 1800 MHz) operating at full power and positioned over the left side of the head. Conditions were single blind. Statistical analysis of spectral parameters of the EEG revealed an effect in only absolute but not relative power in one frequency band in one of four brain regions investigated, for one of the analog phones. The authors attributed this to chance.

Lebedeva et al (2000) recorded EEG from 24 subjects during sham exposure and exposure to a 900 MHz signal directed at the back of the head. An index representing the ‘dimensional complexity’ of the EEG signals was reported to vary significantly as a function of exposure condition, with a more pronounced difference between exposure and sham under ‘eyes-closed’ conditions than under ‘eyes open’. The authors concluded that their chosen measure of EEG was more sensitive to the effects of RF signals than conventional indices. A definition of their index and a comparison with conventional indices was not provided, however. There is also almost no information about how the data were analyzed statistically, and no information about levels of statistical significance.

Borbély et al (1999) reported that exposure to a 900 MHz ‘pseudo GSM signal’ immediately prior to sleep increased resting EEG power in the 11-11.5 Hz range only. Exposure and sham were double-blind, randomized and given at 1-week interval, and only 14 of the total sample of 24 participants had sufficient data for the analysis. An extended analysis was provided in Huber et al (2003). In a follow-up study, Huber et al (2000) exposed 16 healthy volunteers for 30 min to a GSM 900 signal immediately before sleep. Resting EEG prior to sleep was reported to be reduced in the 10.5-11 Hz range. An extended analysis of these data was also provided in Huber et al (2003). Huber et al (2002) investigated the effects of GSM 900 signals resembling that of a handset, and of a CW 900 MHz signal on waking EEG. The left side of each subject’s head was exposed to each of these signals for 30 minutes on 3 separate evenings at weekly intervals, before they went to sleep. Power in the alpha band was found to increase for pulse-modulated but not CW exposure.

Freude et al (2000) performed a single-blind study, where subjects were exposed for the duration of a series of cognitive tests to a signal from a GSM 900 phone. Analysis of the EEGs revealed a decrease of EEG power in all regions except frontal during a visual monitoring task. These effects were stronger in the exposed hemisphere. Croft et al (2002) exposed 24 volunteers to GSM mobile phone type RF radiation and recorded the spontaneous EEG and subsequently phase-locked responses from the EEG during the performance of an auditory discrimination task (results briefly summarized below). The study was single-blind and counterbalanced with a crossover design. Spectral analysis of the spontaneous EEG revealed decreases in the theta (4-8 Hz) EEG frequency band and increases in the alpha (8-12 Hz) frequency band. However, the strength of the dosimetry in that study has been subsequently questioned by the investigators (Croft et al 2008).

D'Costa et al (2003) made EEG recordings from 10 subjects during exposure to a GSM phone positioned behind the head, the antenna pointing towards the head. Two experimental trials were conducted. In the first trial, the GSM phone had its speaker disabled and was configured to transmit at full power. In the second trial, the mobile phone was in active standby mode. For each trial, subjects were exposed under single-blind conditions in 5-min intervals to a randomized, interrupted sequence of 5 active and 5 sham exposures. The average EEG band power in active exposure recordings was compared to the corresponding sham recordings. The EEG alpha (8-12 Hz) and beta (13-30 Hz) bands showed significant differences when the full power mode was on. However, it is difficult to directly compare these results to others as a unique recording method was employed which assessed the 8-12 Hz fluctuations of the 'difference between EEG activity in left and right hemispheres', rather than fluctuations of the activity itself, and no control for type I error was employed although numerous statistical tests were conducted.

Kramarenko and Tan (2003) recorded EEG changes during the exposure of adults and children (12 years old) to a GSM phone on standby. They claim to have suppressed the interference caused by emission from the phone by transmission of the EEG signal by telemetry. They observed changes in EEG patterns: after 20-40 s, a slow-wave delta (2.4-6.0 Hz) appeared in areas on the side of the phone, in periods lasting several seconds. After turning off the mobile phone, slow wave activity disappeared. They observed similar changes in children, but the slow-waves with lower amplitude (1-2.5 Hz) appeared earlier in children. According to the authors, these results suggest that cellular phones may induce abnormal slow waves in the EEG of conscious subjects. However, the dosimetry was not well described and the transmission of the signal by telemetry raises doubts about the interpretation of this study. Also the study appears not to have been performed blinded to the subjects, no sham exposure was performed, and no details of appropriate statistics are provided.

Hinrikus et al (2004) exposed 20 healthy volunteers in a single-blind setup to 450-MHz microwaves with 7-Hz on-off modulation. RF stimulation caused changes in the EEG in the frontal region which varied strongly from subject to subject but overall were not statistically significant.

In a study comparing effects in males and females, Papageorgiou et al (2004) exposed healthy volunteers to a GSM-like signal and measured the EEG during the initial anticipatory phase of a memory test. They observed that the baseline EEG power was greater in males than in females and that exposure decreased the power in males and increased it in females. They found no effect of exposure on performance in the memory test. This study suffered from a lack of adequate details of the experimental setup; the exact type of signal and the level and duration of exposure are not given. It is also not clear whether any blinding was observed. Moreover, no actual EEG data are presented, only the differences in overall EEG energies, and these EEG changes are not comparable to those in the other studies, as it was recorded while participants performed a cognitive task.

Curcio et al (2005) used a GSM 900 phone to expose 20 volunteers for 45 min under double-blind conditions. In half of the subjects they measured the EEG after completion of the exposure, in the other half during the last 7 minutes of exposure. They observed a small increase in some frequencies in the alpha band, which was stronger when measured during exposure than after. Maby et al (2006a) exposed healthy volunteers and epileptic patients to a signal from an undefined GSM mobile phone (single blind). In the healthy volunteers they observed a decrease in EEG power in the theta, alpha and beta bands and a decrease in the variations in the delta band. In contrast, in the epileptic patients an increase in power in all EEG bands was observed. Although the authors provide an elaborate description of the methods of

analysis of the EEG signals, they fail to give sufficient details on the experimental design. Regel et al (2007a) assessed the EEG in awake volunteers in a double-blind counterbalanced exposure setup. They used 900 MHz signals, either GSM-type, or CW, applied for 30 min from a planar antenna. The EEG was recorded immediately and 30 and 60 min after exposure during both eyes-closed and eyes-open conditions. An increase in alpha band activity was observed 30 min after exposure to the pulsed signal with the eyes-closed condition. No effects were seen at other times, neither with eyes open nor after the continuous signal.

A replication of the study by Huber et al (2002) described earlier was published by Perentos et al (2007). They performed an effectively single blind study on healthy volunteers of changes in four specified EEG bands resulting from exposure to either a signal similar to that generated by a 900 MHz GSM mobile phone or a 900 MHz continuous wave. No effect of either type of signal on any EEG band was observed. The authors suggest that the failure to replicate the Huber et al (2002) study might be associated with the very small sample size (n=10) or the differences in exposure pattern. Whereas in the current study a modified phone was used, Huber et al (2002) used a patch antenna, effectively exposing a larger area of the brain.

Croft et al (2008) exposed 120 adult volunteers in a double-blind counterbalanced crossover design to an 875 MHz GSM phone and assessed the EEG in the first and last 10 min of a 30-min exposure. The phones were positioned on either the left or the right side of the head. Comparisons were made between ipsi- and contra-lateral effects and anterior and posterior scalp regions. An increased power in the alpha band was found which was larger on the ipsilateral compared to the contralateral side in posterior regions. This is a well-performed study, with a large number of participants, appropriate control of a number of variables and adequate statistics.

Table II.5.1.: Electrical activity of the brain: spontaneous EEG

Assay endpoint	Exposure Conditions	Response	Comment	References
EEG in awake healthy volunteers (n=36)	Mega-wave 150/1 therapy instrument (9.6 Hz pulsed 150 kHz) or 904 MHz mobile phone, 40 cm behind head; output power 8 W EEG for 1 h, exposure during 2nd quarter	Mega-wave: increase in power in alpha (9.75-12.5 Hz) and beta bands (12.75-35 Hz); mobile telephone: increase in beta2 power (18.75-35 Hz) after 15 min delay.	Measures taken to protect against interference. Unclear statistics.	Reiser et al 1995
EEG in awake healthy volunteers 21-35 y (n=34)	GSM at 40 cm from vertex; power density 0.5 W m^{-2} EEG 2 x 10 min; exposure or sham for 3.5 min midway; awake, eyes closed	No effect of exposure; no sensitive subgroup detected.		Roschke and Mann 1997
EEG immediately prior to sleep in healthy volunteers 20-25 y (n=14)	GSM signal, base station-like, 900 MHz, 2, 8, 217, 1736 Hz modulation, 3 antennas 30 cm from the head; max SAR: 1 W kg^{-1} EEG continuous; awake, eyes closed	Increase in power for 11-11.5 Hz band.	Measures taken to protect against interference.	Borbély et al 1999

Assay endpoint	Exposure Conditions	Response	Comment	References
EEG immediately prior to daytime sleep in healthy volunteers 20-25 y (n=16)	GSM signal, base-station-like,900 MHz, 2, 8, 217, 1736 Hz modulation, planar antennas, peak SAR: 1 W kg^{-1} Exposure for 30 min before sleep	Reduction in power for 10.5-11 Hz band.	Measures taken to protect against interference.	Huber et al 2000
EEG in awake healthy volunteers 28-57 y (n=19)	Analog and digital mobile phones 1 cm from head; peak 1-2 W EEG 5 x 30 min; exposure or sham for 20 min midway; awake, eyes closed	No effect, except for difference in absolute (but not relative) power in delta band of 1 of 4 brain regions with one analog phone.	Measures taken to protect against interference.	Hietanen et al 2000
Evoked potentials in healthy volunteers; exp.1: 21-30 y (n=20); exp.2: 21-26 y (n=19)	916 MHz GSM phone, SAR: 0.88 W kg^{-1} EEG recording during exposure	No effect on performance; decreased EEG power in central and parieto-tempero-occipital regions, stronger in exposed hemisphere.	No correction for interference.	Freude et al 2000
EEG in awake healthy volunteers 20-30 y (n=24)	Mobile phone signal to back of head EEG 60 min, exposure after 15 min for 15 min; change eyes open / closed every 5 min	Change in index representing EEG 'dimensional complexity'; larger with eyes closed than with eyes open.	EEG machine shielded. Validity of used index uncertain because of lack of definition.	Lebedeva et al 2000
EEG in adult awake healthy volunteers 19-48 y (n=24)	900 MHz GSM phone; 20 min EEG during 4x2 min resting (eyes open) + 3 min auditory task	Decrease in theta and increase in alpha band for resting EEG, progressing with exposure time. Decrease in theta and beta and increase in gamma activity during auditory EEG.	No testing for interference.	Croft et al 2002
EEG immediately prior to daytime sleep in healthy volunteers 20-25 y (n=16)	GSM signal, mobile phone-like, 900 MHz, 2, 8, 217, 1736 Hz modulation, planar antennas, SAR: 1 W kg^{-1} Exposure for 30 min before sleep	Increased power in alpha band prior to sleep for pulse-modulated but not continuous RF field.	Measures taken to protect against interference. Spectral power of modulation higher than in base station-like signal from previous studies (Borbély et al 1999, Huber et al 2000)	Huber et al 2002

Assay endpoint	Exposure Conditions	Response	Comment	References
EEG in awake healthy volunteers 18-30 y (n=10)	900 MHz GSM, antenna pointed at the back of the head; full power (250 mW) or standby EEG 10 x 5 min, random exposure / sham, at 10-15 min intervals	Significant decrease in alpha (8-12 Hz) and beta (13-30 Hz) bands power with full power mode only.	No interference found upon testing.	D'Costa et al 2003
Awake EEG in awake healthy adults (n=10) and children, 12 y (n=10)	900 MHz GSM, 100 MHz radio Awake, eyes open Wireless EEG recording system	Appearance of seconds-long periods of slow waves (2.5-6.0 Hz in adults; 1.0-2.5 Hz in children) 20-40 sec after turning on phone.	Incomplete experimental details; no statistics. No exposure-sham comparison.	Kramarem-ko and Tan 2003
EEG in awake healthy volunteers 19-23 y, (n=20)	450 MHz,7 Hz modulated; SAR: 0.0095 Wkg^{-1} EEG during 10 60-sec periods, at 60-sec intervals; awake, eyes closed	Changes in alpha waves, high inter-individual variability, overall not significant. Less changes in theta waves. Effects increase in subsequent exposure periods.	No mention of measures taken against interference.	Hinrikus et al 2004
EEG during memory task in healthy volunteers 23.3±2.2 y; males (n=9) and females (n=10)	900 MHz GSM signal, mean power 64 mW EEG recording during anticipatory period of memory test	Baseline EEG power greater in males than in females; exposure decreased power in males and increased power in females. No effect on memory.	Incomplete description of experimental design.	Papageorgi ou et al 2004
EEG in awake healthy volunteers 22-31 y (n=20)	900 MHz GSM at max power; SAR: 0.5 Wkg^{-1}; 45 min EEG after or during last 7 min of exposure; during EEG eyes closed	Increased power in alpha band (9-10 Hz).	No mention of measures taken against interference.	Curcio et al 2005
EEG in healthy volunteers (n=9) and epileptic patients (n=6)	GMS mobile phone signal EEG before and during exposure or sham	Healthy subjects: decrease in variation in delta band; decrease in power in theta, alpha and beta bands. Epileptics: power increase in all bands.	Incomplete description of experimental design.	Maby et al 2006a
EEG in awake healthy volunteers19-25 y (n=24)	900 MHz, GSM pulsed or continuous, 30 min, SAR:1 W kg^{-1}; planar antenna EEG immediately, 30 min, 60 min after exposure, during 3 min eyes closed and 3 min eyes open	Increase 10.5-11 Hz power 30 min after GSM with eyes closed only; no effects continuous signal		Regel et al 2007a

Assay endpoint	Exposure Conditions	Response	Comment	References
EEG in awake healthy volunteers19-32 y (n=12)	900 MHz GSM and continuous wave signals, peak SAR: 1.56 W kg^{-1} Recording during 2-h period that included 3 15 min exposure periods; only pre- and postexposure analyzed	No effects of either pulsed or continuous exposure.	Replication of Huber et al (2002)	Perentos et al 2004
EEG in awake healthy volunteers 18-69 y (n=120)	875 MHz GSM, max SAR in brain: 0.11 W kg^{-1} Exposure 30 min, EEG in first 10 min and in 10 min following exposure; eyes open	Increased power in alpha band; greater effect ipsilateral.	Well performed large study with appropriate control of interference.	Croft et al 2008

Sleep EEG

Recent reviews of studies of sleep EEG have been performed by Hamblin and Wood (2002) and Mann and Röschke (2004).

Mann and Roschke (1996) exposed volunteers during sleep to a GSM 900 signal and determined effects on the EEG and on sleep architecture. The order of sham and exposure was randomized, but the interval between sessions is not provided. They reported that exposure to GSM-like signals reduced the latency to sleep onset and the percentage of REM sleep. Also the power density of the EEG was increased during REM sleep, mainly in the alpha band. No changes in well-being or mood were reported by the subjects, so it is not possible to conclude that the observed effects had any influence on health. In a subsequent study by the same group (Wagner et al 1998; also briefly reported by Mann et al 1998) 22 volunteers were exposed to the same GSM 900 signal, but at slightly lower field strength. Exposure and sham were given on consecutive nights. In this study a planar antenna was used, so the dose distribution also differed from that in the previous study where a mobile telephone was used. This study failed to replicate the findings of the Mann and Röschke (1996) study. A third study from this group (Wagner et al 2000) employed a much stronger exposure (a power density of 50 W m^{-2}, vs 0.5 and 0.2 W m^{-2} in the previous studies). Sham or exposure conditions were given on two consecutive nights, with at least 1 week between conditions. They observed no effects on sleep architecture or EEG spectral power density. The authors suggest that there might be a difference in the effects of linearly polarized fields, such as used in the first study (Mann and Röschke 1996), and the circularly polarized fields used in the two subsequent studies.

Borbély et al (1999), described above, reported that exposure to a 900 MHz 'pseudo GSM signal' during sleep in a 15 min on / 15 min off schedule was associated with a reduced number of wakings after sleep onset and changes in EEG power spectra during the first of the night's episodes of non-REM sleep. No effects on sleep latency and sleep state were observed. Exposure and sham were double-blind, randomized and given at 1 week interval. In a follow-up study, Huber et al (2000), described above, exposed healthy volunteers for 30 min to a 900 MHz GSM signal immediately before a 3-hour morning sleep episode. Again, exposure and sham were double-blind, randomized and given at 1 week interval. They observed an increased spectral power in alpha and beta bands (9.75-11.25 Hz and 12.5-13.25 Hz) in the first non-REM sleep phase. The effect subsided later during sleep. There were no differences in effect between right or left-sided exposure. Sleep stages and sleep latency were not changed and the subjects did not indicate any changes in sleep quality. An extended analysis of the data first published in two previous studies (Borbély et al 1999; Huber et al 2000) was given by Huber et al (2003). The conclusions from the original papers were not changed and authors interpret the effects as originating from a structure below the cortex such as the thalamus which was similarly exposed.

Huber et al (2002), described above, investigated the effects of 900 MHz GSM signal resembling that of a handset, and of a continuous wave 900 MHz signal on both sleeping and waking EEG. The left side of

each subject's head was exposed to each of these signals for 30 minutes on 3 separate evenings at weekly intervals, before they went to sleep. Subjects then slept while their EEG was monitored. Pulse-modulated, but not CW RF, produced a significant increase in the 12.25-13.5 Hz band of the EEG activity in ensuing sleep, without changing other aspects of EEG or sleep behavior. However, the effects of pulse-modulated RF on the EEG, though statistically reliable, were small relative to the normal variation in EEG activity during sleep. Loughran et al (2005) performed an experiment very similar to that of Huber et al (2002) but with a larger sample (n=50). They also exposed healthy volunteers for 30 min to a GSM signal immediately before sleep. Exposure and sham conditions were randomized and given at 1-week intervals. In contrast to Huber et al (2002) who used a planar antenna, Loughran et al used an 894.6 MHz mobile telephone. They positioned the EEG electrodes after the exposure, which introduced a 20-min delay between the end of the exposure and sleeping time. They tested three specific frequency bands reported in the literature to be increased in the first non-REM sleep phase (11.5-12.25 Hz, 12.25-13.5 Hz and 13.5-14 Hz; Borbély et al 1999, Huber et al 2002). They found an increase in spectral power in the exposure condition for the 11.5-12.25 Hz band only. The latency until REM sleep was delayed, but there were no changes in other sleep parameters. Also using similar methods to Huber et al (2002), Regel et al (2007b) exposed healthy volunteers for 30 min to a 900 MHz GSM signal immediately before sleep. Again, exposure and sham were double-blind, randomized and given at 1 week interval, but importantly they looked for a dose-response relation using 0.2 and 5 W kg-1 peak spatial SAR. They observed a dose-related increase in spectral power in the 10.75-11.25 Hz and 13.5-13.75 Hz bands during non-REM sleep, which increased during the night. Sleep stages and sleep latency were not changed.

Fritzer et al (2007) exposed 10 healthy subjects during sleep in a similar exposure design as used by Borbély et al (1999). The subjects slept in the laboratory for 8 consecutive nights. The first night was for adaptation; the second night was for collecting unexposed baseline data and the 3rd through 8th nights, real or sham exposure took place. The authors compared sleep parameters and EEG of the 1st and 6th exposure night with those of the baseline night that immediately preceded the exposure nights. No differences in any parameter were detected, except that some effects were seen in EEG power in the first non-REM sleep phase for some frequency bands. The authors state, however, that empirical values indicate changes in power only if a high amplitude in the spectral differences (at least two power-units) was paired with a low p-value over a certain minimum frequency range (at least a band of 2.5 Hz), which was not the case in this study. The combination of a between-subjects design and very small sample size makes these results difficult to interpret.

Hung et al (2007) studied the effect of GSM 900 signals with different ELF pulse modulations on sleep onset and sleep architecture. Ten healthy subjects were exposed under carefully controlled conditions for 30 min at weekly intervals to signals simulating 'talk' mode (with 8 and 217 Hz modulation), 'listen' mode (with 2, 8 and 217 Hz modulation) and 'standby' mode (with 1-32 Hz modulation), and to sham exposure. Directly following exposure lights went off and subject could sleep. Following talk mode, an increase in sleep latency was observed, but no increase was seen in 1-4 Hz EEG power that had been found with the other two conditions. The authors point out that increases in the power in this delta band more accurately reflect transitions from waking to sleeping and back than the other frequency bands. They conclude that the 8 and 217 Hz modulation might be responsible for the effect on sleep latency and that the additional 2 Hz component in the listen mode may negate this effect. It is worth noting, however, that the slow rolling eye movements that produce delta artifacts (in addition to blinking) are just as large (if not larger) with eyes closed as open in waking.

In a study into possible therapeutic effects of RF exposure, Reite et al (1994) exposed 52 volunteers to a 27.12 MHz field modulated at 42.7 Hz. The signal was applied through a mouthpiece. The SAR in the oral mucosa was calculated to be approximately 10 W kg^{-1}, but in brain tissue only 100 mW kg^{-1}. Application decreased the sleep latency by approximately 2 min and increased the deep sleep time by about 1 min. Pasche et al (1996) performed a follow up study with 97 patients suffering form psychophysiological insomnia. They were treated for 20 min, 3 times per week for four weeks in a double blind, randomized placebo-controlled study design. In patients receiving the active treatment, an increase in total sleep time, a decrease in sleep latency, and a 30% increase in number of sleep cycles per night were observed. The authors conclude that this treatment might be of benefit to people suffering form chronic insomnia.

Table II.5.2.: Electrical activity of the brain: sleep EEG

Assay endpoint	Exposure Conditions	Response	Comment	References
EEG and sleep parameters in healthy volunteers 18-53 y (n=52)	Low energy emission therapy device (27.12 MHz modulated at 42.7 Hz); intrabuccal applicator; max brain SAR:100 mW kg^{-1} EEG 35 min, exposure 15 min, starting at 5 min after EEG	Decrease in sleep latency and increase in sleep duration.		Reite et al 1994
EEG and sleep parameters in healthy volunteers 21-34 y (n=12)	GSM mobile phone, 900 MHz, 217 Hz modulation, at 40 cm from vertex, 0.5 W m^{-2} EEG and exposure continuous for 8 h	No effect on sleep efficiency; sleep latency onset and % REM sleep reduced. Increased spectral power density during REM sleep, mainly in alpha band.	Control for interference.	Mann and Röschke 1996
Sleep parameters in volunteers with psychophysiological insomnia 21-55 y (n=97)	Low energy emission therapy device (27.12 MHz modulated at 42.7 Hz); intrabuccal applicator; max brain SAR: 100 mW kg^{-} 20 min, 3x per week for 4 weeks	Increase in total sleep time, decrease in sleep latency, 30% increase in number of sleep cycles per night.		Pasche et al 1996
EEG and sleep parameters in healthy volunteers 18-37 y (n=22)	GSM signal from planar antenna, 900 MHz, 217 Hz modulation, 40 cm below pillow, 0.2 W m^{-2}, max SAR: 0.6 W kg^{-1} EEG and exposure continuous for 8 h	No effects on sleep architecture and EEG.	Control for interference.	Wagner et al 1998
EEG and sleep parameters in healthy volunteers 20-25 y (n=24)	GSM signal, base station-like, 900 MHz, 2, 8, 217, 1736 Hz modulation, 3 antennas 30 cm from the head; max SAR: 1 W kg^{-1} EEG continuous and exposure 15 min on / 15 min off for 8 h	Reduced waking during sleep only with sham before exposure. No effect on sleep latency and sleep state. Increase in power for 10-11 Hz and 13.5-14 Hz band during non-REM sleep.	Control for interference.	Borbély et al 1999
EEG and sleep parameters in healthy volunteers 19-36 y (n=20)	GSM signal from horn antenna, 900 MHz, 217 Hz modulation, 40 cm below pillow, 50 W m^{-2}, max SAR: 1.8 W kg^{-1} EEG and exposure continuous for 8 h on 2 consecutive nights	No effects on sleep architecture and EEG.	Control for interference.	Wagner et al 2000

Assay endpoint	Exposure Conditions	Response	Comment	References
EEG and sleep parameters in healthy volunteers 20-25 y (n=16)	GSM signal, base-station-like,900 MHz, 2, 8, 217, 1736 Hz modulation, planar antennas, peak SAR: 1 W kg^{-1}Exposure for 30 min before sleep	Spectral power in 9.75-11.25 Hz and 12.5-13.25 Hz band increased in first non-REM phase. No difference between right or left exposure. No effect on sleep stage or sleep latency.		Huber et al 2000
EEG in sleeping volunteers (n=20)	Mobile phone EEG during 8-h sleep; continuous exposure	Increase in alpha band power and changes in sleep pattern during exposure	No information on experimental setup and methods of data analysis	Lebedeva et al 2001
EEG and sleep parameters in healthy males, 20-25 y (n=13).	GSM signal, mobile phone-like, 900 MHz, 2, 8, 217, 1736 Hz modulation, planar antennas, SAR: 1 W kg^{-1} Exposure for 30 min before sleep	Increased power in alpha band prior to sleep and in the 12.25-13.5 Hz band during non-REM sleep; no fading during sleep. No effects of non pulse-modulated field.	Spectral power of modulation higher than in base station-like signal from previous studies (Borbély et al 1999, Huber et al 2000)	Huber et al 2002
EEG and sleep parameters in healthy volunteers, 18-60 y (n=50).	894.6 MHz GSM mobile phone, peak SAR: 0.29 W kg^{-1}, for 30 min	Decrease in REM sleep latency; increase in power in 11.5-12.25 Hz band during first 30 min of 1st non-REM sleep period.	EEG electrodes positioned after exposure.	Loughran et al 2005
EEG and sleep parameters in healthy males, 22-36 y (n=10).	GSM signal, 900 MHz, 2, 8, 217, 1733 Hz modulation, 3 antennas 30 cm from the head; max SAR: 1 W kg^{-1} EEG continuous and exposure during sleep for 6 nights	Comparison between unexposed baseline night and 1st and 6th exposure night. No effect on sleep parameters or on EEG power in either night.	Possibly too strict criteria for EEG power changes to become statistically significant.	Fritzer et al 2007
EEG and sleep parameters in healthy volunteers, 18-28 y (n=10).	GSM signal, 900 MHz, pulse modulated with 8, 217 Hz (talk), 2, 8, 217 Hz (listen), or 1-32 Hz (standby); mean SAR resp.: 0.133, 0.015, <0.001 W kg^{-1}, for 30 min EEG continuous and during exposure and sleep	Delayed sleep latency after talk mode; no similar increase in 1-4 Hz (delta) power as in other conditions.		Hung et al 2007
EEG and sleep parameters in healthy volunteers 20-26 y (n=15)	GSM signal, base-station-like, 900 MHz, 2, 8, 217, 1736 Hz modulation, planar antennas, peak SAR: 0.2 and 5 W kg^{1} Exposure for 30 min before sleep	Dose-response increase in 10.75-11.25Hz and 13.5-13.75 Hz bands in non-REM, Stage 2 and slow-wave sleep. No effect on sleep stage or sleep latency.		Regel et al 2007b

Event-related (evoked) potentials

These studies have examined RF (mostly GSM signals) effects on auditory, visual and somatosensory event-related potentials (ERPs). In addition, one group focused in ERPs related to the performance of cognitive tasks and another examined cortical excitability using transcranial magnetic stimulation applied to the motor cortex before and after RF exposure in order to generate muscle contraction. Most studies were carried out on adults; one study was carried out using children.

Studies with adult subjects

A number of studies have reported that acute exposure to GSM RF radiation can affect auditory ERPs. However, the interpretation of the conflicting results from many of these studies is often weakened by various methodological limitations such as questionable dosimetry, small sample size and single-blinding techniques.

Eulitz et al (1998) observed an increase of high frequency (approximately 18-30 Hz) spectral power in auditory ERP waveforms elicited by infrequent auditory 'oddball' stimuli interspersed among a more frequent class of auditory stimulus. The effect was observed only in the left hemisphere, the side of the exposure. This study in 13 volunteers was single blind and no control of possible interference of the GSM signal with the electrodes or leads was reported. Croft et al (2002; described above) exposed 24 volunteers to GSM mobile phone type RF radiation and recorded phase-locked responses from the EEG during the performance of an auditory discrimination task. The study was single-blind and counterbalanced with a crossover design. A decrease in power of the theta (1-4 Hz) and beta (12-30 Hz) bands and an increase in power of the gamma band (30-45 Hz) in the phase-locked EEG were reported. The strength of the dosimetry in that study was subsequently questioned by the investigators (Croft et al 2008).

Arai et al (2003) studied the neuronal pathways mediating auditory stimulus, from ear to midbrain, by recording the auditory brainstem-evoked responses (ABR). They exposed healthy volunteers for 30 min to a mobile phone operating at full power. ABRs were recorded before and after exposure. They performed three experiments at 1 week intervals to study three different parameters. No effects of the exposure were observed. Bak et al (2003) exposed 45 volunteers to signals of three types of mobile phones operating at different frequencies. Exposure was intermittent at four times per minute at 1-s intervals, for 20 min on both the right and left side of the head. Dosimetry was performed, but exposure levels are unclear. ABRs were not influenced by RF exposure.

A pilot study by Hamblin et al (2004) in 12 subjects reported a decrease in the amplitude and latency of a sensory component of an auditory ERP, and an increase in the latency of a later more cognitive component. This study had a single-blind, counterbalanced crossover design. Sievert et al (2005) reported a lack of effect of exposure to a CW or GSM signal on ABRs before, during and after exposure in a study comprising 12 volunteers. It is not clear whether the study was blinded in any way.

Maby et al (2004) studied auditory ERPs in two groups of 14 subjects: healthy volunteers and epileptic patients. They reported a reduction in the amplitude and latency of the early sensory component of the auditory ERP in the healthy subjects, and an increase in latency and decrease in amplitude for the epileptic patients. In the healthy subjects only, they also observed an increase in the amplitude of a later component. Unfortunately the experimental design was not clearly described. Therefore it is difficult to draw any conclusions on the effect of exposure. Maby et al (2005, 2006b) examined the effects of GSM RF radiation on auditory ERPs evoked by two different sound stimuli in both normal and epileptic subjects. In both studies, nine healthy volunteers and six patients suffering from temporal lobe epilepsy were exposed or sham-exposed to GSM-type RF whilst auditory ERPs were recorded. The authors calculated in each individual the temporal and frequency correlation variations for the auditory ERP responses to the different pairs of stimuli recorded from 14 (out of 32) selected electrodes, with or without RF exposure. Each subject acted as its own control and the study was a single-blind design. Variable exposure-related differences in the correlation coefficients were observed in both healthy and epileptic subjects, but it was not possible to determine the significance of this observation for health.

Oysu et al (2005) measured ABRs in 18 healthy volunteers. It is not clear whether the subjects were blinded to the exposure conditions. ABRs were determined before and after a 15-min exposure to the signal from a 900 MHz mobile phone. No differences were observed between the before and after measurements.

In an attempt to avoid the methodological weaknesses of previous studies, Hamblin et al (2006) further investigated their earlier study (Hamblin et al 2004) which reported a decrease in the amplitude and latency of a sensory component of an auditory ERP, and an increase in the latency of a later, more cognitive component, using larger number of subjects (120), better dosimetry and a double-blind, counterbalanced, crossover design. Two experimental sessions were held, 1 week apart; in each session subjects were initially sham exposed, and then either exposed or sham-exposed to GSM 895 RF. The authors measured the reaction times for cognitive responses to an auditory and a visual cognitive (oddball) task and recorded the early and late components of ERPs resulting from the auditory and visual stimuli. There were no statistically significant effects on the early or late components of the ERPs, and no effect on reaction times. The authors concluded that there is currently no clear evidence in support of a mobile phone related EMF effect on ERPs or reaction times.

Several studies have examined RF effects on visual ERPs. Freude et al (1998) measured slow response-related brain potentials in a visual monitoring task in a single-blind study in 16 volunteers. They observed a small reduction in the amplitude of potentials in the central and temporo-parieto-occipital regions. In contrast to the study of Eulitz et al (1998), the effect was strongest in the right (contralateral) hemisphere. No effects were found in the potentials preceding spontaneous movements, and neither were there any exposure effects on task performance.

Freude et al (2000) performed a follow-up of this study. They measured visual ERPs in two groups of 20 and 19 volunteers, respectively. In the second group, other evoked potentials were measured. In a single blind design, the subjects were exposed for the duration of the tests to a signal from a GSM 900 phone. Exposure had no effect on this performance, nor on a simple finger tapping task or a more complex task involving two visual stimuli. Urban et al (1998) found no effects in a pilot study of GSM mobile phone radiation effects on the visual ERP evoked by reversal of a checkerboard pattern in 20 volunteers. However, visual ERPs were only measured after exposure and the subjects were aware of whether the phone was on or off.

Jech et al (2001) studied visual ERPs in 22 patients with narcolepsy. In 17 of these patients ERPs were studied during a visual ‘oddball’ task. In this task, rare horizontally-striped ‘targets’ (the oddballs) were interposed among presentations of more frequent non-targets (vertical stripes). Both classes of stimulus could occur either in full-field, or restricted to one or other side of the visual field. Exposure was double-blind, with sham and exposure conditions occurring on separate days (ordering of conditions was counterbalanced). Recordings were obtained during exposure to a GSM 900 signal. Exposure was found to enhance the amplitude of two components of the brain’s response to the oddball stimuli, but only when the stimuli were presented to the right half of the visual field. This effect was most marked in waveforms from right hemisphere electrodes. In addition, exposure was found to shorten reaction time to both stimulus classes by approximately 20 ms. It should be noted however that the majority of the narcoleptic patients were medicated, possibly restricting the generality of these findings. In addition, since stimuli presented to the right visual hemifield project to the left hemisphere, it is not clear that the effect reported over the right hemisphere related to the experimental task.

Yuasa et al (2006) studied the effects of mobile phone RF radiation on somatosensory ERPs in 12 subjects. The experiment was single-blinded. Exposure or sham exposure was to 800 MHz RF radiation from a digital mobile phone held by hand for 30 min within 4 cm of the head. The authors recorded the ERP in the sensory region of the right cortex evoked by median nerve stimulation of the left arm before during and after exposure. They reported that the RF exposure did not affect the somatosensory ERP or its recovery function, suggesting that neither the neural pathways mediating somatosensory stimuli nor the large neurons of the sensory cortex are affected by mobile phone radiation.

Ferreri et al (2006) investigated the effects of GSM mobile phone RF radiation on cortical excitability in fifteen right-handed young male volunteers. Transcranial magnetic stimulation was applied to the motor cortex before and after RF exposure in order to generate motor-evoked potentials in a target muscle in the

hand. This approach differs from the other ERP studies where a sensory stimulus evoked electrical potentials in the sensory cortex of the exposed or sham-exposed brain. All subjects underwent two trials, separated by one week, in a double-blind cross-over experimental design. The left side of the subject's head was exposed or sham-exposed to RF radiation for 45 min; the right side served as a control. The effect of main interest was the triple interaction of time, exposure condition and hemisphere. This had a probability of 0.07, which is not statistically significant but is sufficiently borderline to be of interest. It indicates a transient decrease in intracortical inhibition and a transient increase in intracortical facilitation in the RF-exposed hemisphere. However, the analysis and interpretation is complex and depends, for example, on the stability of the base-line response to a single pulse, as indicated by the authors.

Krause et al (2000a; 2000b) investigated the effects of a GSM 900 signal on event-related desynchronization/synchronization (ERD/ERS) of four narrow EEG frequency bands: 4-6 Hz, 6-8 Hz, 8-10 Hz and 10-12 Hz, during performance of a cognitive task. Although not referred to as evoked potentials *per se*, different EEG frequencies are associated with different mental processes and synchronization or desynchronization of these reflects event-related increases or decreases respectively in the relative EEG power of the different frequency bands. Krause et al (2000a) examined ERD/ERS in 16 subjects during the performance of an auditory memory task during exposure. A counterbalanced, single-blind procedure was used. RF exposure significantly increased the ERD/ERS responses in the 8-10 Hz frequency band only, which is associated with attention and memory functions. They concluded that RF exposure can influence neural oscillatory systems associated with memory retrieval. The second study (Krause et al 2000b) examined ERD/ERS elicited by the visual presentation of letters during an 'n-back' working memory task. Twenty-four subjects were employed, using an on/off single-blind procedure with a 900 MHz GSM signal. Exposure effects were observed in two specific bands of the EEG spectrum, at 6-8 and 8-10 Hz. The authors concluded that, as in their previous study, the findings suggested that RF effects on EEG are most prominent during active cognitive processing.

A replication of the Krause et al 2000a study using an auditory memory task was published by Krause et al (2004). In this double-blind study, all 24 subjects performed the memory task both with and without exposure in a counter-balanced order. Although the authors found some effects of RF on the ERD/ERS responses in the 4-8 Hz EEG frequency range, they were not able to replicate the findings from their earlier study. In contrast to the previous study, they did observe an increase in the number of incorrect answers in the memory task during exposure. They concluded that GSM 900 effects on EEG and on the performance of memory tasks are variable and not easily replicable for unknown reasons.

Further puzzling observations were made by the Krause group in a subsequent study (Krause et al 2007). Here they used a double blind crossover experimental design to expose two groups of 36 volunteers to both GSM 900 and CW signals. One group performed a visual memory task during exposure, the other one an auditory memory task. No effects of exposure to either type of signal on performance were observed. In both groups, some small, but inconsistent differences were found in EEG power in the alpha band (8-10 Hz) between CW and GSM conditions, but not with sham. The authors conclude that EMF effects on the EEG are either non-existent or so susceptible to many other factors in standard EEG experiments (such as normal variations in the EEG, attentional effects, random variance, etc.), that they are difficult to capture systematically.

Hinrichs & Heinze (2004) exposed healthy volunteers for 30 minutes to a GSM 1800 signal during the learning (encoding) phase of a memory test. They subsequently measured the total EEG power during memory retrieval, where subjects had to indicate whether the words they were presented were part of the list they had seen during the encoding phase. The reaction time and percentages of correct answers were not influenced by the exposure. In some parts of the left hemisphere (the exposed side) differences in EEG power were detected at some specific time windows after the start of the stimulus. It is not clear, however, whether these differences were increases or decreases in total EEG power. It is also not clear whether any blinding of the subjects to the exposure condition was performed.

Studies with children

Krause et al (2006) used the same experimental design as in previous studies (Krause et al 2000a, 2001, 2004) to examine ERD/ERS responses in 15 subjects aged 10-14 years performing an auditory memory

task. The study design was counterbalanced and double-blind. The authors reported that RF exposure resulted in statistically significant differences in the responses associated with encoding and recognition in the ~ 4-8 Hz EEG frequency range, and ~15 Hz, also associated with recognition. They note that these results are congruent with their previous studies, described above, although they caution that the actual changes that occur (increases or decreases in response) are not consistent between studies, for reasons that are unclear.

Table II.5.3.: Electrical activity of the brain: event-related potentials

Assay endpoint	Exposure Conditions	Response	Comment	References
Adults				
Auditory ERP after auditory stimuli in volunteers 21-27 y (n=13)	Mobile phone, 916 MHz, 2.8 W peak power ERP measurement without and during exposure	Increase in 18-30 Hz spectral power in left (exposure side) hemisphere only	No control for interference	Eulitz et al 1998
Slow brain potentials in volunteers 21-26 y (n=16)	Mobile phone, 916 MHz, peak SAR: 0.88 W kg^{-1} SP measurement with / without exposure (8 min)	Decrease of SP in central and temporo-parieto-occipital regions; effect stronger in right (contralateral) hemisphere	No control for interference	Freude et al 1998
Visual ERPs in healthy volunteers 19-70 y (n=20)	GSM mobile phone 4 consecutive VEP measurements, 5-min phone call between 2nd and 3rd	No effects.	Insufficient experimental data. No blinding.	Urban et al 1998
Visual ERPs in healthy volunteers; exp.1: 21-30 y (n=20); exp.2: 21-26 y (n=19)	916 MHz GSM phone, SAR: 0.88 W kg^{-1} EEG recording during exposure	No effect on performance; decreased EEG power in central and parieto-tempero-occipital regions, stronger in exposed hemisphere.	No correction for interference.	Freude et al 2000
EEG effects during an auditory memory task in healthy volunteers (mean age 23.3 y; n=16)	902 MHz GSM phone, power 0.25 W Exposure during task performance (~30 min)	Increased power in alpha band.	No correction for interference.	Krause et al 2000a
EEG effects during an auditory memory task in healthy volunteers (mean age 23.3 y; n=16)	902 MHz GSM phone, power 0.25 W Exposure during task performance (~30 min)	Increased power in alpha band.	No correction for interference.	Jech et al 2001
Evoked spectral power in adult awake healthy volunteers 19-48 y (n=24)	900 MHz GSM phone; 20 min EEG during 4x3 min auditory task	Decrease in evoked theta and beta and increase in gamma activity during auditory EEG.	No testing for interference.	Croft et al 2002
Auditory brainstem responses (ABR) in volunteers 26-50 y (n=15)	Mobile phone 800 MHz, operated at maximum power, for 30 min ABR directly after exposure	No effects.		Arai et al 2003

Assay endpoint	Exposure Conditions	Response	Comment	References
ABRs in volunteers 21-28 y (n=45)	Mobile phones, 450, 935, 1800 MHz 4x14 sec min^{-1}, for 20 min, both left and right	No effects.	Controlled for interference. Unclear dosimetry.	Bak et al 2003
Auditory ERP after auditory stimuli in volunteers 19-44 y (n=12)	Mobile phone, 895 MHz, 0.25 W mean power Exposure 1 h, ERP recording after 30 min	Decrease in amplitude and latency of stimulus-bound ERP components and increase in latency of cognitive components.	Controlled for interference.	Hamblin et al 2004
Auditory ERPs in healthy volunteers (n=14) and epileptic patients (n=14)	GSM signal, maximum SAR: 1.4 W kg^{-1} Four recording phases: no RF, minimal power, maximal power, minimal power; 3 sessions: first two for right / left ear, 3rd for missing data or placebo	Healthy subjects: decrease in latency and amplitude of early ERP component, increase in latency of later component. Epileptics: increase in latency and decrease in amplitude of early component.	Inadequate description of experimental design.	Maby et al 2004
EEG effects during an auditory memory task in healthy volunteers 24.3±8.1 y (n=24)	902 MHz GSM phone, SAR: 0.65 W kg^{-1} Exposure during task performance (~30 min)	No effects on EEG, but increase in incorrect answers.	Replication of Krause et al (2000a)	Krause et al 2004
EEG during memory task in healthy volunteers 18-20 y (n=12)	1870 MHz GSM signal, SAR: 0.61 W kg^{-1} 30 min exposure during memory encoding; EEG measured during retrieval; average power over 0-50 Hz	No effect on reaction time and error rate; differences in total EEG power in left (exposed) hemisphere during retrieval.	Blinding not clear.	Hinrichs and Heinze 2004
ABRs in healthy volunteers 19-57 y (n=12)	GSM phone, 890 MHz, continuous or with 217 Hz modulation, SAR: 1.9 W kg^{-1}	No effects.		Sievert et al 2005
ABRs in healthy volunteers 20-28 y (n=18)	900 MHz GSM phone, SAR: 0.82 W kg^{-1} Measurement before / after 15 min exposure	No effects.	Unclear whether blinding occurred.	Oysu et al 2005
Auditory ERPs in healthy volunteers 21-32 y (n=9) and epileptic patients 25-39 y (n=6)	900 MHz GSM mobile phone, max SAR: 1.4 W kg^{-1} Auditory ERP recording during control exposure, minimal and maximal power	Difference in correlation coefficients between control and experimental sessions.	Complex parameters used in the analysis; low number of subjects.	Maby et al 2005

Assay endpoint	Exposure Conditions	Response	Comment	References
Auditory ERPs in healthy volunteers 21-23 y (n=9) and epileptic patients 25-39 y (n=6)	900 MHz GSM mobile phone, max SAR: 1.4 W kg^{-1} Auditory ERP recording during control exposure and maximal power.	Healthy subjects: amplitude increase slow response in frontal area. Epileptics: lengthening of fast response in frontal area contralateral to exposure.	Same study as Maby et al (2005) with different endpoints.	Maby et al 2006b
Auditory ERPs in healthy volunteers 18-69 y (n=120)	895 MHz GSM phone, SAR: 0.1 W kg^{-1} Measurements before / during 21 min exposure	No effects.	Control for interference.	Hamblin et al 2006
Somatosensory ERPs in healthy volunteers 20-55 y (n=12)	800 MHz GSM phone, SAR: 0.054±0.02 W kg^{-1} SEP recording before and after 30 min exposure	No effects.	No control for interference. Very low SAR	Yuasa et al 2006
Brain excitability in healthy volunteers 20-36 y (n=15)	902 MHz GSM phone, SAR: 0.5 W kg^{-1} Recording of muscle contraction after TMS before and after 45 min exposure	Indication of transient decrease in intracortical inhibition and transient increase in intracortical facilitation.		Ferreri et al 2006
EEG effects in healthy volunteers during a visual memory task (age 22.9±2.4 y; n=36) and an auditory memory task (age 23.6±2.4 y; n=36)	902 MHz GSM or continuous wave signal, SAR: 0.74 W kg^{-1} Exposure during task performance (~30-40 min)	No effects on performance. Some differences in alpha band between CW and pulsed conditions, but not with sham.		Krause et al 2007
Children				
EEG effects during an auditory memory task in children 10-14 y (n=15)	902 MHz GSM phone, peak SAR: 1.98 W kg^{-1} Exposure during task performance (~30 min)	Modulation of EEG power in theta and beta bands.	No control for interference.	Krause et al 2006

II.5.1.2. Auditory and vestibular systems

As mobile phones are held close to the ear, various studies have checked for possible effects of exposure to GSM type mobile-phone RF on the vestibular (balance) organs and cochlear (auditory) that comprise the inner ear. The hair cell receptors present in each organ respond to head movement or to audible sound.

The semi-circular canals of the vestibular organ respond to angular head movement, the inertia of the endolymph within the semicircular canal displacing a flap-like ampulla within each canal which, through effects on hair cell receptors, provides a neural signal to appropriate brain centers, especially those involved in the control of eye movement. Pau et al (2005) measured an eye movement called nystagmus, in 13 volunteers during exposure or sham exposure to a CW or to a GSM 900 signal. Nystagmus is normally generated by horizontal head movement but is also generated by localized warming of the horizontal semi-circular canal by more than 0.1°C. The description of the experimental protocol and dosimetry was incomplete. However, the local SAR at the position of the horizontal semi-circular canal was estimated at about 1.9 W kg^{-1}. GSM exposure did not induce nystagmus, suggesting that neither CW nor GSM exposure-induced temperatures in the vestibular region of the head rose by more than 0.1°C and that there was also no direct stimulation of the vestibular organ by the GSM signal.

Several authors have examined the effect of mobile phone RF radiation on auditory function in volunteers which can be studied by measuring otoacoustic emission (OAE). This is sound recorded in the outer ear canal thought to result mostly from outer hair cell activity in the cochlea, either spontaneous or following auditory stimulation. Ozturan et al (2002) determined transiently evoked (TE) and distortion product (DP) OAEs in 30 healthy adult volunteers. The authors did not find any effect of a 10 min exposure to the signal from a 900 MHz GSM phone. The level of exposure was not clear, however, because it was not indicated at which output level the mobile phone operated.

Monnery et al (2004) reported that OAEs were unaffected in 12 volunteers during transmission from a mobile phone placed in close proximity to the ear. They did not provide, however, any information on the type of mobile phone used, and on the level and duration of exposure. Kerekhanjanarong et al (2005) studied auditory function in 98 subjects that underwent clinical hearing evaluations and correlated those with reported intensity of mobile phone use. No differences were found in audiometry between the dominant and non-dominant ear. In the eight subjects with the highest telephone use, uncorrelated differences in OAE and ABR were observed between the two ears. These could not be analyzed statistically.

Using a double blind study protocol, Uloziene et al (2005) measured baseline audiological parameters and transient evoked otoacoustic emissions (TEOAE). They exposed healthy volunteers for 10 minutes to GSM 900 or 1800 mobile phones operating at maximum power. Measurements performed directly before and after exposure were not significantly different, nor was there any difference between responses after real and sham exposure.

Two studies have examined possible effects on distortion product OAEs (DPOAEs) which result from the intermodulation products generated by the responses to two tones applied simultaneously. Janssen et al (2005) measured DPOAEs in 28 subjects between consecutive GSM signal pulses from a monopole antenna held 5 cm away from the subject's ear. No statistically significant changes in DPOAEs were seen in this single blind experiment. However, after correction for variation resulting from effects other than EMFs, DPOAEs were observed to be increased in a few subjects by a physiologically trivial amount (< 1.0 dB compared to a normal dynamic range of 120 dB) but these data were not subject to statistical analysis. Parazzini et al (2005) used mathematical techniques in order to examine two separate components of the DPOAE signal, increasing the sensitivity of the investigation. Fifteen subjects participated; DPOAEs were measured immediately before and after exposure or sham exposure to a GSM signal at 900 MHz or at 1800 MHz; SARs to the cochlea were estimated as 0.41 and 0.19 W kg^{-1} respectively. The study was double-blind with a within-subject design. No statistically significant effects of mobile phone RF radiation were seen. In a follow-up study by the same group, Paglialonga et al (2007) studied transient evoked otoacoustic emissions (TEOAE) in a group of 29 healthy volunteers. The design of the experiment was identical to the previous one. Also for the TEOAE, no effects of a 10 min exposure were observed.

Oktay and Daşdağ (2006) performed audiometry in three groups of 20 volunteers: one group that had used a mobile phone for more than 2 h daily for 4 years (heavy users); one group that used it daily for 10-20 min for 4 years (moderate users) and a group of non-users. Some control was made for confounding by excluding subjects that had been subjected to loud noises from acoustic devices. No effects were observed on brainstem evoked response audiometry, an objective measure of auditory function. On a more subjective measure, pure tone audiometry, a decrease in hearing was observed in the heavy users group, at 400 Hz for the right ear and 500 and 400 Hz for the left ear. The authors state that no correction could be made for laterality, because 13 of the 20 subjects indicated that they used the phone on both ears. An explanation for the difference between left and right ear is not provided. However, the sound level of the phones was not measured; therefore it is possible that long-term exposure to loud noises from the phones might be the cause of the observed hearing loss.

In a double-blind crossover study with a group of subjects with complaints attributed to using a mobile phone (cases) and a group of control subject without such complaints, Bamiou et al (2008) studied the effect of a 30 min exposure from a modified handset capable of producing GSM 882 and CW signals. Both auditory and vestibular functions were measured, in separate experiments 2-4 weeks apart. No

effects were observed of either sham, CW or GSM signals, in both cases and controls, and for both auditory and vestibular functions.

Table II.5.4.: Auditory and vestibular systems

Assay endpoint	Exposure Conditions	Response	Comment	References
Transiently evoked (TE) and distortion product (DP) otoacoustic emissions (OAEs) in volunteers19-36 y (n=30)	GSM phone, 900 MHz Measurements before / after 10 min exposure	No effects.	Exposure level not clear ('activated phone').	Ozturan et al 2002
OAE in volunteers (n=12)	Mobile telephone set to transmit outgoing call	No effects.	No information on type of phone and level and duration of exposure.	Monnery et al 2004
Effect on vestibular organ in healthy volunteers 29-58 y (n=13)	GSM signal, 890 MHz, 217 Hz modulation, SAR: 1.9 W kg^{-1}	No effect on nystagmus from continuous or pulsed field.	Incomplete description of experimental conditions. No statistics.	Pau et al 2005
Audiometry, OAE and ABR in patients 20-67 y (n=98)	Reported intensity of mobile phone use	No effects, except uncorrelated effects on OAE and ABR in subjects with highest use.	No statistics.	Kerekhanjan arong et al 2005
Audiology and OAE in healthy volunteers 18-30 y (n=30)	GSM signal, mobile phone, 900 or 1800 MHz, maximum power (2 resp. 1 W), concurrent speech sound OAE before / after 10 min exp; 24 h interval between exp / sham	No effects.		Uloziene et al 2005
Distortion product OAEs in healthy volunteers 16-30 y (n=28)	900 MHz signal, 41 Hz modulation, mean power 0.465 W , SAR: 0.1 W kg^{-1} DPOEA measurements during interval between pulses	Slight, but physiologically irrelevant, increase in DPOAE in some subjects after exposure.		Janssen et al 2005
Distortion product OAEs in healthy volunteers 18-30 y (n=15)	GSM signal, mobile phone, 900 or 1800 MHz, SAR: 0.41 resp 0.19 W kg^{-1}, concurrent speech sound OAE before / after 10 min exp; 24 h interval between exp / sham	No effects.		Parazzini et al 2005

Assay endpoint	Exposure Conditions	Response	Comment	References
Transient evoked OAEs in healthy volunteers 23-30 y (n=29)	GSM signal, mobile phone, 900 or 1800 MHz, SAR: 0.41 resp 0.19 W kg^{-1}, concurrent speech sound TEOAE before / after 10 min exp; 24 h interval between exp / sham	No effects.		Paglialonga et al 2007
Audiology in healthy volunteers 22-53 y (n=60)	3 groups of 20: heavy users (>2 h d^{-1} for 4 y); moderate users (10-20 min d^{-1} for 4 y); non users	No effect on brainstem evoked response audiometry (BERA); hearing loss in heavy users at 4000 Hz (right ear) and 500 and 4000 Hz (left ear).	Some control for confounding (loud noise from acoustic devices). No correction for laterality possible.	Oktay and Daşdağ 2006
Transient evoked OAEs and nystagmus (video-oculography) in volunteers 20-55 y (subjects with complaints: n=9; controls: n=21)	GSM or continuous signal, 882 MHz, SAR: 1.3 W kg^{-1}, Measurements before / after 30 min exposure; auditory and vestibular exps 2-4 wk apart	No effects on auditory and vestibular function in either group.		Bamiou et al 2008

II.5.1.3. Regional cerebral blood flow

It is generally assumed that changes in regional cerebral blood flow reflect localized changes in neural activity. Huber et al (2002) studied the effects of EMF signals similar to a GSM phone on regional cerebral blood flow (rCBF) measured by positron emission tomography (PET). A 900 MHz signal simulating that of a GSM mobile phone was delivered by a planar antenna to the left side of the head. The peak SAR was estimated at 1 W kg^{-1}. Thirteen subjects were tested in exposed and sham exposed conditions in counterbalanced order, using a within-subjects double-blind design. At least 1 week elapsed between the two tests. In each test, rCBF was measured over three 1 minute periods, starting 10, 20 and 30 minutes after completion of a 30 minute exposure to pulse-modulated EMF or sham exposure. Subjects were asked to count silently during the scans, to balance cognitive function across scans. The results showed a significant increase in rCBF in the dorsolateral prefrontal cortex of the left (exposed) brain hemisphere. In a follow-up study (Huber et al 2005), the effects of 900 MHz mobile-telephony signals on rCBF were investigated in 12 healthy male volunteers, again using the PET technique. Two types of exposure were used: base-station-like and mobile-phone-like, with a similar exposure setup as in the previous study. The exposure of one side of the head lasted 30 min and resulted in a peak SAR of 1 W kg^{-1} for both exposure conditions. Following exposure, an increase in rCBF was observed in the dorsolateral prefrontal cortex on the side of exposure only following the mobile phone-like exposure. The authors interpreted this finding, supporting their previous observations, that only pulse-modulated RF (as in the mobile phone-like signal, in contrast with the CW base-station-like signal) is necessary to induce changes in brain physiology.

Haarala et al (2003a) examined the effects of exposure and sham exposure to a GSM 900 signal on rCBF in 14 volunteers, also using PET, under double blind conditions using a counterbalanced order of exposure/sham exposure. In contrast to the experiments of Huber et al (2002, 2005) exposure took place during PET scanning, while the subjects performed a visual working memory task. The main effect of mobile phone exposure was a bilateral decrease in rCBF in the auditory cortex, which the authors attributed to a high frequency auditory signal emitted by the battery of an active phone, since preliminary results from a follow-up study, reported here, indicated that there was no effect with a phone using a

silent, external power source. There was no effect on the performance of the visual working memory task, including the reaction times and accuracy of the responses. In a follow-up study Aalto et al (2006) improved upon the design of Haarala et al (2003a) by ascertaining that no auditory clues at all were present. They observed a reduced rCBF close to the antenna, and an increase at various other locations deeper in the brain. There was no effect on reaction time. It is not clear from either study whether the changes observed in rCBF were immediately present following the onset of exposure, or that it took some time for them to develop. The papers give no clear indication of the sequence of events.

Table II.5.5.: Regional cerebral blood flow

Assay endpoint	Exposure Conditions	Response	Comment	References
Cerebral blood flow in healthy males, 20-25 y (n=13). PET scan 10 min after exposure.	902 MHz, simulating mobile phone, SAR: 1 W kg^{-1}, for 30 min, 1 wk between exposure and sham.	Increased relative rCBF in dorsolateral prefrontal cortex on the side of the exposure.	Exposure less localized than when using mobile telephone.	Huber et al 2002
Cerebral blood flow in healthy males, 20-25 y (n=12). PET scan 10 min after exposure.	902 MHz, simulating mobile phone or base station, SAR: 1 W kg^{-1}, for 30 min, 1 wk between conditions.	Increased relative rCBF in dorsolateral prefrontal cortex on the side of the exposure with mobile phone signal, not with base station signal. No relationship with SAR distribution.	Exposure less localized than when using mobile telephone.	Huber et al 2005
Cerebral blood flow (rCBF) during memory task in healthy volunteers, 21-35 y (n=14). PET scan during exposure.	902 MHz GSM phone, operating at 0.25 W (SAR~1.2 W kg^{-1}), for 45 min; no time between exposure and sham.	Relative decrease in rCBF in auditory cortex, not in area of maximum RF exposure.	Possibly auditory signal. Sequence of events not clear.	Haarala et al 2003a
rCBF by PET scan during a cognitive task and mobile phone exposure in healthy males, 25±2 y (n=12)	902 MHz from a mobile phone operated at 0.25 W, for 30 min.	Reduced rCBF close to antenna, increase at various other locations. No effect on reaction time.	Sequence of events not clear.	Aalto et al 2006

II.5.1.4. Cognitive performance

Cognitive studies have been carried out in healthy adult volunteers, in adults who report experiencing a variety of symptoms such as headaches in the vicinity of RF sources and, following the recommendations of IEGMP (2000), in children, and also in adolescents. Giedd (2004) for example, notes that there are dynamic changes in brain anatomy throughout childhood and adolescence. The amount of white matter (the myelination of nerve axons), which is related to the speed of neuronal processing, increases linearly throughout adolescence. Changes in grey matter content, thought to reflect changes in size and complexity in neurons such as the number of synaptic connections rather than changes in cell number and considered to be related to the maturation of behavior, are more complex but continue into an individual's early twenties.

Healthy adults

Preece et al (1999) investigated the performance of 36 volunteers on a wide range of tasks, including short- and long-term memory, simple and choice reaction time, and sustained attention, which, together, yielded a total of 15 dependent variables. Using a double-blind, counterbalanced, randomized cross-over

design, volunteers were exposed or sham exposed to a continuous or a pulsed 915 MHz GSM-type signal for about 30 min. A statistically significant shortening of reaction time during exposure to the continuous signal in the choice reaction time task was reported. The effect was not accompanied by a reduction in the accuracy of responding, suggesting that it did not reflect a speed-accuracy trade-off. Simple reaction times were unaffected and there were no changes in word, number or picture recall, or in spatial memory. There was no significant effect of exposure to the pulsed GSM signal.

Koivisto et al (2000a) studied 48 volunteers, also using a wide range of cognitive tests. Using a single-blind counterbalanced crossover design, volunteers were exposed or sham-exposed to 902 MHz GSM signal. Koivisto et al reported decreased response times in simple reaction time and vigilance tasks. In addition, the time needed to accomplish a mental arithmetic subtraction task was decreased during exposure. However, the effect of exposure on the choice reaction time task analogous to that employed by Preece et al (1999) was far from significant. In addition, no allowance had been made for multiple testing (IEGMP 2000). Nevertheless, in a second study (Koivisto et al 2000b) using a similar experimental design to investigate GSM RF effects on the performance of a task where working memory load was varied, Koivisto and colleagues reported a statistically significant reduction of reaction time when the memory load was particularly demanding. However, an attempt by the same group to confirm and extend the results from both studies was not successful (Haarala et al 2003b, 2004). Using an improved experimental design by increasing the sample size, performing the study in two independent laboratories and implementing a double-blind design, no consistent field-dependent effects were observed on reaction times or error rates during performance of any of the cognitive tasks.

Lass et al (2002) studied the effect of exposure or sham exposure of the head to a 450 MHz RF signal, amplitude modulated at 7 Hz, on the performance in 3 cognitive tasks by 100 students, randomly allocated to either group. Exposed subjects made significantly fewer errors on the memory recognition task than sham-exposed subjects. In contrast, performance of the other tasks by the exposed group, which were more demanding, produced small and non-significant effects in the opposite direction, showing worse performance and greater inter-subject variability. Edelstyn and Oldershaw (2002) employed a single-blind, between-subjects experimental design to assess the effects of GSM 900 signals on the performance of 19 subjects in six widely-used cognitive tasks. Exposure to a mobile phone held to the left ear was for a total of 30 min; there was no dosimetry. Testing was undertaken in a pre-exposure baseline period, and 15 and 30 min after exposure. The authors reported that exposure facilitated cognitive tasks involving attentional capacity and one task which involved processing speed. Smythe and Costall (2003) also examined the effect of RF exposure from a mobile phone held to the left ear on the performance on a verbal memory task by 62 students, randomly assigned to one of three groups. The authors reported a significant improvement in immediate recall in males but not females. There were no effects of exposure on delayed testing.

Maier et al (2004) studied the effect of exposure to a GSM 900 RF on the performance of an auditory discrimination task. This task required participants to determine whether two successive auditory stimuli were temporally separate and on which side the two stimuli were presented. It was carried out before and immediately after the double-blind exposure or sham exposure of 11 volunteers for 50 min to GSM RF from a mobile phone held 4 cm away from the left ear. The authors reported that exposure significantly reduced the subsequent performance of the task. Cinel et al (2007) replicated this study with a larger number of subjects. Two groups of 84 healthy volunteers were exposed for 40 min to either a 888 MHz GSM signal or a continuous signal. They did not find an effect on performance of the auditory task from either signal type. Both studies do not indicate the type of blinding used, however. Curcio et al (2004) also examined the effect of exposure to a GSM 900 signal on subsequent cognitive task performance. Twenty subjects were randomly assigned to two groups, one group exposed for 45 min before, and the other exposed during a 45 min experimental session. Each subject was tested on four performance tasks: an acoustic simple-reaction time task, a visual search task, an arithmetic descending-subtraction task and an acoustic choice-reaction time task. Using a counter-balanced double-blind paradigm, subjects were subjected to three separate trials separated by 48 h: baseline exposure, real exposure and sham exposure. A significant reduction of both simple and choice reaction times was seen in subjects exposed to a GSM signal. In addition, subjects exposed before testing performed faster than those exposed during testing.

The authors suggested that RF exposure for a minimum time of around 25-30 min was required for these effects to become manifest.

A different experimental protocol was used by Besset et al (2005) which attempted to better emulate real-life exposure. In a double-blind study, 55 subjects were assigned to GSM 900-exposed or sham-exposed groups, matched for age, sex and IQ. Over the 45-day experimental period, there was a baseline period of 3 days, an exposure period of 28 days during which exposures or sham exposures took place, and a recovery period of 14 days, during which both groups were sham exposed. Exposures or sham exposures were carried out for 2 h per day, five days per week. Subjects held the phone adjacent to their preferred ear during this time; the localized SAR was approximately 0.54 W kg^{-1}. A neuropsychological battery of 22 tasks screened information processing, attention, memory, and executive function on 4 days during the 45-day period, 13 hours after the previous exposure or sham exposure. Statistically significant decreases in reaction time were seen for all tasks between the baseline and exposure periods, indicating a learning effect, but there was no significant effect of RF exposure on task performance. The authors note that, in contrast to other studies, there was a 13 h delay between exposure and testing, which included an overnight sleep. This indicates that there are no lasting effects of exposure, but it precludes any meaningful comparison with studies that looked at short-term effects.

Russo et al (2006) investigated the effects on cognitive performance of exposure to 888 MHz CW or GSM RF using a large number (168) of male and female volunteers, compared to the earlier studies, increasing the statistical power of the study. The subjects were exposed or sham-exposed in two sessions, separated by one week. Half of the subjects had the left side of the head exposed, and half the right side, irrespective of their handedness. Unlike most previous studies, the RF exposure was carried out under double-blind procedures. Cognitive performance was assessed using similar tasks to those used previously, i.e., reaction time task, 10-choice serial reaction time task, subtraction task and vigilance task, which were administered in a counterbalanced order. The authors found no significant effects of RF exposure on task performance, irrespective of whether the left or right side of the head was exposed.

Keetley et al (2006) investigated the effect of exposure to GSM RF radiation on the cognitive performance of 120 male and female volunteers using a double-blind crossover design. The subjects were exposed or sham-exposed in two sessions, separated by one week. Cognitive performance was assessed using a battery of eight cognitive tests: Rey's audio-visual learning test, digital span test, digital symbol substitution test, speed of comprehension test, trail making task, reaction time task, choice reaction time task and inspection time task, which were administered in a counterbalanced order. After adjusting for known covariates (gender, age and education), simple and choice reaction times showed significant impairment, in contrast to earlier studies (Preece et al 1999; Koivisto et al 2000b), whereas performance on the trail-making task, which involves working memory, significantly improved. However, this study involved numerous comparisons with no adjustment for multiple comparisons (type I error). The authors point out that neither of the earlier studies corrected for known covariates, and that the study of Koivisto et al (2000b) used only a single-blind study design.

Eliyahu et al (2006) examined, in 36 young, right-handed male subjects, the effects of GSM RF exposure of the right or left side of the head on four cognitive tasks selected for high cerebral hemisphere specificity. The authors' intention was to examine the effect of RF exposure of a specific part of the brain on associated cognitive functions. These were a spatial item recognition task (activating the right premotor cortex), a verbal item recognition task (activating the left posterior parietal cortex and supplementary motor and premotor cortex), and two spatial compatibility tasks (a visual stimulus on the left side of the test screen activating the left posterior parietal cortex, and on the right side activating the right posterior parietal cortex). Each task required right- and left-handed responses. The subjects performed 3 series of tests, with exposure to the right or the left hemisphere or with sham exposure, in either of two 1-h sessions, separated by 5 minutes. The study was conducted under single-blinded conditions, and the exposure regime and task sequence were counterbalanced. The authors analyzed the reaction times for correct responses to each task, comparing the exposure condition (left, right or sham) for left hand or for right hand responses. Generally, right-hand responses were faster than left-hand responses (the subjects were right-handed subjects) and strong training effects (reaction times faster in the second session) were present in most sham responses. The authors reported that exposure of the left hemisphere of the brain resulted in slower left-hand responses in the second session compared to the first,

for two tasks: the spatial item recognition task, thought to activate the right premotor cortex, and one spatial compatibility task, where left-handed responses are thought to activate the left parietal cortex. Thus, no correlation was seen between exposure of the left hemisphere and the hemisphere-dependence of the two affected tasks.

Haarala et al (2007) also compared responses of exposure of the left and right hemisphere. They exposed 36 healthy male volunteers to a continuous or GSM signal operating at 0.25 W. In a double blind, crossover design, subjects were exposed in 90 min sessions at 1 week intervals. Different cognitive functions tasks were performed: simple reaction time, 10-choice reaction time, subtraction, verification, vigilance, and memory (n-back test). No difference on response was observed for any task for both continuous wave and GSM exposure between exposure to either the left of right hemisphere and sham exposure. The absence of a difference in response with a control group tested without the exposure equipment indicated that the presence of the exposure equipment also was of no influence. The authors comment that they used fairly simple and hemisphere-aspecific behavioral tasks and that it cannot be excluded that more complex or hemisphere-specific cognitive tasks could be more sensitive to effects of RF exposure.

Terao et al (2006) exposed 16 healthy volunteers for 30 min to an 800 MHz mobile phone signal in a double-blind crossover study. Immediately before and after actual or sham exposure a precued choice reaction time test was performed. Exposure did not have any effect on reaction time or accuracy. Schmid et al (2005) exposed 58 healthy volunteers to UMTS signals resulting in SAR values of 0.37 or 0.037 W kg^{-1}, thus simulating a UMTS phone in transmitting or receiving mode. A double-blind crossover design was used. Four visual perception tests were applied: the Critical Flicker Fusion Frequency test, a visual pursuit test, the Tachistoscopic Traffic test Mannheim, and a contrast sensitivity threshold test. The duration of exposure is not given, but was most likely the time it took to perform the tests. Since for all subjects the test procedures were applied on one single day, carry-over effects might be present. However, in none of the tests was an effect of either level of exposure observed.

Regel et al (2007a) investigated reaction time and memory in 20 subjects exposed to either a 900 MHz continuous or GSM-type signal for 30 min. In this double-blind, randomized, counterbalanced cross-over study no effects were observed in single or 2-choice reaction time tests. An improvement in accuracy in the 3-back memory test was found after GSM-type (pulsed field) exposure, but not after CW exposure. Regel et al (2007b) exposed healthy volunteers for 30 min to a GSM 900 signal immediately before sleep while performing cognitive tasks. Exposure and sham were double-blind, randomized and given at 1 week interval, and importantly they looked for a dose-response relation using 0.2 and 5 W kg^{-1} peak spatial SAR. They observed a dose-related reduction of reaction time with increasing field strength for the 1-back task, and similar relations at trend level for the 2-back task and the choice reaction time task, but no effect on the simple reaction time or 3-back task.

Adults with EMF-attributed symptoms

In a study using exposure similar to that from mobile phone base stations, Zwamborn et al, (2003) investigated subjective feeling and cognitive functions in a group of 36 subjects claiming to experience symptoms in connection with living near a GSM base station and a group of 36 healthy subjects. The groups differed in terms of age and gender distribution and therefore no comparisons could be made between the groups, only within groups for periods with and without exposure. The subjects were exposed to a 1 V/m field at 900 and 1800 MHz (GSM signal), and 2100 MHz (UTMS signal). Each subject participated in three sessions, one of which was unexposed, using a double-blind design. Exposure groups therefore consisted of 24 subjects. Each session took 45 min including exposure (during which cognitive functions were tested), questionnaire, and break. Cognitive function tests included reaction time, memory comparison, dual-tasking, selective visual attention and filtering irrelevant information. A corrected analysis of the data was presented in a report of the Health Council of the Netherlands (2004). In this reanalysis, only one statistically significant result was found with the cognitive function tests. In the control group without symptoms UMTS exposure resulted in an increased completion of the memory comparison test. This could be a chance effect. The results with respect to symptoms have been discussed in the chapter on electrosensitive people.

The follow-up study by Regel et al (2006) investigated the effect only of the 2140 MHz UMTS base-station-like RF signal, identical to that used by Zwamborn et al, on well-being and cognitive performance in 33 RF-sensitive subjects and in 84 non-sensitive subjects. There were three experimental sessions held at one-week intervals; subjects were randomly assigned to one of six possible sequences of three exposure conditions, each lasting 45 min: 0 V/m (sham), 1 V/m (identical to that used by Zwamborn et al), and 10 V/m (in order to assess any possible dose-response relationship). The study was double-blinded with a randomized crossover design. Cognitive performance was assessed using a simple-reaction time task, a 2-choice reaction time task, the N-back task and the visual selective attention task, the latter also used by Zwamborn et al (2003). No effect of either exposure level was observed on cognitive performance. Again, the results with respect to symptoms have been discussed in the chapter on electrosensitive subjects.

Another recent study (Wilén et al 2006) investigated the effects of mobile phone radiation on various physiological parameters such as heart-rate variability, electrodermal activity, and respiration rate, measured before, during and after exposure, in 20 RF-sensitive subjects and in 20 non-sensitive controls. In addition, tests of arousal and vigilance, short-term memory and reaction times were performed before and after exposure. The subjects were exposed or sham exposed to GSM 900 RF for 30 min on two separate days. The study was single-blinded. No significant effects of RF radiation on any physiological or cognitive variable were found. The results with respect to physiological parameters have been discussed in the chapter on electrosensitivity.

Children and adolescents

Haarala et al (2005) and Preece et al (2005) both exposed children to GSM 900 signals in a double-blind cross-over design. In the study by Haarala et al (2005), 32 children (10-14 years old) performed a battery of cognitive tests that were the same as in previous work of the same group on adults (Haarala, et al 2004). There were no significant differences between the exposure conditions in reaction times and accuracy over all tests. In the study by Preece et al (2005), 18 children (10-12 years of age) were tested using the Cognitive Drug Research cognitive assessment system. The two exposure levels were 0.025 or 0.25 W. There were no significant alterations in any of the tests and in particular in reaction times, which had been found to decrease in adults under exposure to more powerful signals (Preece, et al 1999). However, there are some experimental weaknesses in these two studies that limit their interpretation, such as low exposure levels, limited power, and high variability of the tests of cognitive function and their applicability to children (SSI 2006).

Lee et al (2001) compared the performance of schoolchildren, segregated according to mobile phone usage into two groups (37 users vs. 35 non-users), on three ‘paper-and-pencil’ tests of cognitive function: symbol-digit matching, stroop test, and trail making. Mobile phone users were selected according to self-reported usage; the controls were age and sex-matched. The authors reported a mild facilitating effect on attention in the user group. However, AGNIR (2003) note that the effect may reflect the influence of one or more variables confounded with phone use, rather than a direct effect of mobile phone signals on cognitive function. In addition, there was no correction for multiple testing (Haarala et al 2003a). A later single blind study by the same group (Lee et al 2003) further investigated their earlier observation of a facilitating effect with increasing duration of mobile phone exposure. The authors randomly assigned 78 undergraduate students to an exposure or sham exposure group. The same cognitive tests used by Lee et al (2001) and an additional sustained attention task were performed during exposure or sham exposure to 1900 MHz from a mobile phone situated over the right cerebral hemisphere. The authors reported that reaction time in the sustained attention task was decreased in the RF-exposed group, supporting their earlier observation.

Table II.5.6.: Human studies cognitive performance

Assay endpoint	Exposure conditions	Response	Comment	References
Healthy adults				
Cognitive function in two groups of healthy volunteers, 21-60 y (n=18), 20-28 y (n=18).	915-MHz simulated mobile phone signal, 1 W for 25-30 min, continuous or pulsed.	Decrease in reaction time, no effect on other functions. Effect stronger with continuous field.		Preece et al 1999
Reaction time in healthy volunteers, 18-49 y (n=48).	902-MHz field from GSM phone, operating at 0.25 W, for1 h.	Decrease in reaction time in simple reaction and vigilance tasks; decrease in time for mental arithmetic.	Single blind study. No correction for multiple testing.	Koivisto et al 2000a
Working memory in healthy volunteers, 18-34 y (n=48).	902-MHz field from GSM phone, operating at 0.25 W, for 30 min.	Decrease in reaction time in 3-back test, not in 0-, 1-, or 2-back tests. No change in accuracy.	Single blind study.	Koivisto et al 2000b
Reaction time and accuracy in healthy volunteers, 20-42 y (in two labs: Finland: n=32; Sweden: n=32).	902 MHz GSM phone, operating at 0.25 W, for 65 min, with 24 h between sessions.	No effects on reaction time and accuracy.	Replication of Koivisto et al 2000a	Haarala et al 2003b
Memory in healthy volunteers, 20-42 y (in two labs: Finland: n=32; Sweden: n=32).	902 MHz GSM phone, operating at 0.25 W, for 65 min, with 24 h between sessions.	No effect on memory.	Replication of Koivisto et al 2000b	Haarala et al 2004
Attention and memory in students (n=100)	450 MHz, 7 Hz, modulated RF, 0.158 mW/cm^2, for 10-20 min	In exposed subjects, decrease in memory errors but worse attention		Lass et al 2002
Various cognitive tests in healthy volunteers, 20-22 y (exposed and controls: n=19.) Testing before, at 15 and 30 min of exposure.	900 MHz from GSM SAR: 1.19 W kg^{-1}, for 30 min.	At 15 min improved memory and attention.	Exposure not clear; no fixed position of phone.	Edelstyn and Oldershaw 2002
Memory in students (male: n=33; female: n=29)	Mobile phone.	Improved memory in males, but not in females		Smythe and Costall 2003
Auditory discrimination task in healthy volunteers (n=11).	900 MHz GSM phone, for 50 min. Test before / after exposure.	Decreased performance in 9 / 11.	Low number of subjects.	Maier et al 2004
Auditory discrimination task in healthy volunteers, 18-42 y (n=84/group).	888 MHz GSM or continuous signal operating at 1.4 W kg^{-1}, for 40 min Test before / after exposure.	No effect of GSM or continuous signal.	Replication of Maier et al 2004. Blinding not clear.	Cinel et al 2007

Assay endpoint	Exposure conditions	Response	Comment	References
Cognitive performance in healthy adults, 22-31 y (n=10/group).	902 MHz GSM phone, operating at 0.25 W, for 45 min before or during testing.	Decreased reaction time, more after than during exposure. Hypothesis: minimum 25 min needed for changes.	Small groups.	Curcio et al 2004
Cognitive functions in healthy volunteers, 18-40 y (exposed: n=28, sham: n=27). Testing before, halfway during and after exposure period.	900 MHz GSM phone, SAR: 0.54 W kg^{-1}, for 2h/d, 5d/wk, 28d.	Strong learning effect, but no effects of exposure.	Testing only 13 h after last exposure.	Besset et al 2005
Attention, reaction time in healthy volunteers, 17-41 y (n=168)	888 MHz GSM or continuous, SAR: 1.4 W kg^{-1}, for 35-40 min.	No effects.	Replication of previous studies (Koivisto et al 2000b; Curcio et al 2004), with larger sample.	Russo et al 2006
Cognitive functions in healthy volunteers, 18-70 y (n=120). Testing started after 30 min exposure.	900 MHz GSM phone operating at 0.25 W, for 90 min.	Increased simple and choice reaction times, improved working memory.	Unlike other studies, correction for age, gender, education.	Keetley et al 2006
Cognitive functions in healthy volunteers, 19-27 y (n=36).	890 MHz GSM phone, operating at 0.25 W. Exposure of left and right hemisphere and sham exposure during two 1 h sessions with 5 min interval.	Increased left-hand response time with left-side exposure. No effect on dominant hand (all subjects right-handed). No effect on accuracy.	No washout period between exposure conditions; carry-over effect possible. Single blind study.	Eliyahu et al 2006
Cognitive function in healthy volunteers, 23±2 y (n=36); no-exposure-equipment controls, 24±3 y (n=16)	GSM signal, pulsed and continuous, from a mobile phone operated at 0.25 W, at 1 wk interval; exposure of left and right hemisphere during each session.	No effects. Mere presence of exposure equipment also did not affect response.		Haarala et al 2007
Visuo-motor reaction time in healthy volunteers, 23-52 y (n=16).	800 MHz mobile phone, operated at 0.27 W, for 30 min, sessions at 7 d interval. Testing before and after exposure session.	No effects.	Small group.	Terao et al 2006
Visual perception in healthy volunteers, 20-40 y (n=58).	1970 MHz UMTS antenna, SAR: 0, 0.37 and 0.037 W kg^{-1}.	No effect.	All tests performed in one afternoon; washout period not given.	Schmid et al 2005

Assay endpoint	Exposure conditions	Response	Comment	References
Reaction time and memory in healthy volunteers, 19-25 y (n=20).	900 MHz continuous or GSM-type, SAR:1 W kg^{-1}, for 30 min at 1 wk intervals.	No effect on reaction time, increased accuracy in memory test with GSM exposure.		Regel et al 2007a
Cognitive performance in healthy volunteers 20-26 y (n=15)	GSM signal, base-station-like, 900 MHz, 2, 8, 217, 1736 Hz modulation, planar antennas, peak SAR: 0.2 and 5 W kg^{-1} Exposure for 30 min before sleep	Dose-response reduction in 1-back reaction time, trend-level reduction in reaction time for 2-back and choice reaction time, no effect on 3-back or simple reaction time, or accuracy for any of the above.		Regel et al 2007b
Self-proclaimed electrosensitive adults				
Cognitive functions in subjects with symptoms attributed to RF exposure, 31-74 y (n=36), and healthy controls, 18-72 y (n=36).	900 MHz, 1800 MHz (GSM) and 2100 MHz (UMTS) at 0.7 V m^{-1} (GSM) and 1 V m^{-1} (UMTS) for 20-25 min at 20-25 min intervals.	In control group faster completion of memory comparison test after UMTS exposure. No effect in other combinations.	Could be chance effect.	Zwamborn et al 2003 Health Council of the Netherlands 2004
Cognitive functions in self-proclaimed electrosensitives, 20-60 y (n=33), and healthy controls, 20-60 y (n=84).	2140 MHz (UMTS) at 1 or 10 V m^{-1} for 45 min at 1 wk intervals.	No effect on cognitive functions in either group at both levels of exposure.	Study has improved design with respect to Zwamborn et al (2003).	Regel et al 2006
Cognitive functions in self-proclaimed electrosensitives, 32-64 y (n=20), and healthy controls, 29-65 y (n=20).	900 MHz from GSM phone, SAR: 1 W kg^{-1}, for 30 min at 1 d interval.	No effect on cognitive functions.	Difference in baseline response between electrosensitives and controls. Single blind study Effects on symptoms reported in next chapter.	Wilén et al 2006
Children and adolescents				
Cognitive functions in children, 10-12 y (n=18)	902 MHz from GSM phone, operating at0.025 and 0.25 W, for 30-35 min at 24 h intervals.	No effects.		Preece et al 2005
Cognitive function in children, 10-14 y (n=32)	902 MHz from GSM phone, operating at 0,25 W, SAR: 0.99 W kg^{-1}, for 50-65 min at 24 h intervals.	No effects.		Haarala et al 2005

Assay endpoint	Exposure conditions	Response	Comment	References
Attention in same-level high school students (mean age 16 y).	Mobile phones users (n=37) vs non-users (n=35)	Better performance in users in 1 of 3 tests.	Large variation in phone use (175 – 27240 min total use time). No correction for multiple testing.	Lee et al 2001
Attention in undergraduate students.	1900 MHz from GSM phone for 25 min, 2 trials 2 min apart.	Decreased reaction time in 2nd trial in users. No effect on accuracy.	Single blind study.	Lee et al 2003

II.5.1.5. Subjective Symptoms

A wide range of subjective symptoms has been attributed to exposure to various sources of RF both at home and at work. Some people report they suffer a variety of subjective complaints, including headaches and migraines, fatigue, skin itches, and sensations of warmth (Frey 1998a & b; Hocking 1998; Chia et al 2000a & b; Hocking and Westerman 2001; Sandström et al 2001; Santini et al 2002, 2003; Rubin et al 2005; Röösli 2008). They attribute these symptoms to exposure from mobile telephones, nearby base stations, DECT cordless phones and, more recently, wireless LAN systems. Less commonly reported symptoms include dizziness, blurred vision, memory loss, confusion and vagueness, toothaches, and nausea. An increasing number of those people considers themselves electrosensitive.

The prevalence of these symptoms, and of the associated self-proclaimed electrical hypersensitivity, has been investigated in several countries. Hillert et al (2002) sent a paper questionnaire to 15000 adult residents of the Stockholm county. With an overall high response rate of 73%, 1.5% of the respondents reported to be sensitive to EMF exposure. The prevalence was higher in women than in men and the highest prevalence was found in the age group 60-69. Eriksson and Stenberg (2006) performed a more general study of symptoms related to the indoor environment, both at home and at work, among 2154 people in Sweden. They found that women reported symptoms more often than men. Symptoms associated with electrical hypersensitivity (EHS) were more frequent among VDU users. Levallois et al (2002) performed telephone interviews with 2072 Californians. They found a self-reported prevalence of 3.2% for being “allergic or very sensitive” to being near electrical devices, which was strongly associated with self-reported environmental illness or multiple chemical sensitivity. EHS only pertained to devices emitting power-frequency fields, however.

Several other studies investigated a possible association between mobile phone use and symptoms through questionnaires. Oftedal et al (2000) sent a questionnaire to 12000 Swedes and 5000 Norwegians. 13% of the respondents in Sweden and 31% in Norway indicated to have at least one of the symptoms questioned. Of those people, 45% indicated that they had taken measures to reduce exposure. The study was not blinded in that it was indicated that it was a study into effects of mobile phone use. This may have influenced the responses. Another problem is that the response rates between the two groups were rather different: 76% in Sweden and 64% in Norway. The main question addressed by this study, however, was whether there would be differences in pattern and types of symptoms between users of analog and digital phones. This was addressed by Sandström et al (2001). It appeared that the prevalence of symptoms was similar in users of either phone type. Overall, longer calls and a higher number of calls were associated with a higher prevalence of warmth behind or around or on the ear and with headaches and fatigue. This observation led to a third analysis that included a subset of 2197 users that made more than 2 calls per day and used one of four different types of GSM phones (Wilén et al 2003). These were selected to result in a range of SAR values. The prevalence of dizziness, discomfort and warmth behind the ear were associated with exposure to SAR values higher than 0.5 W kg^{-1} and long calling times. According to Sandström et al (2001) confounding factors such as psychosocial workload, occupation, and gender might affect the prevalence of symptoms. In this subset study the authors found that it was not possible, however, to correct for these factors because of the relatively small numbers of cases. The same group also studied heart function and mood in 14 self-proclaimed EHS and an equal-size control group

(Sandström et al 2003). In the EHS group they observed larger heart rate variability. They also performed ELF measurements in the homes and found that the ELF levels were not associated with mood.

Provocation studies provide the most direct way of studying a possible effect of RF exposure on the occurrence of symptoms. A setback is that such studies only investigate direct, short-term interactions, while symptoms may only occur after some longer exposure time. In a single-blind provocation study, Koivisto et al (2001) presented two groups of 48 individuals without symptoms with either real or sham exposure to a pulsed 902 MHz field. Two experiments were performed. In the first, exposure and sham were in two 1-h sessions separated by 24 h. In the second, 30-min sessions were given without interval. Subjects were asked to rate subjective symptoms and sensations during the sessions. No significant differences were found between exposure conditions, although fatigue and headaches increased toward the end of sessions. In another single-blind provocation study, Hietanen et al (2002) challenged 20 volunteers who reported themselves to be sensitive to RF using analogue or digital phone signals. Blood pressure, heart rate and breathing rate were measured every 5 min and subjects were asked to report any abnormal feelings. Nineteen of the subjects reported symptoms, most of which were sensations in the head of pain or warmth. However, more symptoms were reported during sham exposure than real exposure. The subjects could not indicate whether they were being exposed and by what type of signal. The physiological parameters showed no relevant trends, although they tended to decrease throughout the day.

In a double-blind provocation study, Rubin et al (2006) investigated the effect of exposure to GSM RF on the severity of the symptoms experienced by 60 EHS, compared to 60 'non-sensitive' subjects. Each subject was exposed or sham exposed for 50 min either to a GSM 900 signal or to a non-pulsed signal, both of which induced a localized SAR in the region of the head adjacent to the phone of 1.4 W kg^{-1}. There were three separate experimental sessions over a two-year period within which the order of presentation was randomized and counter-balanced. All subjects were asked to score on visual analogue scales before, during and after exposure, the severity of headaches and various other symptoms such as nausea, fatigue, dizziness. The authors found that the proportion of sensitive participants who believed a signal was present during GSM exposure (60%) was similar to the proportion (63%) who believed one present during sham exposure. In addition, the prevalence of various symptoms experienced during exposure or sham exposure in people who reported themselves as GSM-sensitive was very much higher than in non-sensitive subjects, but this occurred irrespective of the exposure condition. In some cases, for sensitive subjects, the symptoms experienced were so severe that the individual withdrew from the study. Rubin et al (2006) suggested that psychological factors, possibly the conscious expectation of such symptoms (the nocebo effect), might have a key role in the etiology of this condition.

Wilén et al (2006) investigated the effects of mobile phone exposure on various physiological parameters such as heart rate variability, electrodermal activity, and respiration rate, measured before, during and after exposure, in twenty self-proclaimed RF-sensitive subjects and in twenty non-sensitive controls. In addition, cognitive function tests were performed; these have been described in the previous chapter. The subjects were exposed to GSM 900 RF for 30 min or sham exposed on two separate days. The study was single-blinded. No significant effects of RF radiation on any physiological parameter were found. However, people who experienced subjective symptoms showed differences in heart-rate variability compared to controls. Wilén et al (2006) suggested that these results might reflect differences between these two groups in autonomic nervous system function. Using the same exposure setup, Oftedal et al (2007) studied headache, discomfort and various physiological parameters in 17 subjects that attributed their symptoms to mobile phone use, using a double blind, sham-controlled design. Exposure was given in four 30-min sessions separated by 2 days. An increase in headache and discomfort was found after sham, but not after real exposure; subjects could not perceive being exposed. There were no effects on heart rate and blood pressure.

All these studies used mobile telephones as sources of exposure. It is however also of interest to study exposure at levels as experienced continuously in daily life that result from base station antennas. This was the idea behind the study of Zwamborn et al (2003). They explored the effects of exposure to GSM and UMTS signals on and cognitive functions (which have been reported in the previous chapter) and self-reported well-being (that will be reported here). A small, but significant, decrease in well-being after UMTS exposure was seen in both study groups (subjects who had previously reported symptoms

attributed to GSM radiation and a control group without such symptoms). No effects were seen using GSM signals either at 900 or 1800 MHz. However, the validity of the questionnaire used to score well-being was challenged in the comprehensive analysis of this study by the Health Council of the Netherlands (2004). A follow-up study by Regel et al (2006) using an improved protocol with greater numbers of subjects investigated the effect only of the 2140 MHz UMTS base-station-like RF signal, identical to that used by Zwamborn et al (2003), on cognitive performance (reported in the previous chapter) and well-being in 33 self-proclaimed RF-sensitive subjects and in 84 non-sensitive subjects. Well-being was assessed using three standard questionnaires, one of which was identical to that used in the Zwamborn et al (2003) study. Well-being was not affected by UMTS radiation at either of two exposure levels. Even though RF-sensitive subjects generally reported more health problems, Regel et al (2006) found no difference between the two groups with respect to the applied field conditions. Subjects were also not able to discriminate between exposure levels, but they reported more health complaints when they suspected exposure, suggesting that, as indicated above, psychological factors may be involved in this condition.

This can also be concluded from the study of Lonne-Rahm et al (2000) who did not use exposure to an RF source, but instead used a VDU which only minimally emits RF EMF. Nevertheless the study is worth brief discussion here, because of the similarity of the results with the studies described above. Lonne-Rahm et al studied a group of 24 EHS subjects and 12 controls. They subjected participants to situations with either low or high stress (by exposing them to flashing lights while trying to solve mathematical problems) with or without exposure to EMF from a VDU. The EHS subjects reported increased skin symptoms when they thought that they were perceiving fields, but there was actually no difference between situations with the VDU field present or absent. No effect was detected on inflammatory mediators and skin mast cells.

Table II.5.7.: Subjective symptoms

Study endpoint	Exposure conditions	Response	Comment	References
Prevalence				
Prevalence of self-reported hypersensitivity to electric or magnetic fields in Stockholm county, (n=15000, 19-80 y) assessed by paper questionnaire.		Response rate 73%. EHS reported by 1.5% of respondents. Highest in women and 60-69 y age group.		Hillert et al 2002
Prevalence of symptoms related to indoor environment in Sweden (n=2154, 18-64 y).		Response rate 70%. High prevalence in VDU users.		Eriksson and Stenberg 2006
Self-reported hypersensitivity to EMF in California, USA (n=2072), assessed by telephone interview.		"Allergic or very sensitive" to being near electrical devices: prevalence = 3.2% (95% CI: 2.8, 3.7).	Alleging environmental illness or multiple chemical sensitivity was the strongest predictor of reporting being hypersensitive to EMFs. Only ELF field exposure questioned.	Levallois et al 2002

Study endpoint	Exposure conditions	Response	Comment	References
Descriptive studies				
Symptoms experienced by mobile phone users, assessed by paper questionnaire (Sweden : n=12000, Norway: n=5000).	Telephone use in daily life by people who own a job-related telephone.)	Response rates 76% (Sweden), 64% (Norway). 13% (Sweden) or 31% (Norway) of respondents had at least one symptom. 45% of those had taken measures to reduce exposure.	Study not blinded.	Oftedal et al 2000
Comparison of symptoms experienced by users of analogue (n=8113) and digital mobile phones (n=8879).	Telephone use in daily life by people who own a job-related telephone.	No difference in prevalence of symptoms. Higher prevalence of warmth behind/around or on the ear, headaches and fatigue with longer calling times or # calls.	Same group of subjects as in Oftedal et al (2000.)	Sandström et al 2001
Symptoms experienced by mobile phone users (n=2197) assessed by paper questionnaire.	Telephone use in daily life by people who own a job-related telephone.	Prevalence of some symptoms higher with SAR > 0.5 W kg^{-1} and long calling times.	Sub group of subjects from Oftedal et al (2000). No correction for possible confounding factors.	Wilén et al 2003
ECG in patients with perceived electrical hypersensitivity (n=14) and controls (n=14). Assessment of mood by paper questionnaire.	Exposure to RF and ELF in daily life; measurement of ELF.	No effect of ELF exposure on mood. Disturbed pattern of heart rate variability in patients.	Small groups.	Sandström et al 2003
Provocation – GSM/UMTS				
Subjective symptoms associated with GSM use in subjects without symptoms (experiment 1: 18-49 y, n=48; experiment 2: 18-34 y, n=48).	GSM 900 phone, operating at a mean power of 0.25 W. Exposures for 60 min at 24 h interval, or for 30 min without interval. Questionnaire at start, middle, end of session.	No effect.	Single blind study.	Koivisto et al 2001

Study endpoint	Exposure conditions	Response	Comment	References
Hypersensitivity symptoms associated with mobile phone use in self-reported electrosensitives, 37-67 y (n=20). Measurement of blood pressure, heart rate and breathing frequency every 5 min.	NMT 900 / GSM 900 and 1800 phones, operating at output power of resp. 1, 0.25 and 0.125 W, for 30 min with minimally 60 min interval.	More symptoms reported during sham exposure. No ability to detect exposure.	Single blind study.	Hietanen et al 2002
Sensitivity to mobile phone signals in subjects with / without symptoms attributed to GSM exposure (n=60/group).	GSM 900, 900 MHz continuous, SAR:1.4 W kg^{-1} ± 30%, for 50 min at intervals of at least 24 h.	No difference in reported symptoms between groups and between exposure and sham. No field perception.	Results suggested as being 'nocebo' effect.	Rubin et al 2006
Mobile phone related symptoms in self-proclaimed electrosensitives, 32-64 y (n=20), and healthy controls, 29-65 y (n=20).	900 MHz from GSM phone, SAR:1 W kg^{-1}, for 30 min at 1 d interval.	No effects of exposure. Indication for difference in autonomous nervous system regulation between cases and controls.	Single blind study. Only subjects with phone-related symptoms, no general electrosensitivity. Effects on cognitive functions reported in previous chapter.	Wilén et al 2006
Headache associated with mobile phone use in subjects with symptoms attributed to mobile phone use, 20-58 y (n=17).	900 MHz from GSM phone, SAR: 1 W kg^{-1}, for 30 min at 2 d interval.	Increase in headache and discomfort higher with sham; no effect on heart rate, blood pressure; no perception of exposure.	Only subjects with phone-related symptoms, no general electrosensitivity. Results explained as 'nocebo' effect.	Oftedal et al 2007
Well being in subjects with symptoms attributed to RF exposure, 31-74 y (n=36), and healthy controls, 18-72 y (n=36).	900 MHz, 1800 MHz (GSM) and 2100 MHz (UMTS) at 0.7 V m^{-1} (GSM) and 1 V m^{-1} (UMTS) for 20-25 min, at 20-25 min intervals.	Slightly decreased well-being after UMTS exposure.	Hypothesis-generating study; small numbers/group; questionable validity of well-being questionnaire.	Zwamborn et al 2003
Well-being in self-proclaimed electrosensitives, 20-60 y (n=33), and healthy controls, 20-60 y (n=84).	2140 MHz (UMTS) at 1 or 10 V/m for 45 min. at 1 wk intervals.	No effect on well-being in either group at both levels of exposure. No ability to detect exposure.	Replication of Zwamborn et al 2003 study with improved design.	Regel et al 2006

Study endpoint	Exposure conditions	Response	Comment	References
Provocation – other				
Provocation with stress and EMF of patients with "sensitivity to electricity" (n=24) and controls (n=12).	30 min high/low stress with/without VDU exposure, at 1 week interval.	Patients reported increased skin symptoms when perceiving fields. In effect, no difference between fields on / off. No effect on inflammatory mediators and skin mast cells.	Small groups. Little RF exposure form VDU.	Lonne-Rahm et al 2000

II.5.1.6. Summary on nervous system

There is some evidence for effects of exposure to a GSM-type signal on the spontaneous EEG. A well-performed large study has confirmed previous smaller studies in finding increased power in the alpha band (8–12 Hz) of brain activity. Effects on other frequency bands of natural brain activity have not been consistently demonstrated. These observations are not corroborated, however, by the results from studies on evoked potentials. Although in some studies some small but inconsistent effects were observed, no effects at all were found when auditory evoked potentials were assessed in the same large study group described above.

A similar conclusion of variable results can be drawn with respect to the effects of exposure to GSM-type signals on sleep, although there is some evidence emerging that suggests there may be an effect on sleep EEG. Some studies, but not all, have indicated effects on EEG power in alpha or beta bands with exposure during sleep. A reported shortening of sleep latency was not subsequently reproduced. Other studies which looked at exposure during 30 min before going to sleep also reported variable results, sometimes reporting increases in alpha and beta band power. In summary, exposure to a GSM-type signal may result in minor effects on brain activity, but it should be stressed that such changes have not been found to relate to any health effects. There are some indications of changes in regional cerebral blood flow during and following RF exposure, but the available data are equivocal. It should be noted that changes in rCBF are not by themselves an indication of health damage. No consistent cognitive performance effects were seen. Studies with larger numbers of subjects generally show no effect. No higher sensitivity was shown in children nor in self-proclaimed electrosensitives compared to healthy adults. If anything, any effect is small and exposure seems to improve performance. It was not possible to derive a dose-response relationship.

The weight of evidence from the studies on auditory and vestibular function indicates that neither hearing nor the sense of balance is influenced by short-term exposure to mobile phone signals.

A wide range of subjective symptoms including headaches and migraine, fatigue, and skin itches have been attributed to various RF sources both at home and at work. However, in provocation studies a causal relation between EMF exposure and symptoms has never been demonstrated. Possibly the conscious expectation of such symptoms may play a role in the etiology of this condition.

II.5.2. Endocrine system

The majority of volunteer studies of the effects of RF radiation on the endocrine system have focused on hormones released into the blood stream by the pineal and pituitary neuroendocrine glands. These are both situated in the head and intimately connected with and controlled by the nervous system. The hormones they release exert a profound influence on body metabolism and physiology, particularly

during development and reproduction, partly via their influence on the release of hormones from other endocrine glands situated elsewhere in the body.

Most studies have investigated the effects of RF exposure on circulating levels of the pineal hormone melatonin or on the urinary excretion of the major metabolite: 6-sulphatoxymelatonin (aMT6s). Fewer studies have been carried out on circulating levels of pituitary hormones or other hormones released from other endocrine glands such as the adrenal cortex.

II.5.2.1. Melatonin studies

Melatonin in humans is produced in a distinct daily or circadian rhythm, peaking during the night and strongly influences circadian physiology and behavior. Night-time peak values of serum melatonin, however, can vary up to ten-fold between different people.

Mann et al (1998) studied the effect of exposure and sham exposure on two successive nights to circularly polarized GSM 900 RF on the nocturnal profiles of melatonin and three other hormones (see below) in 22 male subjects. The treatments were carried out from 23.00 to 07.00 the following morning in a randomized order of presentation under single-blind conditions. Blood samples were withdrawn from an indwelling catheter every 20 min throughout this period. There was no statistically significant effect on night-time serum melatonin levels.

De Seze et al (1999) evaluated the effect on the serum melatonin levels of 2-h daily exposure, 5 days per week, of 19 male volunteers to GSM 900 and 19 others to GSM 1800 mobile phone radiation over a period of four weeks. Exclusion criteria included night-shift work, endocrine disorders and other factors. However, it is not clear whether confounders such as alcohol, coffee or light-at-night were controlled. Blood samples were taken at regular intervals throughout a 24-h period before exposure, after 2 and 4 weeks exposure, and 2 weeks following exposure. Each subject acted as their own control; sham exposures were carried out on the days of the pre-exposure and post-exposure sampling sessions. There was no effect of exposure to either mobile phone signal on the serum melatonin profiles, suggesting that there was no overall cumulative or persistent effect on melatonin secretion.

Radon et al (2001) investigated the effect of circularly polarized GSM 900 signal on salivary melatonin (and cortisol; see below) in 8 volunteers. The RF signal was transmitted by an antenna positioned 10 cm behind each subjects head. In double-blind trials, each subject underwent a total of 20 randomly allotted 4-h periods of exposure and sham exposure between 12.00 and 16.00 or 22.00 and 02.00 the following morning, over a five month summer period. Each treatment period was separated by 2-3 days. Saliva samples were taken at regular times on the day of treatment and on wakening the following morning. There were no significant differences in salivary melatonin concentrations between the exposed and sham exposed conditions. The data were, however, rather variable, a possible consequence of the small number of subjects.

Bortkiewicz et al (2002) exposed or sham exposed 9 male volunteers to GSM 900 RF between 18.00 and 19.00 h emitted from a mobile phone positioned near the subject's head. Urine samples were collected at 19.00, 24.00 and 07.00 the following day and were analyzed for aMT6s content, normalized to creatinine content (a standard procedure to account for errors in the estimation of urine volume). Exposure and sham exposure of all 9 subjects took place on separate days more than one week apart. There was no significant change in aMT6s excretion at these three time-points. There was, however, considerable variation in aMT6s excretion between individuals, reducing the power of the study to detect any effect.

Jarupat et al (2003) studied the effect of exposure and sham exposure to 1906 MHz RF from a mobile phone (Japanese signal) on salivary melatonin levels in eight female subjects. The study was a crossover design. The subjects entered a climatically controlled chamber at 10.00 and held the phone to their left ear for 30 min every hour from 19.00 to 01.00 over a two day period. The authors state that the subjects were unaware whether the phone emitting RF radiation or not, but it is not clear whether this sequence was randomized. Salivary samples were taken at the start of the treatment, and one hour after the treatment terminated. Salivary melatonin levels were reported to be significantly depressed following RF exposure compared to levels taken after sham exposure. The volunteers had led 'a well-regulated life' for a week

before the study, but the authors acknowledge there may have been uncontrolled factors and recommended a larger study.

Wood et al (2006) examined the effect of exposure to GSM 900 RF from a mobile handset on the night-time excretion of aMT6s in 55 adult volunteers. The study was a double-blind crossover design; the subjects were both exposed or sham exposed for 30 min in random sequence on 2 successive Sunday nights. Urine collection was taken shortly after exposure, prior to retiring to bed, and on rising next morning. The authors reported that, after normalization to creatinine concentration, the pre-bedtime aMT6s concentrations were significantly reduced (by about 27%) in the exposed group compared to the sham-exposed group. There was no difference between the post-bedtime measures. The authors acknowledge that the significant result may be spurious, but speculate that it may indicate a delay in onset of the night-time rise in circulating melatonin levels, possibly in a sensitive sub-group of 4 individuals. However, given that the pre-bedtime urine sample was taken shortly after the end of the 30 min exposure, it is difficult to see how there would have been sufficient time for any exposure-induced change in the circulating melatonin levels to have significantly influenced the aMT6s urinary content. This suggests that the effect is more likely to be spurious, perhaps a result of possible lifestyle confounders that were uncontrolled.

II.5.2.2. Pituitary and other hormones

The main pituitary hormones investigated in EMF studies include several hormones involved in the control of growth and body physiology, particularly thyroid-stimulating hormone (TSH) that controls the function of the thyroid gland and the release of thyroxin, adrenocorticotrophic hormone (ACTH), which regulates the function of the adrenal cortex and particularly the release of cortisol, and growth hormone (GH). Hormones released by the pituitary which have important sexual and reproductive functions have also been studied, particularly follicle stimulating hormone (FSH), luteinizing hormone (LH) and prolactin (PRL).

Mann et al (1998), as part of a wider study of the effects of circularly polarized GSM 900 RF on the neuroendocrine system, studied effects on the nocturnal profiles of cortisol, GH and LH, as well as melatonin (see above) in 22 male subjects. Exposure and sham exposure were carried out on two successive nights from 23.00 to 07.00 the following morning in a randomized order of presentation under single-blind conditions. Blood samples were withdrawn from an indwelling catheter every 20 min throughout this period. The authors reported no significant effects on GH or LH levels, but there was a slight, transient elevation of cortisol levels immediately after the onset of RF exposure, suggesting a transient activation of the pituitary adrenal axis.

De Seze et al (1998) evaluated the effect on the hormones of the anterior pituitary gland of 2-h daily exposure, 5 days per week, of 18 male volunteers to GSM 900 RF over a period of four weeks. Subjects acted as their own controls. Blood samples were taken during 9 weeks, 3 weekly samples before exposure, 4 during exposure and 2 after exposure. Most sample were taken on a Monday; thus, during the exposure period, blood samples were taken 48 h after GSM exposure on the previous Friday except following the last exposure (week 7), when a blood sample was taken the day after (Saturday). Because some hormone levels respond quickly to the stress of having a blood sample taken a 15-min rest period after skin puncture but before blood withdrawal was instituted. The samples were tested for 6 hormones: ACTH, TSH, GH, PRL, LH and FSH. Statistical analysis was adjusted for multiple comparisons. All mean hormone concentrations remained within the limits of physiological variation; although for some individuals, aberrant levels of ACTH, GH and PRL, which are known to be affected by stress, suggested that puncture stress hadn't been completely eliminated. Otherwise, there was no significant weekly variation in five of the six hormones studied, but TSH levels showed tendency to decrease from the baseline (i.e. mean pre-exposure value) during exposure, reaching statistical significance (21% decrease) on the 7^{th} sampling, i.e. on the day following the last day of GSM exposure, but recovered to the baseline value during the post-exposure period. The authors concluded that there was no long-lasting or cumulative effect of GSM radiation.

As part of the melatonin study described above, Radon et al (2001) investigated the effect of circularly polarized GSM 900 signal on cortisol levels, directed to the back of the head of 8 volunteers. In double-

blind trials, each subject underwent a total of 20 randomly allotted 4-h periods of exposure and sham exposure over a five-month period and saliva samples were taken at regular times on the day of treatment and on wakening the following morning. There were no significant differences in salivary cortisol concentrations between the exposed and sham exposed conditions. Again however, the data were rather variable, a possible consequence of the small number of subjects.

As part of a study of RF effects on the cardiovascular system (see below) Braune et al (2002) investigated effects on serum levels of cortisol, epinephrine and norepinephrine in 40 young male subjects in a single-blind, randomized crossover study design. Successive periods of sham exposure and exposure to a GSM 900 signal emitted over the right side of the head were given in a randomized order once on each of two different days. No effects of RF exposure were seen.

Table II.5.8.: Endocrine responses

Assay endpoint	Exposure Conditions	Response	Comment	References
Hormones and sleep parameters in healthy volunteers 18-37 y (n=22)	GSM signal from planar antenna, 900 MHz, 217 Hz modulation, 40 cm below pillow, 0.2 W m^{-2}, EEG and exposure continuous for 8 h	Slight elevation of cortisol serum immediately after onset of exposure, persisting for 1 h. No effects on GH, LH and melatonin.	Control for interference. Effect on sleep parameters described in Wagner et al 1998	Mann et al 1998
ACTH, TSH, GH, PRL, LH and FSH in the morning two days after last exposure session (n=18)	GSM 900 MHz mobile phone at max power, 2 h daily, 5 days per week. SAR: 0.3 W kg^{-1}	21% TSH decrease on Only significantly different in the 7th sampling	n=18. One difference in Possible chance effect (9 samplings of 6 hormones)	De Seze et al 1998
Serum melatonin chronobiological rhythm, once every two weeks, 4 sessions (n=19 at each frequency)	GSM 900 and 1800 MHz mobile phone at max power, 2 h daily, 5 days per week. SAR: 0.3 W kg^{-1}	No effect	n=19 at each frequency. No effect on melatonin and no cumulative effect	De Seze et al 1999
Salivary melatonin and cortisol. Samplings during the day and the morning following the exposure session (n=8)	Circularly polarized GSM 900 RF. Antenna 10 cm behind the head for 4 h periods in the day and in the night	No effect	Low number of subjects	Radon et al 2001
Serum levels of cortisol, epinephrine and norepinephrine (n=40)	GSM 900 mounted over the right side of the head, single-blind, randomized cross-over study design	No effect		Braune et al 2002
Urine aMT6s content, normalized to creatinine content (n=9)	GSM 900 RF; mobile phone near the head for 1h	No effect	Low number of subjects	Bortkiewicz et al 2002
Salivary melatonin levels before and after the exposure period (n=8)	1906 MHz RF from a mobile phone, exposure period of 7 x 0.5 h in the evening	Decrease of 42% after exposure	Low number of subjects	Jarupat et al 2003

Assay endpoint	Exposure Conditions	Response	Comment	References
Urine aMT6s content, normalized to creatinine content, on the evening following exposure and on the following morning (n=55)	GSM 900 RF from a mobile handset for 0.5 h - double-blind crossover design	Decrease of 27% on the evening sampling following exposure.	Not physiologically relevant (no time for urine excretion to occur after exposure)	Wood et al 2006

II.5.2.3. Summary on endocrine system

No cumulative effect seems to occur upon repeated chronic exposure for one month on serum melatonin or pituitary hormones. Most studies did not report effects after acute exposure, but often, statistical power was insufficient because of the low number of volunteers. Only one study with acutely repeated exposure seems worth confirming, showing a melatonin decrease in saliva samplings in the morning after 7 consecutive 0.5-h sessions every hour in the evening.

II.5.3. Cardiovascular function and thermoregulation

Volunteer studies have investigated the effects of mobile phone type RF radiation at levels generally assumed to be too low to induce significant heating. A number of studies have been carried out investigating possible effects on heart rate, heart rate variability and on blood pressure. In addition, there is an established literature on cardiovascular responses to RF heating, such as those involved in thermoregulation, and a number of studies address these endpoints. Indeed, such thermoregulatory responses are mediated primarily through well-understood changes in cardiovascular system dynamics and this topic is therefore included here along with a discussion of heat stress disorders and the effects of localized heating in order to place the possible health consequences of RF heating into a broader occupational and environmental context. In particular, a full evaluation of the possible health effects of an RF heat load should also take into account all sources of heat, such as rate of physical work, and the ease with which heat can be lost from the body, which in turn depends to some extent on climatic conditions, clothing etc.

II.5.3.1. Heart rate and blood pressure changes

Braune et al (1998) have reported acute effects on blood pressure in 10 human volunteers exposed to a conventional GSM digital mobile phone positioned close to the right side of the head. After 35 min of exposure, heart rate, blood pressure and capillary perfusion were measured with the subject either supine or standing for 60 s. They found that the heart rate during these tests was slightly lower after exposure to RF than following non-exposed control sessions, and both systolic and diastolic blood pressure were elevated by 5-10 mm Hg. Since capillary perfusion (blood flow through capillaries of the hand) was decreased, the authors concluded that the effects on blood pressure were due to excessive vasoconstriction. This study has been criticized on the basis of both its design and the statistical analysis (Reid and Gettinby 1998). In particular, the 'placebo' (sham exposure) session preceded the test session for all subjects, and therefore the small cardiovascular changes might have been resulted simply from the lengthy period of the experiment.

Braune et al (2002) further investigated these effects on 40 young male subjects in a single-blind, 'randomized crossover study design. Successive periods of sham exposure and exposure to a GSM 900 source mounted over the right side of the head were given in a randomized order once on each of two different days. As in the previous study, systolic and diastolic pressure showed a slow continuous increase of about 5 mm Hg throughout the 50-min protocol; heart rate remained constant. This change in blood pressure was however independent of RF exposure.

Huber et al (2003) reported on an extended analysis of data first published in two previous studies (Borbély et al 1999; Huber et al 2000), focusing on further analysis of EEG recordings (see Chapter

II.5.1.1) and on recordings of the ECG, particularly heart rate and heart rate variability. In these previously published studies, volunteers were exposed to GSM 900 signals either during sleep or during the waking period preceding sleep. In the first experiment, subjects were exposed intermittently during an 8-h night-time sleep period and, in the second experiment, on one side of the head for 30 min before a 3-h daytime sleep period. RF exposure prior to sleep reduced the heart rate during waking and stage-1 sleep, but not during RF exposure itself. Heart rate variability was affected during sleep in both experiments, showing both increases and decreases in the spectral power content prior to and during sleep. The authors speculate that this might indicate changes in sympathetic or vagal activity.

Tahvanainen et al (2004) measured heart rate and blood pressure responses in 32 volunteers during and after a randomized double-blind 35 min exposure to 900, 1800 MHz and sham exposure in three separate exposure sessions. Cardiovascular responses were evaluated in terms of blood pressure and heart rate during four different tests of autonomic regulation of these end-points: a spontaneous breathing test, a deep breathing test, a head-up tilt table test and an expiratory breath test. There were no effects of exposure to either RF frequency on diastolic or systolic blood pressure, or on heart rate, either during or after exposure.

Nam et al (2006) investigated the effects of 30-min sham exposure followed, after a break of 30 min, by exposure to CMDA 835 MHz RF, on blood pressure and heart rate, along with respiration rate and skin resistance (measured on two fingers), in a group of 21 teenagers, and in a group of 21 adults. These parameters were measured after an initial 10 min rest, after 15- and 30-min RF exposure or sham exposure, and 10 min after exposure termination. There were no significant changes in heart rate, diastolic or systolic blood pressure, or respiration rate during any part of the study. However, skin resistance reportedly decreased in the teenagers after 15 and 30 min of RF exposure, and in all males, when grouped together for analysis, after 30 min; in both cases returning to baseline levels within 10 min after the cessation of RF exposure. The authors suggest that the result indicates possible teenage and male susceptibility to CDMA RF radiation. However, the study design, in which sham exposure is always followed by RF exposure, mirrors that of the study by Braune et al (1998), criticized for its lack of randomization of the sham/exposure sequence.

Parazzini et al (2007) focused their investigation of possible GSM 900 RF effects on heart rate variability in 26 volunteers. Frequency and time-domain analysis of heart rate variability is thought to provide quantitative information regarding the sympathetic and parasympathetic control of heart rate by the autonomic nervous system. Heart rate variability data were collected during two different sessions, one with a real RF exposure and the other with a sham exposure. These sessions were performed on separate days in a random order following a double blind experimental design. During each 26 min session, the subject underwent a standard rest-to-stand protocol, thought to elicit sympathetic activity. Data analysis revealed that RF exposure did not affect most heart rate variability parameters; however, a few weak but statistically significant changes were seen in some minor indices of heart rate variability such as an increase the low frequency component as subjects moved into the stand position. The authors suggest this might indicate an augmentation of sympathetic activity. However, it is not clear whether the analysis allowed for multiple testing, and so the significance of these minor changes may have been overestimated.

Barker et al (2007) examined the effects of both TETRA and GSM signals on blood pressure and heart rate variability in 120 subjects. In this study, the subjects were seated and blood pressure and heart rate were recorded during a 20-min pre-exposure period, and a 40-min double-blind RF exposure or sham-exposure session. Four different sets of RF signals were applied in addition to the sham exposures: GSM modulated signals, GSM carrier wave, TETRA modulated, and TETRA carrier wave. The authors found no effect of any RF signal on mean arterial blood pressure, or on any measure of heart rate variability, either in the low frequency or high frequency bands. However, mean arterial blood pressure was reduced (by ~ 0.7 mm Hg) for GSM sham exposures and the authors speculate that this might have resulted from a slight increase in the operating temperature of the handset when in this mode.

Table II.5.9.: Heart Rate and Blood Pressure

Assay endpoint	Exposure Conditions	Response	Comment	References
Heart rate, blood pressure and capillary perfusion (n=10)	35 min exposure to GSM digital mobile phone	Lower heart rate and elevated systolic and diastolic blood pressure by 5-10 mm Hg	No cross-over design with risk of systematic bias due to order	Braune et al 1998a
Systolic and diastolic pressure (n=40)	GSM 900 source mounted over the right side of the head	Heart rate constant; 5 mm Hg pressure increase, not related to exposure	Confirm a bias in the previous experiment	Braune et al 2002
Heart rate and heart rate variability (ECG) (n= 14; Borbély et al 1999; n=16; Huber et al 2000)	GSM 900 signals	Heart rate variability affected suggesting changes in vagal or sympathetic activity	Extended analysis of data from Borbély et al 1999 and Huber et al 2000.	Huber et al 2003
Heart rate and blood pressure (n=32)	Randomized double-blind 35 min exposure to 900, 1800 MHz and sham exposure in three separate exposure sessions	No effect		Tahvanainen et al 2004
Blood pressure and heart rate, along with respiration rate and skin resistance (n=21 adults; n=21 teenagers)	30 min sham exposure followed, after a break of 30 min, by exposure to CMDA 835 MHz RF	Decreased skin resistance in teenagers after 15 and 30 min of RF exposure, and in males after 30 min. Not persistent 10 mn after end of exposure	Lack of randomization of the sham/exposure sequence	Nam et al 2006
Heart rate variability (n=26)	GSM 900 RF radiation for 26 min	Increase the low frequency component as subjects moved into the stand position	Augmentation of sympathetic activity? Not clear if checked for multiple testing.	Parazzini et al 2007
Blood pressure and heart rate variability (n=120)	TETRA and GSM mobile handset signals. 40 min double-blind RF or sham exposure	No effect of RF. mean arterial blood pressure reduced (by ~ 0.7 mm Hg) for GSM sham exposures	Increase in the operating temperature of the handset when in sham mode	Barker et al 2007

II.5.3.2. Cardiovascular responses during thermoregulation

RF energy is absorbed by the body resulting in heat due to an increase in molecular rotational and translational kinetic energy. The absorbed heat energy is distributed throughout the body by the circulation of blood and is eventually lost to the external environment. Significant whole-body heating has a major impact on cardiovascular physiology. In addition, the ability to carry out cognitive tasks is also likely to be compromised before physiological limits of tolerance are reached. Thermoregulatory responses to heat and heat-related disorders in humans have were discussed at a recent WHO Workshop (Kheifets et al 2003) and been reviewed by WHO (1993), Adair and Black (2003), Donaldson et al (2003) and McKinlay et al (2004), and are briefly summarized here in order to place the possible health consequences of RF heating into a broader occupational and environmental context.

Cardiovascular responses to heat and exercise are central to body temperature regulation in humans (Adair and Black 2003; Donaldson et al 2003). Except in various pathological conditions and during heavy exercise, the ‘core’ body temperature is maintained under a wide range of environmental

conditions at a value of about 37°C with a circadian fluctuation of about ±0.5°C. Heat gained at rest, during exercise or exposure to RF, has to be compensated by heat loss and is often accompanied by a small increase in heat storage. The principal heat loss mechanisms in humans are radiant and convective heat loss from the skin through increased skin blood flow and evaporative heat loss from sweat. Heat storage reflects shifts in both peripheral and core temperatures and occurs, for example, during heavy exercise or in hot, humid environments. Prolonged rates of increase in heat storage, such as 0.5–1.0 W kg^{-1} for 1–2 hours, will lead to unacceptable rises in body temperature (Gordon 1984). In moderate conditions, however, increased skin blood flow will increase heat storage through an increase in the temperature of the peripheral tissues of the body, increasing heat loss without necessarily increasing core temperature.

These relationships can be expressed more formally (Bligh and Johnson 1973) as:

$$M \pm W_o = E \pm C \pm R \pm K \pm S \qquad \textit{Eqn. 1}$$

where M = rate of metabolic heat production, SAR = specific energy absorption rate of internally absorbed RF radiation, W_o = rate of physical work, E = rate of evaporative heat loss, C = rate of convective heat loss, R = rate of radiant heat loss, K = rate of conductive heat loss, and S = rate of heat storage in the body (All values can be expressed in eg W (ie Watts or Joules s^{-1}), W m^{-2}, or W kg^{-1}).

Generally, values for the rate of whole-body metabolic heat production in humans vary between about 1 W kg^{-1} and 10 W kg^{-1}; typical average values for many industrial jobs for example vary between about 2.5 W kg^{-1} for light work and 6 W kg^{-1} for heavy manual labor (NIOSH 1980). The degree to which humans can increase skin blood flow and sweat confers marked thermoregulatory advantages compared to other mammals, allowing excessive heat loads to be dissipated more effectively (Adair and Black 2003; Adair 2008). In particular, skin blood flow can increase from approximately 0.2-0.5 liters min^{-1} in thermally neutral conditions, to values exceeding 7-8 liters min^{-1} during hyperthermia, a dynamic range which is much higher than in other species (Donaldson et al 2003).

The main physical difference between children and adults affecting thermoregulation is the much higher surface-area-to-mass ratio of children (Falk 1998). In a warm environment this allows them to rely more upon increased skin blood flow and heat loss through convection and radiation, and less upon evaporative cooling. The lower sweating rate of children is partly due to a lower sensitivity of the sweating mechanism to thermal stimuli. Nevertheless, during exercise in thermally neutral or warm environments, children thermoregulate as effectively as adults. When ambient temperatures exceed body temperature, however, children are more liable to have a higher rate of heat absorption compared with adults. Also, whilst neither children nor adults sufficiently replace fluid loss during exercise in the heat, dehydration may have a more detrimental effect on children because of their greater reliance upon elevated skin blood flow to dissipate heat.

Whole-body and localized RF heating

RF radiation absorbed by the body provides an additional source of heat that has to be lost through the normal heat loss mechanisms described above. The heat balance equation (1) given above can be modified to account for the absorption of RF radiation (Adair 1996) as follows:

$$M \pm W_o + A_{rf} = E \pm C \pm R \pm K \pm S \qquad \textit{Eqn. 2}$$

where A_{rf} = the whole body SAR, ie the rate of absorption of RF per unit body mass averaged over the whole body.

The constraints described above apply to the cardiovascular responses and heat loss during exercise in hot environments would also apply to additional heat loads generated by exposure to RF radiation, except that, in the latter case, heating is passive rather than the result of muscular activity, avoiding the potential conflict between the demands for skin and blood flow. However, individuals may of course be exposed to RF radiation whilst engaging in physical activity in hot environments.

The physiological responses of seated or supine volunteers acutely exposed to RF radiation have been studied mostly by two groups of researchers, namely Shellock et al (1989, 1994), considering mostly the safety of clinical magnetic resonance diagnostic procedures, and by Adair et al (1998, 1999, 2001a, 2001b, 2003, 2005) investigating whole or partial body exposures.

Shellock et al (1989) exposed six volunteers to 64 MHz RF magnetic fields for 30 min; the RF antenna was situated over each subject's abdomen resulting in a partial body exposure. SARs averaged over the whole body mass ranged between 2.7 and 4.0 W kg^{-1} with a mean value of 3.3 W kg^{-1}. Over the 30 min exposure, body temperature rose by an average of 0.1^{o}C, although this response was variable. Cutaneous blood flow and skin temperature in the abdominal region were significantly increased (and were still rising at the end of exposure). All of the subjects reported that they felt warm during the procedure and each of them had visible signs of perspiration on their forehead, chest and abdomen. Subsequently, Shellock et al (1994) exposed six volunteers to 64 MHz RF magnetic fields for 16 min; exposure was centered over the abdomen as in the previous study. Whole-body SARs were estimated as about 6 W kg^{-1}. The ambient room temperature was 22.3^{o}C, the relative humidity was maintained at 45 and air movement was kept to less than 0.1 m s^{-1}. Tympanic membrane temperature rose significantly by an average of 0.4^{o}C; heart rate rose significantly by 13 bpm. In addition, skin blood flow on the abdomen more than doubled. Each subject had signs of perspiration, especially on the forehead, face, abdomen and chest and there were statistically significant increases in skin temperature on the abdomen, upper arm, hand, chest and thigh.

The physiological responses of healthy volunteers, most of whom engaged in regular exercise programs, given whole or partial body exposure to RF radiation at 450 or 2450 MHz, has been investigated in a series of experiments by Adair et al (1998, 1999, 2001a, 2001b). The RF was directed to the back of each seated volunteer; each exposure lasted 45 min and followed a 30 min equilibration period. In the first study, Adair et al (1998) exposed 7 adult volunteers to 450 MHz at two power densities (180 and 240 W m^{-2}) and at three environmental temperatures (24, 28 and 31^{o}C). Peak surface SARs on the back of 6 and 7.7 W kg^{-1} were estimated from measurements in phantoms; there was no measure of whole-body heat load. Vigorous increases in sweating rate on the back and chest, directly related to power density, peak SAR and environmental temperature were reported; core body (oesophageal) temperature and metabolic rate were essentially unchanged during exposure.

An experiment by Adair et al (1999) compared the physiological responses induced in the volunteers by exposure at 450 MHz in the previous study (Adair et al 1998) with those induced by a similar exposure at 2450 MHz in the present study, using a different group of 7 subjects (one subject participated in both studies) in a different laboratory. The experimental procedures were similar to those described above except that the power densities at 2450 MHz were adjusted (270 and 350 W m^{-2}) to produce similar peak surface SARs (6.0 and 7.7 W kg^{-1}) to those induced by 450 MHz. There was no difference in metabolic heat production, or in core body temperature at the different RF frequencies. However, skin temperature in the irradiated area was greater at 2450 MHz than at 450 MHz, probably reflecting the better stimulation of thermal skin receptors by the higher frequency, less penetrative, RF fields. Local sweat rates were more variable within and between the different groups, and related to both local SAR and environmental temperature. A later study (Adair et al 2001a) reported that exposure to 2450 MHz at higher power densities (500 or 700 W m^{-2}), where local peak SARs were 11 and 15.4 W kg^{-1}, resulted in increased skin temperatures in the exposed region increased of up to 4.0 ^{o}C, and more vigorous local sweating and increased local skin blood flow compared to results from the previous study. There was no statistically significant effect of exposure on core body temperature or metabolic rate; hole body SARs were estimated as approximately 0.7 and 1.0 W kg^{-1}.

Adair et al (2001b) compared the effects of exposure to pulsed and continuous wave 2450 MHz in two separate experiments, carried out one and a half years apart, on the thermoregulatory responses of two different groups of 7 subjects (four subjects participated in both experiments). Similar levels of exposure resulted in peak SARs of 6.0 and 7.7 W kg^{-1} for both CW and pulsed RF; whole body SARs were estimated as 0.4 and 0.5 W kg^{-1}. The authors reported that there was little change in core body temperature and metabolic heat production in all test conditions with no reliable differences between CW and pulsed RF. The increase in skin temperature in the exposed region of the back was greater following pulsed RF compared to CW RF. Otherwise, there were no reliable differences between pulsed and CW

RF in skin temperature responses measured in other areas, or in local skin blood flow and sweat rate. These latter were more variable within and between groups and may have obscured interpretation of these responses to different RF frequencies and other waveform parameters.

Two later studies investigated the thermophysiological effects of exposure to 100 MHz RF (Adair et al 2003), which is close to resonance for a seated person, and exposure to 220 MHz RF (Adair et al 2005), which lies in a critical transition range from deep heating to more superficial energy deposition. The experimental protocols were similar to those described above: each study was carried out on 6 or 7 volunteers at three different environmental temperatures; thermophysiological measurements were made before, during and after each 45 min exposure.

Exposure at 100 MHz resulted in whole body SARs were estimated to be 0.27, 0.41 or 0.55 W kg^{-1} (Adair et al 2003). Metabolic heat production was unaffected at any level of exposure; changes in core body temperature were small (< 0.2^{o}C). Unlike the studies carried out at 450 and 2450 MHz, local skin temperatures did not rise significantly except for the ankle, as predicted by dosimetric calculation for a seated person (Findlay and Dimbylow 2006), where temperatures increased by up to 4^{o}C. There was nevertheless an increase in local sweat rate on the back and chest and, to a lesser extent, a small increase local skin blood flow, indicating that the more deeply penetrating RF radiation had stimulated thermal receptors lying deeper within the body, initiating these responses. The subjects were unaware of the onset and termination of exposure, probably because of the failure to increase skin temperatures, although most reported increased thermal discomfort at high levels of exposure and ambient temperature due to increased sweating.

Whole-body SARs during exposure to 220 MHz were estimated as 0.4, 0.54 or 0.67 W kg^{-1} (Adair et al 2005). Metabolic heat production was unaffected at any level of exposure; changes in core body temperature were small (< 0.35^{o}C), as occurred at 100 MHz. Body temperature was controlled by vigorous sweating, greater than that seen at 100 MHz and by minor changes in skin blood flow. Dosimetric modeling predicted that heating would occur in neural tissues such as the brainstem and spinal cord, suggesting that it was the activation of thermal receptors in these tissues rather than in the skin that initiated increased sweating and skin blood flow. These internal thermoreceptors transmit information to the preoptic area of the anterior hypothalamus, which regulates body temperature.

Table II.5.10.: Thermoregulatory responses of volunteers to RF radiation

Assay endpoint	Exposure Conditions	Response	Comment	References
Body temperature, skin temperature, skin blood flow, in 6 supine male subjects before, during and after exposure.	Partial body exposure to 64 MHz in a 1.5 T MRI system for 30 min; whole body SARs of 2.7-4.0 W kg^{-1}	No effect on body temperature; increased cutaneous blood flow and skin temperature in exposed regions.	Sweating on forehead, chest and abdomen.	Shellock et al 1989
Body temperature, skin temperature, cutaneous blood flow, heart rate, blood pressure, in 6 supine male subjects before, during and after exposure.	Partial body exposure to 64 MHz in a 1.5 T MRI system for 30 min; whole body SARs of 6.0 W kg^{-1}	Body temperature rose by an average of 0.4^{o}C; heart rate rose by 13 bpm; increased skin blood flow and temperatures in exposed regions.	Sweating on forehead, chest and abdomen. Blood pressure unaffected.	Shellock et al 1994
Body temperature, metabolic rate, local skin temperature, and sweat rate in 7 seated subjects before, during and after exposure.	Dorsal exposure to 450 MHz for 45 min; peak SARs on the back of 6.0 and 7.7 W kg-1; no whole-body SAR given.	Body temperature and metabolic rate unchanged. Increases in sweat rate in the exposed region.	Effects exacerbated with increasing environmental temperatures: 24, 28 and 31oC.	Adair et al 1998

Assay endpoint	Exposure Conditions	Response	Comment	References
Body temperature, metabolic rate; skin temperature, and sweat rate in 7 seated subjects before, during and after exposure.	Dorsal exposure to 2450 MHz for 45 min; peak SARs on the back of 6.0 and 7.7 W kg-1; no whole-body SAR given.	Body temperature and metabolic rate unchanged. Increases in skin temperature in exposed areas greater at 2450 MHz.	Increase in local sweat rate slightly lower in exposed region at 2450 MHz, but were variable within and between groups.	Adair et al 1999
Body temperature, metabolic rate; skin temperature, skin blood flow and sweat rate in 7 seated subjects before, during and after exposure.	Dorsal exposure to 2450 MHz for 45 min; peak SARs on the back of 11.0 and 15.4 W kg-1; whole-body SARs of 0.7 and 1.0 W kg^{-1}.	Body temperature and metabolic rate unchanged. Increases in skin temperature, blood flow and sweat rate in exposed region.	Individual skin temperature, blood flow and sweat rate responses variable, particularly at high local SARs and environmental temperatures.	Adair et al 2001a
Body temperature, metabolic rate; skin temperature, blood flow and sweat rate; in two different groups of 6 – 7 seated subjects before, during and after exposure.	Dorsal exposure to CW 2450 MHz or pulsed (65 μs pulses at 104 pps; SA of 0.77 mJ per pulse) 2450 MHz for 45 min; peak SARs on the back of 6.0 and 7.7 W kg-1; whole-body SARs of 0.4 and 0.5 W kg^{-1}.	Body temperature and metabolic rate unchanged in both groups. Skin temperature in exposed region increased more by pulsed RF than by CW.	Increases in skin blood flow and sweat rate variable within and between groups.	Adair et al 2001b
Body temperature, metabolic rate; skin temperature, skin blood flow and sweat rate in 7 seated subjects before, during and after exposure.	Dorsal exposure to 100 MHz for 45 min; whole-body SARs of 0.27, 0.41 and 0.54 W kg^{-1}.	Metabolic rate unchanged. Changes in core temperature small (< 0.2°C). Skin temperature largely unchanged by exposure; but increased in the ankle. Sweat rate increased in exposed region and chest.		Adair et al 2003
Body temperature, metabolic rate; skin temperature, skin blood flow and sweat rate in 6 seated subjects before, during and after exposure.	Dorsal exposure to 100 MHz for 45 min; whole-body SARs of 0.4 and 0.54, or 0.67 W kg^{-1}.	Metabolic rate unchanged. Changes in core temperature small (< 0.3°C). Skin temperature largely unchanged by exposure. Sweat rate increased in exposed region and chest.	Several 'hot-spots' identified. Dosimetric modeling suggests heating in brainstem and spinal cord.	Adair et al 2005

Adverse health effects of whole-body and localized heating

There is increasing evidence that cognitive function can be adversely affected by whole-body heat stress, resulting in increased levels of unsafe behavior and reduced task performance (Hancock and Vasmatzidis 2003). For example, Ramsey et al (1983) found a clear correlation between heat stress and unsafe behavior in workers in two industrial plants. A large number of volunteer studies have been carried out over the past 40 years. Most have been in laboratory settings where subjects have performed a variety of cognitive tasks during exposure to a series of thermally stressful conditions. Overall, it appears that simple tasks, such as reaction time and mental calculations, are less vulnerable to heat stress than more complex tasks, such as vigilance, tracking and multiple tasks performed together. [Similar results can be seen in studies with primates (D'Andrea et al 2003a); reduced performance of operant tasks occurs

reliably at body temperature elevations of 1°C or more.] However, with regard to the volunteer studies, a number of other variables will affect performance of these tasks including the level of skill and acclimatization of the subjects. In addition, core body temperature rises were not measured in the experiments reviewed but extrapolated from other data. The precise relationship between increased body temperature and cognitive performance in humans cannot therefore be defined at present (Goldstein et al 2003c); changes in response from small temperature increments would be particularly difficult to judge.

Heat-related disorders such as heat exhaustion are not uncommon in healthy people unaccustomed to hot environments. Heavy exercise either through work or recreation will further exacerbate any problem, particularly if water and salts lost through sweat are not replenished. In addition, people with a history of heat illness, heat injury or heat intolerance and previous difficulty in acclimatizing to the heat are likely to be at increased risk. A number of drugs and chemicals have direct effects on the control of body temperature, or on metabolism or heat production of the body (NIOSH 1986). Almost any drug that impairs central nervous system activity, cardiovascular reserve or body hydration can reduce heat tolerance (NIOSH 1986). For example, drugs such as barbiturates or phenothiazines depress reflex regulation of body temperature generally, while anticholinergic drugs specifically suppress sweating and vasodilation.

The most important adverse consequence of heat stress is death, and in practice the great majority of excess deaths in hot weather are not due to hyperthermia but to the cardiovascular consequences of heat stress in elderly and vulnerable people (Donaldson et al 2003). The main causes of death are heart failure and stroke. In addition, the elderly appear less effective at maintaining normal body temperature compared to the young, due to declines in sweating and blood flow responses, as well as from decline in the neural control of these responses. Cardiovascular diseases that compromise the circulation, such as peripheral vascular disease, are also highly prevalent in older people (Lakatta 2002). Few of the heat-related deaths are specifically attributed to heat in death certificates and national statistics. Accordingly, they can only be assessed by analysis of mortality statistics at the population level. Donaldson et al (2001) note that such calculations cannot be used directly to quantify mortality, but they imply that a substantial increase in heat load might increase mortality in the elderly in hot weather.

The extent to which RF absorption in tissues or organs of the body results in localized peaks of temperature rise in relation to the average rise in core body temperature depends not only on the local SAR but also on the vascularity and flow of blood through the tissue or organ in question which can vary considerably. Localized heating, for example, usually results in vasodilation and increased blood flow but this response may be compromised by cardiovascular responses to whole-body heating. Whilst cardiac output is maintained in healthy elderly people, total peripheral resistance is increased (Ferrari 2002). Cardiovascular diseases, which will further compromise the circulation, such as peripheral vascular disease, which may be caused, for example, by atherosclerosis or heart failure, are also highly prevalent in the elderly (Corti et al 2001; De Sanctis 2001; Lakatta 2002). In addition, people taking medications such as beta-blockers that affect the peripheral distribution of blood flow may also be compromised in this respect.

There are few studies of localized heating in human subjects. Male germ cells in the testes have been known to be heat sensitive for some time; testicular temperatures in most mammalian species are normally several degrees below body temperature. Repeated heating of the human testis by 3–5°C will result in a decreased sperm count lasting several weeks (Watanabe 1959); similar results have been seen in animal studies. Historically, cataracts have been associated with chronic, occupational exposure to infrared radiation (e.g. Lydahl and Phillipson 1984).

Otherwise, information about the damaging effects of localized increases in tissue temperature can be derived from a number of studies of acute exposure have been carried out both in vitro and in vivo, investigating ‘dose-response’ relationships for tissue damage resulting from localized tissue or whole-body heating in order to determine safe but effective hyperthermia regimes in the treatment of cancer. Temperatures have usually ranged between 40 and 45°C, sometimes up to 50°C or more, for periods lasting from a few minutes to several hours. The results of animal studies and a very small number of human studies (mostly of skin damage) have been summarized by Dewhirst et al (2003). The results from different studies are variable but in many cases lesions occurred when temperatures exceeded 42°C or so

for periods of more than about 1 hour, occurring with increasing rapidity as temperatures rose further. CNS tissue seems particularly susceptible (Sharma and Hoopes 2003).

II.5.3.3. Summary on cardiovascular function and thermoregulation

The evidence from the few studies examining the effects of low level mobile phone type radiation on blood pressure and heart rate variability was somewhat equivocal: both positive and negative data were reported. In general, most studies report an absence of effects on blood pressure or heart rate and only weak evidence from two studies for effects on some indices of heart rate variability. However, the small number of studies coupled with weaknesses in the experimental design of some of them, preclude definite conclusions being drawn.

The thermoregulatory studies involving whole-body or localized RF-induced heating indicate that adequately hydrated, resting healthy volunteers exposed to RF in laboratory conditions will accommodate whole-body RF heat loads of between approximately 1 W kg^{-1} for 45 min at environmental temperatures of up to 31°C to 6 W kg^{-1} for at least 15 min at ambient temperatures with minimal changes in core body temperature. With regard to localized heating of the skin, increased skin blood flow and profuse localized sweating increase in skin temperature by up to 4°C in response to a local peak SAR of about 15 W kg^{-1} at the irradiated site.

With regard to the possible health consequences of occupational and/or public exposure to RF, a full assessment of the whole-body heat stress can only be properly derived from a consideration of all sources of heat and from the ease with which heat can be dissipated from the body. Heat gain through solar radiation or other sources of radiant heat may also have to be taken into account. The main adverse health effects expected to result from excessive heat loads are heat-related disorders such as heat exhaustion and, in elderly people, an increase in the risk of heat-related mortality. These effects are well documented in people exposed to hot environments and in elderly people during prolonged periods of hot weather, but have not been associated with RF exposure. In addition, adverse effects on cognitive function may be expected to result from increased body temperature with the potential to increase accident rates but this has proved difficult to quantify in volunteer studies.

A number of studies of acute exposure have been carried out on the adverse effects of raised tissue temperature using animals, often in the context of providing guidance on ultrasound use or hyperthermia in clinical practice. Generally, lesions, including those resulting from cell death, occur when temperatures exceed about 42°C for more than about one hour. The CNS and testes appear particularly susceptible to heat induced damage and show significant changes in cell numbers following exposures at 40–41°C and above.

II.5.4. Summary on human studies

The advantage of laboratory studies using human volunteers is that the results indicate the likely response of other people exposed under similar conditions, but the disadvantages include the often short duration of investigation, the small number and larger heterogeneity of volunteers compared to inbred animal strains. One consequence is the often low power to detect any effect. Furthermore, the subjects are usually chosen to be healthy and are therefore unlikely to reflect the range of responses encountered within a population. For example, the very young and the elderly, or people on medication, have rarely been included within experimental study groups. Nevertheless, within this limited context, volunteer studies can give valuable insight into the physiological effects of exposure in normal, healthy people.

The most consistent effects of acute RF exposure on human subjects are the thermoregulatory responses to RF-induced heating. Cardiovascular responses are particularly important in this context, increasing heat loss from the skin through increased skin blood flow and evaporative heat loss from sweat. Overall, volunteer studies indicate that exposed subjects can accommodate whole body RF heat loads of up to several (< 6) watts per kilogram with minimal changes in core temperature. Increased skin blood flow and profuse localized sweating minimize skin temperature rises (< 4°C) in response to high (< 15 W kg^{-1}) local peak SARs.

Most volunteer studies have investigated the effects of RF exposures characteristic of mobile phone use, usually to the head, on a number of physiological parameters including brain electrical activity and blood flow, cognition, and more generally on the endocrine and cardiovascular systems. The majority of studies have been conducted using healthy human adult subjects. Children and adolescents have become an increasingly important focus of RF studies, given the increasing awareness of the continued maturation of the brain into late adolescence, and a several recent studies using school children have been carried out. In addition, some studies have addressed adults who report themselves to be 'electrosensitive'.

Some evidence suggests that exposure to a GSM-type signal may affect the spontaneous EEG in volunteers(increased power in the alpha band (8–12 Hz) of brain activity). Effects on other frequency bands of natural brain activity have not been consistently demonstrated. However, these observations are not corroborated by the results from studies on evoked potentials. In addition, there are some indications of changes in regional cerebral blood flow, thought to correlate to changes in neural activity, during and following RF exposure, but the available data are equivocal.

A similar conclusion of variable and inconsistent results can be drawn with respect to the effects of exposure to GSM-type signals on EEGs generated during sleep, although there is some evidence emerging that suggests there may be an increase in sleep alpha and beta band activity, either with exposure during sleep or following exposure before going to sleep. In one study this was observed only after exposure to a modulated but not a continuous signal, while in another study a dose-dependent increase in alpha and beta power was observed. Other studies have reported an increase in time to fall asleep, but no other effects on sleep architecture.

The small changes seen in brain electrical activity and possibly in regional cerebral blood flow may not have any functional significance. Despite there having been a large number of studies of cognitive function, no consistent effects on cognitive performance have been found, although the use of a large variety of techniques to assess cognitive performance increases the difficulty with which the results of different studies may be directly compared. When effects have been found, more often in smaller rather than larger studies, they are of small magnitude and exposure generally seems to improve performance, but it has not been possible to derive any dose-response relationship.

With regard to children and adolescents, several recent studies of brain electrical activity and cognitive performance have been published. The results of the spontaneous EEG studies were somewhat equivocal; no effects were seen in two studies of cognitive performance during mobile phone exposure and two studies comparing cognitive performance in mobile phone users versus non-users report a slight facilitation of performance in the users, although this may of course be due to other uncontrolled variables. Overall, there is no robust evidence of any effect of mobile phone type RF on children or adolescents. With regard to possible thermally significant RF exposures, children have a similar thermoregulatory ability to adults, but may be more vulnerable to dehydration because of their larger surface area to volume ratio.

Otherwise, with regard to more general physiological end-points, the weight of evidence from the studies on auditory and vestibular function indicates that neither hearing nor the sense of balance is influenced by short-term exposure to mobile phone signals. In addition, there is no clear evidence of mobile phone type RF exposure on resting heart rate or blood pressure, nor is there consistent evidence of any effect on serum melatonin, or on pituitary hormone levels. However, small but inconsistent changes in heart rate variability were reported in two studies.

A wide range of subjective symptoms including headaches and migraine, fatigue, and skin itches have been attributed to various RF sources both at home and at work. However, the evidence from double-blind provocation studies suggests that the reported symptoms are not causally related to EMF exposure.

II.6. SUMMARY AND CONCLUSIONS

II.6.1. Summary

The mechanisms by which RF exposure heats biological tissue are well understood, and the most consistent effects of acute RF exposure on human subjects are the thermoregulatory responses of the cardiovascular system to RF-induced heating, increasing heat loss from the skin through increased skin blood flow and evaporative heat loss from sweat. Children are known to thermoregulate as well as adults in response to exercise and/or hot environments, but may be more vulnerable to dehydration.

Similar cardiovascular responses to RF-induced heating such as increased skin blood flow occur in laboratory animals. However, animals are less effective at dissipating excess heat than humans, being in general less able to increase skin blood flow and sweat although heat loss can also occur via other mechanisms such as panting. The evidence from volunteer studies suggest that cognitive function can be adversely affected by whole-body heat stress, resulting in increased levels of unsafe behavior and reduced task performance, but this has not yet been explored using RF-exposed subjects. However, laboratory animals show a consistent reduction in the performance of learned behaviors when RF exposure increases core body temperatures by about 1°C or more. Similar RF-induced rises in body temperature also result in significantly enhanced plasma corticosterone or cortisol levels in rodents and primates respectively and transient changes in immune function and hematology, generally consistent with the acute responses to non-specific stressors. Again, these thermal effects have not been systematically explored in RF volunteer studies.

Most recent studies of human subjects, including adults, children and adolescents, have focused on the possible effects of essentially non-thermal exposures to mobile phone type RF, often simulating mobile phone use and so only involving localized exposure of part of the head. A number of non-thermal interaction mechanisms have been proposed but to date none have been experimentally verified. Several volunteer studies using adult subjects report that exposure to a GSM-type signal may result in increased power in the alpha band of the spontaneous EEG. Effects on EEGs generated during sleep were more variable and inconsistent although there is some evidence emerging that suggests there may be an effect on alpha and beta band activity. In addition, there are some indications of changes in regional cerebral blood flow, thought to correlate to changes in neural activity, during and following RF exposure, but again the available data are equivocal. Whether these small changes have any functional significance is unclear; no consistent effects on cognitive performance have been found in a large number of volunteer studies. In addition, regarding possible mobile phone type RF effects on EEG and cognitive function in children and adolescents, there is overall no robust evidence of any effect.

In animals, despite there being sporadic reports of positive effects on brain physiology, most studies have not reported any field-dependent responses either in gene expression or in increased permeability of the blood brain barrier. Several studies indicate that changes may be induced by relatively intense RF exposure in cholinergic activity in the brain, but the evidence of any functional consequence for the performance of some behavioral tasks is equivocal.

A wide range of subjective symptoms including headaches and migraine, fatigue, and skin itches have been attributed to various RF sources both at home and at work. However, the evidence from double-blind provocation studies suggests that the reported symptoms are not causally related to EMF exposure.

Otherwise, with regard to more general physiological endpoints, there is no clear evidence of RF exposure on resting heart rate or blood pressure in human subjects, nor is there consistent evidence of any effect on serum melatonin, or on pituitary hormone levels. However, small but inconsistent changes in heart rate variability have been reported. Animal studies report an absence of effects of pulsed RF radiation characteristic of mobile phone use on circulating serum melatonin levels and other measures of body melatonin.

The evidence from the studies on auditory and vestibular function indicates that neither hearing nor the sense of balance is influenced by short-term exposure to mobile phone signals. The evidence from

laboratory animal studies is rather consistent and suggests that mobile phone type RF exposure has no effect on auditory function. It is also clear that, like humans, animals can hear the pulsed RF characteristic of radar above given thresholds through a thermoelastic expansion mechanism. Studies of the effects of high peak power RF pulses and ultrawide band (UWB) RF has been somewhat diverse and sporadic. Acute exposure to either does not appear to elicit any cardiovascular changes in anesthetized rats.

Overall, the results of recent animal carcinogenicity studies are rather consistent and indicate that such effects on rodents are not likely at SAR levels up to 4 W kg^{-1}. *In vivo* and *in vitro* genotoxicity studies also generally indicate a lack of effect. With regard to *in vitro* studies of non-genotoxic effects such as cell signaling, gene and protein expression, the results are more equivocal. The evidence from studies using measurements of calcium ion concentration, does not support the earlier positive reports of modulated RF effects on calcium ion efflux. There is insufficient research regarding RF effects on nitric oxide signaling, gap junctions and receptor clustering to be conclusive. Recent studies suggest that the RF exposure has no or very little effect on the expression of cancer-related genes (proto-oncogenes and tumor suppressor genes). However, the results of studies of RF exposure on stress protein expression, particularly on hsps, have so far been inconsistent, with both positive and negative outcomes. Heating remains a potential confounder and may account for some of the positive effects reported. More recently, studies using powerful, high-throughput screening techniques, capable of examining changes in the expression very large numbers of genes and proteins, have often shown a limited number of alterations where some genes were up-regulated and others down-regulated, and the expression and phosphorylation of some proteins were changed. However, the magnitude of reported changes was very small and may be of limited functional consequence. In terms of effects on cell behavior, the results of studies on cell proliferation and differentiation, apoptosis and cell transformation are mostly negative.

Thermally significant RF exposure can impair male fertility and cause increased embryo and fetal losses and increase the incidence of fetal malformations and anomalies. Such effects have not been consistently shown at exposure levels that do not induce temperature elevation of 1°C or more. The studies that have addressed postnatal developmental indices or behavior after prenatal exposure to low level RF radiation have generally reported lack of effects. Effects resulting from long-term exposure during the development of juvenile animals have been addressed in only a few studies, and the data are insufficient for conclusions.

Cataract in the eyes of anesthetized rabbits remains a well-established thermal effect of RF exposure. However, primates appear less susceptible to cataract induction than rabbits, and opacities have not been observed in primates following either acute or prolonged exposures.

II.6.2. Conclusions

Overall, it is concluded that:

- The mechanisms by which RF exposure heats biological tissue are well understood and the most marked and consistent effect of RF exposure is that of heating, resulting in a number of heat-related physiological and pathological responses in human subjects and laboratory animals. Heating also remains a potential confounder in *in vitro* studies and may account for some of the positive effects reported.
- Recent concern has been more with exposure to the lower level RF radiation characteristic of mobile phone use. Whilst it is in principle impossible to disprove the possible existence of non-thermal interactions, the plausibility of various non-thermal mechanisms that have been proposed is very low.
- Concerning cancer-related effects, the recent *in vitro* and animal genotoxicity and carcinogenicity studies are rather consistent overall and indicate that such effects are unlikely at SAR levels up to 4 W kg^{-1}. With regard to *in vitro* studies of RF effects on non-genotoxic end-points such as cell signaling and gene/protein expression, the results are more equivocal, but the magnitudes of the reported RF radiation induced changes are very small and of

limited functional consequence. The results of studies on cell proliferation and differentiation, apoptosis and cell transformation are mostly negative.

- There is some evidence of small changes in brain physiology, notably on spontaneous EEG, and somewhat more variable evidence of changes in sleep EEG and regional cerebral blood flow but these may be of limited functional consequence; no changes were seen in cognitive function. With regard to more general physiological end-points, the evidence suggests that there are no consistent effects of non-thermal RF exposures on cardiovascular physiology, circulating hormone levels or on auditory or vestibular function, except for the auditory perception of pulsed RF such as that characteristic of radar.
- The evidence from double-blind provocation studies suggests that subjective symptoms, such as headaches, that have been identified by some individuals as associated with RF exposure, whilst real enough to the individuals concerned, are not causally related to EMF exposure.
- The experimental data do not suggest so far that children are more susceptible than adults to RF radiation, but few relevant studies have been conducted.
- Studies of the effects of RF modalities such as high peak power pulses have been somewhat diverse and sporadic; no effects have been seen other than those associated with heating and with acoustic perception.

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III. Epidemiology

A. Epidemiology of health effects of radiofrequency exposure

B. Epidemiologic evidence on mobile phone and tumor risk

ICNIRP Standing Committee I – Epidemiology

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III.A. EPIDEMIOLOGY OF HEALTH EFFECTS OF RADIOFREQUENCY EXPOSURE*

ABSTRACT

We have undertaken a broad review of epidemiological knowledge about the effects of RF on human health in order to summarize the current state of knowledge, to explain the methodological issues that are involved, and to aid in the planning of future studies. We have looked at epidemiological studies on chronic disease causation; for completeness we have also included epidemiological studies on symptoms although such studies are usually better conducted by laboratory volunteer experiments. For the purpose of this review we have divided the literature into studies of RF exposure from occupational sources, from transmitters, and from mobile phones.

Results of epidemiological studies to date give no consistent or convincing evidence of a causal relation between RF exposure and any adverse health effect. On the other hand, these studies have too many deficiencies to rule out an association. A key concern across all studies is the quality of assessment of RF exposure. Despite the rapid growth of new technologies using RF, little is known about population exposure from RF sources and even less about the relative importance of different sources. An important element in improving future studies would be the use of a meter to monitor individual exposure. The need for better exposure assessment is particularly strong in relation to transmitter studies, because the relation between distance and exposure is very weak. Although the likelihood is low fields emanating from base stations would create a health hazard, because of their weakness, this possibility is nevertheless a concern for many people. Another general concern in mobile phone studies is that the lag periods that have been examined to date are necessarily short. The implication is that if a longer period is required for a health effect to occur, the effect could not be detected in these studies. The majority of research has focused on brain and head and neck tumors but studies on other health effects may be equally justified. Another gap in research is children. Children are increasingly heavy users of mobile phones, they may be particularly susceptible to harmful effects, and they are likely to accumulate many years of exposure.

* This review was first published in Environmental Perspectives 112 (17): 1741-1754; Dec. 2004 and is reproduced here with updates drafted in 2009.

III.A.1. INTRODUCTION

The advent of mobile phones, now used by about 1.6 billion people worldwide, has been accompanied by an upsurge in public and media concern about the possible hazards of this new technology, and specifically of radiofrequency electromagnetic field (RF) exposure. Although some epidemiological research was conducted several decades ago on RF in occupational settings, in general the effects of RF in humans are an emerging area of investigation, and most studies are recent or not yet published. Furthermore, although the results of studies of mobile phone risks have received widespread public attention, their interpretation is not straightforward because of methodological difficulties. In particular, because RF is invisible and imperceptible individuals cannot directly report on their exposure, and therefore the quality of exposure assessment needs particularly careful consideration when interpreting epidemiological studies. In order to summarize the current state of knowledge, to explain the methodological issues that need to be considered when assessing studies, and to aid in planning future studies, we have undertaken a broad review of epidemiological knowledge about the effects of RF on human health. We have divided the literature, for this purpose, into studies of RF exposure from occupational sources, from transmitters, and from mobile phones.

This review covers the possible effects of long-term exposure to RF - defined as 100 KHz to 300 GHz - on the risk of diseases: for instance, cancer, heart disease and adverse outcomes of pregnancy. We have not reviewed the health consequences of communications technology that are indirect or unlikely to be due to radiation. In particular, RF can interfere with implanted medical devices, such as cardiac pacemakers, but the effects on health are a consequence of this interference, rather than a direct effect on the body; phone conversations by drivers of moving vehicles appear to raise the risk of motor vehicle accidents, but this is probably related to distraction rather than RF exposure. While anxieties and psychosomatic illnesses might be caused by knowledge of the presence of phones or phone masts, again this would not be an effect of RF and is not discussed.

As well as epidemiological studies of chronic disease causation some studies have been published that use an epidemiological design to investigate whether mobile phones can affect acute symptoms, such as headaches. For completeness we have included these in this review, although such investigations are usually better conducted by laboratory volunteer experiments rather than by observational epidemiology, given the high degree of susceptibility to biased reporting in response to concerns.

Because this is primarily an epidemiological review we have not detailed the physics and dosimetry of RF from different sources, which are described elsewhere (Hitchcock & Patterson 1995; Mantiply et al 1997; IEGM 2000). However, because understanding of mobile phone-related epidemiology is critically dependent on understanding of mobile phone technology, we have included some information explaining this technology. We have also included, because of its importance to future research advance, some comments on the interface between physics and epidemiology, and the gaps to be bridged between these disciplines if more rigorous investigation of potential RF effects is to be achieved.

As the review was written initially in 2004, we have added addenda for the current book, outlining the subsequent literature and its impact on the conclusions. The references include all years of the literature.

III.A.2. EXPOSURE

III.A.2.1. Sources of exposure

Communications sources have increased greatly in recent years, and there is continuing change in the frequencies used and variety of applications. The first mobile phone systems were analogue and utilized 450 and 900 MHz. Digital systems, operating at somewhat higher frequencies (1800-1900 MHz) and using different modulation techniques, became prevalent in the early 1990s. Currently, the

third generation systems (3G) using the Universal Mobile Telecommunication System (UMTS) are being introduced, which will operate in the 1900-2200 MHz frequency range. Occupational RF exposures occur to workers engaged in a number of industrial processes, particularly when using dielectric heaters for wood lamination and the sealing of plastics and industrial induction heaters. Relatively high levels of exposure to RF fields can occur to workers in the broadcasting, transport and communications industries, and the military, when they work in close proximity to RF transmitting antennas and radar systems. Medical exposures can come from medical diathermy equipment to treat pain and inflammation, electrosurgical devices for cutting tissues, and diagnostic equipment such as Medical Resonance Imaging (MRI).

III.A.2.2. Distribution of exposure in the population

Despite the rapid growth of new technologies using RF, little is known about population exposure from these and other RF sources and even less about the relative importance of different sources. In a typical house, non-occupational exposure could come from external sources, such as radio, TV, and cellular base stations, as well as internal sources, such as a faulty microwave oven, in-house bases for cordless phones, or use of mobile phones.

Radio and TV transmitters have a large coverage area and therefore operate at relatively high power levels up to about 1MW (Dahme 1999). Although these transmitters could generate fairly high fields at ground level, most are not located in heavily populated areas and thus do not lead to high exposure of the population.

Cellular phone base stations are low-powered radio antennas that communicate with users' handsets. In early 2000, there were about 20,000 base stations in the United Kingdom and about 82,000 cell sites in the United States. Base stations can transmit power levels of 100 W or more (Schüz & Mann 2000). It is expected that the number of base stations will roughly double to accommodate new technology and a larger percentage of sites will have to be shared between operators, complicating exposure assessment. The power density levels inside a building can be from 1 to 100 times lower than outside, depending on the type of building construction (Schüz & Mann 2000). In addition, exposure can vary substantially within the building. For example, exposure was found to be about twice as high (and more variable) in the upper compared with the lower floors of a building (Anglesio et al 2001). Driven by a typical pattern of use, the exposure from base stations shows a distinct diurnal pattern, characterized by lowest values during the night and by two maxima during the day, the first from 10 a.m. to 1 p.m. and the second from 6 to 10 p.m. (Silvi et al 2001). Compared with spatial variations, however, these variations are normally less than one order of magnitude (COST 2001).

There have been few and limited efforts to characterize population exposures; all of them have been small (usually areas around 10-20 base stations) (Cost 2000; Schüz & Mann 2000; Anglesio et al 2001). The total power density from the base stations was slightly higher than, but comparable with, the background power density from all other RF sources combined. Mobile phones operate at a typical power of 0.25W. Analogue systems operated at higher power levels than the newer digital systems. Similarly older cordless phones operated to the analogue standard, while modern ones operate to the digital with a transmitted power of a base around 0.09W in a home but higher in a business setting. The actual exposure of the user depends on a number of factors such as characteristics of the phone, particularly the type and location of the antenna; on the way the phone is handled; and most importantly, on the adaptive power control (APC), which may reduce the emitted power by orders of magnitude (up to a factor of 1,000). Factors that influence APC include distance from the base station, the frequency of handovers and traffic conditions. Thus the emitted power is higher in rural than in urban areas and when the user is moving (e.g. in a car). In areas where there is a great deal of phone use, phones may operate more than half of the time at the highest power levels. To compensate for the shielding effect of materials, power levels of phones are, on average, higher when a phone is used indoors than outdoors. Handheld phones constitute the highest source of concentrated exposure to the brain. RF absorption is maximal on the side of the head to which the phone is held, greatest close to the antenna, and falls off to less than a tenth on the opposite side of the head (Dimbylov & Mann 1999).

In an occupational setting, higher exposures occur, albeit infrequently; for example, radar exposed workers in the US Navy had potential for exposures greater than 100 mW/cm^2 (Groves et al 2002).

III.A.2.3. Epidemiological considerations in exposure assessment

General: In the absence of information on what biological mechanism is relevant, it is unclear what aspect of exposure needs to be captured in epidemiologic studies. Because thermal heating is the only known effect of RF, most research has assumed that the metric of choice must be a function of the Specific Absorption Rate (SAR). Metrics proven to be useful in epidemiologic studies of other agents, such as cumulative exposure, average exposure over specific time intervals, and peak exposure need to be considered. Given the uncertainty about the relevant interaction mechanism, the dose needs to be assessed not just as external field intensity, but also as cumulative exposure, as well as SAR for specific anatomical sites. Integrating exposure over time is further complicated by the fact that sources vary markedly over very brief time periods relative to the time periods of interest.

Epidemiologic studies thus far have relied on rather crude proxies for exposure, such as job title, proximity to a base station, or use of a mobile telephone. Refinement of exposure assessment is critical to improved epidemiology. This requires a bridge between the rather disparate worlds of epidemiology and physics. While it is of interest to know about sources of variation or uncertainty in general, the critical need in epidemiological studies is to identify those variables that are most important in determining exposure levels and most amenable to capture within populations.

A key element in linking the complexity of the exposure sources and patterns with the needs of epidemiology is a meter that is capable of monitoring individual exposure. Such meters have now been developed (HPA-NRPB 2003).

Ideally, the dose, time pattern, and frequencies (wavelengths) of exposure from all key sources should be estimated for each individual in the study. Dose- and duration-response analyses are important to assessment of etiology, but have often been absent in the existing literature (Swerdlow 1999). In addition, the possible lag period between exposure and disease manifestation needs to be considered. Hand-held mobile phones were not used regularly until the 1990s. Thus, studies published to date have had little power to detect possible effects with long induction periods or latencies, or effects from long-term heavy exposure to mobile phones or base stations.

Methodologically, it would be desirable to conduct studies to clarify the relative contributions of different spheres of life. Such knowledge would allow epidemiologists to design studies that incorporate all important sources of RF exposure, or at least determine how much it matters that the occupational studies to date have taken no account of residential or mobile phone exposures and vice versa.

Occupational exposures: Most occupational epidemiological studies have based their exposure assessments simply on job titles and have included no measurements (see Tables III.A.1, 2, 3, 4). It is possible that some jobs, e.g., radar operator, are adequate indicators of RF exposure. However, many job titles that have been previously considered to indicate exposure may often provide a poor proxy for RF exposure.

In addition to improving exposure assessment in individual studies, there is the potential to develop job-exposure matrices, with the rows corresponding to relatively homogeneous groups with respect to RF exposure, defined by job title, perhaps specific work location, calendar time, and other recordable work history, and the columns corresponding to RF exposure metrics.

Transmitter exposures: All published epidemiological studies of transmitter exposures have based exposure assessment solely on distance from the transmitter. The relation between exposure and distance from the antenna is usually very complex, especially in urban areas. Close to the antenna, the field is very low due to the directional antenna characteristics. As one moves away, the field pattern can be complicated, with peaks and valleys in field intensity with increasing distance from the antenna.

Estimation of community exposure to RF from transmission towers may, however, be amenable to refinement. Geographic information systems allow for precise assignment of residence, topography, and other likely determinants of exposure. Historical information on power output from the base towers may well be available. This information combined with personal measurements may provide refined measures of exposure that can be applied retrospectively, with empirical validation.

Mobile *phones exposures*: Studies on mobile phones have used the simple dichotomy of user versus non-user, with some incorporating information on years of use, number of phone calls per day and duration of calls. Some studies have separated analogue and digital phone use. Few have included use of cordless phones, from which exposure pattern is different and exposure generally much lower.

Ongoing studies are attempting to incorporate information on intensity of use, place of use, position of the telephone, type of telephone, and calendar period of use. Each of these extensions need to be evaluated, however, to determine (a) whether they are truly important determinant of exposure and (b) whether they are amenable to accurate historical reconstruction through recall or some type of written record There is little benefit in knowing that the intensity of exposure varies by a parameter that cannot be captured, or gathering relatively precise information about, say, model of mobile phone, if no useful exposure variable can be derived from it.

III.A.3. MECHANISMS

Heating of cells and tissues from RF exposure can have benign or adverse biological effects. These effects, which reflect an imbalance in the amount of heat built up in the body and the effectiveness of mechanisms to remove it, can be due to either elevated temperatures or increased physiological strain from attempts to remove the heat. Of particular concern for whole body heating are effects in the elderly, people taking certain kinds of drugs, and the embryo and fetus? Cardiovascular mortality, birth defects and impaired ability to perform complex tasks are among the outcomes that have been associated with whole body heating. The sensitivity of various tissues and cells to thermal damage to both localized and whole body heating varies. The central nervous system, testis and lens of the eye seem to be particularly sensitive, the last due to a limited capacity to dissipate heat rather than due to a greater sensitivity of its cells to heat-induced damage.

Laboratory studies suggest that adverse biological effects can be caused by temperature rises in tissue that exceed 1°C above their normal temperatures (Goldstein et al 2003). In addition to the absolute increase in temperature, duration of heating and thermoregulatory capacity of the body are important determinants of the harmful levels of tissue heating. High rates of physical activity, and warm and humid environments, will reduce tolerance to the additional heat loads.

There has been concern about possible carcinogenic effects of RF below levels that cause detectably harmful heating. Since RF is not sufficiently energetic to destabilize electron configurations within DNA molecules, there is no direct link between RF exposure and genotoxic effects such as DNA mutations, DNA strand breaks, or other genetic lesions. Experimental evidence from animal and laboratory studies at the cellular level confirm the lack of genotoxic effect of RF (Moulder et al 1999; Krewski et al 2001). Similarly, investigations in rodents do not support the suggestion that growth of tumors induced by other agents may be promoted by RF from mobile phone signals (Imaida et al 2001; Mason et al 2001). However, no data exist that examine the carcinogenic risks of chronic thermal exposures below the threshold for detectable tissue damage, either alone or in combination with known carcinogens.

Repacholi et al (1997), evaluated the effects of radiofrequency fields on tumorigenesis in a moderately lymphoma-prone Eμ-*Pim1* oncogene-transgenic mouse line. Exposure was associated with a statistically significant, 2.4-fold increase in the risk of developing lymphoma. Utteridge et al (2002), recently repeated this study with a larger number of mice and with several refinements in the experimental design and failed to demonstrate any difference in the incidence or type of lymphomas that developed between control and treated groups. Questions have been raised about the conduct and reporting of both studies and the inconsistency has not been resolved (Goldstein et al 2002). Additionally, extrapolating the transgenic model to humans remains controversial.

III.A.4. OUTCOMES

The greatest public concern appears to be that the use of hand-held mobile phones may be linked to the occurrence of malignant disease, especially brain cancer and, to a lesser extent leukemia. Other tumors such as acoustic neuroma that occur in the head and neck region have also been investigated. Each of these conditions is rare. The incidence of malignant tumors of the brain in the general population is around 10 to 15 per 100,000 each year (Behin et al 2003), the annual incidence of benign extra cerebral tumors such as meningiomas is about 3 per 100,000, and benign tumors of the cranial nerves such as acoustic neuromas, are rarer still. Because tumor incidence is so low, investigators have so far relied on case-control studies or, in a few instances, retrospective cohort studies. In addition, different tumor subtypes are likely to have different causes, as evidenced among brain tumors by the different molecular pathways leading to malignant astrocytomas on the one hand and benign meningiomas and acoustic neuromas on the other (Inskip et al 1995). Similarly there are a variety of types of leukemia each probably with differences in causation, making it even more difficult to ascertain sufficient numbers of homogeneous tumors for study. Epidemiological assessments have been further complicated because the environmental risk factors for malignant and benign brain tumors (Inskip et al 1995), and hence potential confounders, are largely unknown beyond high-dose ionizing radiation. For leukemia (Petridou et al 2002) knowledge of potential confounders is greater, but still limited: other risk factors, as well as ionizing radiation, include exposure to chemotherapy, cigarette smoking, benzene, and constitutional chromosomal abnormalities among children in particular.

Available evidence suggests that induction of a proportion of brain tumors occurs over decades following tumorigenic exposures early in life. Latency of tumors varies from months to years depending on how aggressive tumor growth is and the location of the tumor. Epidemiological studies should therefore in principle allow for a lead time between potentially causal exposure and disease, although in the absence of biological or epidemiological evidence it is unclear what length this should be for potential RF effects.

Other chronic diseases such as cardiovascular disease, as well as symptoms, both acute and chronic, have been studied in relation to RF exposure. Headaches and other cranial discomforts including sensations of local warmth or heating, dizziness, visual disturbances, fatigue and sleeplessness are the main symptoms volunteered by users of mobile phones. All of these are common symptoms in humans.

III.A.5. REVIEW OF STUDIES ON OCCUPATIONAL EXPOSURE

III.A.5.1. Cancer

Information on cancer risks in relation to occupational RF exposure comes from three types of epidemiological study: cohort studies, investigating a wide range of cancer (and non-cancer) outcomes in groups with potential RF exposure (Tables III.A.1. and 2.); case-control studies of specific cancer sites, investigating occupational RF as well as other exposures (Table III.A.3.); and analyses of routinely collected datasets on cancer incidence or mortality, in which risks of cancer have been assessed in relation to job title (Table III.A.4.). The most extensive literature addresses brain tumors and leukemia.

Considering study size, design, and likely quality of RF assessment, the most informative studies (Groves et al 2002; Milham 1988; Morgan et al 2000) provide little evidence of an association with either brain tumors or leukemia. The one possible exception was a raised risk of non-lymphocytic leukemia in radar-exposed navy veterans (Milham 1988) restricted to only one of three highly exposed occupations (aviation electronics technicians), but this finding was divergent from that of an earlier study of US naval personnel (Garland et al 1990). Two US case-control studies of brain tumor

etiology have shown elevated odds ratios of around 1.5 in relation to jobs believed to have RF exposure RF exposure RF exposure. However, the study by Thomas et al (1987) was based on interviews with relatives of dead cases, and hence was unable to identify exposure with much certainty. The other study (Grayson 1996) assessed exposures by a job exposure matrix based on historical reports of incidents of exposure above permissible limits (10 mW/cm^2). No clear or consistent trend was found in risk of brain tumor in relation to exposure score. A widely cited study of US Moscow embassy staff and their dependents with possible RF exposure was only published as a précis by a third party (Goldsmith 1995); this leaves the study methods unclear, but few brain tumors or leukemia occurred, and half were in dependents who lived outside the embassy.

A key concern across all these studies is the quality of assessment of RF exposure, including the question of whether it was truly present at all, and if so, for what proportion of the cohort. Although the published studies do not give consistent evidence for a raised leukemia or brain cancer risk, they cannot be counted as substantial evidence against a possible association. Most of the studies suffer from severe imprecision, with the cancers of greatest interest rarely found in cohort studies of modest size and the exposure of interest rarely found in geographically based case-control studies. The cohort studies generally lack data on other relevant exposures, including non-RF frequencies of radiation, as well as on RF exposures outside the workplace (e.g., mobile phones). The studies based on routine data are vulnerable to publication bias given the many datasets worldwide that could be used to address this issue. Several of these studies did not follow workers after they left the job of interest (Garland et al 1990; Grayson 1996; Szmigielski et al 2001), with the potential for bias if individuals left employment because of health problems that subsequently turned out to be due to cancer – this might especially be a problem for some types of brain tumor, which can be present for long periods before diagnosis. In addition, several studies have had substantial methodological inadequacies – for instance one study that found apparently raised risks for many different cancers used more sources of exposure information for cancer cases than for non-cancer subjects, and was analyzed improperly (Tynes et al 1996).

Several studies have investigated the risk of breast cancer in relation to RF exposure. A cohort study of radio and telegraph operators in Norwegian merchant ships by Tynes et al (1996) found a relative risk of breast cancer of 1.5 (1.1 – 2.0), based on 50 cases in women working in this occupation, stronger for women aged 50 and above (2.6 (1.3 – 5.5)). An elevated relative risk found also for endometrial cancer suggests that reproductive and hormonal factors (for which full adjustment could not be made), not RF, may have been responsible for the raised breast cancer risk. A large case-control study based on job titles from death certificates in the US found no trend in risk of breast cancer in relation to probability or to level of occupational RF exposure (Cantor et al 1995). A case-control study in the US of men with breast cancer found an odds ratio of 2.9 (0.8 – 10) in radio and communication workers (Demers et al 1991), based on 7 cases in exposed men, and with a low response rate in controls. A study of US embassy personnel with potential RF exposure found 2 breast cancers with 0.5 expected (Goldsmith 1995). Other studies of male (Groves 2002) and female Morgan et al 2000; Lagorio et al 1997) breast cancers, with few cases, did not report increased risks. The available data are insufficient to reach any conclusion on whether RF exposure is related to breast cancer risk, but the results of Tynes et al (1996) do support continued evaluation of the possibility.

Testicular cancer was considered in a US case-control study (Hayes et al 1990). A significantly raised risk was found for self-reported occupational exposure to microwave and other radio waves (0R = 3.1) but not for self-reported radar exposure nor for radar or other microwave exposure assessed by an occupational hygienist based on job history. A cluster of testicular cancer (observed/expected ratio = 6.9) was reported in 6 police officers in Washington State, US, who routinely used hand-held traffic radar guns (Davis & Mostofi 1993) (exposure levels are usually less than 20 W/cm^2 (Lotz et al 1995). In a large US Navy cohort with radar exposure, testicular cancer mortality was lower than expected (SMR 0.6 (0.2 – 1.4), n = 5) in the group with potential for high exposure (Groves et al 2002).

Ocular melanoma was associated with self-reported exposure to microwaves (excluding domestic microwave ovens) or radar (0R 2.1 (1.1 – 4.0)) in a case-control study (Holly et al 1996). Stang et al (2001) found a raised risk of ocular melanoma in subjects with self-reported occupational exposure for at least 6 months and several hours per day to RF (14% of cases, 10% of controls) and for

occupational exposure several hours per day to radio sets (0R 3.3 (1.2 – 9.2)). There was no relation of risk to duration of this exposure, however, and risk was not raised for radar exposure (0R 0.4 (0.0 – 2.6)). The study was small, and combined subjects from two different study designs.

A nested case-control study of electrical utility workers thought to be exposed to pulsed electromagnetic fields found a significant excess of lung cancer (Armstrong et al 1994) and a dose-response gradient with increasing cumulative exposure. Adjustment for crude indicators of smoking and other factors left the results little changed. In an attempt to address a similar exposure in a cohort of US electric utility workers, limited due to the ill-defined agent addressed in the previous study, no increased risk of lung cancer was found (Savitz et al 1997), and no other studies of RF have reported associations with lung cancer (Groves et al 2002; Milham 1988; Morgan et al 2000; Szmigielski 1996 and 2001; Tynes 1996; Lagorio 1997; Milham 1985; Muhm 1992).

In conclusion, there is no cancer site for which there is consistent evidence, or even an individual study providing strong evidence, that occupational exposure to RF affects risk. The quality of information on exposure has generally been poor, however, and it is not clear that the heterogeneous exposures studied can be regarded as a single etiological entity. This, combined with imprecision and methodological limitations, leave unresolved the possibility of an association between occupational RF and cancer.

Addendum: One further study of brain tumor risk, from German population-based case-control data, showed no significant risk of glioma or meningioma from occupational RF exposure, based on self-reported exposure data, although a non-significant increase in risk was found for 10+ years of high exposure (Berg et al 2006). An Australian population-based interview case-control study found no significant risk or trend in risk for exposure-matrix derived occupational RF exposure, although there was a non-significant raised risk, based on small numbers, in the highest exposure category (Karipidis et al 2007). Our conclusions above still stand.

III.A.5.2. Other outcomes

Adverse Reproductive Outcomes

A wide range of potential reproductive consequences of RF exposure have been investigated (Table III.A.5.), with a focus on exposures of physiotherapists to therapeutic short wave diathermy (typically 27.12 MHz). Depending on the type of equipment used and the location of the operator in relation to the equipment, substantial peak exposures can occur (Larsen et al 1991a). Many of the studies analyzed levels of exposure, on the basis of duration of work and type of equipment used (shortwaves or microwaves).

There are isolated suggestions of an association between RF exposure and delayed conception (Larsen et al 1991b), spontaneous abortion (Ouellet-Hellstrom and Stewart 1993; Taskinen et al 1990), stillbirth (Larsen et al 1991b), pre-term birth with exposure to fathers (Larsen et al 1991b), birth defects in aggregate (Larsen 1991), and increased male to female sex ratio (Larsen et al 1991b). Almost always, however, either the finding was not corroborated in other studies of comparable quality or there are no other studies available. The evidence is strongest for spontaneous abortion (based on two independent studies with some support) and perhaps sex ratio (based on a single study with rather striking findings). Potential confounding by other aspects of work activity (e.g., physical exertion) needs to be considered, however.

Semen parameters have been examined among men with varying forms of military exposure to microwaves and radar (Table III.A.5.). Three of these studies found reductions in sperm density, (Lancranjan et al 1975; Weyandt et al 1996; Hjollund 1997), with variable results for other semen parameters, but one did not report such an association (Schrader et al 1998; Grajewski et al 2000). Several of these reports were based purely on volunteers with no attempt to sample from a defined population (Lancranjan et al 1975; Weyandt et al 1996 Schrader et al 1998), and those that did provide information about response proportions (Hjollund et al 1997; Grajewski et al 2000) had substantial

non-response. However, given the well-known susceptibility of spermatogenesis to even subtle heating, the possibility of reduced fertility in exposed men is reasonable to evaluate.

Overall, problems of exposure assessment temper any conclusions regarding reproductive outcomes, and no adverse effects of RF have been substantiated.

Addendum: Since 2004 two studies of reproductive health have been conducted in men occupationally exposed to RF fields, both in Norway (Mjoen et al, 2006; Mollerlokken and Moen 2008). There were no differences in the numbers of children born to exposed men and their unexposed counterparts despite some positive associations with reported difficulty in conceiving (Mollerlokken and Moen 2008; Baste et al, 2008). Furthermore there were no associations between paternal occupational exposure to RF and poor obstetric outcomes or overall occurrence of birth defects (Mjoen et al 2006). Similar conclusions were drawn from an investigation carried out in two villages in Cyprus situated near a military air base with visible antennae, which found no associated increase in adverse obstetric outcomes or birth defects (Preece et al 2007).

Cardiovascular Disease

Several methodologically weak studies from the Soviet Union addressed microwave exposure and acute effects on cardiovascular physiology (e.g., hypotension, bradycardia, tachycardia) as part of a set of ill-defined conditions (Jauchem 1997). Additional studies of indirect relevance considered symptoms among a range of potentially exposed groups including radar workers, pilots, radio broadcasting workers, and electronics industry workers. The variability in research methods, exposure characteristics, and outcome measures makes it difficult to draw conclusions: there are sporadic reports of symptoms among some groups of workers, but no obvious pattern is present.

Major clinical outcomes have been examined less frequently. In a mail survey of US physical therapists (Hamburger et al 1989 men more highly exposed to microwave and shortwave radiation, based on indices including length of employment and frequency of treatments, tended to report a significantly greater prevalence of heart disease, with odds ratios of 2-3. Selective response to this survey must be considered among possible explanations for the associations that were observed. In US Navy veterans potentially exposed to radar (Groves et al 2002) and in a cohort of nearly 200,000 Motorola workers (Morgan et al 2000), heart disease SMRs were well below 1.0, and analyses of mortality (Groves et al 2002), hospital admissions and disability compensation (Robinette et al 1980) did not support greater risk with greater potential exposure. Other cohorts reporting cardiovascular mortality have had small numbers (Lagorio et al 1997; Muhm 1992).

Overall, the literature on RF and cardiovascular symptoms and disease provides little suggestion of an association, but is at too rudimentary a level to draw firm conclusions.

Addendum: Putative alterations in some cardiovascular parameters with RF exposure in an epidemiologic setting have not been replicated in exposed volunteers under experimental conditions (Jauchem 2008), and a neurological study in Sweden found no measurable differences in blood-brain barrier integrity among frequent users of wireless telephones (short- or long-term) compared with infrequent users (Soderqvist et al 2008). All-cause mortality among Belgian military personnel who were radar operators for many years showed no increase compared with their counterparts who were never exposed to radars (Degrave et al 2005). Finally two recent and extensive reviews have found no substantive evidence of adverse health outcomes arising as a result of high levels of RF exposure (Valberg et al 2007; Jauchem 2008).

Cataract

Laboratory research indicates that the lens of the eye is highly sensitive to heat, and damage can occur from even a single acute exposure. Hence there is a potential mechanism for RF to lead to increased cataract incidence. Epidemiologic research has been limited, however, especially with regard to exposure assessment.

Based on hospital records of US military veterans (Cleary et al 1965), men with cataracts were no more likely than men with other medical conditions to have been radar workers (OR 0.67, p>0.10). Age was adjusted using broad groupings, with little change to the result.

In two studies in the US military, ocular examinations were conducted on microwave-exposed and unexposed workers, without knowledge of exposure status by the examiner. In one (Cleary et al 1966) a tendency towards increased minor lens changes was found among exposed workers, characterized as the equivalent of 5 years advanced ageing in the exposed compared with unexposed workers around age 60. In the other (Shacklett et al 1975), prevalence of lens opacities was similar in exposed and unexposed individuals matched on age.

In an Australian study of workers who built and maintained radio and television broadcasting towers, compared with unexposed workers from the same geographic regions (Hollows & Douglas 1984), posterior subcapsular opacities were in excess in exposed workers (borderline significant) but nuclear sclerosis prevalence was similar in exposed and unexposed workers. It was not specified whether evaluators were aware of exposure history. Exposures were estimated to be from 0.08 to 3956 mW/cm^2, with brief, intense exposures thought to be quite common.

The study designs above are limited with respect to exposure assessment and selection of unexposed workers. Solar radiation exposure, a known risk factor for cataracts, was not considered and could have differed between RF exposed and unexposed workers. Not all of the opacities were of direct clinical importance, but they would be pertinent to a pathway that could lead to cataract later in life. The plausibility of a causal relation supports more extensive investigation.

III.A.6. REVIEW OF STUDIES ON ENVIRONMENTAL EXPOSURE FROM TRANSMITTERS

The primary concern with transmitters has been with cancer risk among populations who live in proximity to broadcast towers, including those that are used for transmitting radio, television, microwave, and cellular telephone communications. There is a long history of public concern and resistance to the siting of such towers, for reasons involving aesthetics and property value, as well as health concerns. Much of the research has been conducted in response to such concerns, either based solely on the exposure source or on a perceived cancer cluster among persons living in the vicinity.

The studies of which we are aware are listed in Table III.A.6. together with some fundamental characteristics and major findings.

The first study, in San Francisco (Selvin et al 1992) was focused on statistical analysis of spatial data and the results are not reported according to standard epidemiologic practice. Indeed the authors did not even report a relative risk. The source of exposure was a large TV tower, and the three statistical methods considered in the paper all showed that the pattern of cancer incidence was essentially random with respect to the tower. A case-control study based on an apparent cluster of childhood leukemia (Maskarinec et al 1994) was prompted by an observation of an unusually high number of childhood leukemia cases in a region of Hawaii. There were 12 leukemia cases, and the odds ratio for having lived within 2.6 miles of the radio towers before diagnosis was 2.0 (95% c.l.: 0.06 – 8.3). Hocking et al compared cancer incidence in three municipalities immediately surrounding three TV towers in northern Sydney to the cancer incidence in six adjacent municipalities, estimating power densities from information on commencement of service of each tower , power and frequency band Hocking et al 1996. For leukemia incidence in adults they found a relative risk of 1.24 (95% c.l. 1.09-1.40) for the inner three municipalities compared with the surrounding municipalities. Their highest relative risk, 1.67 (1.12-2.49), was for the subcategory *other leukemia*. For childhood leukemia they observed a relative risk of 1.58 (1.07-2.34). Neither for adults nor for children were there any risk elevations for brain tumor.

Dolk et al reported on an apparent cluster of leukemia and lymphomas near a UK radio and TV transmitter at Sutton Coldfield (Dolk et al 1997a). The study area was defined as a 10 km radius circle

around the transmitter. Ten bands of increasing distance from the antenna were defined as the basis of testing for declining incidence with increasing distance. The relative risk of adult leukemia within 2 km was 1.83 (95% c.l.: 1.22-2.74) and there was a statistically significant decline in risk with increasing distance from the antenna. In children, under 15 years, there were 2 cases compared with 1.1 expected within the 2 km radius circle. The authors concluded that there was an excess risk of adult leukemia in the vicinity of the transmitter. Field strength measurements in the vicinity of the transmitter showed a maximum total power density at any one point of 0.013 W/m^2 for TV and 0.057 W/m^2 for FM radio with considerable variability between different measurement points.

A second investigation with a similar design to the first one was extended to include 20 high power TV and FM radio transmitters (Dolk et al 1997b). Inside the 2 km radius circle the O/E ratio for adult leukemia was 0.97 (95% c.l.: 0.78 – 1.21) and for childhood leukemia was 1.12 (95% c.l.0.61-2.06). Thus these results gave no more than very weak support to the original results.

McKenzie et al re-examined the Sydney results discussed above (McKenzie et al 1998). They found that the excess risk reported by Hocking et al (1996) was mainly limited to one local government area within the studied region.

The Sutton Coldfield results have also been followed up by another group (Cooper et al 2001). They used more recent cancer data to reanalyze cancer incidence around the transmitter and found considerably weaker results than the original.

An Italian study occasioned by local concerns investigated leukemia incidence in children and mortality in adults within a 10 Km circle around the Vatican radio station (Michelozzi et al 2002). The station consists of numerous transmitters with different transmission powers ranging from 5 to 600 kW and with different frequency ranges. In adults of both sexes taken together the SMR within 2 km of the station was 1.8 (95% c.l.: 0.3-5.5) based on 2 cases. Stone's test for trend in rates over successive 2 Km bands around the station gave a p-value of 0.14. The excess risk and the trend were essentially confined to males. In children the SIR for those living within the 2 km radius circle was 6.1 (95% c.l.: 0.4-27.5) based on one case. Elevated rates were observed for all cumulative bands up to 10 km but all had wide confidence intervals and the total number of cases within the 10 km radius circle was 8. The Stone test for trend was reported as p=0.004. No systematic RF measurements have been made in the area and the epidemiologic analyses are based on the simplistic proxy, distance from the source. The numbers of cases were small, especially for children, which precludes firm conclusions. For adults the results are somewhat inconsistent in that the risk elevations were largely confined to males.

Addendum: All studies available at the time of the previous review were ecological studies, with no individual exposure assessment. Since then, two studies on childhood leukemia in relation to environmental RF exposure have been published (Ha et al 2007; Merzenich et al 2008; Schüz et al 2008). The study from South Korea (Ha et al 2007; Schüz et al 2008) included 1,928 childhood leukemia cases diagnosed between 1993 and 1999, and one hospital based control per case. Exposure assessment for each individual child was made through calculations of the RF fields generated by nearby AM radio transmitters. There was no association between childhood leukemia and estimated RF fields; OR=0.83, 95% CI: 0.63-1.08 in the highest exposure quartile. A study from Germany (Merzenich et al 2008) included 1,959 childhood leukemia cases diagnosed between 1984 and 2003 and 5,848 population-based controls. Individual exposure assessment was made through calculations of the RF exposure from AM and FM radio and television broadcast transmitters. An OR of 0.86 (95% CI: 0.67-1.11) was observed for the upper $\geq$95% quantiles compared to the <90% quantiles of the exposure distribution. Stratification of the analyses according to time period revealed no difference in the results before and after the introduction of mobile phones. These studies provide evidence against an association between RF exposure from broadcast transmitters and the risk of childhood leukemia.

Symptoms

A number of cross-sectional studies on the occurrence of subjective symptoms and well-being in relation to RF exposure from mobile phone base-stations or mobile phone use have been published since the 2004 review (Abdel-Rassoul et al 2007; Berg-Beckhoff et al 2009; Blettner et al 2009; Hutter et al 2006; Preece et al 2007; Soderqvist et al 2008; Thomas et al 2008). Methodological limitations inherent in the cross-sectional design make it difficult to draw conclusions about cause and effect based on these studies. Particular difficulties relates to the nature of the studied outcomes, which can only be estimated through self-reports. In addition, exposure to RF fields has rarely been measured, but has often been based on self-reports of mobile phone use or distance to base stations, assessed at the same time as the studied outcomes, or on ecological data, which makes the results prone to bias. Some of the later studies, however, have improved the exposure assessment. An Austrian study was one of the first to perform RF measurements in homes (Hutter et al 2006), but the actual measurements were not used in the analyses of associations with symptoms. Instead the maximum exposure from the base station was computed based on measurements of broadcast channels. Statistically significantly increased 1.3 to 1.6-fold prevalence of three out of 14 subjective symptoms (headaches, cold hands or feet and concentration difficulties) was reported in the group with the highest exposure. No effect was found on sleep quality, although concern for adverse effects of base stations was associated with poorer sleep quality. A German study measured distance to base stations through geo-coding (Blettner et al 2009), and found a slightly higher prevalence of health complaints among people living within 500 meters of a base station. People who were concerned about or attributed adverse health effects to exposure from mobile phone base stations reported a higher prevalence of health complaints. The German study also included a component where RF exposure in the homes of a subset of participants were estimated through individual RF measurements of the background RF-EMFs from mobile phone base stations and other external sources (Berg-Beckhoff et al 2009). People who attributed adverse health effects to mobile phone base stations reported significantly more sleep disturbances and health complaints, but the actual measurements of the RF fields were not associated with health complaints. Another German study used personal measurements of RF fields from mobile communication systems during waking hours to estimate exposure (Thomas et al 2008), which includes also mobile phone use, and found no associations between exposure levels and chronic or acute symptoms.

Generally, studies of symptoms and well-being find a higher prevalence of symptoms and less well-being among persons who are concerned about exposure from base-stations, whereas there is little evidence for an association between measured RF levels and the studied outcomes.

Discussion

The research on community exposures to radiofrequency fields and cancer gives a very weak test of the possibility of a relation. Diverse exposure sources, poorly estimated population exposures, small numbers of cases, and selective investigation in response to cluster concerns have resulted in a literature that is of limited value. Despite apparent positive relations between proximity and leukemia incidence in some analyses (Hocking et al 1996; Michelozzi 2002), the results have not been consistent within or between studies, and do not show relations to RF exposure levels . It seems to us that a prerequisite for a new generation of informative studies to emerge is the use of an RF meter.

Some of the concern about health risks from living near transmitter towers is directed toward symptoms such as fatigue, sleep disturbances, and frequent headaches. It may be tempting to address such issues in a cross-sectional study on people living near transmitters in which subjects are asked to report their symptoms. Indeed, such studies have been done, as discussed above. However, this is a design in which exposure is often poorly characterized and reporting bias with respect to symptoms of profound concern. Experimental designs easily overcome these biases and thus would be preferable, although they have their own limitations such as difficulty in practice in detecting effects present in a small percentage of a population or when the effect is not immediate. In these latter situations, an observational study would be the design of choice, but only if a design was found that avoided reporting bias.

III.A.7. REVIEW OF STUDIES ON MOBILE PHONE USE

Most studies of association between cancer and mobile phone use have evaluated the risk of brain tumors (Table III.A.7.); though in a few instances the risks of other tumors have been explored. Also studies of symptoms in relation to mobile phone use have been conducted (Table III.A.8.). The first case-control study of brain tumors was conducted in Sweden (Hardell et al 1999; 2000; 2001) and included adult cases diagnosed in two regions in Sweden between 1994 and 1996 and still alive, with two controls per case matched for region of residence. Details of intensity and duration of mobile phone use, preferred side (ear) of use and whether phones were analogue or digital, and handheld or hands-free, were gathered by postal questionnaire followed by telephone interview (Hardell 1999). 209 cases (only about a third of the malignant cases occurring in the study geographical area in the period (Ahlbom et al 1999) took part along with 425 controls (a reported 91% response rate – extraordinarily high for a contemporary population-based study). There was no association of phone use with brain tumors (Hardell et al 1999), though later re-analysis of side of use in relation to tumor site suggested a possible relationship (Hardell et al 2001). A second larger study a few years later by the same authors (2002; 2003) was similar in design to the first. It involved 1303 living cases (51% of all brain tumors diagnosed 1997 – 2000) and their controls. Cumulative phone use for over 85 hours, 10 years before case diagnosis, gave ORs for brain tumors of 1.9 (1.1-3.2) and 3.0 (0.6-14.9) respectively for analogue and cordless phones, but not raised for digital. There was no adjustment for confounding variables. Ipsilateral use of analogue phones was related to temporal tumors, OR=2.5 (1.3-4.9), and general analogue phone use was associated with acoustic neuroma, OR= 3.5 (1.8-6.8) (Hardell et al 2002; 2003).

Muscat et al conducted two hospital-based case-control studies in the USA, one of malignant brain tumors (Muscat et al 2000), the other of acoustic neuroma (Muscat et al 2002) using the same ascertainment and data collection procedures (Table III.A.7.). The first study included 469 cases of brain cancer (70% response rate), and 422 matched controls from the same hospitals (90% response rate) with a variety of malignant and benign conditions. Information about mobile phone use was obtained by standard interview (of proxies for 9% of cases and 1% of controls). No raised risks were seen relating to frequency or duration of use, or for site or histologic subtype of brain cancer. An excess of brain cancer was found on the same side of the head as reported phone use among 41 cases with assessable data ($p = 0.06$), compared with a deficit on the side of mobile phone use for tumors specifically located in the temporal lobe ($p = 0.33$). In the acoustic neuroma study, 90 cases were compared with 86 controls, and no associations were seen with level or laterality of phone use.

In another US hospital-based case-control study (Inskip 2001) interview data were obtained from 782 cases with brain tumors (92% response rate; via proxies for 16% and 3% of glioma and acoustic neuroma patients respectively) and 799 matched hospital controls with non-malignant conditions (88% response; 3% by proxy). Results adjusted for potential confounders showed no association between cumulative use of mobile phones (mainly analogue) and brain tumor overall or by histological subtype or anatomical location.

Subscription records of national network providers were used to characterize mobile phone users in a Finnish case-control study (Auvinen et al 2002). All people (398) diagnosed with brain tumors in 1996, ascertained from the National Cancer Registry, were matched with 5 controls per case drawn from the national population register (Table III.A.7.). The OR for brain tumors with ever-subscription to phones was 2.1(1.3 - 3.4) for analogue phones and 1.0 for digital, and the OR for glioma was 1.5 (1.0 – 2.4) (null for other brain tumor histologies) for any phone subscription. The average duration of subscription was 2-3 years for analogue phones and less for digital. Adjusting for potential confounders did not alter results. No information was available about the frequency or duration of calls or about corporate subscriptions.

Of two cohort studies, an early US study (Rothman et al 1996; Dreyer et al 1999) analyzed one year of follow-up of mortality in a cohort of 285,561 non-corporate users of mobile phones with at least 2 billing cycles from two US carriers. No relation was found between mortality from brain cancer (based on 6 cases ascertained from the National Death Index) and the use of handheld versus non-handheld phones. The overall mortality of the cohort was less that in the general population. The

second cohort study was in Denmark (Johansen et al 2002a) and comprised 420,095 private cellular network subscribers (80% of all subscribers), with average follow-up for analogue and digital subscribers of 3.5 and 1.9 years respectively. Standardized incidence ratios comparing cancer rates in phone users with national rates allowing for sex, age and period, showed no relation to risk of brain and nervous system cancers (SIR= 0.95, 0.81 – 1.21) and reduced risk of smoking related-cancers. Risks did not vary by age at, or time since, first subscription, phone type or tumor location. Again no information was available about the frequency or duration of calls or about corporate subscriptions.

Regarding other head and neck cancers, no association with parotid gland tumors (34 cases) was seen in the Finnish case-control study (Auvinen 2002), or in the Danish cohort study (Johansen et al 2002a). A mixed population and hospital-based case-control study of uveal melanoma (Stang et al 2001) included 118 cases and 475 controls. Occupational exposure to mobile phones for several hours a day for 6 months or more assessed by interview gave a raised OR (4.2, 1.2 – 14.5), reflecting result in the hospital-based participants (OR = 10.1), although there was no raised risk of uveal melanoma in the Danish mobile phone user cohort (Johansen et al 2002b). Finally, leukemia was assessed in both cohort studies, but no relation with phone use was found.

The first report from the multicentre Interphone study has recently been published. This study focused on acoustic neuroma and was negative; however, the number of long term users was small (Christensen et al 2004).

Subjective symptoms, including tinnitus, headache, dizziness, fatigue, sensations of warmth, dysesthesia of the scalp, visual symptoms such as flashes, memory loss and sleep disturbance have been investigated in relation to mobile phone use (Chia et al 2000; Oftedal et al 2000; Sandstrom 2001) - see Table III.A.8. for details. As discussed above in relation to transmitter studies, such research is highly susceptible to recall bias. For completeness we have also added a table with experimental studies on mobile phone use and symptoms.

Discussion

Handheld mobile phones were not used regularly until the 1990s, so published studies at present can only assess relatively short lag periods before cancer manifestation. The relevant lag periods are unknown. Even in the large Danish study (Johansen et al 2002a), long-term (15 years) subscribers to analogue phones comprised only a small proportion of users.

Another issue relates to choice of study population. No study populations to date have included children, yet children are increasingly heavy users of mobile phones and they are potentially highly susceptible to harmful effects (although some of these effects might not manifest until adulthood). So far study populations have been ascertained from population registers in Nordic studies, hospital in-patients in the US case-control studies, and cellular network private subscribers in the two cohort studies and the Finnish study. While the population-based studies should have avoided the selection biases inherent in the hospital based studies, this was not so in population-based case-control studies of prevalent living cases with low participation rates (Hardell et al 1999; 2002) since inter alia those with high grade tumors tend to be excluded. While rapid recruitment of incident brain tumor cases was facilitated in the hospital-based studies, loss due to death was still greater for malignant than benign tumors as reflected in differential proxy response rates by tumor type (Inskip et al 2001), and there is a major weakness in using hospital controls with a variety of conditions of unknown relationship to mobile phone use.

Differential recall of mobile phone use among those with and without a cerebral tumor in case-control studies is a major potential source of bias, exacerbated by differential timing of data collection from cases and controls in the hospital studies. Reporting bias is also likely since presence of a brain tumor may distort both memory and hearing. Bias is also likely introduced by the use of proxies, especially as use of proxies was more common for cases than controls. Relying on private cellular network subscription as a proxy for mobile phone use would also have resulted in substantial misclassification because subscribers bear only a modest relation to users (Funch et al 1996) and because corporate users, likely to be among the earliest and heaviest users of mobile phones, were either excluded or

included in the unexposed group. Until there is some objective measure of RF exposure, or at least validation of self-reported records, the validity of self-reported indices of phone use e.g. average minutes of use per day (Hardell et al 2002; Inskip et al 2001) or minutes/hours per month as indicators of RF exposure, remains unknown.

Overall, while occasional significant associations between various types of brain tumor and analogue mobile phone use have emerged (often seen after multiple testing), no single association has been consistently reported across population-based studies. The timing of epidemiological studies and the lack of knowledge about actual RF exposure to the brain from mobile phone use to date (Gandhi et al 1999) mitigate strongly against current ability to detect any true association. Thus current evidence is inconclusive regarding cancer risk following heavy RF exposure from mobile phones. Similarly the studies of symptoms to date do not suggest that a single exposure to RF from a mobile phone results in immediately identifiable symptoms, but there are no adequate population-based data available about the symptomatic effects of repeated mobile phone use, especially among those who claim hypersensitivity to RF.

III.A.8. GENERAL CONCLUSIONS AND RECOMMENDATIONS

Results of epidemiological studies to date give no consistent or convincing evidence of a causal relation between RF exposure and any adverse health effect. On the other hand, these studies have too many deficiencies to rule out an association.

A key concern across all studies is the quality of assessment of RF exposure, including the question of whether such exposure was present at all. Communication sources have increased greatly in recent years, and there is continuing change in the frequencies used and the variety of applications. Despite the rapid growth of new technologies using RF, little is known about population exposure from these and other RF sources and even less about the relative importance of different sources. Certain studies that are currently under way have made serious attempts to improve exposure assessment, based on attempts to learn more about determinants of RF exposure levels. A key element in improving future studies would be the use of a meter that monitors individual exposure. In the absence of information on what biological mechanism is relevant, if any, it is unclear what aspect of exposure needs to be captured in epidemiological studies. Ideally, the dose needs to be assessed not just as external field intensity, but also as cumulative exposure, as well as SAR, for specific anatomical sites.

The need for better exposure assessment is particularly strong in relation to transmitter studies, because the relation between distance and exposure is very weak. There is no point in conducting such studies unless it has been established that exposure levels vary substantially within the study area, and measurements of these RF levels are available. In the future, methods need to be developed to infer exposure based on some combination of knowledge regarding the sources of exposure, the levels of exposure, and location of people in relation to those sources, ideally informed by selective measurements.

Although the likelihood is low that fields emanating from base stations would create a health hazard, because of their weakness, this possibility is nevertheless a concern for many people. To date no acceptable study on any outcome has been published on this. On the one hand, results from valid studies would be of value in relation to a social concern; on the other hand, it would be difficult to design and conduct a valid study, and there is no scientific point in conducting an invalid one.

Another general concern in mobile phone studies is that the lag periods that have been examined to date are necessarily short. The implication is that if a longer lag period is required for a health effect to occur, the effect could not be detected in these studies. Only in the few countries where mobile phones were introduced very early has it been possible to look at ten years of usage or more. Much longer lag periods have been examined for occupational RF exposures, however. The published studies include some large occupational cohorts of good design and quality, except that there has been poor assessment of the degree of RF exposure, which render the results difficult to interpret.

The majority of research has focused on brain tumors and to some extent on leukemia. However, because the RF research questions are not driven by a specific biophysical hypothesis but rather by a general concern that there are unknown or misunderstood effects of RF fields, studies on other health effects may be equally justified. Examples are eye diseases, neurodegenerative diseases, and cognitive function. Given the increase of new mobile phone technologies, it is essential to follow various possible health effects from the very beginning, particularly since such effects may be detected only after a long duration, due to the prolonged latency period of many chronic diseases. Thus, research is needed to address long-term exposure, as well as diseases other than those included in the ongoing case-control studies.

Another gap in the research is children. No study population to date has included children, with the exception of studies of people living near radio and TV antennas. Children are increasingly heavy users of mobile phones, they might be particularly susceptible to harmful effects (although there is no evidence of this), and they are likely to accumulate many years of exposure during their lives..

Table III.A. 1.: Cohort studies of risk of cancer in relation to occupational or hobby RF exposure - description of studies

Authors Year	Occupational group	Sex	No of subjects	Measure of exposure	Outcome
Milham 1988	Amateur radio operators	Male	67,829	Hobby title	Mortality
Garland et al 1990	Navy personnel: electronics technicians, aviation electronics technicians, fire control technicians*	Male	- **	Job title	Incidence
Muhm et al 1992	Electromagnetic pulse test workers	Male	304	Job title	Mortality
Tynes et al 1996	Radio & telegraph operators on merchant ships	Female	2,619	Measures in radio rooms of 3 ships	Incidence
Szmigielski 1996	Military career personnel	Male	128,000 total‡ 3,700 exposed‡	Military health records; representative exposure levels given, based on measurements (no. not stated)	Incidence
Szmigielski et al 2001	Military career personnel	Male	124,500 total 3,900 exposed		
Lagorio et al 1997	Dielectric RF heat sealer operators	Female	481	Unclear – stated that >10W/m^2 'frequently exceeded'	Mortality
Morgan et al 2000	Motorola employees	56% male 44% female	195,775 total 24,621 exposed	Job title, with expert assessment (not measured) of usual exposures	Mortality
Groves et al 2002	Navy personnel with potential radar exposure	Male	40,581 total 20,021 high exposure	Job title, plus expert assessment on potential for high exposure, and information on type and power of radar units	Mortality
Lilienfeld cited by Goldsmith 1995	US embassy personnel	Males & Females	Not stated	Moscow embassy service	Mortality

*We have extracted from the published paper data on those jobs stated by Groves et al (2002) to have greatest RFR exposure. **Not stated.

†Not strictly a cohort study – there does not appear to be any follow-up. Design appears to be calculation of annual rates, based on annual incidence and counts of employed population, and then averaging of these rates.

‡"Mean count each year ". Presumably many but not all of the personnel will have been the same individuals from year to year of the study.

Table III.A.2.: Cohort studies of risk of cancer in relation to occupational RF exposure : results for brain tumor and leukemia

Author Year	Type of analysis	Brain tumor		Leukemia		Comment
		n	Relative risk (95% CI)	n	Relative risk (95% CI)	
Milham 1988	SMR, cohort *cf* general population	29	1.4 (0.9 – 2.0)	36	1.2 (0.9 – 1.7)	In a sample, 31% of subjects worked in EMF-exposed occupations. Analyses by license class, a proxy for duration of licensing, showed no consistent trend in risk (Milham, 1988b)
Garland et al 1990	SIR, cohort *cf* general population	- [*]		5	1.1 (0.4 – 2.5)	
	Electronics techn	- [*]		<3	0.3 (0.0 – 1.9)	
	Aviation tech.	- [*]		<3	0.5 (0.0 – 2.5)	
	Fire control tech.					
Muhm et al 1992	SMR, cohort *cf* general population,	0	-	1	4.4 (0.1 – 24.3)	One of the leukemia cases may have been allocated to this work because of his leukemia.
	underlying cause				7.7 (0.9 – 28.0)	
	SMR, cohort *cf* general population,	0	-	2	5.4 (0.7 – 19.7)	
	mentioned cause	-	-	2		
	SIR, cohort *cf* general population					
Tynes et al 1996	SIR, cohort *cf* general population	5	1.0 (0.3 – 2.3)	2	1.1 (0.1 – 4.1)	
Szmigielski 1996*	Average crude incidence rate in	*	1.9[**](1.1 – 3.5)	19	7.7[†] ([*])	Poorly conducted and reported study. Apparently more exposure data sources for cases than controls. 'Expected' rates in the 1996 paper appear to be incorrect, according to the Royal Society of Canada (The Royal Society of Canada, 1999). Significant excesses reported for several cancer sites not seen in other studies, and for cancer overall, suggesting possible bias. Analyses of risk in relation to exposure level presented only for total cancer, not specific cancer sites.
Szmigielski et al 2001	exposed *cf* average crude rate in unexposed.	7	2.7[**](p<0.01)		6.5[†](p<0.01)	

Table III.A.2.: Cohort studies of risk of cancer in relation to occupational RF exposure : results for brain tumor and leukemia

Author Year	Type of analysis	Brain tumor		Leukemia		Comment
		n	Relative risk (95% CI)	n	Relative risk (95% CI)	
Lagorio et al 1997	SMR, cohort *cf* general population	1	10	1	5	Potential confounding by chemical exposures. Losses to follow-up treated as alive to end of study period.
Morgan et al 2000	SMR, exposed workers *cf* general population	17	0.5 (0.2 – 1.1)	21	0.8 (0.4 – 1.4)	No duration-response trend
	Rate ratio exposed *cf* unexposed in cohort, cumulative exposure					
	None					
	<median	34	1.0	66	1.0	
	≥median	7	1.0 (0.4 – 2.2)	8	0.6 (0.3 – 1.3)	
		10	0.9 (0.4 – 1.9)	13	0.6 (0.3 – 1.0)	
Groves et al 2002	SMR, overall cohort *cf* general population	88	0.9 (0.7 – 1.1)	113	1.0 (0.8 – 1.2)	Significant raised risk for nonlymphocytic leukemia in high exposure cohort, but only raised in one of 3 high-exposure occupations.
	SMR, high exposure cohort *cf* general population	37	0.7 (0.5 – 1.0)	69	1.1 (0.9 – 1.4)	
	Relative risk, exposed *cf* unexposed in cohort	37/51	0.6 (0.4 – 1.0)	69/44	1.5 (1.0 – 2.2)	
Lilienfeld cited by Goldsmith 1995[#]	Observed and expected, but source of latter unclear		Adults: 2/1.9 Children: 0/-		2/2.0 2/4.0	Data also presented for other US embassies in Eastern Europe, but unclear whether they were exposed. Both brain tumors and one leukemia in a child were in dependents who lived out of the embassy.

[*]No data published. For Szmigielski it is implied that there were 2-3 brain tumors and X leukemiae in the exposed group, in which case we imply that the CI for brain tumor is incorrect.
[**]Nervous system. [†]Calculated from data in the paper.
Notes: Study not published by Lilienfeld, and too little information given in précis by Goldsmith for understanding or evaluation of the methods. [#]Small numbers of cancers, and several of the cancers occurred in persons who lived out of the embassy (i.e. presumably were in the embassy little of the time, especially children). Breast cancer in employees 2 observed, 0.5 exp. Cancers of female genitalia 4 observed, 0.8 expected. Exposures estimated to range from 5 – 18μ W/cm^2 – basis of estimate not stated.

Table III.A.3.: Case-control studies of risk of brain tumor and leukemia in relation to occupational RF exposure

Authors Year	Sources of cases and controls*	Measure of exposure	Exposure data collection method	Mortality or incidence	Nos. of cases/ controls	Type of analysis	Results	
							Brain tumor OR (95% CI)	Leukemia OR (95% CI)
Thomas et al 1987	Cases: death certificates Controls: death certificates for deaths from other causes, except epilepsy, stroke, suicide, homicide	Job title & industry	Interview with relatives	Mortality	435/386	Odds ratio *cf* never occ. exposed	1.6 (1.0-2.4)	-
Amstrong et al 1994	Electrical utility workers (nested case-control)	Job exposure matrix based on 1 week meter measurements at 5-20 MHz** for >1000 workers, assessing exposure to pulsed electromagnetic fields	Company records	Incidence	84/325	Odds ratio for ≥median exposure	0.8 (0.5 – 1.5)† 1.9 (0.5 – 7.6)†	-
					95/374	Odds ratio for ≥90th percentile	-	0.7 (0.4 – 1.2)
						Odds ratio for ≥median exposure	-	0.8 (0.2 – 3.4)
						Odds ratio for ≥90th percentile		
Grayson 1996	USAF (nested case-control)	Job title & whether reports of incidents of high exposure for each job title	Military records	Incidence	230/920	Odds ratio *cf* never-exposed	1.4 (1.0-1.9)	-

*All studies restricted to men.

**But it was subsequently found that the meters also responded to fields of 150 and 300 MHz and to radio transmissions.

†Malignant brain tumors.

Table III.A.4.: Analyses of routinely collected data on brain tumor and leukemia risk in relation to occupational RF exposure

Author Year	Type of analysis	Exposed group*	Comparison cohort/ control group	Mortality or incidence	Brain tumor		Leukemia	
					n**	RR (95% CI)	n**	RR (95% CI)
Wright et al 1982	Proportional incidence	Radio & TV repairmen. Telephone linesmen.	All other cancers	Incidence	-†		1	1.2 (-†)
					-†		2	3.1 (-†)
Calle& Savitz 1985	Proportional mortality	Radio & telegraph operators.	All causes of death	Mortality	-†		6	2.3 (-†)
		Radio & TV repairmen			-†		3	0.9 (-†)
Lin et al 1985	Case-control	Electric & telephone linemen, servicemen	Non-cancer deaths	Mortality	27			(-†)
Milham 1985	Proportional mortality	Radio & telegraph operators.	All causes of deaths	Mortality	1	0.4 (-†)	5	1.0 (-†)
		Radio & TV repairmen			2	0.6 (-†)	7	1.8 (-†)
Pearce et al 1989	Case-control	Radio & TV repairmen	All other cancers	Incidence	-†		2	7.9 (2.2 – 28.1)
Tynes et al 1992	Cohort	Radiofrequency exposed occupations	Economically active males	Incidence	3	0.6 (0.1 – 1.8)	9	2.8 (1.3 – 5.4)

*All studies are of males. Exposure assessment for all is based solely on job title, with no measures of exposure.
**No. in exposed group.
†No data published.

Table III.A.5.: Summary of literature on radiofrequency exposure and reproductive health outcomes

Outcome Measure	Reference	Geographic Setting	Population Size & Source	Results: Exposure & Outcome	Comments
Semen parameters					
	Lancranjan et al 1975	Romania	Microwave exposure (31) vs. controls (30)	Sperm count: 50 (Exp), 60 (Ctl) million/ml Motility: 36% (Exp), 54% (Ctl)	
	Weyandt et al 1996	US	Military intelligence (20) vs. controls (30)	Sperm density: 13 (Exp), 35 (Ctl) Percent normal: 69 (Exp), 73 (Ctl) Percent motile: 32 (Exp), 43 (Ctl)	
	Hjollund & Bonde 1997	Denmark	Military: missile operators (19), other (489)	Sperm density: 40 (Exp), 62 (Ctl) Immotile %: 52 (Exp), 33 (Ctl) Percent normal: 61 (Exp), 68 (Ctl)	
	Schrader et al 1998	US (Texas)	Military: radar operators (33), controls (103)	Sperm density: 29 (Exp), 32 (Ctl) Percent normal: 46 (Exp), 42 (Ctl) Percent motile: 46 (Exp), 45 (Ctl)	
	Grajewski et al 2000	US (Maryland)	RF heater operators	Sperm density: 47 (Exp), 45 (Ctl) Sperm count: 73 (Exp), 93 (Ctl) Motile (%): 67 (Exp), 52 (Ctl) Normal morphology: 81 (Exp), 79 (Ctl)	
Fertility					
	Larsen et al 1991	Denmark	Physiotherapists 49 time to pregnancy over 6 mos	TWA Exposure & TTP >6 months RR = 1.0, 0.8 (0.2-2.2), 1.7 (0.7-4.1):	

Outcome Measure	Reference	Geographic Setting	Population Size & Source	Results: Exposure & Outcome	Comments
Spontaneous abortion					
	Taskinen et al 1990	Finland	Physiotherapists 204 Spontaneous abortions	SAb <=10 Deep heat 1.0,1.3,0.7, Shortwaves 1.0,1.2,0.7, Microwaves 1.0,0.7 SAb >10 Deep heat 1.0, 1.3, 2.6, Shortwaves 1.0,2.5,2.4; Microwaves 1.0,2.4	
	Larsen et al 1991	Denmark	Physiotherapists 146 Spontaneous abortions	TWA Exposure & Sab: RR = 1.0, 1.0 (0.5-1.8), 1.4 (0.7-2.8)	
	Ouellet-Hellstrom & Stewart 1993	US	Female physical therapists 1664 Spontaneous abortions	Microwave Diathermy Exposures/mo.: RR=1.0, 1.1(0.8-1.4), 1.5 (1.0-2.2), 1.6 (1.0-2.6) Shortwave Diathermy Exposures/mo: RR=1.0, 1.2 (1.0-1.5), 1.1(0.9-1.4), 0.9 (0.6-1.2)	
Stillbirth					
	Larsen et al 1991	Denmark	Physiotherapists 17 perinatal deaths	TWA Exposure & Perinatal Death: RR = 1.0, 1.5 (0.3-5.3), 2.9 (0.6-10.7)	
Preterm Birth					
	Larsen et al 1991	Denmark	Physiotherapists 37 male, 45 female	TWA Exposure & Preterm Birth: Male: RR=1.0, 1.4 (0.4-4.7), 3.2 (0.7-13.2) Female: RR=1.0, 0.9 (0.4-2.1), 0.9 (0.3-2.8)	

Outcome Measure	Reference	Geographic Setting	Population Size & Source	Results: Exposure & Outcome	Comments
Low Birth Weight					
	Larsen et al 1991	Denmark	Physiotherapists 15 male, 24 female	TWA Exposure & Low Birthweight: Male: RR=1.0, 0.0, 5.9 (1.0-28.2) Female: RR=1.0, 1.2 (0.4-3.3), 0.7 (0-3.2)	
	Guberan et al 1994	Switzerland	Physiotherapists 11 male, 14 female	No association with shortwaves (RR not reported)	
Birth Defects					
	Logue et al 1985	US	Physical therapists (male) 192 birth defects	Observed:expected range -- "appears to be higher than expected"	
	Taskinen et al 1990	Finland	Physiotherapists 51 birth defects	Deep heat 1.0, 2.4 (1.0-5.3), 0.9 (0.3-2.7) Shortwaves 1.0, 2.7 (1.2-6.1), 1.0 (0.3-3.1) Microwaves 1.0, 0.5 (0.1-3.9)	

Table III.A.6.: Summary of studies on transmitters and cancer

Reference	Source of exposure	Comparison	Endpoints	Nr. Cases	Results	Setting	Comments
Selvin et al 1992	MW tower	Internal	Childhood leukemia	123 52	Random pattern	San Francisco	Analysis of spatial data; no epi param.
Maskarinec et al 1994	LF radio (23.4 kHz)	<2.6 miles	Childhood leukemia	12	2.0: 0.06-8.3	Hawaii; case-control	SIR analysis on same cases: 2.09: 1.08-3.65
Hocking et al 1996	TV towers	Inner/ outer	All age leukemia Childhood leukemia		1.24: 1.09-1.40 1.58: 1.07-2.34	Northern Sydney	8 –0.2μW/cm^2
Dolk et al 1997 I	TV and FM radio	<2 km	Adult leukemia	23	1.83:1.22-2.74	Sutton Coldfield	
Dolk et al 1997 II	TV and FM radio	<2 km	Leukemia	79	0.97: 0.78-1.21	All GB	
McKenzie et al 1998	TV transmission antennas	Cont. μW/cm^2 model	Childhood leukemia			Sydney	Reanalysis of Hockings; concl. One LGA explains
Cooper D et al 2001	TV and FM radio	<2 km	All age leukemia Childhood leukemia	20 1	1.32. 0.81-2.05 1.13: 0.03-6.27	Sutton Coldfield	Reanalysis, more timely cancer data
Michelozzi et al 2002	Radio station	<6 km	Childhood leukemia Adult leukemia	8 23	2.2: 1.0-4.1 1.2: .8-1.8	Vatican	

Table III.A.7.: Summary of studies of mobile phone use and risk of brain tumors

Authors Year (study design)	Study population	Tumor type (numbers cases/ controls)	Exposure assessment	Phone type; Duration of use in controls	Phone ever-use RR (95% CI)
Hardell et al 1999 (case-control)	Sweden. Cases: 20-80 yr. Controls: regional population registers, Uppsala-Orebro 1994-96, Stockholm 1995-96	All tumors (209/ 425) Acoustic neuroma	Recalled mobile phone use by questionnaire and interview	Mainly analogue, 450 or 900 MHz; 16% >5 yr.	1.0 (0.7 – 1.4)* 0.8 (0.1 – 4.2)
Muscat et al 2000 (case-control)	USA: Hospital inpatients, NY, Providence, Boston. Cases:18-80 yr ,1994-98. Controls: Malignant and non-malignant conditions.	Malignant brain tumor (469/ 422)	Recalled mobile phone use via interview	Mainly analogue 800 – 900 MHz; 5%>4 yr.	0.9 (0.6 – 1.2)
Inskip et al 2001 (case-control)	USA: Hospital inpatients, Boston; Phoenix; Pittsburgh. Cases:18+ yr, 1994-98. Controls: non-malignant conditions	All tumors (782/799) Glioma (489/799) Meningioma (197/ 799) Acoustic neuroma (96/799)	Recalled mobile phone use via interview	Mainly analogue 800 – 900 MHz; 8% >3 yr.	0.9 (0.7 – 1.1) 1.0 (0.7 – 1.4) 0.8 (0.5 – 1.2) 0.8 (0.5 – 1.4)
Muscat et al 2002 (case-control)	USA: Hospital inpatients, New York. Cases:18+ yr, 1997-99. Controls: Non-malignant conditions.	Acoustic neuroma (90/86)	Recalled mobile phone use via questionnaire	Mainly analogue 800 – 900 MHz; 7% 3-6 yr.	0.9

Authors Year (study design)	Study population	Tumor type (numbers cases/ controls)	Exposure assessment	Phone type; Duration of use in controls	Phone ever-use RR (95% CI)
Auvinen et al 2002 (case-control)	Finland. Cases: 20-69 yr, 1996. Controls: National population register.	All tumors (398/1986) Glioma (198/989) Benign (129/643) Salivary gland (34/170)	Duration of private cellular network subscription	Analogue, average 2-3 yr subscription; digital, average <1 yr subscription.	1.3 (0.9 – 1.8) 1.5 (1.0 – 2.4) 1.1 (0.5 – 2.4) 1.3 (0.4 – 4.7)
Hardell et al 2002 (case-control)	Sweden. Cases: 20-80 yr. 1997-2000. Controls: 4 regional population registers.	All tumors (1303/1303)	Recalled mobile phone use via questionnaire	Analogue 450 or 900 MHz, median 8 yr. Digital 1900 MHz, median 3 yr.	1.3 (1.0 – 1.6)*
Hardell et al 2003 (case-control)		Acoustic neuroma(159/422)		Analogue Digital	1.0 (0.8 – 1.2) 3.5 (1.8 – 6.8) 1.2 (0.7 – 2.2)
Dreyer et al 1999 (cohort)	USA. Subscribers of 2 large cellular networks. 1993. Cases: ≥20 yr deaths 1994	Malignant brain tumor (6).	Duration of subscription	Analogue. 1 yr follow-up	–
Johansen et al 2002 (cohort)	Denmark. Private cellular network subscribers. 1982-95. Cases: ≥18 years. 1982-96.	All tumors (154) Glioma (66) Meningioma (16)	Duration of subscription	Analogue (450 or 900 MHz) or digital. Up to 15 yr follow-up	SIR 1.0 (0.8-1.1) (0.7 – 1.2) 0.9 (0.5 – 1.4)
Christensen et al 2004	Denmark Population-based case-control	Acoustic neuroma (106) Population controls (212)	-	-	0.90 (0.51 – 1.6)

Table III.A.8.: Summary of studies of mobile phone use and symptoms

Authors Year (study design)	Study population	Analyses	Exposure assessment	Outcome assessment	Results
Hocking 1998 (case-series)	Australians with symptoms on mobile phone use who responded to notice in medical journal or media publicity N=40)	Description of type of symptoms reportedly due to mobile phone use	No formal assessment of amount or frequency of mobile phone use	Questionnaire about details of symptoms associated with mobile phone use	Most respondents reported unusual sensations affecting the head, such as dull pain, unpleasant warmth. **
Oftedal et al 2000 (cross-sectional)	Swedish and Norwegian mobile phone users, selected from network operator registers. Only included people who used phone for job. N=10631	Number of respondents with any symptom attributed to mobile phones Number of respondents who had taken steps to reduce symptoms	Not well described, but one table reports number of calls and calling time per day, suggesting reported in a questionnaire.	Self-reported frequency of symptoms. Patient considered to have symptom if occurred at least once per week.	1.13% of participants in Sweden and 31% in Norway reported at least 1 symptom in connection with use of a mobile phone. Most common, warmth around ear. 22% of Norwegians and 7% of Swedes experienced symptom other than warmth. 2. 45% of people experiencing symptoms had taken steps to reduce them, such as reduced calling time, use of hands free kit, changing side phone used.

Authors Year (study design)	Study population	Analyses	Exposure assessment	Outcome assessment	Results
Sandstrom et al 2001 (cross-sectional)	Swedish and Norwegian mobile phone users, selected from network operator registers. N=16,992	Comparison of GSM versus NMT mobile phone users Trends with increasing time of phone usage	Self-completed questionnaire, variables: transmitter system, calling time per day and number of calls per day	Self-reported frequency of range of symptoms. Participant considered to have symptoms if occurred at least once per week.	OR among GSM cf NMT phones: No increased risk for any symptoms. GSM users at lower risk of warmth behind ear (OR: 0.64, 95% CI 0.51-0.80) or on ear (OR: 0.68, 95% CI 0.53-0.86). GSM users in Sweden at lower risk of headaches (OR: 0.73, 95% CI 0.56-0.95) and fatigue (OR: 0.80, 95% CI 0.65-0.99) With increasing minutes of phone use there was an increased odds of reporting fatigue, headaches, warmth, burning and tightness at least once per week. *
Chia et al 2000 (cross-sectional)	Random sample of 635 households in housing estate in Singapore. 808 respondents (NB response rate less than 60%).	Prevalence ratio of headache in mobile phone users vs non-users Association between minutes phone use and headache	Interviewer-administered questionnaire. Purpose of study masked. Classified as MP user if used at least once/day.	Questionnaire concerning nature and severity of 'CNS symptoms.' (headache, dizziness, warmth, tingling, visual disturbances). NB the frequency of headaches required before a respondent was classified as a headache sufferer was not specified.	1. 45% mobile phone users 3% experienced CNS problems Adjusted prevalence ratio for headache among users cf non-users 1.31 (95% CI 1.00-1.70). No significant differences for any other symptoms. 2. Significant positive trend for increasing time spent on the mobile phone and prevalence of headache (p=0.04).

Table III.A.9.: Summary of experimental studies of mobile phone use and symptoms

Authors	Participants	Exposure source	Protocol	Symptoms reported	Results
Hietanen et al 2002 (experimental)	20 volunteer subjects, mean age 51 for women and 47 for men, all of whom classified themselves as hypersensitive to RF fields	Analogue NMT phone, transmitting at 900MHz. 900 and 1800MHz GSM phones.	Phones mounted near but not touching subject's ear. 3 or 4 experimental sessions lasting 30 minutes each, one of which was a sham exposure (random order)	Subjects asked to describe symptoms experienced during exposure. Blood pressure, heart rate and breathing frequency monitored. Follow-up form used to measure symptoms over subsequent days.	19/20 participants reported symptoms during the tests. Compared with women during sham exposure, relative number of symptoms reported by female subjects during NMT exposure was 0.82, GSM 900 0.79, GSM 1800 0.72. Among men, number of symptoms during any RF exposure situation was 0.85 compared with sham exposure.
Koivisto et al 2001 (experimental)	48 volunteers, students at University of Turku, Finland. Mean age 26 years	GSM 900 phone	2 exposure sessions, one with mobile phone on and one with off. Subjects blinded to whether phone was off or on. Half participants had phone on first and half off first.	Questionnaire assessing symptoms administered in the beginning, middle and end of session. Subjects asked to rate strength of sensations on 4 point scale. Symptoms assessed were dizziness, headache, fatigue, tingling, redness, warmth.	There were no significant differences between mean values for subjective ratings between exposure on and exposure off situations.

III.A.9. REFERENCES

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III.B. EPIDEMIOLOGIC EVIDENCE ON MOBILE PHONES AND TUMOR RISK: A REVIEW*

ABSTRACT

This review summarizes and interprets epidemiologic evidence bearing on a possible causal relation between radiofrequency field exposure from mobile phone use and tumor risk. In the last few years epidemiologic evidence on mobile phone use and the risk of brain and other tumors of the head in adults has grown in volume, geographic diversity of study settings, and the amount of data on longer-term users. However, some key methodologic problems remain, particularly with regard to selective non-response and inaccuracy and bias in recall of phone use. Most studies of glioma show small increased or decreased risks among users, although a subset of studies show appreciably elevated risks. We considered methodologic features that might explain the deviant results, but found no clear explanation. Overall the studies published to date do not demonstrate an increased risk within approximately ten years of use for any tumor of the brain or any other head tumor. Despite the methodologic shortcomings and the limited data on long latency and long-term use, the available data do not suggest a causal association between mobile phone use and fast-growing tumors such as malignant glioma in adults (at least for tumors with short induction periods). For slow-growing tumors such as meningioma and acoustic neuroma, as well as for glioma among long-term users, the absence of association reported thus far is less conclusive because the observation period has been too short.

* This review was first published in Epidemiology 20(5):639-652; 2009 and is reproduced here with the kind permission of the Journal.

Mobile phone use has increased with extraordinary rapidity, and is now nearly universal in some countries, with over two billion subscribers worldwide. The rise in use has generated concerns about safety, particularly potential cancer risk. When we reviewed this subject several years ago, we concluded that the studies at that time gave no consistent or convincing evidence of a causal relation between radiofrequency (RF) exposure and any adverse health effect. However, we could not rule out an association because of deficiencies in the research (Ahlbom et al 2004). Mobile phone studies at that time had been able to address only relatively short induction and latency periods, and included a relatively small number of heavy users. In the last five years, the volume of literature has more than doubled. We have therefore conducted a new review of the cumulated evidence on tumor risk in mobile phone users.

The emphasis of our review, and of the majority of recently published studies, is on tumors of the brain and other sites in the head that have the highest exposure from mobile phones held against the ear. These include the glial and meningeal tissue close to the surface of the head, the vestibular portion of the eighth cranial nerve where acoustic neuromas (vestibular Schwannomas) develop, and the parotid gland. For the rest of the human body the exposure is negligible except for the skin, hand and other potential sites where hands-free devices are placed. We first discuss the key methodologic issues, then review in sequence the study methods, results, and interpretation of findings for each of the cancers for which there is a substantial literature: glioma, meningioma, acoustic neuroma, and salivary glands.

III.B.1. METHODOLOGIC CONSIDERATIONS

III.B.1.1. Exposure Characteristics

The first mobile phone systems were analog and operated at 450 and 900 MHz. Digital systems, operating at higher frequencies (1,800–1,900 MHz) and using different modulation techniques, became prevalent in the early 1990s. Around 2004, third-generation systems using the Universal Mobile Telecommunication System, which operates in the 1,900–2,200 MHz frequency range, were introduced.

The systems differ also in other parameters that can influence radiofrequency exposure, including maximum power output and patterns of handovers (the manner in which the phone's connection is handed over from one base station to another). Analog systems operated at higher power levels than digital systems and probably resulted in a higher exposure per unit of use. Adaptive power control (a technology to adapt the transmission power to what is required given actual conditions, such as distance between the phone and base station) may reduce the emitted power by as much as a thousand-fold. With adaptive power control, exposure is generally higher at greater distance from the base station (e.g., in rural areas), when the user is moving (e.g., in a car), and in places where there is intensive use with frequent handovers (Hillert et al 2006; Lonn et al 2004a). To compensate for the shielding effect of building materials, power levels of phones are, on average, higher when a phone is used indoors than outdoors (Hillert et al 2006; Lonn et al 2004a). The importance of the various usage circumstances may vary with geographic location and over time (Hillert et al 2006; Lonn et al 2004a). In addition to system characteristics, the radiofrequency exposure also depends on the characteristics of the phone itself, including the type and location of the antenna (e.g., pull-out rod or built-in) and the tilt of the phone relative to the head. The spatial distribution of RF energy in the brain has been studied using measurements made on phantoms (Cardis et al 2008).4 It appears that nearly all of the energy (97-99%) is absorbed in the brain hemisphere on the side where the phone is used, mainly (50-60%) in the temporal lobe. Hands-free devices substantially reduce exposure to the head.

Most studies of mobile phones and cancer have asked the participants (or their proxies) directly about their history of use, including frequency and duration of calls. Some studies have also asked for more detail, including questions about types of phones. A few studies have instead used information on calls recorded by network operators for billing purposes. Each approach has advantages and disadvantages. More detailed data can be collected when information is obtained directly from the participants, but at the price of compromised accuracy and increased potential for recall and reporting bias. Validation

studies have shown that healthy individuals have a tendency to overestimate the length of their calls and to underestimate the frequency (Vrijheid et al 2009a; Vrijheid et al 2006). This pattern was dependent on the amount of use; heavy users tended to overestimate, whereas light users underestimated their use. A validation study including both brain tumor cases and healthy controls (Vrijheid et al 2009a) found a similar pattern among cases; however, the overestimation by cases increased with increasing time before interview, which was not seen among controls. The potential differential exposure misclassification in studies using self-reported phone use, especially for more distant time periods, may cause positive bias in estimates of disease risk. Network operator information is presumably more accurate and objective, but may be lacking in validity: some networks have information only about outgoing calls, and the information they have refers to subscribers rather than actual users. Neither self-report nor records provide all the relevant or completely accurate data. Thus, all studies based on phone use are affected by exposure misclassification, which (if non-differential) could dilute risk estimates. This is in addition to the errors inherent in inferring radiofrequency radiation exposure even from accurate information on use, for the reasons noted above.

III.B.1.2. Tumor location and laterality of tumor in relation to habitual side of phone use

When a mobile phone is held to the ear, maximum RF energy absorption occurs within the lobes of the brain or other sites near the ear that are within a few centimeters of the phone antenna. Thus, tumors in these locations are more plausibly associated with RF exposure from mobile phones than tumors at other locations.

Some case-control studies have asked about the habitual side of mobile phone use when the phone is hand-held, and have sought to investigate the association with ipsilateral and contralateral brain tumors. However, there is no evidence of consistency over time in a person's preferred side of use. Retrospective self-report of preferred side of use may be subject to bias. If cases believe that mobile phone use may have caused their tumor, they might overreport mobile phone use on the same side as the tumor. In addition, analysis of data regarding laterality of phone use presents analytic problems. First, a method is needed for handling cases and controls who say they have no preferred side of use. Second, the analysis of control data regarding laterality of mobile phone is problematic because controls have no tumor to determine a reference side. Several techniques have been employed to deal with this issue (Inskip et al 2001; Lonn et al 2004b; Takebayashi et al 2006). One should keep in mind that the one employed by Inskip et al (2001) results in a relative risk that cannot be compared with other relative risks. If a causal effect were operative, one would expect null findings for contralateral use and elevated risk for ipsilateral use, with an overall elevation in risk for all users. On the other hand, if individuals with cancer believed that phone use caused their tumor and overreport use on the affected side, this would result in an apparent excess risk of brain tumor on the side of reported phone use and a deficit in risk on the other side.

III.B.1.3. Induction and latency periods

Because mobile phones are a new technology, there is epidemiologic evidence on cancer risk only for relatively short periods since first exposure; data on exposures more than 10 years before cancer diagnosis are still limited. Most types of cancer occur many years, or even decades, after initial exposure to known carcinogens. A widely expressed view has been that it is therefore too soon to know whether mobile phones have an effect on cancer risk. However, the important issue is not how long it takes for maximum risk to occur, but how long before detectable risk is present. Even for asbestos, a carcinogen that has a notoriously long induction period, detectable elevations in risk occur 10-14 years after first exposure (Walker 1984). Futhermore, it has been argued that RF fields cannot plausibly initiate cancer since they do not damage DNA, and that if RF acts at a later stage in carcinogenesis, the effects on tumor occurrence should be relatively rapid. However, epidemiologic studies are based on diagnosed tumors, whose identification depends not just on the induction period (period between exposure and initiation of disease) but also on their latency (i.e., how long they are present before being detected). Latency is likely to be short for fast-growing malignancies, but could be decades for less-aggressive tumors such as acoustic

neuromas and benign meningiomas. Hence for glioma (or at least the subset of gliomas that are fast-growing) information on risks 10 or 15 years after first exposure could provide meaningful information for determining whether mobile phone use has an etiologic effect, although this may not be true for slower-growing tumors.

III.B.1.4. Definition of Cases

The constitution of case groups has differed across studies, in some instances in clear and logically defined ways. For example, cases may be restricted to malignant or benign tumors or defined by histologic grade or anatomic location to create the subgroup of interest. Comparison of results across studies is challenging when the diagnostic groups are overlapping but not entirely consistent. Also, the varying ways of handling attrition from the target case group of interest - eg losses due to death, inability to provide exposure or covariate information, and refusal - can be problematic methodologically.

III.B.1.5. Selection of Controls

The goal of identifying controls who are a representative sample from the population that gave rise to the cases is straightforward in principle, but it is not easily achieved in practice. For studies that identify cases comprehensively from a geographically-defined population, the desired composition of the control group is clear, although such controls are not necessarily easy to recruit and interview, as shown in two Nordic studies (Auvinen et al 2002; Lonn et al 2005). For hospital-based case-control studies, the health conditions of controls that resulted in their inclusion in the study need to be scrutinized for potential associations with mobile phone use, as seen for example in two US studies (Inskip et al 2001; Muscat et al 2000).

III.B.1.6. Response rates

Reported participation proportions have varied across studies, with inconsistent methods of calculation distorting comparisons (eTable 1). While attrition from the intended study population is fully reported in some studies, incomplete reporting makes assessment of the potential effect of selection difficult in many studies.

The cohort studies and the registry-based case-control study did not require active subject participation, allowing essentially all of the subjects to be included. Other studies required personal contact and the completion of an interview, with lower participation rates. Participation has been highest in the Scandinavian countries, with reported rates above 70% for both cases and controls in Sweden, and generally worse in other countries.

In several studies, there were indications that non-participation was related to exposure status, with mobile phone users more willing to participate than non-users (Vrijheid et al 2009b). To evaluate the potential magnitude of selection bias, most of the study centers of one study (Interphone; mentioned later) sought a short interview with non-participants (Vrijheid et al 2009b). They were able to elicit responses from 57% of control refusers and 41% of case refusers. In all centers, a lower rate of regular mobile phone use was found in controls who refused the full interview (56% overall) compared with controls who were full participants (69%), regardless of whether the study was presented as a “mobile phone” study or not. The same pattern was found for cases: 50% of case refusers were regular mobile phone users, compared with 66% among full participants. Selection bias introduced by non-participation was estimated to cause a downward bias of around 10% in odds ratios for regular mobile phone use (Vrijheid et al 2009b). It is not known if such a bias would be present differentially among various categories of users (eg between regular versus infrequent users).

III.B.1.7. Precision of risk estimates

Precision is a concern in research on rare health outcomes, which applies to all the cancers of interest here. Nonetheless, large numbers of cases have been identified for study through population registries. The other determinant of precision is the prevalence of the exposure, i.e., mobile phone use. The dramatic increase in mobile phone use over the past 20 years has implications for the power of epidemiologic studies to detect an association, with the optimal exposure prevalence for maximum power being 50%. For long-term exposure, which requires early usage given the secular trends, the numbers remain small and result in limited precision of effect estimates.

III.B.2. METHODS OF STUDIES

eTable 1 summarizes the methods of studies to date, conducted in ten countries. Aside from a group of early studies conducted in the US (Inskip et al 2001; Muscat et al 2000; Dreyer et al 1999; Muscat et al 2002; Warren et al 2003) the vast majority of publications have come from Scandinavia. One set of studies within Scandinavia was conducted by Hardell and coworkers: three on brain tumors (Hardell et al 2005a; 2006a; 2002a; 1999) and one each on salivary gland tumors (Hardell et al 2004), non-Hodgkin's lymphoma (Hardell et al 2005b), and testicular cancer (Hardell et al 2007), as well as pooled analyses of two of the brain tumor studies (Hardell et al 2006b;c). In addition, a large number of re-analyses of the brain tumor studies have been published. In this review we have considered the original publications; re-analyses were considered only if they provided relevant information not available in the original publication (Hardell et al 2002b; 2001). A third set of studies was conducted within the Interphone collaboration. Interphone consisted of a series of 16 coordinated case-control studies conducted in 13 countries. While the overall results have not been published, results of several of the national analyses (Lonn et al 2004b; Takebayashi et al 2006; Lonn et al 2005; Christensen et al 2005; Christensen et al 2004; Hepworth et al 2006; Klaeboe et al 2007; Schlehofer et al 2007; Schuz et al 2006a; Takebayashi et al 2008; Sadetzki et al 2008; Lonn et al 2006; Hours et al 2007) and pooled studies from the Nordic countries and UK (Lahkola et al 2007; 2008; Schoemaker et al 2005) have been published and are considered here. A group of independent studies the two Nordic studies (Auvinen 2002; Johansen et al 2001; Schuz et al 2006b) using subscriber data for exposure assessment and one German study (Stang et al 2001) on uveal melanoma-comprise the fourth group.

The tables in this manuscript are organized in the sequence of the preceding paragraph: Early US studies, Hardell studies, Interphone studies, and Subscriber list based studies.

Only two studies have been cohort studies (Dreyer et al 1999; Johansen et al 2001; Schuz et al 2006b) with the rest being case-control studies. All of the studies were limited to adults, although the age ranges varied somewhat. Most of the case-control studies were population-based, except for the US studies, which were hospital-based. Proxies were used to varying degrees for some of the deceased and ill cases (generally less than 10%).

The US Studies and some of the Swedish studies were based on case ascertainment that started as early as 1994, while the Interphone studies ascertained cases from 2000 through 2004. Therefore lifetime exposure prevalence among controls has varied substantially from <10% to 65%. In addition, exposure definitions and methods of categorization (ever/never use of mobile phones; definition of regular, heavy, and long-term use; and the exposure cutpoints) were inconsistent across studies, making direct comparison difficult. Tables III.B.1-5 present all the published original studies, plus published pooled analyses of the two sets of related studies (Hardell, Interphone). Pooled estimates across the overall literature are also presented. There are numerous further papers in the literature that at first sight appear to present different material but are in fact the same data analyzed in different ways or combinations. Figures 1-4 display the key results of the studies graphically. For details about the figures, refer to the footnotes in the corresponding tables.

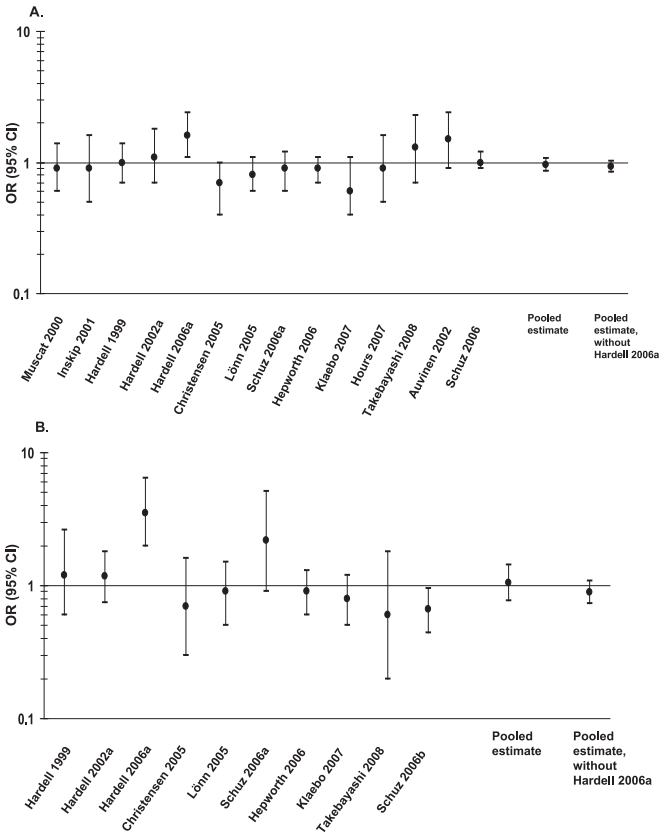

Figure III.B.1. : Mobile phone use and risk of glioma.
A, short-term use (for pooled estimate, *P* for homogeneity = 0.138; without Hardell et al (2006a) *P* = 0.443); B, long-term use (for pooled estimate, *P* for homogeneity = 0.001; without Hardell et al (2006a), *P* = 0.251.

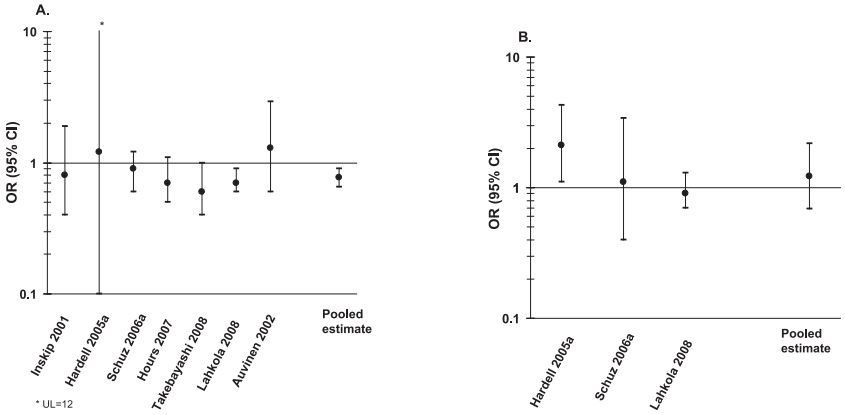

Figure III.B.2. : Mobile phone use and risk of meningioma.
A, short-term use (for pooled estimate, *P* for homogeneity = 0.602); B, long-term use (for pooled estimate, *P* for homogeneity = 0.119). * Upperlimit = 12

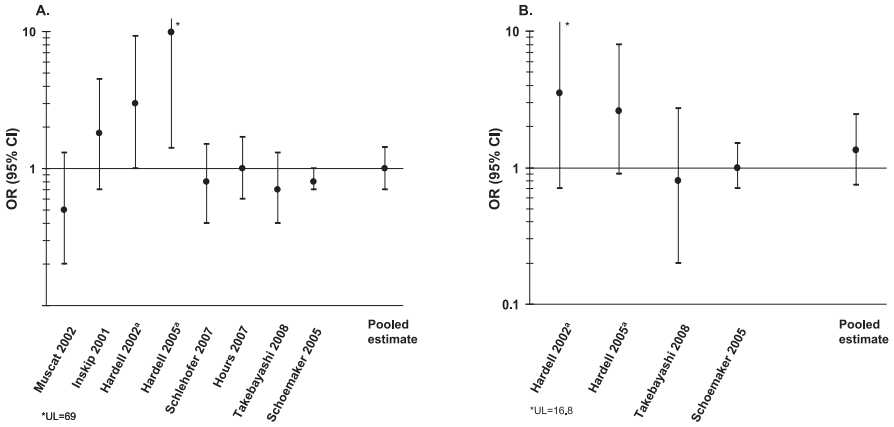

Figure III.B.3. : Mobile phone use and risk of acoustic neuroma.
A, short term use (for pooled estimate, *P* for homogeneity = 0.028); B, long term use (for pooled estimate, *P* for homogeneity = 0.191). *Upperlimit =16.8.

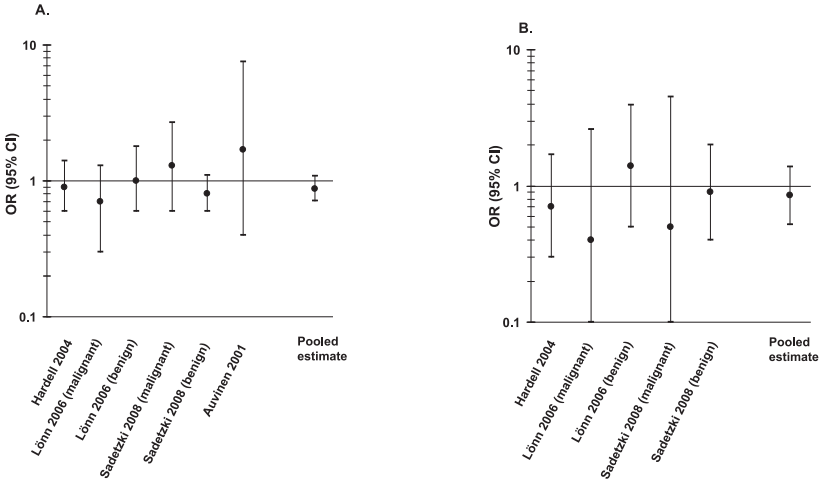

Figure III.B.4. : Mobile phone use and risk of salivary gland tumors.
A, short-term use (for pooled estimate, *P* for homogeneity = 0.667); B, long-term use (for pooled estimate, *P* for homogeneity = 0.743).

In the studies by Hardell, which provide results for both digital and analogue phones, we have chosen to present the analog results in the figures in order to avoid multiple representation and because analog phones give rise to higher exposure levels and were introduced earliest. For the Interphone group of

studies we have chosen the results by Lahkola and Schoemaker instead of the original studies for tumor types (meningioma, acoustic neuroma) where they include data that are not presented in a separate publication.

III.B.3. GLIOMA: RESULTS AND INTERPRETATION

Among the 14 original studies addressing mobile phone use and risk of glioma (Table III.B.1), most found risk estimates close to or below unity with ever-use of mobile phones (Inskip et al 2001; Lonn et al 2005; Muscat et al 2000; Hardell et al 2002a; Hardell et al 1999; Christensen et al 2005; Hepworth et al 2006; Klaeboe et al 2007; Schuz et al 2006a; Takebayashi et al 2008; Hours et al 2007; Schuz et al 2006b), while two did not (Auvinen et al 2002; Hardell et al 2006a). These two studies found risk increases after short-term exposure; Auvinen (2002) found odds ratios (ORs) ranging from 1.2 to 1.7 across indices of mobile phone exposure, with the maximum exposure category (more than 2 years of use) giving an OR of 1.7(95% CI = 0.9-3.5). The most recent study by Hardell (2006a) found increased risks in all categories of time since first use, with an OR of 1.6 (1.1-2.4) within five years based on 100 exposed cases. Hours (2007) found an OR of 2.0 (0.7-5.2) for 3.8 or more years since first use, which was the maximum exposure category analyzed in this French Interphone study. Takebayashi (2008) also reported an elevated OR after intermediate term exposure duration, but found a reduced OR after longer term exposure (more than 6.5 years). Both the Hours and Takebayashi studies included few exposed cases. For at least 10 years since first exposure, Hardell (2006a) found a more than threefold risk increase (OR = 3.6[1.7-7.5] for digital use) and Schuz (2006a) reported a twofold risk increase based on 12 exposed cases (2.2 [0.9-5.1]). Most studies, however, tended to find no evidence for an association based on duration of use or cumulative exposure (Inskip et al 2001; Lonn et al 2005; Muscat et al 2000; Hardell et al 2002a;b; Hardell et al 2001; Christensen et al 2005; Hepworth et al 2006; Klaeboe et al 2007; Schuz et al 2006b). The pooled analysis of Nordic and UK Interphone studies (Lahkola et al 2007), which to date includes the largest number of glioma cases, found an OR of 1.0 (0.7-1.2) based on 143 exposed cases, among persons who started to use a mobile phone 10 or more years prior to diagnosis. Pooling all original studies gave summary risk estimates close to unity in all exposure duration categories (OR = 1.2 [0.9-1.7] for long-term use), as well as for ever-use of mobile phones (1.0 [0.9-1.2]) (Table III.B.1). A sensitivity analysis shows that if the third Hardell et al (2006a) study were excluded, the long-term pooled OR would be 0.9 (0.8-1.1) and the heterogenity across studies would vanish (p= 0.21). This could not be achieved by, for example, excluding the Interphone studies.

Laterality of phone use in relation to laterality of tumor is a potentially important aspect of study results, but, as discussed above, there are methodologic problems with this approach. In particular, if the ipsilateral risk is raised without a raised overall risk, biased recall of side of use is implicated. Similarly, an increased ipsilateral risk together with a decreased contralateral risk also suggests that recall bias operates. This pattern is commonly found in the laterality results presented in Table III.B.2.

Lobe-specific results did not differ substantially from the corresponding overall results (Inskip et al 2001; Auvinen et al 2002; Lonn et al 2005; Muscat et al 2000; Hardell et al 2006a; Hardell et al 2002a; Schuz et al 2006b).

The overall pattern of results does not support the presence of an association between mobile telephone use and glioma. However, two issues call for clarification: (1) the basis for the discrepancy between the predominantly null findings and the few studies suggesting a positive association and (2) the tendency for studies not finding an association to report relative risks for ever-use slightly below the null value rather than dispersed symmetrically around it.

Non-differential exposure misclassification could in principle produce these negative results even in the presence of a causal effect. Might the few positive studies have resulted from a markedly superior assessment of exposure compared with studies by other investigators? The studies by Hardell et al. differed most notably in considering wireless phones in homes (DECT phones) in addition to mobile telephones (2002b; 2006a-c; 2007). However, the association between DECT phone use and glioma risk was investigated by the Swedish and German Interphone studies (Lonn et al 2005; Schuz et al 2006a;c),

without finding an increased risk of glioma. The exposure assessment methods of Auvinen et al (2002) are similar to the ones used in Schuz et al (2006b), and the methods of Schuz et al (2006a) and Hours et al (2007) are indistinguishable from those of other Interphone studies. Another potential reason for the discrepant results is selection bias through non-response among controls who did not use mobile phones, as discussed above. However, selection bias within the Interphone study was estimated to cause a downward bias in risk estimates of approximately 10% (Vrijheid et al 2009b); if this estimate is correct, this source of selection bias does not appear large enough to explain the differences in results.

If the series of negative studies is correct, it is appropriate to consider the potential reasons, including random error, for spurious positive findings in the studies generating positive results. The positive studies do not appear to have structural features with regard to case and control group constitution that would bias associations in a positive direction. The basic approach to exposure assessment does not appear to differ from that of other studies, with most studies based on self-report of use and various derived indices of exposure. While on the surface, the positive studies, including those by Hardell et al., are very much like the studies that obtained quite different results, subtle aspects of data collection and methods of analysis may be responsible for the apparent discrepancies. Investigators must make decisions regarding the exact constitution of the case groups, such as, whether to restrict by anatomic location, histology, stage, or malignancy. Exposure assignment requires even more complex decisions, including analog or digital phone use; how to define regular use; how to categorize hours of use or cumulative exposure; consideration of laterality of use and tumor location; and selection of reference dates of use for controls in relation to the timing of disease diagnosis. There is potential for differing recruitment methods to affect the magnitude and pattern of non-response, for interviewer training and monitoring to affect reporting tendencies of cases and controls, and even for the wording of questions to have subtle effects on the resulting data. Every team of investigators faces these decisions, and, presuming that there are compensating practices, the series of studies in the literature overall is expected to converge on a valid result. These decisions represent a major reason why replication of results by different research groups is needed before results can be considered as established.

The studies by Hardell and colleagues are particularly problematic because of variation across their publications in the exact constitution of case groups, criteria for exclusion, exposure definitions, and the selection of results for presentation in the multiple overlapping publications. In our view, the series of decisions in methods, analysis, and presentation provide the most plausible explanation for the deviation of the findings of the Hardell studies from those of other investigators. This does not address the other positive reports, but they seem to fit more in the distribution of results expected given random error across studies.

In summary, the complete array of available data does not suggest a causal association of mobile phone use with risk of glioma. However, there remains some uncertainty due to inconsistencies across the studies, as well as the recognized problems of exposure misclassification and potential for bias due to selective participation. As discussed previously, non-participation in the Interphone studies has been estimated to result in a 10% downward bias of the odds ratios, which can not explain all of the observed risk reduction. In addition, the period between exposure to a causal agent and manifestation of glioma may range from 5 to 20 years or more, judging from the intervals observed between ionizing radiation exposure and tumor diagnosis. Symptoms depend on the site and nature of the tumor, with slowest onset for low-grade tumors and rapid onset for highly malignant and swiftly-growing tumors. The data for long-term phone use of more than 10 years are still sparse, and any increased risk of slow-growing tumors may not yet have become manifest.

III.B.4. MENINGIOMA: RESULTS AND INTERPRETATION

Eleven original case-control studies (Inskip et al 2001; Auvinen et al 2002; Lonn et al 2005; Hardell et al 2005a; 2002a; 1999; Christensen et al 2005; Klaeboe et al 2007; Schuz et al 2006a; Takebayashi et al 2008; Hours et al 2007), one cohort study (Johansen et al 2001; Schuz et al 2006b), and two pooled analyses (Hardell et al 2006c; Lahkola et al 2008) have investigated the association between mobile phone use and meningioma. With the exception of the most recent study by Hardell (2005a), all studies

found risk estimates close to or below unity, regardless of time since first mobile phone use (Table III.B.3). The study by Hardell (2005a) found an increased risk with ever-use of an analog mobile phone (OR = 1.7 [1.0-3.0]), with the highest risk estimate for more than 10 years since first use (2.1 [1.1-4.3]). The largest study so far- the pooled analysis of the Nordic and UK Interphone studies - found an OR of 0.9 (0.7-1.3) for long term use. Pooling all original studies gave risk estimates close to or below unity (Table III.B.3). Thus, there is no consistent evidence of an increased risk of meningioma among mobile phone users.

Many of the methodologic concerns discussed above for glioma apply also to meningioma, since they were typically evaluated within the same epidemiologic studies. A particular consideration in the interpretation of studies of mengioma is the long latency for this disease. Unlike gliomas, meningiomas are typically very slow-growing tumors with probable latencies of up to 30 yrs or more (Choudhary et al 2006). Cases may have no symptoms for a long period before detection of their tumor because meningiomas compress rather than invade the brain. A proportion of patients diagnosed with meningiomas in the 1990s and included in early studies could well have had the tumor present prior to any substantive exposure to mobile phones. Thus, the negative results give weaker evidence regarding an absence of association than the corresponding negative results for glioma.

III.B.5. ACOUSTIC NEUROMA: RESULTS AND INTERPRETATION

The 13 original studies of acoustic neuroma (Inskip et al 2001; Lonn et al 2004b; Takeyabashi et al 2006; Muscat et al 2002; Warren et al 2003; Hardell et al 2005a; 2002a; 1999; Christensen et al 2004; Klaeboe et al 2007; Schlehofer et al 2007; Hours et al 2007; Johansen et al 2001; Schuz et al 2006b) (Table III.B.4) generally included small numbers of cases. The pooled analyses are larger (Hardell et al 2006c; Schoemaker et al 2005), especially the Nordic-UK pooled analysis (Schoemaker et al 2005). Response rates for cases have been relatively high, reflecting the benign nature of this tumor, but control response rates have generally been lower. For ever-use of a mobile phone, all studies found risk estimates close to or below unity, except the two most recent studies by Hardell et al (2005a; 2002a), where up to fourfold risk increases were reported. It is notable that Hardell et al (2005a; 2002a; 2006c) observed considerably increased risks also within a short time period since first use. Acoustic neuroma is a very slow-growing tumor (Thomsen et al 1990) and it seems likely that the majority of cases diagnosed within five years of their first mobile phone use would have had their tumor already present before they started to use the mobile phone. Two of the US studies (Inskip et al 2001; Muscat et al 2002) also reported somewhat elevated ORs relatively soon after first mobile phone use, but these were based on small numbers of exposed cases (Table III.B.4).

For long durations of exposure (10 years or more), the Nordic-UK pooled analysis included the largest number of cases, and reported an OR of 1.0 (0.7-1.5). Most studies found risk estimates below one, sometimes with a considerable risk reduction (eg Christensen (2004), with an OR of 0.2 [0.2-1.1], although the Swedish Interphone study (Lonn et al 2004b) found an OR of 1.9 (0.9-4-1). The two recent Hardell studies (2005a; 2002a) generated results that are discrepant from the other studies, with increased ORs of 3.5 (0.7-16.8) and 2.6 (0.9-8.0) for long-term analog phone use. Pooling all studies gave summary risk estimates of 1.2 (0.8-2.0) for long-term use, and 1.1 (0.8-1.4) for ever-use. Analyses in relation to cumulative hours of use or cumulative number of calls likewise indicated no clear associations except in one of the Hardell studies (2005a).

The risk of acoustic neuroma after reported regular ipsilateral phone use was not increased in the Nordic-UK analysis (OR 0.9 [0.7-1.1]). The same was true in the other datasets (Inskip et al 2001; Lonn et al 2004b; Takebayashi et al 2006; Muscat et al 2002; Klaeboe et al 2007; Hours et al 2007) except one by Hardell (2005a), in which there were ORs of 5.1 (1.9-14) for analog use and 2.9 (1.4-6.1) for digital use. There was, however, a raised risk associated with first ipsilateral phone use at least 10 years prior to diagnosis in the study by Lonn (OR = 3.9 [1.6-9.5]). The corresponding result in the Nordic-UK pooled analysis was 1.3 (0.8-2.0), although a raised risk was associated with at least 10 years of use (OR = 1.8 [1.0-3.3]) (Schoemaker et al 2005). Handedness has not been associated with ipsilateral tumor risk (Schoemaker et al 2005).

Acoustic neuroma can cause unilateral deafness, which could lead to cessation of phone use (and hence spuriously reduced risks). Alternatively, the deafness could lead to the diagnosis of an otherwise unrecognized tumor and hence lead to spuriously increased risks. Hearing loss associated with acoustic neuromas may influence the side of phone use as the tumor progresses, resulting in preferred contralateral phone use relative to the tumor. This is not predictable, however, since hearing can be preserved in the presence of large vestibular schwannomas and, conversely, hearing loss can frequently occur as the result of radiologically static, small tumors (Rutherford et al 2005). Potential effects on the side of mobile phone use or earlier detection of tumors should, however, affect all available studies similarly; this cannot explain the discrepancies in the results.

Unlike the situation for gliomas and meningiomas, laterality virtually defines the anatomical position of acoustic neuromas, and all ipsilateral acoustic neuromas arise close to the mobile phone handset position. Therefore if reliable unbiased information on side of exposure could be obtained, it would be possible to conduct a powerful unbiased analysis of the effect of mobile phone exposure on acoustic neuroma risk. This analysis, however, is hampered by inconsistency in side of phone use, reporting bias resulting from the tumor diagnosis, and the symptom-based changes in use noted above. The results indicating an increased risk associated with ipsilateral phone use but no overall raised risk again raise questions about the contribution of reporting bias. Thus, the elevated ipsilateral risk beyond 10 years in the large Nordic-UK analysis seems more likely to represent reporting bias than a causal effect, because the latter should lead to a raised risk (although diluted) for users overall beyond 10 years - a finding that was not seen in the overall Nordic-UK data.

As was the case for meningioma, acoustic neuromas are often present for years before diagnosis. Thus, the only data about phone use that are of any potential relevance to acoustic neuroma etiology may be the exposure occurring many years before diagnosis. The available data make it unlikely that there is any substantial raised risk of acoustic neuroma in relation to mobile phone use in the ten years preceding the diagnosis of the tumor. The results leave uncertainty as to whether there are raised risks beyond 10 years from initial use.

III.B.6. SALIVARY GLAND TUMORS: RESULTS AND INTERPRETATION

There is no consistent evidence of an increased risk of salivary gland tumors among mobile phone users (Table III.B.5, Fig. III.B.4) based on four case-control studies (Auvinen et al 2002; Hardell et al 2004; Sadetzki et al 2008; Lonn et al 2006) and one cohort study (Schuz 2006b). One study (Auvinen et al 2002) showed an increase in risk for ever-use compared with never-use and for greater cumulative years of exposure, but the results were based on few cases and had very wide confidence intervals. There was no indication of a raised risk in any of the other studies including that of Hardell. Pooling the results from all studies gave risk estimates slightly below unity in all exposure categories (Table III.B.5). Both publications from the Interphone study reported higher risk estimates associated with ipsilateral phone use at least 10 years prior to diagnosis, with an OR of 2.6 (0.9-7.9) in the Lonn study (2006), and 1.6 (0.7-3.7) in the study by Sadetzki et al (2008). Corresponding ORs for contralateral use were, however, considerably reduced in both studies: 0.3 (0.0-2.3) and 0.6 0.2-2.3), respectively. Thus, reporting bias seems likely to explain these findings.

Single studies of tumors at other sites (pituitary adenoma (Takebayashi et al 2008), non-Hodgkin's lymphoma (Hardell et al 2005b), testicular cancer (Hardell et al 2007), uveal melanoma (Stang et al 2001) are not discussed here. The main results for these cancer sites are shown in eTable 2.

III.B.7. CONCLUSIONS

In the last few years the epidemiologic evidence on mobile phone use and risk of brain and other tumors of the head has grown considerably. In our opinion, overall the studies published to date do not demonstrate a raised risk within approximately ten years of use for any tumor of the brain or any other

head tumor. However, some key methodologic problems remain - for example, selective non-response and exposure misclassification. Despite these methodologic shortcomings and the still limited data on long latency and long-term use, the available data do not suggest a causal association between mobile phone use and fast-growing tumors such as malignant glioma in adults, at least those tumors with short induction periods. For slow-growing tumors such as meningioma and acoustic neuroma, as well as for glioma among long-term users, the absence of associations reported thus far is less conclusive because the current observation period is still too short. Currently data are completely lacking on the potential carcinogenic effect of exposures in childhood and adolescence.

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Table III.B.1.: Results of studies on mobile phone use and risk of glioma[a]

	Time since first use						Ever/never use	
	Short-term use		Intermediate-term use		Long-term use			
Reference	**No.**	**OR (95% CI)**	**No.**	**OR (95% CI)**	**No.**	**OR (95% CI)**	**No.**	**OR (95%**
US Studies								
Muscat et al 2000 (Malignant brain)	49	0.9 (0.6-1.4) [b]	17	0.7 (0.4-1.4)			66	0.7 (0.5-1.1)
Inskip et al 2001 (Glioma)	31	0.9 (0.5-1.6)	11	0.5 (0.2-1.3)			201	1.0 (0.7-1.4)
Hardell Studies								
Hardell et al 1999 (All brain)	78	1.0 (0.7-1.4)	34	0.8 (0.5-1.4)	16	1.2 (0.6-2.6)	78	1.0 (0.7-1.4)
Hardell et al 2002a,b (All	36 (analog)	1.1 (0.7-1.8)			43 (analog)	1.2 (0.8-1.8)	79 analog	1.1 (0.8-1.6)
Hardell et al 2006a (All malignant)	0 (analog)	-	20 (analog)	1.8 (0.9-3.5)	48 (analog)	3.5 (2.0-6.4)	68 analog	2.6 (1.5-4.3)
Hardell pooled analysis								
Hardell et al 2006b[c] (All malignant)	39 (analog)	1.2 (0.8-1.8)	57 (analog)	1.1 (0.8-1.6)	82 (analog)	2.4 (1.6-3.4)	178 analog	1.5 (1.1-1.9)
Interphone Studies								
Christensen et al 2005 (Glioma)[d]	43	0.7 (0.4-1.0)	42	0.6 (0.4-1.0)	14	0.7 (0.3-1.6)	106	0.7 (0.5-1.0)
Lonn et al 2005 (Glioma)	112	0.8 (0.6-1.1)	75	0.7 (0.5-1.0)	25	0.9 (0.5-1.5)	214	0.8 (0.6-1.0)
Schuz et al 2006a (Glioma)	82	0.9 (0.6-1.2)	39	1.0 (0.6-1.5)	12	2.2 (0.9-5.1)	138	1.0 (0.7-1.3)
Hepworth et al 2006 (Glioma)	271	0.9 (0.7-1.1)	170	1.0 (0.8-1.3)	66	0.9 (0.6-1.3)	508	0.9 (0.8-1.1)
Klaeboe et al 2007 (Glioma)	27	0.6 (0.4-1.1)	64	0.5 (0.3-0.8)	70	0.8 (0.5-1.2)	161	0.6 (0.4-0.9)
Hours et al 2007 (Glioma)	38	0.9 (0.5-1.6) [e]	21	2.0 (0.7-5.2)			59	1.2 (0.7-2.1)
Takebayashi et al 2008 (Glioma)	32	1.3 (0.7-2.3) [f]	17	1.9 (0.8-4.4)	7	0.6 (0.2-1.8)	56	1.2 (0.6-2.4)
Interphone pooled analysis								
Lahkola et al 2007[g] (Glioma)	384	0.8 (0.7-0.9)	342	0.8 (0.6-0.9)	143	1.0 (0.7-1.2)	867	0.8 (0.7-0.9)
Subscriber list Studies								
Auvinen et al 2002 (Glioma)	25	1.5 (0.9-2.4)[h]	11	1.7 (0.9-3.5)			36	1.5 (1.0-2.4)
Schuz et al 2006b (Nervous system)	266	1.0 (0.9-1.2)	235	1.0 (0.8-1.1)	28	0.7 (0.4-1.0)	580	1.0 (0.9-1.0)
Pooling all studies[i]		1.0 (0.9-1.1)		0.9 (0.8-1.1)		1.1 (0.8-1.4)		1.0 (0.8-1.2)
P for homogeneity		0.138		0.010		0.001		0.001

[a] All studies are case-control studies except Schuz et al 2006b
[b] Pooled result for 1 year and 2-3 years
[c] Data from Hardell 2002b and 2006a
[d] Pooled results for low grade and high grade glioma
[e] Pooled result for <1.3 years, 1.3-2.25 years, and 2.25-3.8 years
[f] Pooled result for <2.2 years and 2.2-4.7 years
[g] Data from Christensen 2005, Lonn 2005, Klaebo 2007, part of Hepworth 2006, and data from Finland not previously published
[h] Pooled result for <1 year and 1-2 years
[i] Pooling all studies except Hardell 2006b and Lahkola 2007, using the random effects model. From Hardell 2002 and 2006, when results for both analogue and digital phone use were available, only the results for analogue phone use were included to avoid including duplicate data.

OR indicates odds ratio; CI, confidence interval.

Table III.B.2.: Results of laterality analyses in studies on mobile phone use and risk of glioma

	Ever/never use		≥10 years since first use		
	Ipsilateral	Contralateral	Ipsilateral	Contralateral	
Reference	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	Comment
Hardell et al 1999/2001	1.1 (0.6-1.8)	0.7 (0.4-1.2)			
Hardell et al 2002a,b	1.9 (1.2-3.0)	0.6 (0.4-1.1)	1.8 (1.0-3.4)[a]	0.7 (0.4-1.6)[a]	Analog
	1.6 (1.1-2.4)	0.9 (0.5-1.4)	2.3 (0.6-8.9)[a]	0.3 (0.0-2.9)[a]	Digital
Hardell et al 2006a	3.1 (1.6-6.2)	2.6 (1.3-5.4)			Analog
	2.6 (1.6-4.1)	1.3 (0.8-2.2)			Digital
Lonn et al 2005	1.1 (0.8-1.5)	0.7 (0.5-1.0)	1.6 (0.8-3.4)	0.7 (0.3-1.5)	
Hepworth et al 2006	1.2 (1.0-1.5)	0.8 (0.6-0.9)	1.6 (0.9-2.8)	0.8 (0.4-1.4)	
Klaeboe et al 2007	1.0 (0.7-1.4)	0.7 (0.5-1.1)	1.3 (0.8-2.1)[b]	0.8 (0.5-1.4)[b]	
Hours et al 2007	1.2 (0.6-2.4)	1.2 (0.5-2.7)			
Takebayashi et al 2008	1.2 (0.7-2.3)	1.1 (0.6-2.0)			
Lahkola et al 2007	1.1 (1.0-1.3)	0.8 (0.6-0.9)	1.4 (1.0-1.9)	1.0 (0.7-1.4)	

[a] >6 years
[b] ≥6 years

Table III.B.3.: Results of studies on mobile phone use and risk of meningioma[a]

Reference	Time since first use						Ever/never use	
	Short-term use		Intermediate-term use		Long-term use			
	No. exposed cases (exposure period)	OR (95% CI)	No. exposed cases (exposure period)	OR (95% CI)	No. exposed cases (exposure period)	OR (95% CI)	No. exposed cases	OR (95% CI)
US Studies								
Inskip et al 2001	12 (0.5-3 years)	0.8 (0.4-1.9)	6 (≥5 years)	0.9 (0.3-2.7)			67	0.8 (0.5-1.2)
Hardell Studies								
Hardell et al 1999							16	1.1 (0.5-2.3)
Hardell et al 2002a							60 analog	1.1 (0.7-1.5)
							78 digital	0.8 (0.6-1.0)
Hardell et al 2005a	1 (analog)	1.2 (0.1-12)	14 (analog)	1.4 (0.7-2.8)	20 (analog)	2.1 (1.1-4.3)	35 analog	1.7 (1.0–3.0)
	96 (digital) (1-5 years)	1.2 (0.8-1.8)	47 (digital) (6-10 years)	1.4 (0.9-2.3)	8 (digital) (>10 years)	1.5 (0.6-3.9)	151 digital	1.3 (0.9–1.9)
Hardell pooled analysis								
Hardell et al 2006c[b]	32 (analog)	1.2 (0.8-1.8)	47 (analog)	1.2 (0.8-1.8)	34 (analog)	1.6 (1.0-2.5)	113 analog	1.3 (1.0-1.7)
	220 (digital) (1-5 years)	1.0 (0.8-1.3)	67 (digital) (6-10 years)	1.1 (0.8-1.6)	8 (digital) (>10 years)	1.3 (0.5-3.2)	295 digital	1.1 (0.9-1.3)
Interphone Studies								
Christensen et al 2005	35 (1-4 years)	0.8 (0.5-1.3)	21 (5-9 years)	0.7 (0.3-1.2)	6 (≥10 years)	1.0 (0.3-3.2)	67	0.8 (0.5-1.3)
Lonn et al 2005	64 (1-4 years)	0.6 (0.4-0.9)	40 (5-9 years)	0.7 (0.5-1.1)	12 (≥10 years)	0.9 (0.4-1.9)	118	0.7 (0.5-0.9)
Schuz et al 2006a	73 (1-4 years)	0.9 (0.6-1.2)	18 (5-9 years)	0.8 (0.5-1.5)	5 (≥10 years)	1.1 (0.4-3.4)	104	0.8 (0.6-1.1)
Klaeboe et al 2007	19 (<2 years)	0.6 (0.3-1.1)	41 (2-5 years)	0.7 (0.4-1.2)	36 (≥6 years)	1.0 (0.6-1.8)	96	0.8 (0.5-1.1)
Hours et al 2007	56 (<3.8 years)	0.7 (0.5-1.1)[c]	15 (≥3.8 years)	0.7 (0.3-1.9)			71	0.7 (0.4-1.3)
Takebayashi et al 2008	35 (<5.2 years)	0.6 (0.4-1.0)[d]	20 (>5.2 years)	1.1 (0.5-2.1)			55	0.7 (0.4-1.2)
Interphone pooled analysis								
Lahkola et al 2007[e]	286 (1-4 years)	0.7 (0.6-0.9)	214 (5-9 years)	0.8 (0.6-1.0)	73 (≥10 years)	0.9 (0.7-1.3)	573	0.8 (0.7-0.9)
Subscriber list Studies								
Auvinen et al 2002	9 ≤2 years	1.3 (0.6-2.9)[f]	2 >2 years	0.8 (0.2-3.5)			11	1.1 (0.5-2.4)
Schuz et al 2006b[3]							68	0.9 (0.7-1.1)
Pooling all studies[g]		0.8 (0.7-0.9)		0.9 (0.7-1.0)		1.2 (0.7-2.2)		0.9 (0.8-1.0)
P for homogeneity		0.602		0.799		0.119		0.232

[a] All studies are case-control studies except Schuz et al. 2006b
[b] Data from Hardell 2002a and 2005a
[c] Pooled result for <1.3 years, 1.3-2.25 years, and 2.25-3.8 years
[d] Pooled result for <1.6 years, 1.6-3.2 years, and 3.2-5.2 years
[e] Data from Christensen 2005, Lonn 2005, Klaebo 2007, and data from UK and Finland not previously published
[f] Pooled result for <1 year and 1-2 years
[g] Pooling all studies, except Hardell 2006c, Christensen 2005, Lonn 2005, and Klaebo 2007, using random effects model. From Hardell 2005, only the results for analogue phone use were included.

Table III.B.4.: Results of studies on mobile phone use and risk of acoustic neuroma[a]

Reference	Time since first use: Short-term use No. exposed cases (exposure period)	Short-term use OR (95% CI)	Intermediate-term use No. exposed cases (exposure period)	Intermediate-term use OR (95% CI)	Long-term use No. exposed cases (exposure period)	Long-term use OR (95% CI)	Ever/never use No. exposed cases	Ever/never use OR (95% CI)
US Studies								
Muscat et al 2002	7 (1-2 years)	0.5 (0.2-1.3)	11 (3-6 years)	1.7 (0.5-5.1)			18	0.8 (0.4-1.7)[b]
Inskip et al 2001	8 (0.5-3 years)	1.8 (0.7-4.5)	5 (≥5 years)	1.9 (0.6-5.9)			40	0.8 (0.5-1.4)
Warren et al 2003							21	1.2 (0.6-2.2)
Hardell Studies								
Hardell et al 1999							5	0.8 (0.1-4.2)
Hardell et al 2002a	12 (analog)	3.0 (1.0-9.3)	19 (analog)	3.8 (1.4-10.2)	7 (analog)	3.5 (0.7-16.8)	38 analog	3.5 (1.8-6.8)
	21 (digital) (1-5 years)	1.2 (0.6-2.2)	2 (digital) (6-10 years)	2.0 (0.2-22.1)	(>10 years)		23 digital	1.2 (0.7-2.2)
Hardell et al 2005a	2 (analog)	9.9 (1.4-69)	11 (analog)	5.1 (1.9-14)	7 (analog)	2.6 (0.9-8.0)	20 analog	4.2 (1.8-10)
	29 (digital) (1-5 years)	1.7 (0.9-3.5)	23 (digital) (6-10 years)	2.7 (1.3-5.7)	1 (digital) (>10 years)	0.8 (0.1–6.7)	53 digital	2.0 (1.1-3.8)
Hardell pooled analysis								
Hardell et al 2006c[c]	16 (analog)	2.3 (1.2-4.1)	33 (analog)	3.4 (2.1-5.5)	19 (analog)	3.1 (1.7-5.7)	68 analog	2.9 (2.0-4.3)
	75 (digital) (1-5 years)	1.4 (1.0-2.1)	29 (digital) (6-10 years)	1.8 (1.1-3.0)	1 (digital) (>10 years)	0.6 (0.1-5.0)	105 digital	1.5 (1.1-2.1)
Interphone Studies								
Christensen et al 2005	23 (1-4 years)	0.9 (0.5-1.6)	17 (5-9 years)	0.9 (0.4-1.9)	2 (≥10 years)	0.2 (0.0-1.1)	45	0.9 (0.5-1.6)
Lonn et al 2004b	44 (1-4 years)	0.8 (0.5-1.3)	30 (5-9 years)	1.1 (0.6-1.8)	14 (≥10 years)	1.9 (0.9-4.1)	89	1.0 (0.6-1.5)
Schlehofer et al 2007	20 (1-4 years)	0.8 (0.4-1.5)	8 (5-9 years)	0.5 (0.2-1.3)	0 (≥10 years)	-	29	0.7 (0.4-1.2)
Klaeboe et al 2007	4 (<2 years)	0.4 (0.1-1.4)	10 (2-5 years)	0.5 (0.2-1.2)	8 (≥6 years)	0.5 (0.2-1.4)	22	0.5 (0.2-1.0)
Hours et al 2007	44 (<3.8 years)	1.0 (0.6-1.7)[d]	14 (≥3.8 years)	0.7 (0.3-1.6)			58	0.9 (0.5-1.6)
Takebayashi et al 2008	26 (<4 years)	0.7 (0.4-1.3)	21 (4-7 years)	0.8 (0.4-1.5)	7 (≥8 years)	0.8 (0.2-2.7)	51	0.7 (0.4-1.2)
Interphone pooled analysis								
Schoemaker et al 2005[e]	231 (1-4 years)	0.8 (0.7-1.0)	96 (5-9 years)	0.9 (0.7-1.2)	31 (≥10 years)	1.0 (0.7-1.5)	360	0.9 (0.7-1.1)
Subscriber list Studies								
Schuz et al 2006b[f]							32	0.7 (0.5-1.0)
Pooling all studies[g]		1.0 (0.7-1.4)		1.3 (0.8-2.1)		1.4 (0.7-2.5)		1.0 (0.8-1.4)
P for homogeneity		0.028		0.002		0.191		0.000

[a] All studies are case-control studies except Schuz et al. 2006b
[b] Pooling of categorical analyses
[c] Data from Hardell 2002a and 2006a
[d] Pooled result for <1.3 years, 1.3-2.25 years, and 2.25-3.8 years
[e] Data from Christensen 2005, Lonn 2004b, Klaebo 2007, and data from Finland, UK-North and UK-South not previously published
[f] Nerve sheath tumours, cranial nerves
[g] Pooling all studies except Hardell 2006c, Christensen 2005, Lonn 2004b, Klaebo 2007[29], using random effects model. From Hardell 2002 and 2005 only results for analogue phone use were included.

Table III.B.5.: Results of studies on mobile phone use and risk of salivary gland tumors

Reference	Time since first use						Ever/never use	
	Short-term use		Intermediate-term use		Long-term use			
	No. exposed cases (exposure period)	OR (95% CI)	No. exposed cases (exposure period)	OR (95% CI)	No. exposed cases (exposure period))	OR (95% CI)	No. exposed cases	OR (95% CI)
Hardell Studies								
Hardell et al 2004	31 (analog)	0.9 (0.6-1.4)	17 (analog)	0.8 (0.4-1.4)	6 (analog)	0.7 (0.3-1.7)	31 (analog)	0.9 (0.6-1.4)
	45 (digital) >1 year	1.0 (0.7-1.5)	8 (digital) >5 years	1.2 (0.5-2.8)	>10 years		45 (digital)	1.0 (0.7-1.5)
Interphone Studies								
Lonn et al 2006	14 (malignant)	0.7 (0.3-1.3)	8 (malignant)	0.7 (0.3-1.7)	2 (malignant)	0.4 (0.1-2.6)	25 (malignant)	0.7 (0.4-1.3)
	47 (benign) (1-4 years)	1.0 (0.6-1.8)	23 (benign) (5-9 years)	0.8 (0.4-1.5)	7 (benign) (≥10 years)	1.4 (0.5-3.9)		0.9 (0.5-1.5)
Sadetzki et al 2008	21 (malignant)	1.3 (0.6-2.7)	11 (malignant)	0.9 (0.4-2.3)	1 (malignant)	0.5 (0.1-4.5)	33 (malignant) 252 (benign)	1.1 (0.5-2.1)
	335 (benign) (1-4 years)	0.8 (0.6-1.1)	246 (benign) (5-9 years)	1.0 (0.7-1.3)	22 (benign) (≥10 years)	0.9 (0.4-2.0)		0.9 (0.6-1.1)
Subscriber list Studies								
Auvinen et al 2002	3 1-2 years	1.7 (0.4-7.5)	1 >2 years	2.3 (0.2-25.3)			4	1.3 (0.4-4.7)
Schuz et al 2006b							26	0.9 (0.6-1.3)
Pooling all studies[a]		0.9 (0.7-1.1)		0.9 (0.8-1.1)		0.9 (0.5-1.4)		0.9 (0.8-1.1)
P for homogeneity		0.667		0.884		0.743		0.957

[a] Using random effects model. From Hardell 2004, only results for analog phone use were included.

eTable 1. Methods of studies on mobile phone use and tumor risk.

Reference	Geog. Location	Period of case ascertain.	Age Range	Hospital or population based	Tumors considered & whether histol; grade; lobe covered	Reported response rate		% proxy interviews	% exposed among controls	Comments
						Cases	Controls			
US Studies										
Dreyer et al. 1999	USA	1994	20+	Population	Total mortality					Cohort based on operator data. Follow up to Rothman 1996. Subjects using handheld phones compared to subjects using bag phones.
Muscat et al. 2000 Muscat et al. 2002	USA	1994-1998	18-80	Hospital	Malignant brain tumours (ICD9-CM codes 191.0-191.9) Lobe Grade Acoustic neuroma	75%	90%	9% brain tumor cases 1.1% AN cases 1.4% controls	18% brain tumor controls 27% AN controls (ever had a mobile phone subscription)	Data collection through personal interviews.
Inskip et al. 2001	USA	1994-1998	18+	Hospital	Glioma, Meningioma, Acoustic neuroma Grade Lobe (ICD & morph-ology codes in paper)	92%	86%	16% glioma 8% meningiom a 3% AN 3% controls	22% (regular use, i.e. at least twice per week) 45% (ever use)	Data collection through personal interviews.
Warren et al. 2003	USA	1995-2000	Not stated	Hospital	Facial nerve Acoustic neuroma	Not stated	Not stated	0%	38% (ever use)	Data collection through telephone interviews.

Hardell Studies

Hardell et al. 1999	Sweden	1994-1996	20-80	Population	All brain tumors Malign+Benign Acoustic neuroma Grade Lobe	90%	91%	Deaths excluded	38% (at least 8 hours of use)	Response proportions exclude deaths, physician refusals from denominator in all Hardell studies. Data collection through postal questionnaires.
Hardell et al. 2002[a,b]	Sweden	1997-2000	20-80	Population	All brain tumors Malign+Benign Acoustic neuroma Grade Lobe	88%	91%	Deaths excluded	Analogue 15% Digital 30% Cordless 27% (ever use)	Repeated interviews of selected subjects; Data collection through postal questionnaires.
Hardell et al. 2005[a] Hardell et al. 2006[a]	Sweden	2000-2003	20-80	Population	Benign brain tumors Acoustic neuroma Meningioma Lobes Malignant brain tumors High grade astrocytomas	89% benign 88% malign	84%	Deaths excluded	Analogue 11% Digital 50% Cordless 44% Any type 66% (ever use)	Data collection through postal questionnaires.
Hardell et al. 2004	Sweden	1994-2000	21-80	Population	Salivary gland Also by localization and histopathology	91%	90%	Deaths excluded	Analogue 13% Digital 16% Cordless 19% Any type 33% (ever use)	Majority of controls from a 2002 brain tumor study Data collection through postal questionnaires.
Hardell et al. 2005[b]	Sweden	1999-2002	18-74	Population	Non Hodgkin Lymphoma B-cell T-cell (further subdivided) Other	91%	92%	Deaths excluded	Analogue 18% Digital 55% Cordless 41% Any type 68% (ever use)	Controls recruited on "several occasions", 30 cases excluded after ascertainment of exposure as NLH not confirmed Data collection through

											postal questionnaires.
Hardell et al. 2007	Sweden	1993-1997	20-75	Population	Testicular cancer Seminoma Non-seminoma	91%	89%	Deaths excluded	Analogue 20% Digital 16% Cordless 19% (ever use)		Data collection through postal questionnaires.
Hardell pooled analyses											
Hardell et al. 2006c Hardell et al. 2006[b]	Sweden	1997-2003	20-80	Population	Benign brain tumors Acoustic neuroma Meningioma Malignant brain tumors High grade astrocytomas			Deaths excluded			Pooled analysis of Hardell 2002[17] and 2005[15]/2006[16] No heterogeneity tests reported
Interphone studies											
Christensen et al. 2004 Christensen et al. 2005	Denmark	2000-2002	20-69	Population	Acoustic neuroma Meningioma Glioma Low grade, High grade (ICD & morph-ology codes in paper)	82% 71% 74%	64%	0% 2% 8%	46% 42% 50% (regular use=at least once per week during 6 months or more)		Proportion of regular use among controls reflects age and sex distribution of cases. Data collection through personal interviews.
Lonn et al. 2004[b] Lonn et al. 2005	Sweden	1999-2002 2000-2002	20-69	Population	Acoustic neuroma Meningioma Glioma Low grade, High grade Lobes (ICD & morph-ology codes in paper)	93% 84% 75%	72% 71%	0% 3% 9%	59% (regular use)		Data collection through personal interviews.

Schlehofer et al. 2007 Schuz et al. 2006[a]	Germany	2001-2003	30-69	Population	Acoustic neuroma Meningioma Glioma Low grade, High grade	89% 88% 80%	55% in AN study 63% in brain tumor study	0% 1% 11%	38% 37% 39% (regular use)	Data collection through personal interviews.
Klaeboe et al. 2007	Norway	2001-2002	19-69	Population	Acoustic neuroma Meningioma Glioma	68% 71% 77%	69%	0% 0% 36%	63% (regular use)	Data collection through personal interviews. Large proportion of interviews was made over the phone.
Takebayashi et al. 2006 Takebayashi et al. 2008	Japan	2000-2004	30-69	Hospital cases Population controls	Acoustic neuroma Meningioma Glioma Pituitary adenoma (ICD & morph-ology codes in paper)	84% 78% 59% 76%	52% 52% 53% 49%	0%	58% 52% 65% 65% (regular use)	Some hospitals did not participate. Controls selected through random digit dialing. Data collection through personal interviews.
Hepworth et al. 2006	UK	2000-2004	18-69	Population	Glioma Low grade, High grade (ICD & morph-ology codes in paper)	51%	45%	7% cases	52% (regular use)	Study includes data from two centers in the UK. Data collection through personal interviews.
Hours et al. 2007	France	2001-2003	30-59	Hospital cases Population controls	Meningioma Glioma AN	60% 78% 81%	75%	4% cases	56% (regular use)	Some hospitals did not participate. Data collection through personal interviews.
Lonn et al. 2006	Denmark and Sweden	2000-2002	20-69	Population (malignant cases and all controls) Hospital	Malignant parotid gland Benign pleomorphic adenoma	85% (Malig n) 88% (Benig n)	60% (Denmar k) 72% (Sweden)	1 Malign case in Sweden	60% (regular use)	Matched controls in Denmark Results presented for two countries combined Data collection through

				cases (benign cases)								personal interviews.
Sadetzki et al. 2008	Israel	2001-2003	18-59	Population	Malignant and benign parotid gland histologically or cytologically confirmed	84% (Malign) 87% (Benign)	66%	4% cases 0.1 % controls	55% (regular use)			Data collection through personal interviews.
Interphone pooled analyses												
Schoemaker et al. 2005	Denmark Finland Norway Sweden UK-South UK-North	1999-2004	18-69	Population	Acoustic neuroma	83% (69-91)	51% (42-69)	0%	54% (regular use)			Age range varied by country. Pooled analysis of six Interphone studies, includes Christensen 2004, Lonn 2004, Klaeboe 2007, and data from Finland and the UK not previously published
Lahkola et al. 2007 Lahkola et al. 2008	Denmark Finland Norway Sweden UK-South	2000-2004	18-69	Population	Meningioma Glioma Glioblastoma (ICD & morphology codes in paper)	60% (37-81) glioma 74% (55-90) meningioma	50% (42-69)	12% of glioma cases 1.6% of meningioma 0.1% of controls	59% (regular use) 92% (ever use)			Pooled analysis of five Interphone studies, includes Christensen 2005, Lonn 2005 and Klaeboe 2007, part of Hepworth 2006, and data from Finland not previously published
Subscriber list based studies and Other Studies												
Auvinen et al. 2002	Finland	1996	20-69	Population	All BT Glioma,				11% (ever had a			Exposure assessment based on operator data

					Meningioma, Salivary gland Microscop Lobe				mobile phone subscription)	in case-control design
Schuz et al. 2006[b] Johansen et al. 2001	Denmark	1982-2002	18-	Population	All cancer Brain Meningioma Glioma Acoustic neuroma Salivary gland Eye, leukemia, testis Lobes				Cohort study	Exposed cohort: mobile phone subscribers. Compared to sex-, age-, and calendar year specific cancer incidence in the general Danish population. Originally published by Johansen 2001, longer follow-up analysed in Schuz 2006.
Stang et al. 2001	Germany	1995-1998	35-74	Part-hospital Part-pop.	Uveal melanoma	84% pop. 88% hosp.	48% pop. 79% hosp.	0%	<10%	

eTable 2. Results of studies on mobile phone use and risk of glioma

Reference	Diagnostic group	No. cases ever/never user	No. controls ever/never user	OR ever* *cf* never (95% CI) user	OR (95% CI) for max yrs exp. (cut point)	OR (95% CI) for max cumulative exposure	OR for ever-analogue use OR (95% CI)	Laterality (ever/never) ipsi/contra
US Studies								
Dreyer et al. 1999	Brain	2/4		No excess Too small numbers for analysis				
Muscat et al. 2000	Non-meningioma brain (mainly malignant) Astrocytic	66/403 41/313	76/346	0.7 (0.5-1.1) 0.8 (0.5-1.2)	0.7 (0.4-1.4) (≥4)	0.7 (0.3-1.4) (>480 hrs)		
Inskip et al. 2001	Glioma	201/285	358/440	1.0 (0.7-1.4)	0.6 (0.3-1.4) (≥5)	0.5 (0.2-1.3) (>500h)		Inskip method: RR=0.9, p=0.77
Hardell Studies								
Hardell et al. 1999/2001	All brain Astrocyt/glioblast	78/131 36/58	161/264	1.0 (0.7-1.4) 1.1 (0.6-1.8)	1.2 (0.6-2.6) (>10)	1.1 (0.3-3.4) (>968h)	0.9 (0.6-1.4)	1.1 (0.6-1.8)/ 0.7 (0.4-1.2)
Hardell et al. 2002[a,b]	All malignant Astrocytoma low grade Astrocytoma high grade	79 analogue 112 digital 12 analogue 16 digital 46 analogue 64 digital ? unexposed	70 analogue 99 digital 8 analogue 19 digital 37 analogue 52 digital ? unexposed	1.1 (0.8-1.6) 1.1 (0.9-1.5) 1.2 (0.8-1.9) 1.2 (0.8-1.8) 1.5 (0.6-3.7) 0.8 (0.4-1.6)	1.2 (0.8-1.8) 1.7 (0.7-4.3) (>6)		1.1 (0.8-1.6)	1.9 (1.2-3.0)/ 0.6 (0.4-1.1) (Analogue) 1.6 (1.1-2.4)/ 0.9 (0.5-1.4) (Digital)
Hardell et al. 2006[a]	All malignant	68 analogue	79 analogue	2.6 (1.5-4.3)	3.5 (2.0-6.4)	4.0 (2.2-7.3)	2.6 (1.5-4.3)	3.1 (1.6-6.2)/

		198 digital 63 unexp.	343 digital ? unexp.	1.9 (1.3-2.7)	3.6 (1.7-7.5)	2.4 (1.6-3.7)		2.6 (1.3-5.4)
	Astrocytoma low grade	5 analogue 24 digital 7 unexposed		1.2 (0.3-4.9) 1.4 (0.5-3.8)	1.2 (0.2-7.7) 1.5 (0.1-15)	1.4 (0.3-7.2) 1.8 (0.6-5.5)	1.2 (0.3-4.9)	2.3 (0.4-14)/ 0.3 (0.0-3.7)
	Astrocytoma high grade	52 analogue 129 digital 43 unexposed		3.6 (1.9-6.5) 2.2 (1.4-3.3)	4.7 (2.4-9.2) 4.5 (2.0-10) (>10)	5.7 (2.8-11) 2.7 (1.6-4.5) (>80h analogue >64h digital)	3.6 (1.9-6.5)	4.2 (1.9-9.4)/ 5.4 (2.2-13) (Analogue, similar pattern for digital)
Hardell pooled analysis								
Hardell et al. 2006[b]	All malignant	178 analogue 402 digital 322 unexp.	297 analogue 776 digital ? unexp.	1.5 (1.1-1.9) 1.3 (1.1-1.6)	2.4 (1.6-3.4) 2.8 (1.4-5.7)	5.9 (2.5-14) 3.7 (1.7-7.7) (>2000h)	1.5 (1.1-1.9)	2.1 (1.5-2.9)/ 1.1 (0.8-1.6)
	Astrocytoma low grade	5 analogue 24 digital 7 unexposed		1.2 (0.6-2.2) 1.4 (0.9-2.3)	1.6 (0.6-4.1) 1.3 (0.2-11)		1.2 (0.6-2.2)	1.8 (1.4-2.4)/ 1.0 (0.7-1.3)
	Astrocytoma high grade	52 analogue 129 digital 43 unexposed		1.7 (1.3-2.3) 1.5 (1.2-1.9)	2.7 (1.8-4.2) 3.8 (1.8-8.1) (>10)		1.7 (1.3-2.3)	(Analogue, similar pattern for digital)
Interphone Studies								
Christensen et al. 2005	Low grade glioma High grade glioma	47/34 59/112	90/65 155/175	1.1 (0.6-2.0) 0.6 (0.4-0.9)	1.6 (0.4-6.1) 0.5 (0.2-1.3) (≥10)	1.2 (0.5-3.1) 0.5 (0.3-1.1) (>467.9h)		
Lonn et al. 2005	Glioma Low grade glioma High grade glioma	214/157 44//29 155/117	399/275	0.8 (0.6-1.0) 0.6 (0.3-1.0) 0.9 (0.6-1.2)	0.9 (0.5-1.5) 1.0 (0.4-2.8) 0.8 (0.4-1.5) (≥10)	0.6 (0.4-1.0) 0.5 (0.2-1.1) 0.7 (0.4-1.1) (≥500h,	0.8 (0.5-1.2)	1.1 (0.8-1.5)/ 0.7 (0.5-1.0)

							handsfree adjusted)		
Schuz et al. 2006[a]	Glioma	138/ 228	283/449	1.0 (0.7-1.3)	2.2 (0.9-5.1) (≥10)	1.0 (0.6-1.6) (>195h)			
Hepworth et al. 2006	Glioma	508/456	898/818	0.9 (0.8-1.1)	0.9 (0.6-1.3) (≥10)	0.9 (0.7-1.2) (>544h)	0.9 (0.7-1.2)	1.2 (1.0-1.5)/ 0.8 (0.6-0.9)	
Klaeboe et al. 2007	Glioma	161/128	227/131	0.6 (0.4-0.9)	0.8 (0.5-1.2) (≥6)	0.7 (0.4-1.3) (≥425h, handsfree adjusted)	0.7 (0.4-1.1)	1.0 (0.7-1.4)/ 0.7 (0.5-1.1)	
Hours et al. 2007	Glioma	59/37	54/42	1.2 (0.7-2.1)	2.0 (0.7-5.2) (≥3.8)	1.8 (0.7-4.3) (≥260h)		1.2 (0.6-2.4)/ 1.2 (0.5-2.7)	
Takebayashi et al. 2008	Glioma	56/27	106/57	1.2 (0.6-2.4)	0.6 (0.2-1.8) (>6.5)	1.7 (0.7-4.3) (>620h)	0.8 (0.2-3.0)	1.2 (0.7-2.3)/ 1.1 (0.6-2.0)	
Interphone pooled analysis									
Lahkola et al. 2007	Glioma Glioblastoma	867/629 368/330	1853/1281	0.8 (0.7-0.9) 0.8 (0.6-0.9)	0.95 (0.7-1.2) 0.9 (0.6-1.2) (≥10)	0.9 (0.7-1.1) 0.9 (0.6-1.1) (>503h, handsfree adjusted)	0.9 (0.7-1.1)	1.1 (1.0-1.3)/ 0.8 (0.6-0.9)	
Subscriber list based Studies									
Auvinen et al. 2002	Glioma	36/360	119/1859	1.5 (1.0-2.4)	1.7 (0.9-3.5) (>2)		2.1 (1.3-3.4)		
Schuz et al. 2006[b]	Nervous system Glioma	580 257		1.0 (0.9-1.0) 1.0 (0.9–1.1)	0.7 (0.4-1.0) (≥10)				

eTable 3. Results of studies on mobile phone use and risk of meningioma

Reference	Diagnostic group	No. cases ever/never user	No. controls ever/never user	OR ever* *cf* never (95% CI) user	OR (95% CI) for max yrs exp. (cut point)	OR (95% CI) for max cumulative exposure	OR for ever-analogue use OR (95% CI)	Laterality (ever/never) ipsi/contra
US Studies								
Inskip et al. 2001	Meningioma	67/130	358/440	0.8 (0.5-1.2)	0.9 (0.3-2.7) (≥5)	0.7 (0.2-2.4) (>500h)		Inskip method: RR=0.9, p=1.0
Hardell Studies								
Hardell et al. 1999	Meningioma	16/30	161/264	1.1 (0.5-2.3)				
Hardell et al. 2002[a]	Meningioma	60 analogue 78 digital ? unexposed	56 analogue 102 digital ? unexposed	1.1 (0.7-1.5) 0.8 (0.6-1.0)			1.1 (0.7-1.5)	
Hardell et al. 2005[a]	Meningioma	35 analogue 151 digital 103 unexposed	79 analogue 343 digital ? unexposed	1.7 (1.0–3.0) 1.3 (0.9–1.9)	2.1 (1.1–4.3) 1.5 (0.6–3.9) (>10)	2.9 (1.1–8.1) 1.5 (0.6–3.9) (>80h analogue >64h digital)	1.7 (1.0–3.0)	1.6 (0.7–3.9)/ 2.6 (1.1–6.0) (analogue) 1.5 (0.9–2.5)/ 1.5 0(.9–2.3) (digital)
Hardell pooled analysis								
Hardell et al. 2006[c]	Meningioma	113 analogue 295 digital 455 unexposed	297 analogue 776 digital ? unexposed.	1.3 (1.0-1.7) 1.1 (0.9-1.3)	1.6 (1.0-2.5) 1.3 (0.5-3.2) (>10)	1.4 (0.5-3.8) 0.7 (0.3-1.4) (>1000h)	1.3 (1.0-1.7)	1.3 (0.9-2.0)/ 1.2 (0.7-1.8) (analogue) 1.4 (1.0-1.8)/ 1.1 (0.8-1.5) (digital)

Interphone Studies								
Christensen et al. 2005	Meningioma	67/108	133/183	0.8 (0.5-1.3)	1.0 (0.3-3.2) (≥10)	0.6 (0.3-1.6) (>467.9h)	0.8 (0.5-1.3)	
Lonn et al. 2005	Meningioma	118/155	399/275	0.7 (0.5-0.9)	0.9 (0.4-1.9) (≥10)	0.7 (0.4-1.2) (≥500h, handsfree adjusted)	0.7 (0.4-1.3)	0.8 (0.5-1.1)/ 0.6 (0.4-0.9)
Schuz et al. 2006[a]	Meningioma	104/277	234/528	0.8 (0.6-1.1)	1.1 (0.4-3.4) (≥10)	1.0 (0.6-1.8) (>195h)		
Klaeboe et al. 2007	Meningioma	96/111	227/131	0.8 (0.5-1-1)	1.0 (0.6-1.8) (≥6)	0.9 (0.4-1.7) (≥425h, handsfree adjusted)	1.2 (0.7-2.3)	0.9 (0.6-1.3)/ 0.9 (0.6-1.3)
Hours et al. 2007	Meningioma	71/74	80/65	0.7 (0.4-1.3)	0.7 (0.3-1.9) (≥3.8)	0.8 (0.3-2.1) (≥260h)		0.9 (0.4-1.8)/ 0.7 (0.3-1.3)
Takebayashi et al. 2008	Meningioma	55/73	118/111	0.7 (0.4-1.2)	1.1 (0.5-2.1) (>5.2)	0.9 (0.4-2.0) (>260h)	1.1 (0.4-3.1)	1.1 (0.7-2.0)/ 0.7 (0.4-1.1)
Interphone pooled analysis								
Lahkola et al. 2007	Meningioma	573/631	1696/1249	0.8 (0.7-0.9)	0.9 (0.7-1.3) (≥10)	0.9 (0.7-1.1) (>514h, handsfree adjusted)	0.8 (0.6-1.0)	0.8 (0.7-1.0)/ 0.7 (0.5-0.8)
Subscriber list Studies								
Auvinen et al. 2002	Meningioma	11/247	48/1238	1.1 (0.5-2.4)	0.8 (0.2-3.5) (>2)		1.5 (0.6-3.5)	
Schuz et al. 2006[b]	Meningioma	68		0.9 (0.7-1.1)				

eTable 4. Results of studies on mobile phone use and risk of acoustic neuroma

Reference	Diagnostic group	No. cases ever/never user	No. controls ever/never user	OR ever* *cf* never (95% CI) user	OR (95% CI) for max yrs exp. (cut point)	OR (95% CI) for max cumulative exposure	OR for ever-analogue use OR (95% CI)	Laterality (ever/never) ipsi/contra
US Studies								
Muscat et al. 2002	AN	18/72	23/63	0.8 (0.4-1.7)*	1.7 (0.5-5.1) (3-6)	0.7 (0.2-2.6) (>60 hrs)		Inskip method: RR=0.9, p=0.07
Inskip et al. 2001	AN	40/56	358/440	0.8 (0.5-1.4)	1.9 (0.6-5.9) (≥5)	1.4 (0.6-3.4) (>100h)		Inskip method: RR=0.9, p=0.63
Warren et al. 2003	AN	21/30	53/88	1.2 (0.6-2.2)				
Hardell Studies								
Hardell et al. 1999	AN	5/8		0.8 (0.1-4.2)				
Hardell et al. 2002[a]	AN	38 analogue 23 digital ? unexposed	11 analogue 19 digital ? unexposed	3.5 (1.8-6.8) 1.2 (0.7-2.2)	3.5 (0.7-16.8) 2.0 (0.2-22.1) (>10 analogue) (>5 digital)		3.5 (1.8-6.8)	
Hardell et al. 2005[a]	AN	20 analogue 53 digital 18 unexp.	79 analogue 343 digital ? unexp.	4.2 (1.8-10) 2.0 (1.1-3.8)	2.6 (0.9-8.0) 2.7 (1.3-5.7) (>10 analogue) (>5-10 digital)	6.0 (2.2-17) 2.5 (1.2-5.2) (>80h analogue >64h digital)	4.2 (1.8-10)	5.1 (1.9-14)/ 4.9 (1.2-21) (analogue) 2.9 (1.4-6.1)/ 1.6 (0.7-3.7) (digital)
Hardell pooled analysis								

Hardell et al. 2006[c]	AN	68 analogue 105 digital 88 unexposed	297 analogue 776 digital ? unexp.	2.9 (2.0-4.3) 1.5 (1.1-2.1)	3.1 (1.7-5.7) 1.8 (1.1-3.0) (>10 analogue) (>5-10 digital)	5.1 (1.9-14) 3.1 (1.5-6.4) (>1000h)	2.9 (2.0-4.3)	3.0 (1.9-5.0)/ 2.4 (1.4-4.2) (analogue) 1.7 (1.1-2.6)/ 1.3 (0.8-2.0) (digital)
Interphone Studies								
Christensen et al. 2005	AN	45/61	97/115	0.9 (0.5-1.6)	0.2 (0.0-1.1) (≥10)	0.7 (0.3-1.7) (>467.9h)	0.3 (0.1-0.8)	
Lonn et al. 2005	AN	89/59	356/248	1.0 (0.6-1.5)	1.9 (0.9-4.1) (≥10)	1.1 (0.6-2.1) (≥450h, handsfree adjusted)	1.6 (0.9-2.8)	1.1 (0.7-1.6)/ 0.9 (0.6-1.4)
Schlehofer et al. 2006	AN	29/68	74/120	0.7 (0.4-1.2)	0.5 (0.2-1.3) (5-9)	0.4 (0.1-1.0) (>195h)		
Klaeboe et al. 2007	AN	22/23	227/131	0.5 (0.2-1.0)	0.5 (0.2-1.4) (≥6)	0.5 (0.2-1.6) (≥425h, handsfree adjusted)	0.8 (0.3-2.2)	0.7 (0.3-1.4)/ 0.9 (0.5-1.9)
Hours et al. 2007	AN	58/51	123/91	0.9 (0.5-1.6)	0.7 (0.3–1.6) (≥3.8)	0.9 (0.4-2.1) (≥260h)		0.6 (0.3-1.2)/ 1.2 (0.6-2.4)
Takebayashi et al. 2006	AN	51/46	192/138	0.7 (0.4-1.2)	0.8 (0.2-2.7) (>8)	0.7 (0.3-1.8) (>900h)	1.2 (0.4-3.8)	0.9 (0.5-1.6)/ 0.9 (0.6-1.6)
Interphone pooled analysis								
Schoemaker et al. 2005	AN	360/316	1934/1612	0.9 (0.7-1.1)	1.0 (0.7-1.5) (≥10)	0.9 (0.7-1.2) (>534h)	0.9 (0.7-1.2)	0.9 (0.7-1.1)/ 1.1 (0.9-1.4)

Subscriber list based Studies							
Schuz et al. 2006[b]	Nerve sheath tumours, cranial nerves	32		0.7 (0.5-1.0)			

* Pooling of categorical analyses

eTable 5. Results of studies on mobile phone use and risk of other tumors

Reference	Diagnostic group	No. cases ever/never user	No. controls ever/never user	OR ever* *cf* never (95% CI) user	OR (95% CI) for max yrs exp. (cut point)	OR (95% CI) for max cumulative exposure	OR for ever-analogue use OR (95% CI)	Laterality (ever/never) ipsi/contra
Hardell Studies								
Hardell et al. 2004	Salivary gland	31 analogue 45 digital ? unexposed	137 analogue 170 digital ? unexposed	0.9 (0.6-1.4) 1.0 (0.7-1.5)	0.7 (0.3-1.7) 1.2 (0.5-2.8) (>10 analogue, >5 digital)		0.9 (0.6-1.4)	
Hardell et al. 2005b	B-cell	141 analogue 422 digital 278 unexposed	178 analogue 559 digital 321 unexposed	0.9 (0.7-1.3) 1.0 (0.8-1.3)	1.0 (0.7-1.4) 1.1 (0.4-3.4)	1.1 (0.7-1.6) 1.1 (0.8-1.5)	0.9 (0.7-1.3)	
	T-cell	14 analogue 31 digital 13 unexposed		1.6 (0.6-3.8) 1.4 (0.7-2.9)	1.5 (0.5-4.3) 3.0 (0.3-34.1) (>10)	1.3 (0.4-3.9) 1.5 (0.6-3.5) (>198h analogue >91h digital)	1.6 (0.6-3.8)	
Hardell et al. 2007	Testicular cancer	175 analogue 164 digital 515 unexposed	173 analogue 137 digital ? unexposed	1.0 (0.8–1.3) 1.1 (0.8–1.5)	2.1 (0.7–6.2) 2.8 (0.8–11) (>10 analogue, >5 digital)	0.7 (0.5–1.0) 0.9 (0.6–1.3) (>160h analogue >182h digital)	1.0 (0.8–1.3)	
Interphone Studies								
Lonn et al. 2006	Parotid, malignant Parotid, benign	25/35 77/35	401/280 202/119	0.7 (0.4-1.3) 0.9 (0.5-1.5)	0.4 (0.1-2.6) 1.4 (0.5-3.9) ≥10	0.6 (0.2-1.8) 1.0 (0.5-2.1) ≥450 hours		1.2 (0.6-2.6)/ 0.5 (0.2-1.1) 1.4 (0.9-2.2)/ 0.7 (0.4-1.1)

Sadetzki et al. 2008	Parotid, malignant Parotid, benign	33/25 252/150	88/106 603/469	1.1 (0.5-2.1) 0.9 (0.6-1.1)	0.5 (0.1-4.5) 0.9 (0.4-2.0) (≥10)	1.2 (0.4-3.5) 1.1 (0.7-1.6) (≥1035 hours)		1.0 (0.8-1.4)/ 0.9 (0.6-1.2) (malign&benign)
Takebayashi et al. 2008	Pituitary adenoma	62/39	105/56	0.9 (0.5-1.6)	0.8 (0.3-1.8) (>7.2)	1.3 (0.6-3.1) (>560h)	0.5 (0.2-1.8)	1.1 (0.7-2.0)/ 0.7 (0.4-1.1)
Subscriber list based Studies and Other Studies								
Auvinen et al. 2002	Salivary gland	4/64	18/322	1.3 (0.4-4.7)	2.3 (0.2-25.3) (>2)		1.0 (0.3-4.0)	
Schuz et al. 2006[b]	Salivary Eye Leukaemia Testis	26 42 351 522		0.9 (0.6-1.3) 1.0 (0.7-1.3)* 1.0 (0.9-1.1)* 1.1 (1.0-1.2)*	1.1 (0.7-1.5) (≥10, Leukemia)			
Stang et al. 2001	Uveal melanoma			4.2 (1.2-14.5) (ever = probable/ certain use at workplace for at least several hours per day!)				

* Pooled results for men and women

IEEE P802.15
Wireless Personal Area Networks

Project	IEEE P802.15 Working Group for Wireless Personal Area Networks (WPANs)	
Title	**Coexistence analysis of IEEE Std 802.15.4 with other IEEE standards and proposed standards**	
Date Submitted	[September, 2010	
Source	[802.15 Working Group] [IEEE 802] []	Voice: [] Fax: [] E-mail: []
Re:	[]	
Abstract	[This document contains the coexistence analysis that was performed forIEEE Std 802.15.4-2003, IEEE Std 802.15.4-2006, IEEE Std 802.15.4a-2007, IEEE Std 802.15.4c-2009 and IEEE Std 802.15.4d-2009. This information was previously an informative annex in the standards, but is now provided as a separate document, as is the convention with current IEEE 802 wireless standards.]	
Purpose	[A coexistence assurance document enable the IEEE 802 LMSC Executive Committee and the IEEE 802 LMSC Coexistence Working Group to determine if a proposed wireless standard has made a reasonable effort to be able to coexist with devices compliant to other IEEE 802 standards in their operating band. Coexistence, however, does not imply that there is no interference. A detailed discussion of coexistence and coexistence methods is found in IEEE Std 802.15.2-2003.]	
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Release	The contributor acknowledges and accepts that this contribution becomes the property of IEEE and may be made publicly available by P802.15.	

Coexistence analysis of IEEE Std 802.15.4 with other IEEE standards and proposed standards

1. Introduction

While not required by this standard, IEEE 802.15.4 devices can be reasonably expected to “coexist,” that is, to operate in proximity to other wireless devices. This annex considers issues regarding coexistence between IEEE 802.15.4 devices and other wireless IEEE-compliant devices. For UWB devices specifically, additional consideration is given to certain non-IEEE standards.

This is the first IEEE 802® standard defining use of the 780 MHz band (779 MHz to 787 MHz) in China and as such coexistence is not a practical issue at this time. However, the two PHYs specified for use in the 780 MHz band use the exact same channel plan; hence they can potentially cause interference to each other. Due to the short duration (burst nature) of IEEE 802.15.4 packets and use of CSMA-CA, coexistence is not considered to be a problem for the two PHYs when they share a common channel. Similar examples of this are shown in 3.

The use of the 950 MHz band (950 MHz to 956 MHz) for LR-WPAN has only been recently allocated by the Japanese Regulatory committee. This is the first IEEE 802® standard defining use of the 950 MHz band (950 MHz to 956 MHz) in Japan and as such coexistence is not a practical issue at this time. However, the two PHYs specified for use in the 950 MHz band can potentially cause interference to each other. The Japanese regulation includes requirements to address coexistence for devices operating in band, e.g., listen before talk, transmission control, and duty cycle restrictions. Together with the short duration (burst nature) of IEEE 802.15.4 packets and the use of CSMA-CA, coexistence is not considered to be a problem for the two PHYs when they share a common channel. Similar examples of this are shown in 3.

1.1 Standards and proposed standards characterized for coexistence

This clause enumerates IEEE-compliant devices that are characterized and the devices that are not characterized for operation in proximity to IEEE 802.15.4 devices.

This standard is specified for operation in the 800 MHz, 900 MHz, and 2400 MHz bands. In the 800/900 MHz bands, there are BPSK, O-QPSK, and ASK PHYs, which can interact with each other. In the 2400 MHz band, there is only an O-QPSK PHY, which can interact with other IEEE 802 wireless devices operating in the 2400 MHz industrial, scientific, and medical (ISM) band.

Standards and proposed standards characterized in this annex for coexistence are as follows:

— IEEE Std 802.11b™-1999 (2400 MHz DSSS)
— IEEE Std 802.15.1™-2002 [2400 MHz frequency hopping spread spectrum (FHSS)]
— IEEE Std 802.15.3-2003 (2400 MHz)

Standards not characterized in this annex for coexistence are as follows:

— IEEE Std 802.11™-2007 , frequency hopping (FH) (2400 MHz FHSS)
— IEEE Std 802.11™-2007, infrared (333 GHz amplitude modulation)
— IEEE Std 802.16™-2004, (2400 MHz OFDM)
— IEEE Std 802.11™-2007, (5 GHz DSSS)

The CSS PHYs for the 2400 MHz ISM band are specified for operation in 14 channels. Channel 0 through channel 13 reside in frequencies from 2412–2484 MHz bands and, therefore, can interact with other IEEE-compliant devices operating in those frequencies.

Standards and proposed standards characterized in this annex for coexistence are as follows:

— IEEE Std 802.11-2007 (ERP)
— IEEE Std 802.11-2007 (2400 MHz DSSS)
— IEEE Std 802.15.1™-2005 [2400 MHz frequency hopping spread spectrum (FHSS)]
— IEEE Std 802.15.3™-2003 (2400 MHz DSSS)
— IEEE Std 802.15.4-2006 (2400 MHz DSSS)
— IEEE Std 802.15.4a-2007 (2400 MHz CSS)

Standards not characterized in this annex for coexistence are as follows:

— IEEE Std 802.11-2007, frequency hopping (FH) (2400 MHz FHSS)
— IEEE Std 802.11-2007, infrared (IR) [333 GHz amplitude modulation (AM)]
— IEEE Std 802.16™-2004 (2400 MHz OFDM)
— IEEE Std 802.11-2007 (5.2 GHz DSSS)

The UWB PHYs for the 250–750 MHz band reside in frequencies that can interact with other IEEE standards in development. UWB PHYs for the 3244–4742 MHz and 5944–10 234 MHz bands can interact with both IEEE-compliant devices and non-IEEE-compliant devices.

Standards and proposed standards characterized in this annex for coexistence are as follows:

— IEEE Std 802.16-2004
— IEEE P802.22
— ECMA 368[1]

1.2 General coexistence issues

This standard provides several mechanisms that enhance coexistence with other wireless devices operating in the 800 MHz, 900 MHz, and 2400 MHz bands. This subclause provides an overview of the mechanisms that are defined in the standard. These mechanisms include

— CCA
— Dynamic channel selection
— Modulation
— ED and LQI
— Low duty cycle

[1]ECMA 368, High Rate Ultra Wideband PHY and MAC Standard (December 2005) (www.ecma-international.org).

— Low transmit power
— Channel alignment
— Neighbor piconet capability

In addition, this standard provides several mechanisms that enhance coexistence of UWB PHYs with other wireless devices operating in the same spectrum.

— UWB modulation with extremely low PSD
— Low duty cycle
— Low transmit power
— Dynamic channel selection
— Coordinated piconet capabilities

These mechanisms are described briefly in the following subclauses.

1.2.1 Clear channel assessment (CCA)

IEEE 802.15.4 PHYs provide the capability to perform CCA in its CSMA-CA mechanism. The PHYs require at least one of the following three CCA methods: ED over a certain threshold, detection of a signal with IEEE 802.15.4 characteristics, or a combination of these methods. Use of the ED option improves coexistence by allowing transmission backoff if the channel is occupied by any device, regardless of the communication protocol it may use.

1.2.2 Modulation

1.2.2.1 2400 MHz band PHY

The 2400 MHz PHY specified for this standard uses a quasi-orthogonal modulation scheme, where each symbol is represented by one of 16 nearly orthogonal PN sequences. This is a power-efficient modulation method that achieves low signal-to-noise ratio (SNR) and signal-to-interference ratio (SIR) requirements at the expense of a signal bandwidth that is significantly larger than the symbol rate. A typical low-cost detector implementation is expected to meet the 1% packet error rate (PER) requirement at SNR values of 5 dB to 6 dB.

Relatively wideband interference, such as IEEE Std 802.11b-1999 and IEEE Std 802.15.3-2003, would appear like white noise to an IEEE 802.15.4 receiver. The detector performance in this case is similar to noise performance, but the overall SIR requirement is 9 dB to 10 dB lower because only a fraction of the IEEE 802.11b or IEEE 802.15.3 signal power falls within the IEEE 802.15.4 receiver bandwidth.

The use of PN sequences to represent each symbol in this standard offers DSSS-like processing gains to interferers whose bandwidth is smaller than the bandwidth of this standard. For example, this processing gain helps to reduce the impact of an IEEE 802.15.1 interferer, whose 20 dB bandwidth is roughly 50% smaller than the bandwidth of this standard. Whereas the SNR requirement is 5 dB to 6 dB for 1% PER in noise, the equivalent SIR requirement for an IEEE 802.15.1 signal centered within the pass band of the IEEE 802.15.4 receiver is only 2 dB.

In terms of interference to others, this standard appears as wideband interference to IEEE Std 802.15.1-2005, and only a fraction (~50%) of the IEEE 802.15.4 signal power falls within the IEEE 802.15.1 receiver bandwidth. Furthermore, due to the bandwidth ratios and to the frequency hopping used in IEEE Std 802.15.1, IEEE 802.15.4 transmissions will interfere with approximately 3 out of the 79 hops, or approximately 4%. To an IEEE 802.11b receiver, this standard looks like a narrowband interferer, and the

processing gain resulting from the spread-spectrum techniques in IEEE Std 802.11b-1999 will help reduce the impact of the IEEE 802.15.4 interferer.

1.2.2.2 800/900 MHz band PHYs

The 800/900 MHz band PHYs specified in this standard each use DSSS modulation. These power-efficient modulation methods achieve low SNR and SIR requirements at the expense of a signal bandwidth that is significantly larger than the symbol rate. A defining feature of systems that use spread spectrum modulation is that they are less likely to cause interference in other devices due to their reduced PSD. For the same reason, spread spectrum devices have some degree of immunity from interfering emitters, making them a good choice for environments where coexistence is an issue.

1.2.2.3 Direct sequence UWB modulation

The UWB PHY specified in this standard uses a UWB direct sequence modulation. This power-efficient modulation method achieves low requirements for signal-to-noise ratio (SNR) and signal-to-interference ratio (SIR) through the use of a signal bandwidth that is significantly larger than the symbol rate. A defining feature of systems that use UWB modulation is that they are less likely to cause interference in other devices due to their reduced PSD. In fact, even the least restrictive regulations for UWB devices today require the emission PSD levels to be at or below the levels allowed for unintentional emissions by other electrical or electronic devices. In some cases, the UWB PSD limits are as much as 35 dB below these same unintentional emissions limits. For the same reason, UWB devices have some degree of immunity from interfering emitters, making them a good choice for environments where coexistence may be an issue.

1.2.3 ED and LQI

The IEEE 802.15.4 PHYs include two measurement functions that indicate the level of interference within an IEEE 802.15.4 channel. The receiver ED measurement is an estimate of the received signal power within an IEEE 802.15.4 channel and is intended for use as part of a channel selection algorithm at the network layer. The LQI measures the received energy level and/or SNR for each received packet. When energy level and SNR data are combined, they can indicate whether a corrupt packet resulted from low signal strength or from high signal strength plus interference.

1.2.4 Low duty cycle

The specifications of this standard are tailored for applications with low power and low data rates (a maximum of 250 kb/s and down to 20 kb/s). Typical applications for IEEE 802.15.4 devices are anticipated to run with low duty cycles (under 1%). This will make IEEE 802.15.4 devices less likely to cause interference to other standards.

In the UWB bands, the data rates have been increased to a nominal mandatory rate of 850 kb/s. Although not designed to provide continuous higher throughputs, the UWB PHY also provides for optional data rates as high as 27 Mb/s. These rates are not designed to support high-rate applications such video transport, but instead are provided to allow devices in close proximity to shorten their transmission duty cycle by as much as a factor of 32 relative to the mandatory rate to further reduce the likelihood that these devices will interfere with or be subject to interference by other devices when conditions allow.

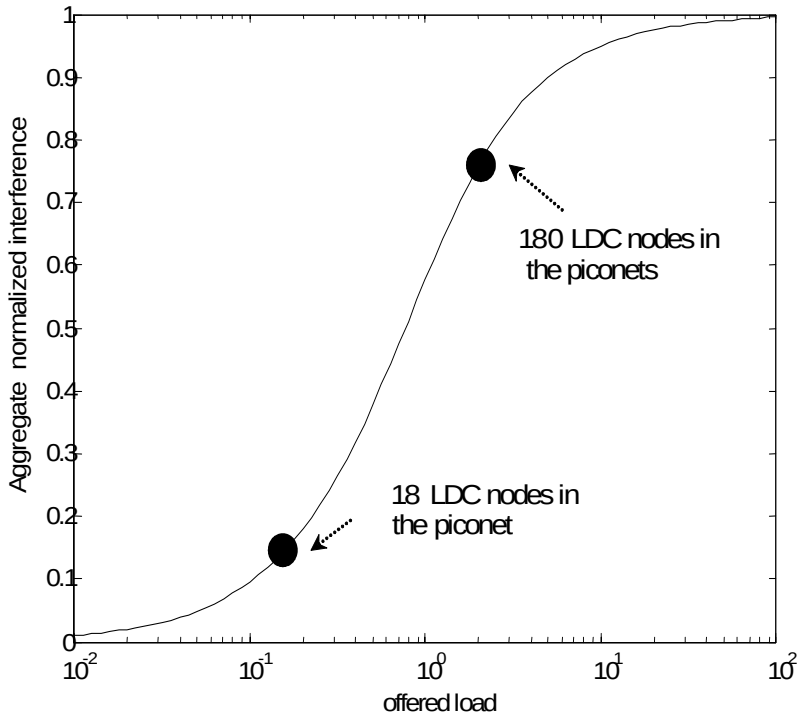
1.2.5 Low-duty-cycle considerations for UWB PHYs

Low-duty-cycle piconet scenarios are used to model the following situations:

— UWB PHY devices are deployed in high density in a limited area, e.g., hot-spot deployment scenarios.

— Some UWB victim systems cover a much larger area than the coverage of a typical UWB PHY piconet, are located well above the local cluster (e.g., IEEE 802.16, radio astronomy service, and satellite service), or are closely located with a piconet coordinator (e.g., devices placed at the same desk or even within the same computer).

In such cases, transmissions from every device in the piconet can affect the victim receiver. For reasons of less complexity, lower power consumption, as well as physical limitations, it is difficult for simple UWB PHY devices to detect victim systems reliably. The aggregate interference from the piconet increases with piconet members. Given 1% average device duty cycle and pure ALOHA protocol, the aggregate interference is 17.6% from a piconet with 18 members, as illustrated in Figure 1. Besides, the channel idle periods are randomly segmented into small pieces. Therefore, it is hard to use the channel effectively. Analyzing the interference in the channel is similar to the collision analysis of a pure ALOHA system.



LDC = low duty cycle

Figure 1—Aggregate normalized interference

The maximal interference level to such kinds of victim systems can be limited by controlling the duty cycle of the piconet through general active/inactive periods, as illustrated in Figure 2. The traffic can occur only in the active period. Victim systems are free of interference in the inactive period. The distribution of active/inactive periods is controlled by the piconet coordinator. This can be implemented by a clock in the application layer. The piconet coordinator defines global time of the piconet and duration of the active period. When a device joins a piconet, it synchronizes its clock with that of coordinator.

The interference level is restricted by the ratio of active period to the total period. The possible packet collision in the active period can be mitigated as follows:

— Adopt CSMA-CA mechanism.

— Adopt channel-dependent ALOHA: The channel-dependent ALOHA is used to set transmission probability related with the channel quality, which can be obtained through listening to a beacon from the coordinator by means of LQI and receiver ED. The function to map channel quality to

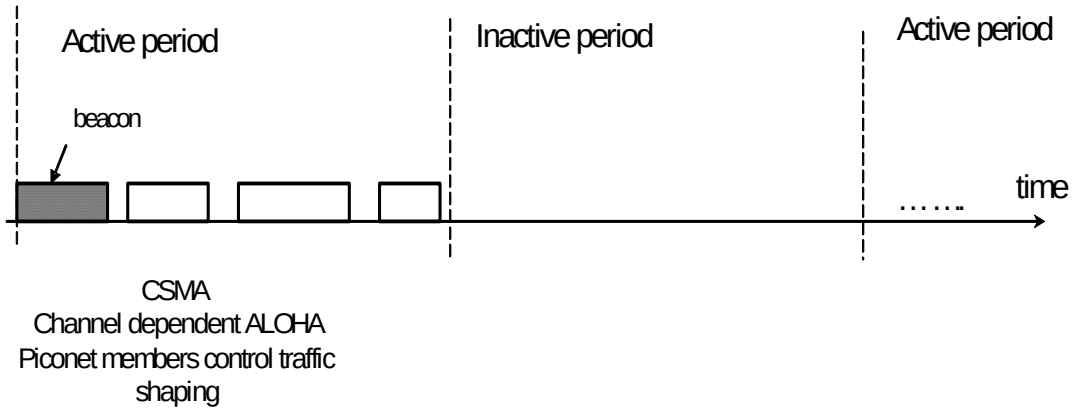


Figure 2—Generalized active/inactive periods

transmission probability is defined at application layer. A simple way is to set a threshold and only enable transmission when the channel quality is above the threshold.

— Limit the number of piconet members through association.

— Use traffic shaping, e.g., a combination of short packet to large packet.

Considering the applications for which the UWB PHY is designed, in application scenarios where a greater number of nodes can be expected, duty cycle (aggregate and individual) can be expected to be orders of magnitude less than the 1% used above. Consider, for example, a sensor application where low-cost sensor nodes are deployed in large number (typically indoors). An individual node may be "awake" only milliseconds per hour. In such scenarios, the aggregate duty cycle would be under the control of the higher layer protocols and very low compared to the 1% used in the above analysis. This observation has two important implications:

— ALOHA is well suited to this application where probability of collision is small and controllable; therefore, the complexity advantage is a good trade-off.

— There is low impact on coexistence due to a large number of IEEE 802.15.4a nodes as the aggregate duty cycle remains very low.

1.2.6 Low transmit power

1.2.6.1 2400 MHz band PHY

Although operation in the 2400 MHz band under Section 15.247 of FCC CFR47 [B5] rules allow transmission powers up to 1 W, IEEE 802.15.4 devices will likely operate with much lower transmit power. A key metric of IEEE Std 802.15.4-2006 is cost, and achieving greater than 10 dBm transmit power in a low-cost system on chip, while feasible, will be economically disadvantageous. Furthermore, European regulations (ETSI EN 300 328 [B3] and [B4]) for out-of-band emissions make it difficult to transmit above 10 dBm without additional, expensive filtering. These factors limit the distribution of devices with greater than 10 dBm transmit power to a few specialized applications.

At the low end, the IEEE 802.15.4 PHY specifies that devices must be capable of at least –3 dBm transmit power. At this level, actual transmit power represents a small fraction of the overall power consumed by the transmitter, so there is little benefit in terms of energy savings to operate below this level. However, this standard does encourage operating with lower transmit power, when possible, to minimize interference.

Thus the majority of IEEE 802.15.4 devices are expected to operate with transmit powers between –3 dBm and 10 dBm, with 0 dBm being typical. IEEE 802.11b devices also operate under Section 15.247 of FCC

CFR47 [B5], where up to 1 W of transmit power is allowed; however, most devices in the market today operate at transmit powers between 12 dBm and 18 dBm. IEEE 802.15.3 devices operate under Section 15.249 of FCC CFR47, which limits transmit power to 8 dBm EIRP. The EIRP measurement for the IEEE 802.15.3 PHY includes the antenna gain; therefore, a 1 dB increase antenna gain requires a 1 dB decrease in transmit power. In contrast, devices operating under Section 15.247 of FCC CFR47 are allowed up to 6 dB of antenna gain without modifications to the transmit power.

Assuming moderate antenna gain (~0 dBi) for typical implementations, the discussion in this subclause implies that a nominal IEEE 802.15.4 transmitter would operate about 8 dB less than the IEEE 802.15.3 transmitter and about 12 dB to 18 dB less than a typical IEEE 802.11b implementation.

1.2.6.2 800 MHz band PHYs

Regulations defined by ERC Recommendation 70-03 [B1] and ETSI EN 300 220-1 [B2] limit transmitter power in the 868 MHz to 25mW (13.9 dBm) maximum. Although devices conforming to IEEE Std 802.15.4-2006 may transmit at this power, the economics of system-on-chip designs will limit the transmit power to around 10 dBm. At the low end, all confirming devices must be capable of at least –3 dBm transmit power. At this power, the transmit power represents a small fraction of the overall power consumed by the device; therefore, there is no significant energy savings for operating below this level. However, this standard does encourage operating with lower power, when possible, in order to minimize interference.

Consequently, it is reasonable to assume that all 868 MHz devices will transmit at a power between –3 dBm and +10 dBm.

1.2.6.3 900 MHz band PHYs

Regulations defined by FCC CFR47 [B5] limit transmitter power in the 868 MHz to 1000 mW (30 dBm) maximum. Although devices conforming to this standard may transmit at this power, the economics of system-on-chip designs will limit the transmit power to around 10 dBm. At the low end, all confirming devices must be capable of at least –3 dBm transmit power. At this power, the transmit power represents a small fraction of the overall power consumed by the device; therefore, there is no significant energy savings for operating below this level. However, this standard does encourage operating with lower power, when possible, in order to minimize interference.

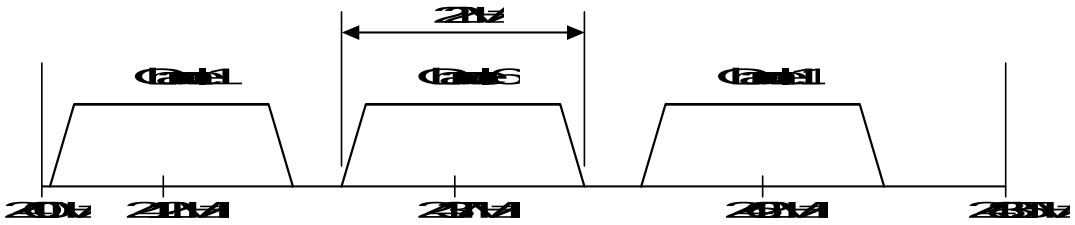
1.2.6.4 UWB PHYs

The UWB PHY operates under strict regulations for unlicensed UWB devices worldwide. The least restrictive regulations for UWB are available under the Federal Communications Commission (FCC) rules, US 47 CFR Part 15, subpart F. Under these rules, the highest allowable limits for UWB emissions are based on an equivalent emission PSD of –41.3 dBm/MHz. Other future UWB regulations in other regions will likely be at this same level or even lower. Under these limits, the allowable transmit power for a 500 MHz bandwidth UWB device would be less than –14 dBm, or about 37 μW transmit power. This transmit power level is at or below the limits for unintentional emissions from other electrical or electronic devices, as well as less than the out-of-band emission limits for other unlicensed devices operating in designated bands such as the 2.4 GHz ISM or 5 GHz UNII bands. Additionally, since this transmit power is spread over at least 500 MHz of bandwidth, the highest power in the operating bandwidth of a typical narrowband 20 MHz victim system is less than –28 dBm, or about 1.5 μW of transmit power per 20 MHz. These very low power levels emitted into the operating band of any potential victim system will reduce the likelihood that these devices might interfere with other systems.

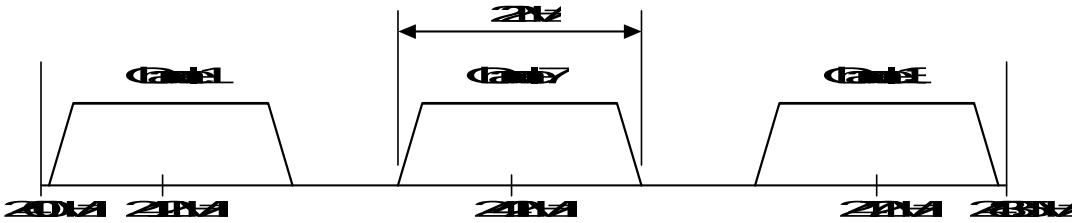
1.2.7 Channel alignment

The alignment between IEEE 802.11b (nonoverlapping sets) and IEEE 802.15.4 2400 MHz band channels is shown in Figure 3. There are four IEEE 802.15.4 channels that fall in the guard bands between (or above)

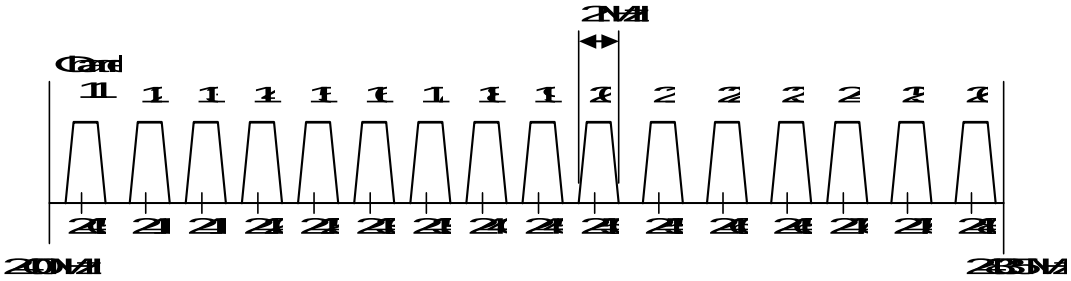
the three IEEE 802.11b channels (n = 15, 20, 25, 26 for North America; n = 15, 16, 21, 22 in Europe). While the energy in this guard space will not be zero, it will be lower than the energy within the channels; and operating an IEEE 802.15.4 WPAN on one of these channels will minimize interference between systems.



a) IEEE 802.11b North American channel selection (nonoverlapping)



b) IEEE 802.11b European channel selection (nonoverlapping)



c) IEEE 802.15.4 channel selection (2400 MHz PHY)

Figure 3—IEEE 802.15.4 (2400 MHz PHY) and IEEE 802.11b channel selection

The alignment between IEEE 802.11 HR/DSSS (nonoverlapping sets) and CSS channels (overlapping sets) is shown in Figure 4. There are 14 CSS channels (n = 0, 2, … , 13). Operating an IEEE 802.15.4 CSS WPAN on one of these channels will minimize interference between systems.

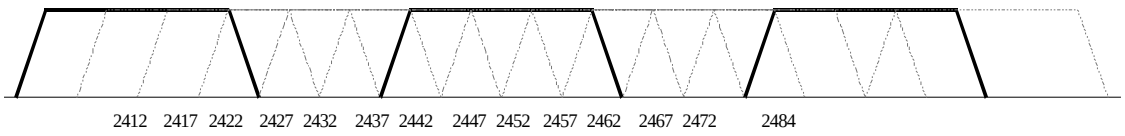


Figure 4—IEEE 802.15.4a CSS channel selection

When performing dynamic channel selection, either at network initialization or in response to an outage, a CSS device will scan a set of channels specified by the ChannelList parameter. For CSS WPANs that are installed in areas known to have high IEEE 802.11 HR/DSSS activity, the ChannelList parameter can be set by the next higher layer in order to enhance the coexistence of the networks.

1.2.8 Dynamic channel selection

When performing dynamic channel selection, either at network initialization or in response to an outage, an IEEE 802.15.4 device will scan a set of channels specified by the ChannelList parameter. For 2400 MHz band IEEE 802.15.4 networks that are installed in areas known to have high IEEE 802.11b activity, the ChannelList parameter can be set by the next higher layer in order to enhance the coexistence of the networks. For 915 MHz IEEE 802.15.4 networks that are installed in areas known to have interference from known sources, the ChannelList parameter can be set by the next higher layer in order to enhance the coexistence of the networks.

When performing dynamic channel selection, either at network initialization or in response to an outage, a UWB device will scan a set of channels specified by the ChannelList parameter. For UWB WPANs that are installed in areas known to have spectrum restrictions, the ChannelList parameter can be set by the next higher layer in order to enhance the coexistence of the networks.

1.2.9 Neighbor piconet capability

Interoperability with other systems is beyond the scope of this standard. However, certain schemes may be envisaged for this purpose, for example, the PAN coordinator can set aside GTSs specifically for use by other systems. This type of neighbor piconet support capability may further alleviate interference with other systems.

2. 2400 MHz band coexistence performance (except for CSS PHYs)

The assumptions made across all standards characterized for coexistence are described in 2.1. Subclauses 2.2 and 2.3 describe the assumptions made for individual standards and quantify their predicted performance when coexisting with IEEE 802.15.4 devices.

2.1 Assumptions for coexistence quantification

2.1.1 Channel model

The channel model is based on IEEE Std 802.11 as adapted by IEEE Std 802.15.2™-2003 and IEEE Std 802.15.3-2003:

$$d = 10^{\frac{(P_t - P_r - 40.2)}{20}} \quad \text{for } d < 8 \text{ m}$$

$$d = 8 \times 10^{\frac{(P_t - P_r - 58.5)}{33}} \quad \text{for } d > 8 \text{ m}$$

2.1.2 Receiver sensitivity

The receiver sensitivity assumed is the reference sensitivity specified in each standards as follows:

a) –76 dBm for IEEE 802.11b 11 Mb/s CCK
b) –70 dBm for IEEE Std 802.15.1-2005
c) –75 dBm for IEEE 802.15.3 22 Mb/s DQPSK
d) –85 dBm for this standard

2.1.3 Transmit power

The transmitter power for each coexisting standard has been specified as follows:

a) 14 dBm for IEEE Std 802.11b-1999
b) 0 dBm for IEEE Std 802.15.1-2005
c) 8 dBm for IEEE Std 802.15.3-2003
d) 0 dBm for this standard

2.1.4 Receiver bandwidth

The receiver bandwidth is as required by each standard as follows:

a) 22 MHz for IEEE Std 802.11b-1999
b) 1 MHz for IEEE Std 802.15.1-2005
c) 15 MHz for IEEE Std 802.15.3-2003
d) 2 MHz for this standard

2.1.5 Transmit spectral masks

The maximum transmitter spectral masks are assumed for the calculations. This assumption is the absolute worst-case scenario; in most cases, the transmitter spectrum will be lower. The transmitter spectral mask for IEEE Std 802.11b is given in Table 1.

Table 1—Transmit mask for IEEE Std 802.11b-1999

Frequency	Relative limit
$f_c - 22$ MHz < f < $f_c - 11$ MHz and $f_c + 11$ MHz < f < $f_c + 22$ MHz	–30 dBr
f < $f_c - 22$ MHz and f > $f_c + 22$ MHz	–50 dBr

The transmit mask for IEEE Std 802.15.1-2005 is given in Table 2

Table 2—Transmit mask for IEEE Std 802.15.1-2005

Frequency offset	Transmit power
± 500 kHz	–20 dBc
$\|M - N\| = 2$	–20 dBm
$\|M - N\| \geq 3$	–40 dBm
The transmitter is transmitting on channel *M*, and the adjacent channel power is measured on channel number *N*.	

The transmit mask for IEEE Std 802.15.3-2003 is given in Table 3

Table 3—Transmit mask for IEEE Std 802.15.3-2003

Frequency offset	Relative limit
7.5 MHz < $\lvert f - f_c \rvert$ < 15 MHz	–30 dBr
15 MHz < $\lvert f - f_c \rvert$ < 22 MHz	$-1/7[\lvert f - f_c \text{ (MHz)} \rvert + 13]$ dBr
22 MHz < $\lvert f - f_c \rvert$	–50 dBr

The transmit mask for IEEE 802.15.4 is given in Table 4

Table 4—Transmit mask for this standard

Frequency	Relative limit	Absolute limit
$\lvert f - f_c \rvert$ >3.5 MHz	–20 dBr	–30 dBm

2.1.6 IEEE 802.11b transmit PSD

Because IEEE 802.11 implementations will generally meet FCC requirements, they will achieve an absolute power of less than –41.3 dBm/MHz at a separation of 22 MHz from the carrier frequency. The reason for this is that there is a restricted band that ends at 2.39 GHz, which is 22 MHz from the center of the lowest channel used for the FCC regulatory domain, as described in 18.4.6.2 in FCC CFR47 [B5]. Thus, the relative power for greater than 22 MHz separation would be +14 dBm – (–41.3 dBm) = 55.3 dB.

2.1.7 Interference characteristics

The effect of the interfering signal on the desired signal is assumed to be similar to additive white Gaussian noise (AWGN) in the same bandwidth.

2.1.8 Bit error rate (BER) calculations

The BER calculations are as described in C3.6 of IEEE Std 802.15.2-2003:

The BER for IEEE Std 802.11b-1999 at 1 Mb/s is given by

$$BER_{802.11,1} = Q(11 \times SINR)$$

The BER for IEEE Std 802.11b at 2 Mb/s is given by

$$BER_{802.11,2} = Q\left(5.5 \times \frac{SINR}{2}\right)^{\frac{1}{2}}$$

The BER for IEEE Std 802.11b at 5.5 Mb/s is given by

$$BER_{802.11,5.5} = \frac{8}{15} \times \left(14 \times Q(8 \times SINR)^{\frac{1}{2}} + Q(16 \times SINR)^{\frac{1}{2}}\right)$$

The BER for IEEE Std 802.11b at 11 Mb/s is given by

$$BER_{802.11,11} = \frac{128}{255} \times \left(24 \times Q(4 \times SINR)^{\frac{1}{2}} + 16 \times Q(16 \times SINR)^{\frac{1}{2}} + 174 \times Q(8 \times SINR)^{\frac{1}{2}} + 16 \times Q(10 \times SINR)^{\frac{1}{2}} + 24 \times Q(12 \times SINR)^{\frac{1}{2}} + Q(16 \times SINR)^{\frac{1}{2}} \right)$$

The BER for IEEE Std 802.15.1-2005 is given by

$$BER_{802.15.1} = 0.5 \times e^{\frac{-SINR}{2}}$$

The BER for IEEE Std 802.15.3-2003 at 11 Mb/s is given by

$$BER_{802.15.3} = Q\left(SINR^{\frac{1}{2}} \right)$$

The BER for this standard is given by

$$BER_{802.15.4} = \frac{8}{15} \times \frac{1}{16} \times \sum_{k=2}^{16} -1^{k} \binom{16}{k} e^{\left(20 \times SINR \times \left(\frac{1}{k} - 1\right)\right)}$$

2.1.9 Packet error rate (PER)

To convert between BER and PER, the following average packet lengths are assumed:

a) Average frame for IEEE Std 802.11b-1999 = 1024 octets
b) Average frame for IEEE Std 802.15.1-2002 = 1024 octets
c) Average frame length for IEEE Std 802.15.3-2003 = 1024 octets
d) Average frame length for this standard = 22 octets

2.2 BER model

This subclause presents the BER for standards characterized for coexistence. The BER results were obtained using the analytical model from IEEE Std 802.15.2-2003. The calculation follows the approach outlined in 5.3.2 of that standard, and the conversion from SNR to BER uses the formulas in 5.3.6 of that standard. Figure 5 illustrates the relationship between BER and SNR for IEEE Std 802.11b-1999, IEEE 802.15.3 base rate, IEEE Std 802.15.1-2005, and this standard.

2.3 Coexistence simulation results

Using the assumptions outlined in 2.2, an analytical simulation tool was developed to quantify the effect of interference between neighboring devices. For each of the cases studied, the receiver under test was presented with a desired signal at 10 dB above the required sensitivity, as described in 2.1.2, and a single interfering device with appropriate transmit power, as described in 2.1.3. The amount of received interference power was determined using the propagation model, as described in 2.1.1, as well as the

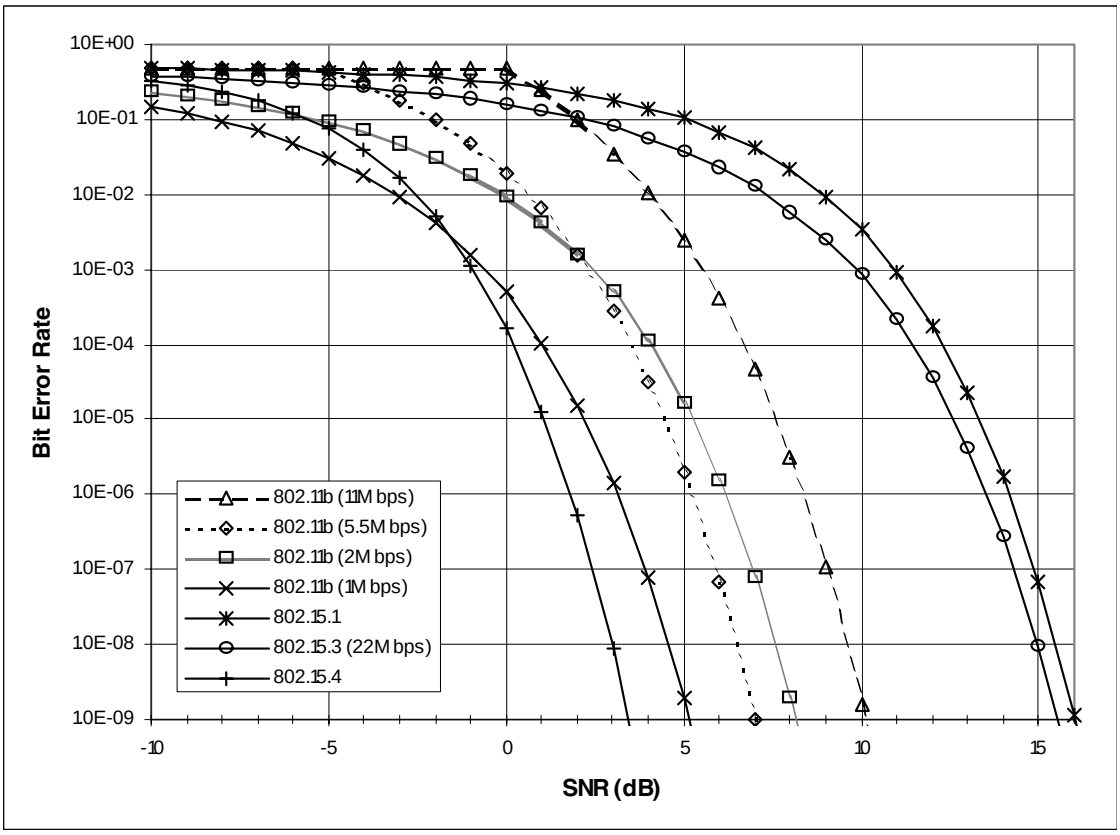


Figure 5—BER results for IEEE Std 802.11b, IEEE Std 802.15.1, IEEE Std 802.15.3, and this standard

transmit PSD, as described in 2.1.5, and receiver bandwidth, as described in 2.1.4, and the resulting SIR level was used to estimate the achievable PER.

The simulation output, illustrated in Figure 6, Figure 7, Figure 8, Figure 9, Figure 10 and Figure 11, shows the PER versus separation distance and frequency offset for various combinations of devices. When comparing the results, some obvious features stand out. First, for the nonhopping systems, large frequency offsets allow close-proximity coexistence (less than 2 m separation), while low-frequency offsets, or co-channel interference, require separation distances in the tens of meters. Therefore, as expected, the ability to detect channel occupancy and perform dynamic channel selection is an important mechanism for coexistence.

A second observation is that transmit power level is the dominant factor in co-channel interference situations. When a low-power IEEE 802.15.4 device is moved toward an IEEE 802.11b or IEEE 802.15.3 device, the IEEE 802.15.4 device is the first to degrade. IEEE Std 802.15.1-2005 and this standard have similar transmit powers, and their interference effects on each other are similar.

Even with its low transmit power level, the results presented here suggest that an IEEE 802.15.4 device can cause degradation to the other devices in co-channel situations with separation distances below 20 m. However, in practice, several IEEE 802.15.4 coexistence features (which were not included in this PHY simulation) will help to further reduce the occurrence and severity of co-channel interference. These include the very low duty cycle operation for typical IEEE 802.15.4 applications, as well as the use of CCA prior to transmission (CSMA-CA).

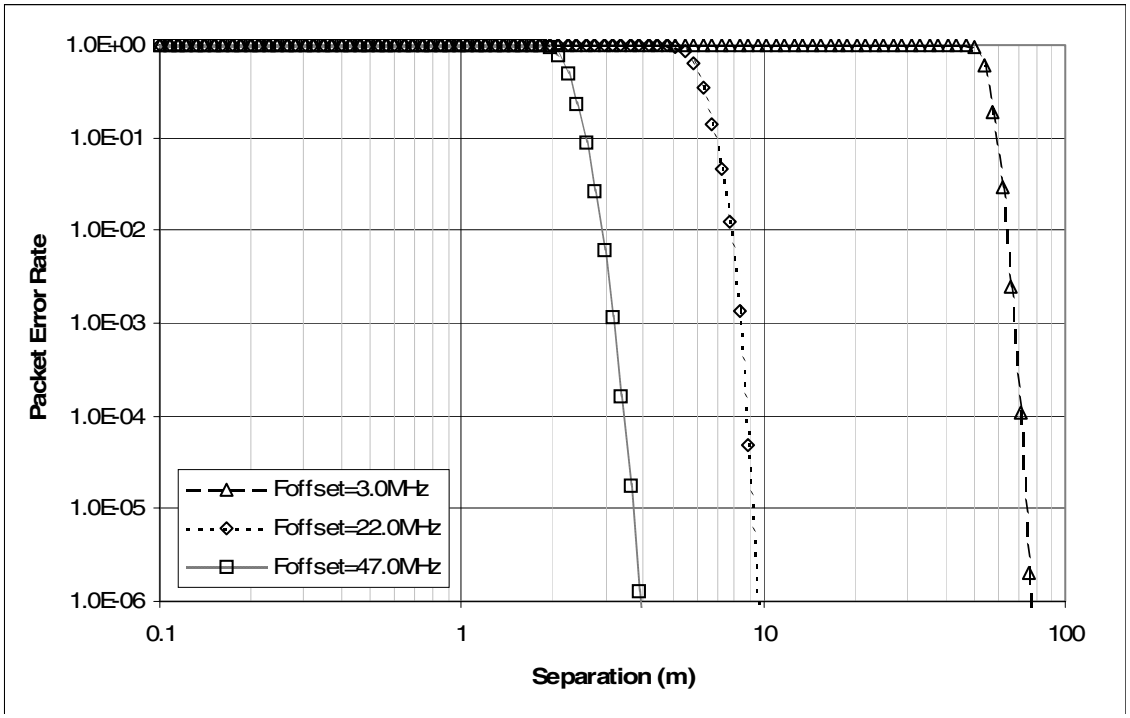


Figure 6—IEEE 802.15.4 receiver, IEEE 802.11b interferer

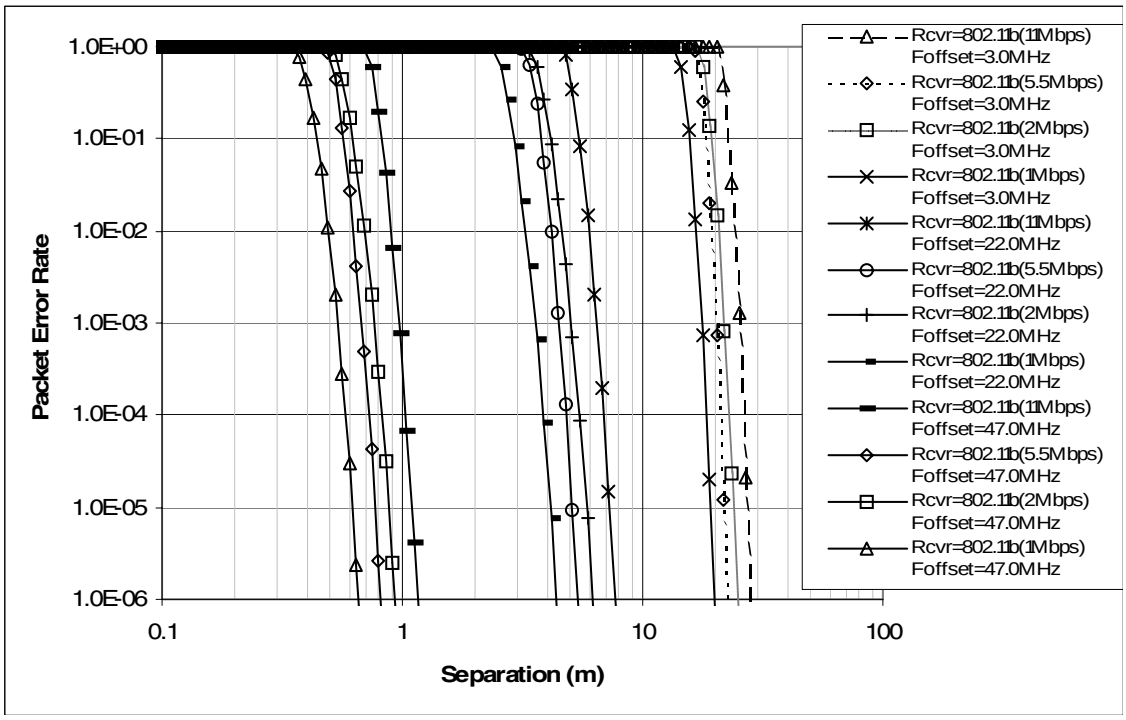


Figure 7—IEEE 802.11b receiver, IEEE 802.15.4 interferer

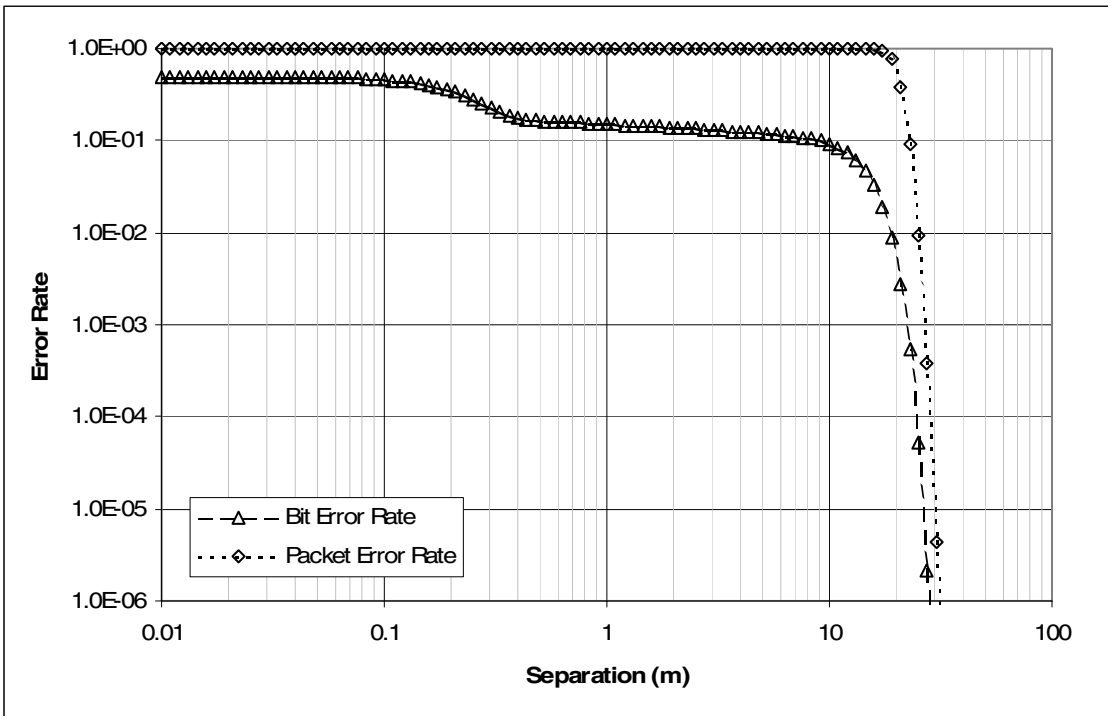


Figure 8—IEEE 802.15.4 receiver, IEEE 802.15.1 interferer

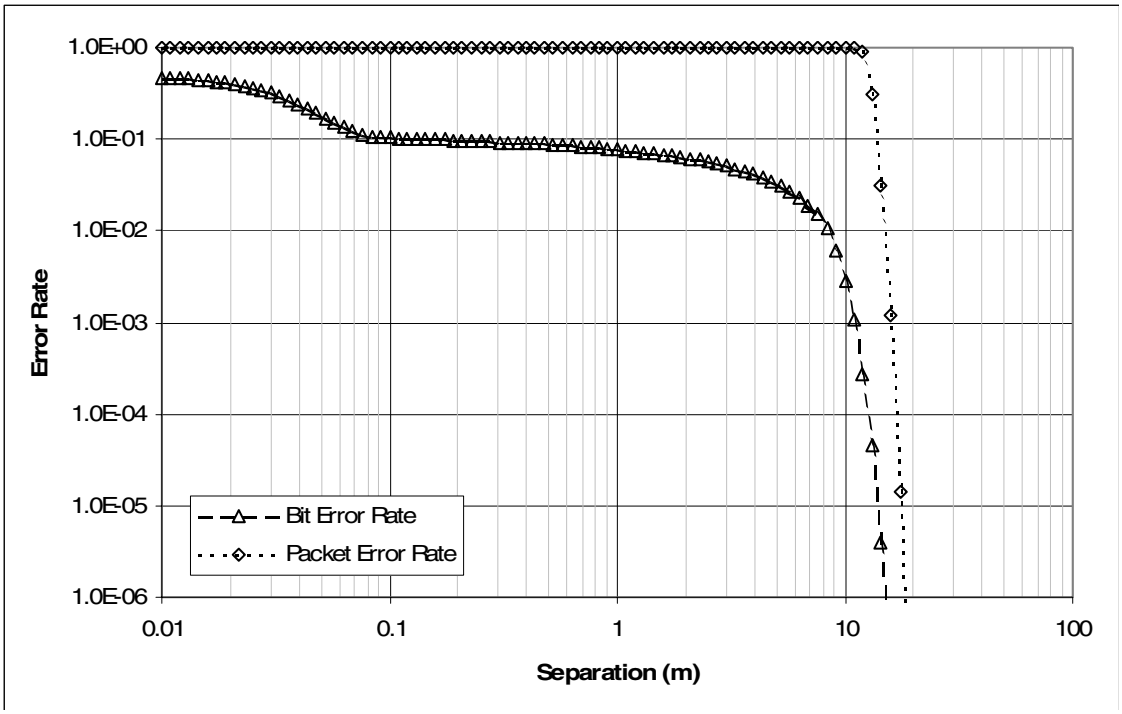


Figure 9—IEEE 802.15.1 receiver, IEEE 802.15.4 interferer

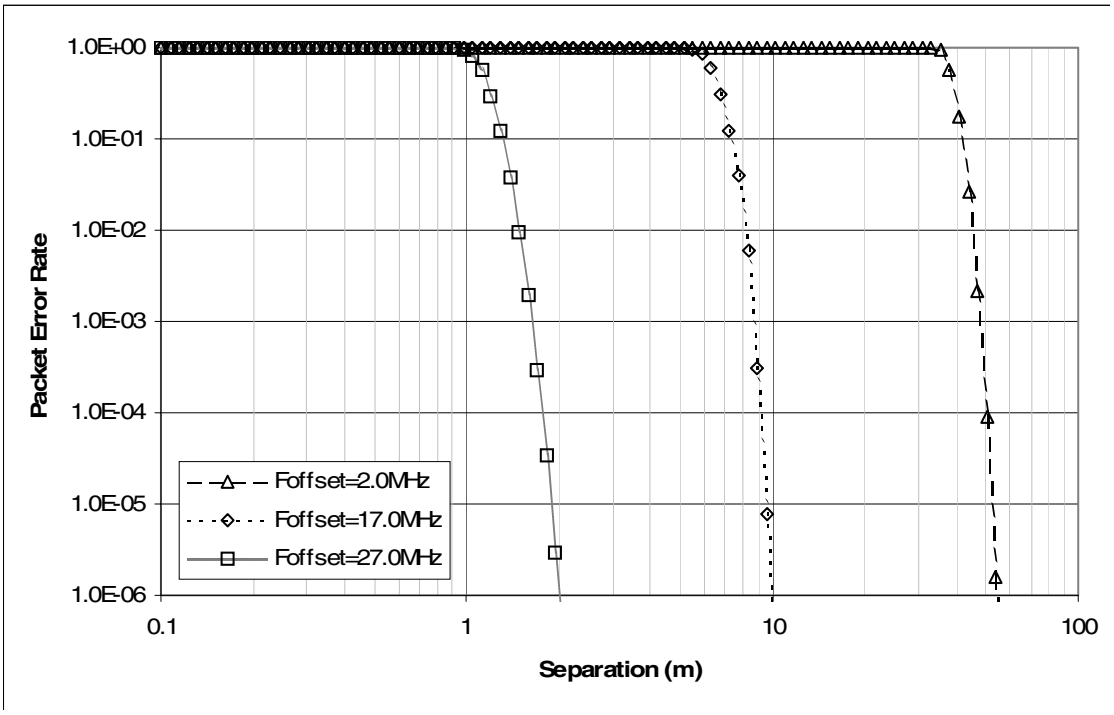


Figure 10—IEEE 802.15.4 receiver, IEEE 802.15.3 interferer

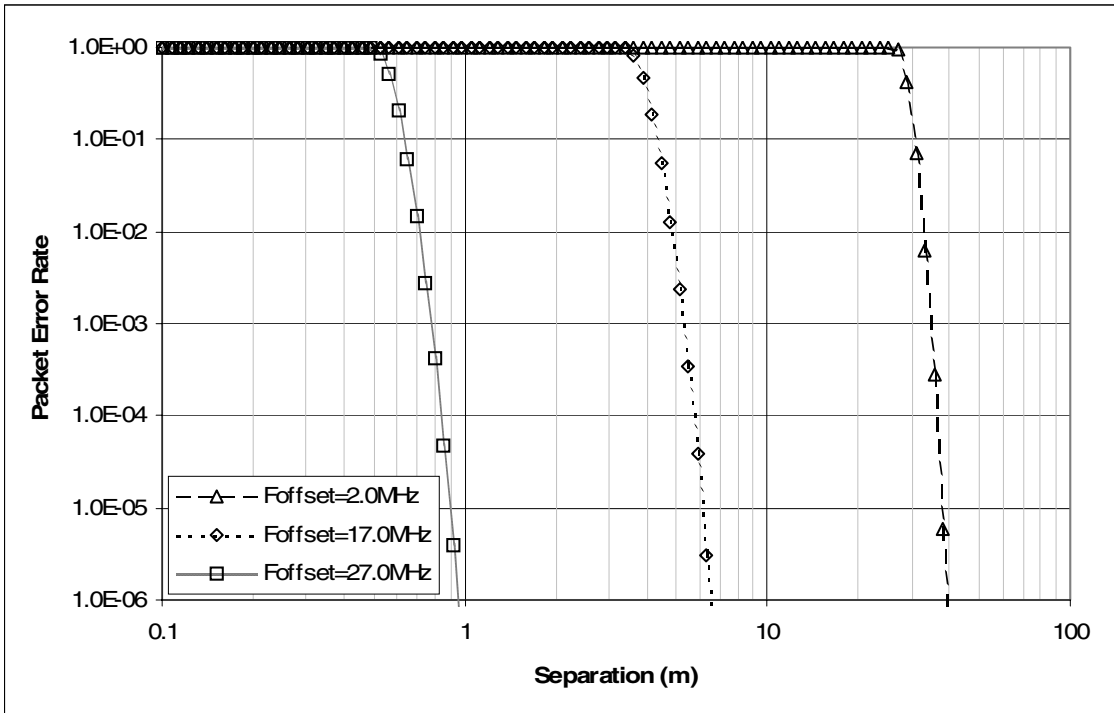


Figure 11—IEEE 802.15.3 receiver, IEEE 802.15.4 interferer

3. 800/900 MHz bands coexistence performance

In order to quantify the coexistence performance of the IEEE 802.15.4 PHYs operating below 1 GHz, the techniques described in Shellhammer [B6] and [B7] have been adopted.

The coexistence assurance methodology predicts the PER of an affected wireless network (AWN, or victim) in the presence of an interfering wireless network (IWN, or assailant). In its simplest form, the methodology assumes an AWN and an IWN, each composed of a single transmitter and a receiver. The methodology takes as input a path loss model, a BER function for the AWN, and predicted temporal models for packets generated by the AWN and for "pulses," i.e., packets generated by the IWN. Based on these inputs, the methodology predicts the PER of the AWN as a function of the physical spacing between the IWN transmitter and the AWN receiver.

The appeal of the coexistence assurance methodology is that multiple networking standards can be characterized and compared with just a few parameters, notably

— Bandwidth of AWN and IWN devices

— Path loss model for the networks

— BER as a function of SIR of AWN devices[2]

The general assumptions made across all six sub-gigahertz PHYs are described in the followin subclauses.

3.1 Victims and assailants

At present, the six PHYs described in this standard are the only wireless networking standards in the 868 MHz and 915 MHz bands covered under IEEE 802. Because other wireless systems are not characterized here, it is assumed that the PHYs will serve as both *victims* (participants in AWNs) and as *assailants* (participants in IWNs).

3.2 Bandwidth

The three IEEE 802.15.4 PHYs that operate in the 868 MHz band have one channel, approximately 600 kHz wide. The coexistence methodology assumes that any 868 MHz device in an AWN will have the same bandwidth as a device in the IWN.

Similarly, the three PHYs that operate in the 915 MHz band have 10 channels, each one 2 MHz wide. The coexistence methodology assumes that any 915 MHz device in an AWN will be operating in the same channel and have the same bandwidth as a device in the IWN.

3.3 Path loss model

The coexistence methodology uses a variant of the path loss model described in IEEE Std 802.15.2-2003, which stipulates a two-segment function with a path loss exponent of 2.0 for the first 8 m and then a path loss exponent of 3.3 thereafter. The formula given in IEEE Std 802.15.2 is

[2]Although the methodology described in Stellhammer [B6] uses symbol error rate (SER) to characterize PHY performance, BER has been used in this standard instead because available error functions are more commonly defined as BER rather than SER.

$$pl(d) = \begin{cases} 40.2 + 20\log_{10}(d) & d \le 8 \text{ m} \\ 58.5 + 33\log_{10}\left(\frac{d}{8}\right) & d > 8 \text{ m} \end{cases}$$

The constants in this formula are based on a 2400 MHz center frequency. To adapt the model to a 900 MHz center frequency, the preceeding equation can be generalized as

$$pl(d) = \begin{cases} pl(1) + 10\gamma_1\log_{10}(d) & d \le 8 \text{ m} \\ pl(8) + 10\gamma_8\log_{10}\left(\frac{d}{8}\right) & d > 8 \text{ m} \end{cases}$$

where

$pl(1)$ is the path loss at 1 m (in dB)
γ_1 is the path loss exponent at 1 m, $\gamma_1 = 2.0$
γ_8 is the path loss exponent at 8 m $\gamma_8 = 3.3$)

The initial condition of $pl(1)$ is computed as.

$$pl(1) = 10\gamma_1\log_{10}\left(\frac{4\pi f}{c}\right)$$

where

$\gamma_1 = 2.0$
$f = 900$ MHz
c = speed of light = 299792458 ms^{-1}

which gives $pl(1) = 31.53$ and $pl(8) = 49.59$. The path loss function modified for 900 MHz is then

$$pl(d) = \begin{cases} 31.53 + 20\log_{10}(d) & d \le 8 \text{ m} \\ 49.59 + 33\log_{10}\left(\frac{d}{8}\right) & d > 8 \text{ m} \end{cases}$$

A plot of the path loss function is shown in Figure 12.

3.4 Temporal model

In this standard, packet overhead is kept to minimum. The maximum PSDU size is 128 octets, and a typical packet may be only 32 octets, including PSDU and synchronization octets. For the coexistence methodology, all packets, whether belonging to the AWN or IWN, are assumed to be 32 octets.

As specified in ERC Recommendation 70-03 [B1] and ETSI EN 300 220-1 [B2], the 868 MHz ISM band is limited by European regulations to operate at or under 1% duty cycle. Therefore, all 868 MHz BPSK devices, whether operating in AWNs or IWNs, can be assumed to be operating at 1% worst case.

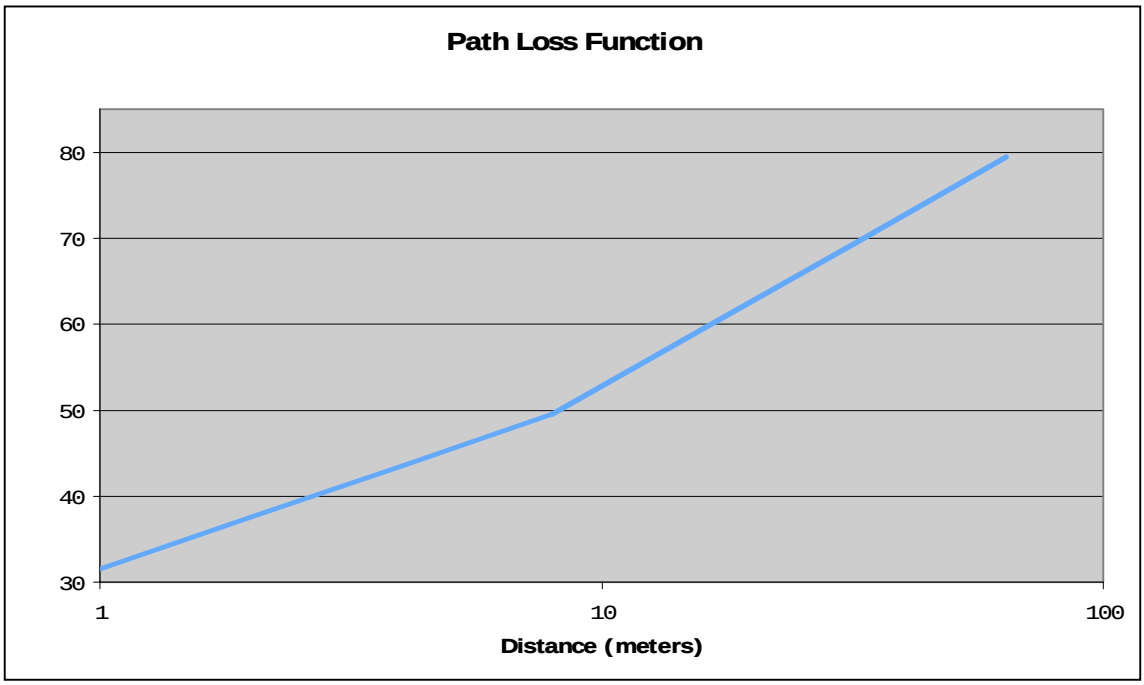


Figure 12—Plot of path loss function for 900 MHz

Although there are no duty cycle limitations in the 915 MHz band, many networks based on this standard are expected to operate at well under 1% duty cycle, particularly devices that are battery powered. It is reasonable to expect that mains-powered devices, such as PAN coordinators and data aggregation points, may operate at duty cycles as high as 10%. For purposes of modeling coexistence, it is assumed that all 915 MHz devices, whether operating in AWNs or IWNs, have a duty cycle of 10%.

3.5 Coexistence assurance results

This subclause describes the parameters that are particular to each PHY covered under this standard and shows the results of the coexistence assurance methodology for each of the sub-gigahertz PHYs.

3.5.1 868 MHz BPSK PHY

3.5.1.1 BER as a function of SIR

IEEE 802.15.4 868 MHz BPSK modulation uses a chip rate R_c of 300 kc/s and a bit rate R_b of 20 kb/s. Conversion from SNR to E_b/N_0 assumes a raised cosine filter which gives

$$\frac{E_b}{N_0} = \frac{0.75R_c}{R_b}SNR = \frac{0.75 \times 300000}{20000}SNR = 11.25 \times SNR \ .$$

BER P_b is computed for noncoherent BPSK, e.g., from Sklar [B8], as

$$P_b = 0.5\exp\left(-\frac{E_b}{N_0}\right).$$

Rolling these together produces the BER function.

$$P_b = 0.5\exp(-11.25 \times SNR)$$

3.5.1.2 Temporal model

With a 1% operating duty cycle and a packet size of 32 octets, the channel will be occupied for

$$\frac{payloadSizex \times 8}{bitsPerSecond} = \frac{256}{20000}S = 12.8 \text{ ms}$$

and the channel will be idle for 99 × 12.8 ms = 1.2672 s.

3.5.1.3 Coexistence methodology results

Figure 13 shows the coexistence methodology results for the 868 MHz BPSK PHY.

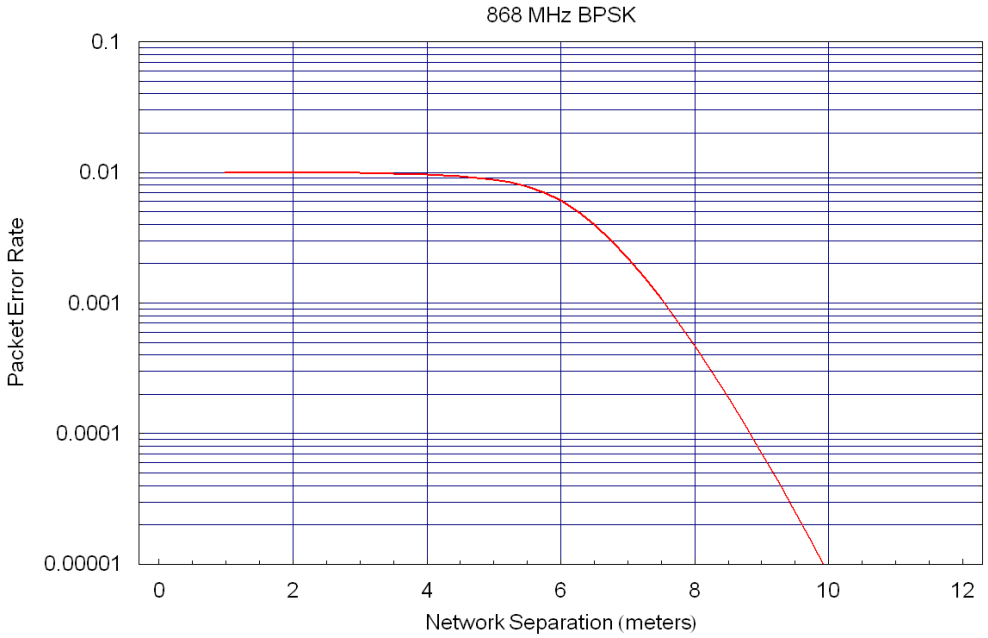


Figure 13—Coexistence methodology results for 868 MHz BPSK PHY

3.5.2 868 MHz O-QPSK PHY

3.5.2.1 BER as a function of SIR

IEEE 802.15.4 868 MHz O-QPSK modulation uses a chip rate R_c of 400 kc/s, a bit rate R_b of 100 kb/s, and a codebook of M = 16 symbols. Conversion from SNR to E_b/N_0 assumes matched filtering and half-sine pulse shaping which results in

$$\frac{E_b}{N_0} = \frac{0.625R_c}{R_b}SNR = \frac{0.625 \times 400000}{100000}SNR = 2.5 \times SNR \ .$$

Conversion from bit noise density E_b/N_0 to symbol noise density E_s/N_0 is given by

$$\frac{E_s}{N_0} = \log_2(M)\frac{E_b}{N_0} = 4\frac{E_b}{N_0}.$$

Symbol error rate (SER) P_s is computed for noncoherent MFSK, e.g., from Sklar [B8], as

$$P_s = \frac{1}{M}\sum_{j=2}^{M}(-1)^j\binom{M}{j}\exp\left(\frac{E_s}{N_0}\left(\frac{1}{j}-1\right)\right).$$

Finally, conversion from SER P_s to BER P_b is given as

$$P_b = P_s\left(\frac{M/2}{M-1}\right) = P_s\left(\frac{8}{15}\right).$$

Rolling these together produces the BER function.

$$P_b = \left(\frac{8}{15}\right)\left(\frac{1}{16}\right)\sum_{j=2}^{M}(-1)^j\binom{16}{j}\exp\left(10\times SNR\times\left(\frac{1}{j}-1\right)\right)$$

3.5.2.2 Temporal model

With a 1% operating duty cycle and a packet size of 32 octets, the channel will be occupied for

$$\frac{payloadSizex8}{bitsPerSecond} = \frac{256}{100000}S = 2.56 \text{ ms}$$

and the channel will be idle for 99 × 2.56 ms = 253.44 ms.

3.5.2.3 Coexistence methodology results

Figure 14 shows the coexistence methodology results for the 868 MHz O-QPSK PHY.

3.5.3 868 MHz PSSS PHY

3.5.3.1 BER as a function of SIR

IEEE 802.15.4 868 MHz PSSS uses a form of ASK modulation, for which the BER function is most easily derived by simulation and curve fitting. For SNR values greater than –8 dB, the BER function is approximated as

$$P_b = 0.4146\exp(-6.0871\times SNR).$$

3.5.3.2 Temporal model

With a 1% operating duty cycle and a packet size of 32 octets, the channel will be occupied for

$$\frac{payloadSizex8}{bitsPerSecond} = \frac{256}{250000}S = 1.024 \text{ ms}$$

and the channel will be idle for 99 × 1.024 ms = 101.376 ms.

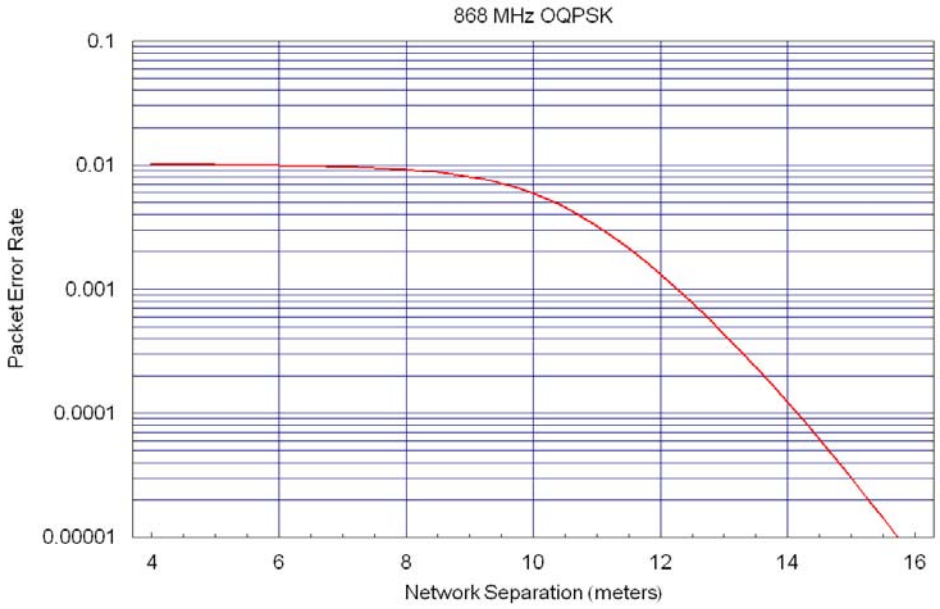


Figure 14—Coexistence methodology results for 868 MHz O-QPSK PHY

3.5.3.3 Coexistence methodology results

Figure 15 shows the coexistence methodology results for the 868 MHz PSSS PHY.

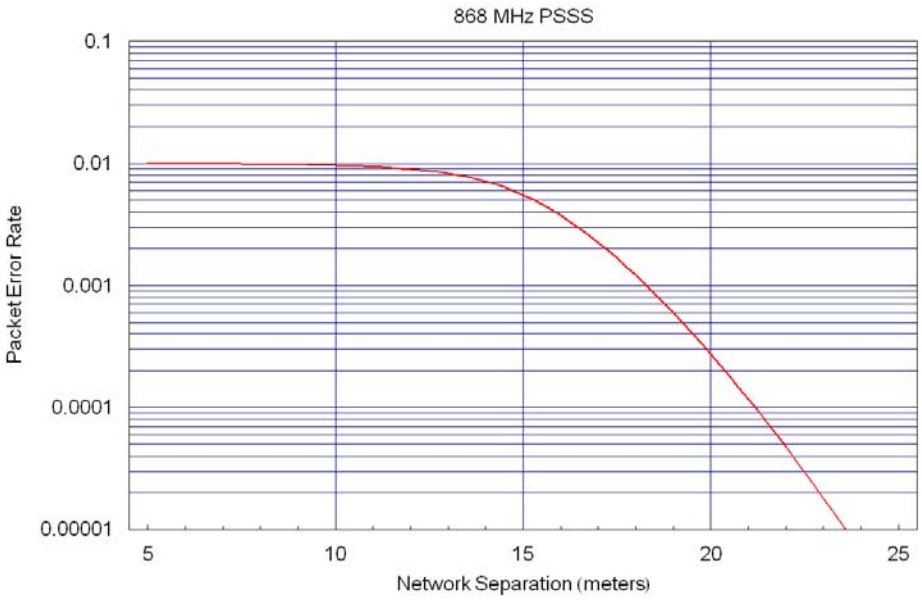


Figure 15—Coexistence methodology results for 868 MHz PSSS PHY

3.5.4 915 MHz BPSK PHY

3.5.4.1 BER as a function of SIR

IEEE 802.15.4 915 MHz BPSK modulation uses a chip rate R_c of 600 kc/s and a bit rate R_b of 40 kb/s. Conversion from SNR to E_b/N_0 assumes a raised cosine filter which results in

$$\frac{E_b}{N_0} = \frac{0.75R_c}{R_b}SNR = \frac{0.75 \times 600000}{40000}SNR = 11.25 \times SNR \ .$$

BER P_b is computed for noncoherent BPSK, e.g., from Sklar [B8], which results in

$$P_b = 0.5\exp\left(-\frac{E_b}{N_0}\right).$$

Rolling these together produces the BER function.

$$P_b = 0.5\exp(-11.25 \times SNR)$$

3.5.4.2 Temporal model

With a 10% operating duty cycle and a packet size of 32 octets, the channel will be occupied for

$$\frac{payloadSizex8}{bitsPerSecond} = \frac{256}{40000}S = 6.4 \text{ ms}$$

and the channel will be idle for 90 × 6.4 ms = 576 ms.

3.5.4.3 Coexistence methodology results

Figure 16 shows the coexistence methodology results for the 915 MHz BPSK PHY.

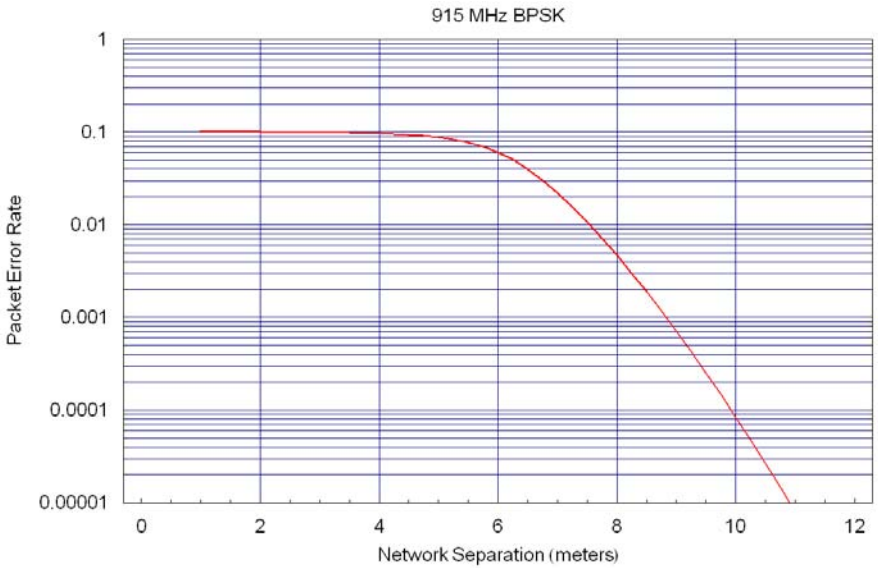


Figure 16—Coexistence methodology results for 915 MHz BPSK PHY

3.5.5 915 MHz O-QPSK PHY

3.5.5.1 BER as a function of SIR

IEEE 802.15.4 915 MHz O-QPSK modulation uses a chip rate R_c of 1000 kc/s, a bit rate R_b of 250 kb/s, and a codebook of M = 16 symbols. Conversion from SNR to E_b/N_0 assumes matched filtering and half-sine pulse shaping which gives

$$\frac{E_b}{N_0} = \frac{0.625R_c}{R_b}SNR = \frac{0.625 \times 1000000}{250000}SNR = 2.5 \times SNR \ .$$

Conversion from bit noise density E_b/N_0 to symbol noise density E_s/N_0 gives

$$\frac{E_s}{N_0} = \log_2(M)\frac{E_b}{N_0} = 4\frac{E_b}{N_0}.$$

SER P_s is computed for noncoherent MFSK, e.g., from Sklar [B8], as

$$P_s = \frac{1}{M}\sum_{j=2}^{M}(-1)^j\binom{M}{j}\exp\left(\frac{E_s}{N_0}\left(\frac{1}{j}-1\right)\right).$$

Finally, conversion from SER P_s to BER P_b is given as

$$P_b = P_s\left(\frac{M/2}{M-1}\right) = P_s\left(\frac{8}{15}\right).$$

Rolling these together produces the BER function as

$$P_b = \left(\frac{8}{15}\right)\left(\frac{1}{16}\right)\sum_{j=2}^{M}(-1)^j\binom{16}{j}\exp\left(10SNR\left(\frac{1}{j}-1\right)\right).$$

3.5.5.2 Temporal model

With a 10% operating duty cycle and a packet size of 32 octets, the channel will be occupied for

$$\frac{payloadSizex8}{bitsPerSecond} = \frac{256}{250000}S = 1.024 \text{ ms}$$

and the channel will be idle for 90 × 1.024 ms = 92.16 ms.

3.5.5.3 Coexistence methodology results

Figure 17 shows the coexistence methodology results for the 915 MHz O-QPSK PHY.

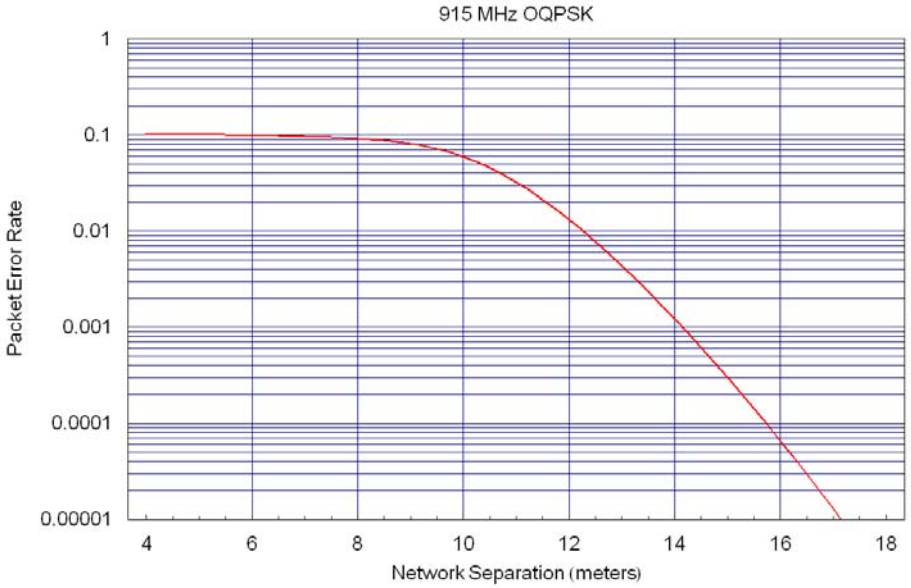


Figure 17—Coexistence methodology results for 915 MHz O-QPSK PHY

3.5.6 915 MHz PSSS PHY

3.5.6.1 BER as a function of SIR

IEEE 802.15.4 915 MHz PSSS uses a form of ASK modulation, for which the BER function is most easily derived by simulation and curve fitting. For SNR values greater than –8 dB, the BER function is approximated as

$$P_b = 7.768\exp(-21.93SNR) - 12.85\exp(-27.53 \times SNR) \ .$$

3.5.6.2 Temporal model

With a 10% operating duty cycle and a packet size of 32 octets, the channel will be occupied for

$$\frac{payloadSizex8}{bitsPerSecond} = \frac{256}{250000}S = 1.024 \text{ ms}$$

and the channel will be idle for 90 × 1.024 ms = 92.16 ms.

3.5.6.3 Coexistence methodology results

Figure 18 shows the coexistence methodology results for the 915 MHz PSSS PHY.

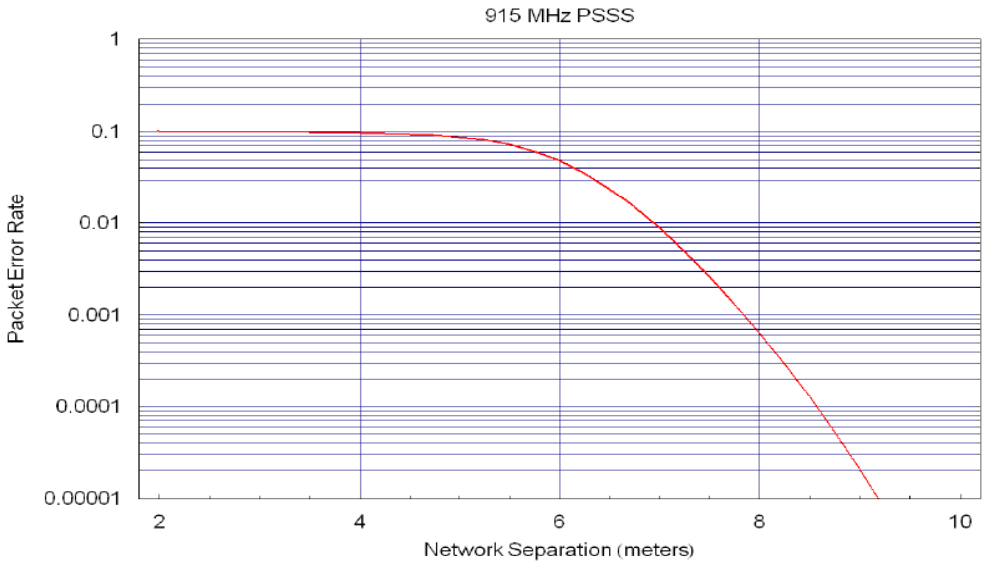


Figure 18—Coexistence methodology results for 915 MHz PSSS PHY

4. 2400 MHz band coexistence performance for CSS PHYs

Subclauses E.3.2 and E.3.4 also describe the assumptions made for individual standards and quantify their predicted performance when coexisting with IEEE 802.15.4a CSS devices.

4.1 Assumptions for coexistence performance

The receiver sensitivity assumed is the reference sensitivity specified in each standard as follows:

— –76 dBm for IEEE 802.11 HR/DSSS 11 Mb/s CCK
— –82 dBm for IEEE 802.11 ERP 6 Mb/s OFDM
— –74 dBm for IEEE 802.11 ERP 24 Mb/s OFDM
— –65 dBm for IEEE 802.11 ERP 54 Mb/s OFDM
— –70 dBm for IEEE 802.15.1 devices
— –75 dBm for IEEE P802.15.3 22 Mb/s DQPSK
— –85 dBm for IEEE 802.15.4 devices
— –85 dBm for IEEE 802.15.4a 1 Mb/s CSS

The transmit power for each coexisting standard has been specified as follows:

— 14 dBm for IEEE Std 802.11 HR/DSSS
— 0 dBm for IEEE Std 802.15.1-2005
— 8 dBm for IEEE Std 802.15.3-2003
— 0 dBm for IEEE Std 802.15.4-2006

— 0 dBm for IEEE 802.15.4a CSS

The bit error rate (BER) calculation for IEEE 802.15.4a CSS is

$$BER_{CSS} = [(M-2) \times Q(\sqrt{SNR_0 \times \log_2(M)}) + Q(\sqrt{SN(R_0 \times 2\log_2(M))})]/2$$

where

1 Mb/s: SNR_0 = SNR × 14 × 1.6667, M = 8
250 kb/s: SNR_0 = SNR × 14 × 1.6667 × 4, M = 64

For the IEEE 802.11 ERP 6 Mb/s: M-PSK, the BER calculation is

$$BER_{802.11,6}(M=2) = Q\left(\sqrt{2 \times \frac{E_b}{N_0} \times 10^{\frac{5.7}{10}}}\right)$$

For the IEEE 802.11 ERP 24 Mb/s and 54 Mb/s QAM modes, the BER calculation is

$$BER_{802.11}(M>2, Cg) = 1 - \left[1 - 2\left(\left(1 - \frac{1}{\sqrt{M}}\right) \times Q\left(\sqrt{\frac{3}{M-1} \times \frac{\log_2(M) \cdot E_b}{N_0} \times 10^{\frac{Cg}{10}}}\right)\right)\right]^{2 \cdot \frac{1}{\log_2(M)}}$$

where M is the number of points in the constellation and C_g is the coding gain. The values for 24 Mb/s and 54 Mb/s are:

24 Mb/s: M = 16, C_g = 5.7 dB
54 Mb/s: M = 64, C_g = 3.8 dB

The relationship between E_b/N_0 and SNR is assumed to be computable from the subcarrier spacing F_s = 0.3125 MHz and the OFDM symbol rate, R_s = 0.25 Msymbol/s as follows:

$$SNR = \frac{E_b}{N_0} \times \frac{F_s}{R_s}$$

The PER is based frame lengths and duty cycles listed in Table 5.

Table 5—Frame length and duty cycles for PER calculations

PHY	Average frame length	Duty cycle
IEEE 802.11 HR/DSSS	1500 octets	50% (average)
IEEE 802.11 ERP	1500 octets	50% (average)
IEEE 802.15.1	2871 bits	50% (average)
IEEE 802.15.3	1024 octets	50% (average)
IEEE 802.15.4	22 octets	1% (normal) 10% (rare, aggregated)
IEEE 802.15.4 CSS	32 octets	0.25%, 1% (normal) 2.5%, 10% (rare, aggregated)

Figure 19 illustrates also the relationship between BER and SNR for IEEE 802.11 HR/DSSS, IEEE 802.15.3 base rate, IEEE 802.15.1, IEEE 802.15.4, and IEEE 802.15.4a CSS PHYs.

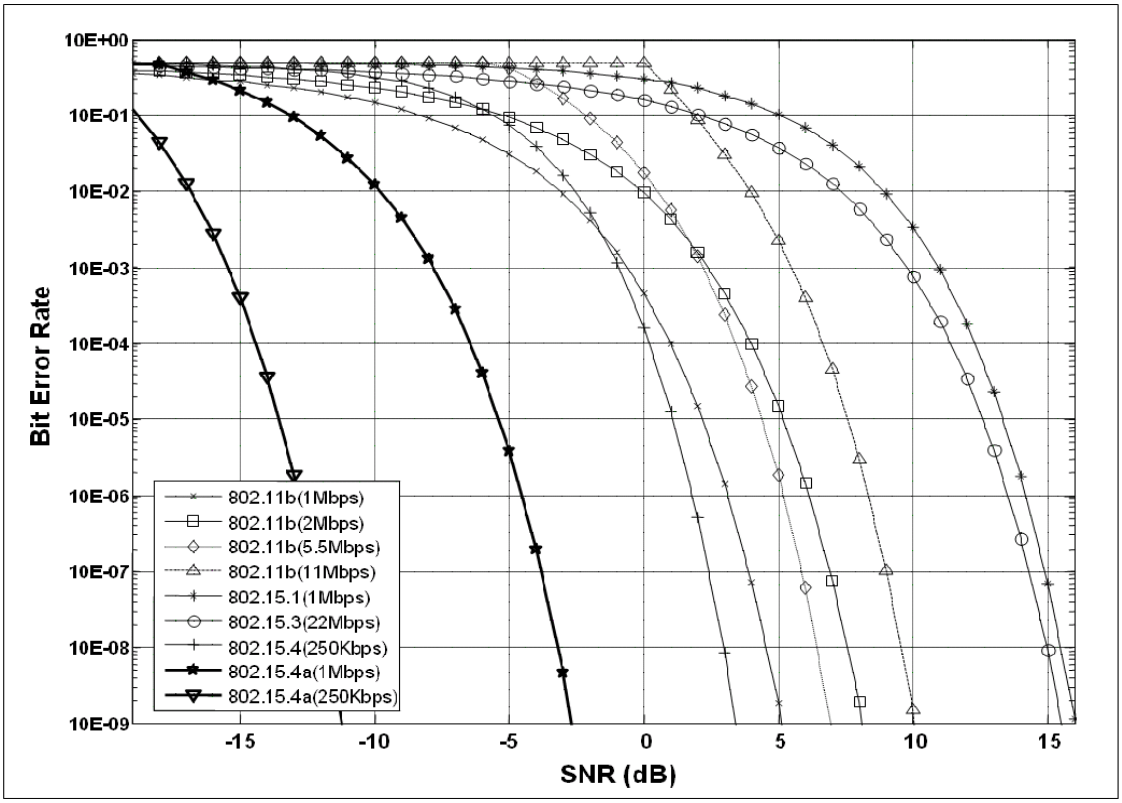


Figure 19—BER results of IEEE 802.11 HR/DSSS, IEEE 802.15.1, IEEE 802.15.3, IEEE 802.15.4 (2400 MHz) and IEEE 802.15.4a CSS PHYs

4.2 Coexistence simulation results

The shapes of the assumed transmit spectra and receive filter shapes are defined in Table 6.

Table 6—Frequency offset (MHz)Attenuation (dB)

IEEE 802	Transmit		Receive	
	Frequency offset (MHz)	Attenuation (dB)	Frequency offset (MHz)	Attenuation (dB)
15.1	0	0	0	0
	0.25	0	0.25	0
	0.75	38	0.75	38
	1	40	1	40
	1.5	55	1.5	55
11 HR/DSSS	0	0	0	0
	4	0	4	0
	6	10	6	10
	9	30	9	30
	15	50	15	50
	20	55	20	55
11 ERP	0	0	0	0
	5	0	5	0
	8	4	8	4
	9	10	9	10
	10	25	10	25
	15	40	15	40
	40	43	40	43
15.3	0	0	0	0
	8	0	8	0
	8	30	8	30
	15	30	15	30
	15	40	15	40
	22	50	22	50

Table 6—Frequency offset (MHz)Attenuation (dB) *(continued)*

IEEE 802	Transmit		Receive	
	Frequency offset (MHz)	Attenuation (dB)	Frequency offset (MHz)	Attenuation (dB)
15.4 (non CSS)	0	0	0	0
	0.5	0	0.5	0
	1	10	1	10
	1.5	20	1.5	20
	2	25	2	25
	2.5	30	2.5	30
	3	31	3	31
	3.5	33	3.5	33
	4	34	4	34
	5	40	5	40
	6	55	6	55
CSS	0	0	0	0
	6	0	6	0
	12	32	12	32
	15	55	15	55

4.3 Low-duty-cycle assumption

In general, IEEE 802.15.4 devices address low-duty-cycle applications. The assumption of 1% duty cycle for IEEE 802.15.4 devices was introduced in 1.2.4. Under the assumption that IEEE 802.15.4 devices are battery-powered and have a lifetime of at least one year, the 1% assumption can be hardened by taking into account state-of-the-art numbers: A typical AA battery has a capacity of 1.8 Ah. A typical IEEE 802.15.4 device operating at 2.4 GHz has a transmit current of 30 mA. If the device only transmits during its entire lifetime, the result would be 30/1800 = 60 h of operation. Over a lifetime of one year (365 × 24 h = 8760 h), the duty cycle would be 0.0068, which is clearly below 1%. In reality, traffic generated by several nodes might accumulate. On the other hand, a significant part of the battery power will be spent in receive mode (which requires more current than the transmit mode for many implementations). Thus the 1% duty cycle also is valid for networks of IEEE 802.15.4 devices. In some rare cases, traffic might aggregate in proximity to coordinator nodes. Thus an aggregated duty cycle of up to 10% can be assumed in rare cases.

4.4 Impact of increased data rate

It should be noted that IEEE 802.15.4 devices will serve applications with similar low required data traffic. Since CSS devices offer a significantly increased data rate (1 Mb/s versus 250 kb/s), the duty cycle of IEEE CSS devices can be expected to be significantly below the duty cycle of other IEEE 802.15.4 devices. Since the 2.4 GHz ISM band has become an extremely busy medium, a low duty cycle achieved by high data rates is crucial for reasonable coexistence performance.

4.5 Co-channel scenario

Operating any two systems at the same location and at the same center frequency is obviously not a desirable situation. As long as no active interference cancellation is provided, the coexistence performance will be determined by the duty cycle behavior of both systems. Applying the duty cycle assumptions on CSS devices as stated above will result in reasonable performance. However, whenever possible, it is recommended that this situation be avoided by using a nonoverlapping channel. When a nonoverlapping channel is not available to the CSS PHY, because other networks (for example, IEEE 802.11 networks) are themselves already using the nonoverlapping channels, the recommendation is to select for the CSS PHY a channel between the channels already in use. It is further recommended that in the case of IEEE 802.11 networks, the CSS center frequency be selected so that the spatially closer IEEE 802.11 network has a frequency offset of at least 15 MHz.

The figures in this subclause show the computed PER versus separation distances (in meters) for co-channel pairings of systems when those systems use the spectra and filter properties given in Table 6.

Figure 20 illustrates the effect on a CSS receiver in the presences of an 802.11 HR/DSSS interferer.

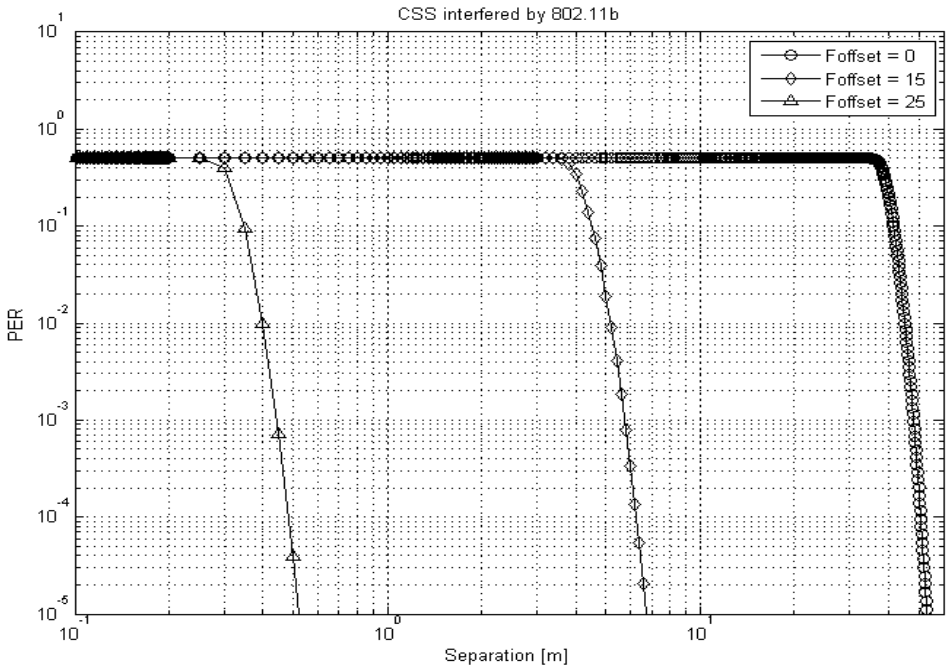


Figure 20—IEEE 802.15.4 CSS receiver, IEEE 802.11 HR/DSSS interferer

Figure 21 illustrates the effect on an 802.11 HR/DSSS receiver in the presence of a CSS interferer with normal duty cycle.

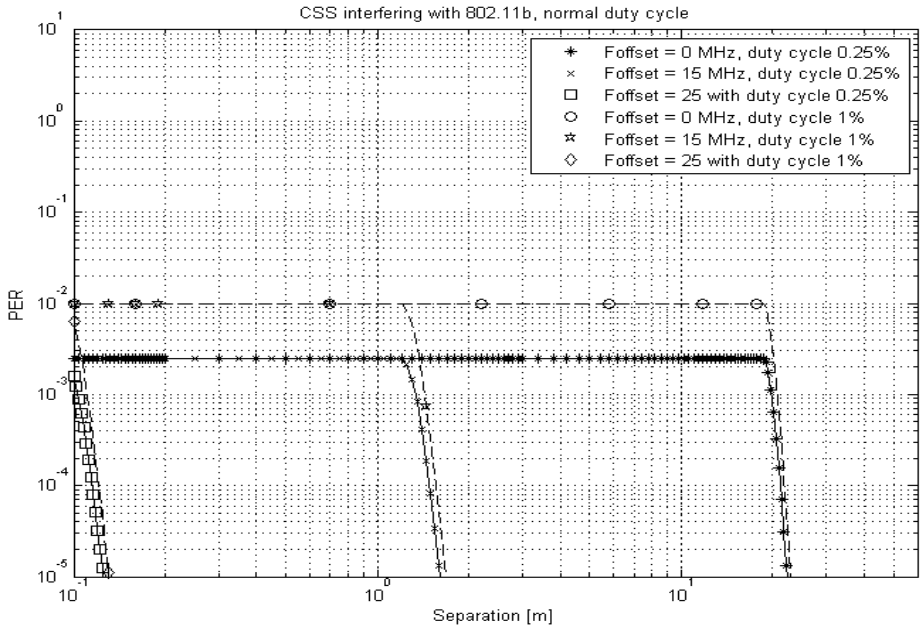


Figure 21—IEEE 802.11 HR/DSSS receiver, IEEE 802.15.4 CSS interferer with normal duty cycle

Figure 22 illustrates the effect on an 802.11 HR/DSSS receiver in the presence of a CSS interferer with rare duty cycle.

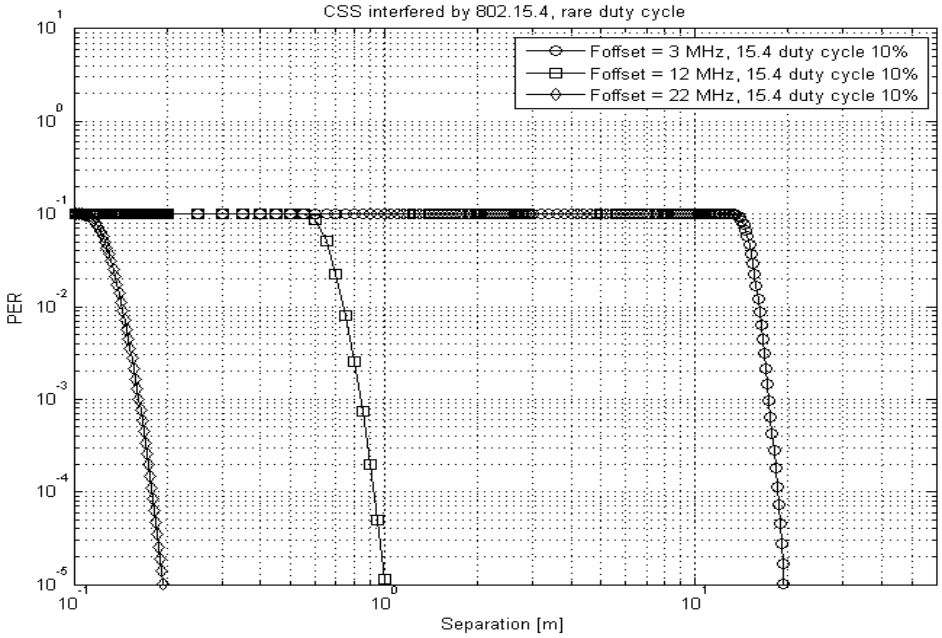


Figure 22—IEEE 802.11 HR/DSSS receiver, IEEE 802.15.4 CSS interferer with rare duty cycle

Figure 23 illustrates the effect on a CSS receiver in the presences of an 802.11 HRP interferer.

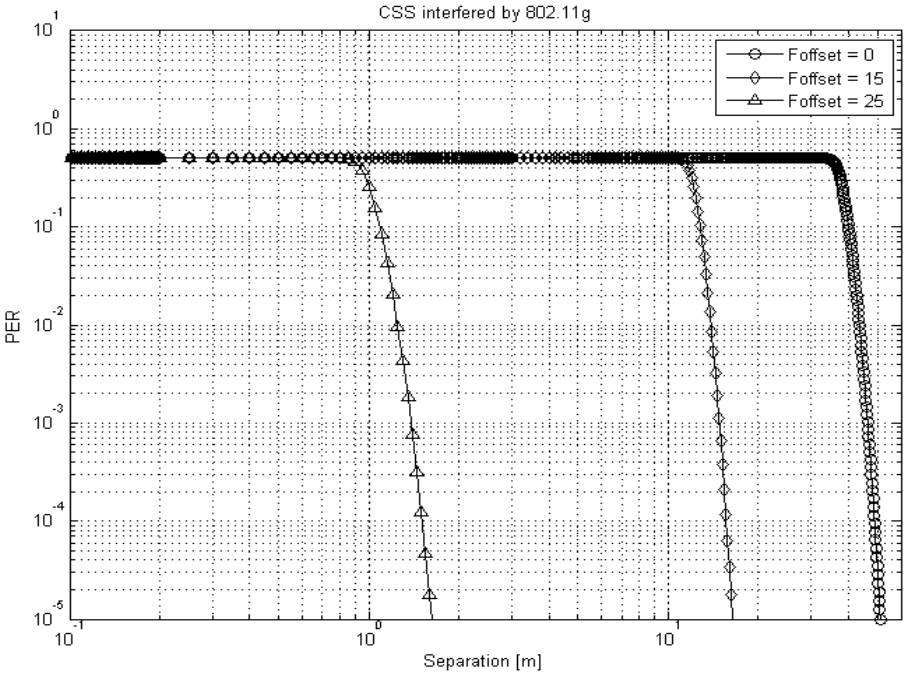


Figure 23—IEEE 802.15.4 CSS receiver, IEEE 802.11 ERP interferer

Figure 24 illustrates the effect on an 802.11 ERP receiver, 6 Mb/s, in the presence of a CSS interferer with normal duty cycle.

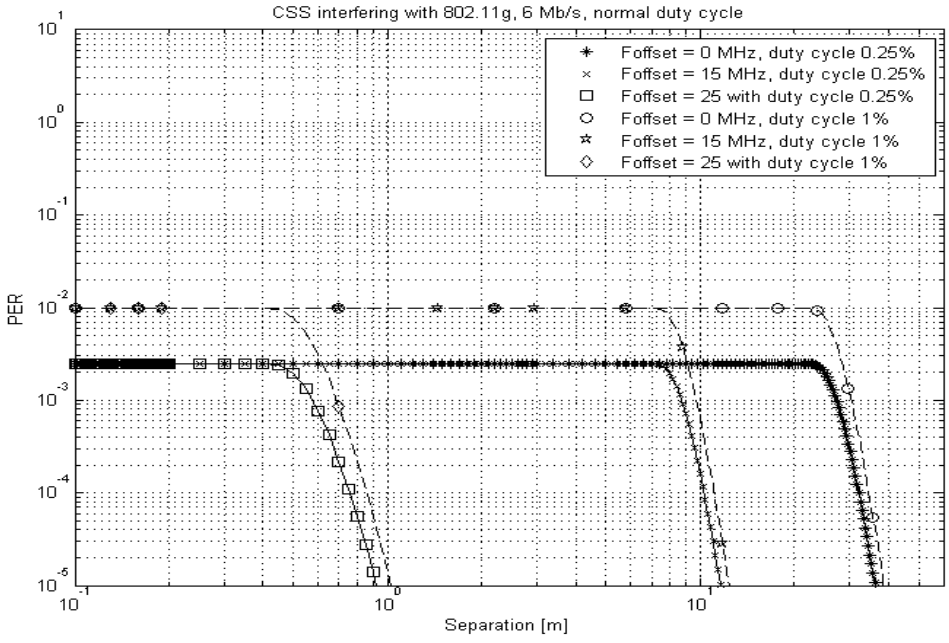


Figure 24—IEEE 802.11 ERP receiver, 6 Mb/s, IEEE 802.15.4a CSS interferer with normal duty cycle

Figure 25 illustrates the effect on an 802.11 ERP receiver, 6 Mb/s, in the presence of a CSS interferer with rare duty cycle.

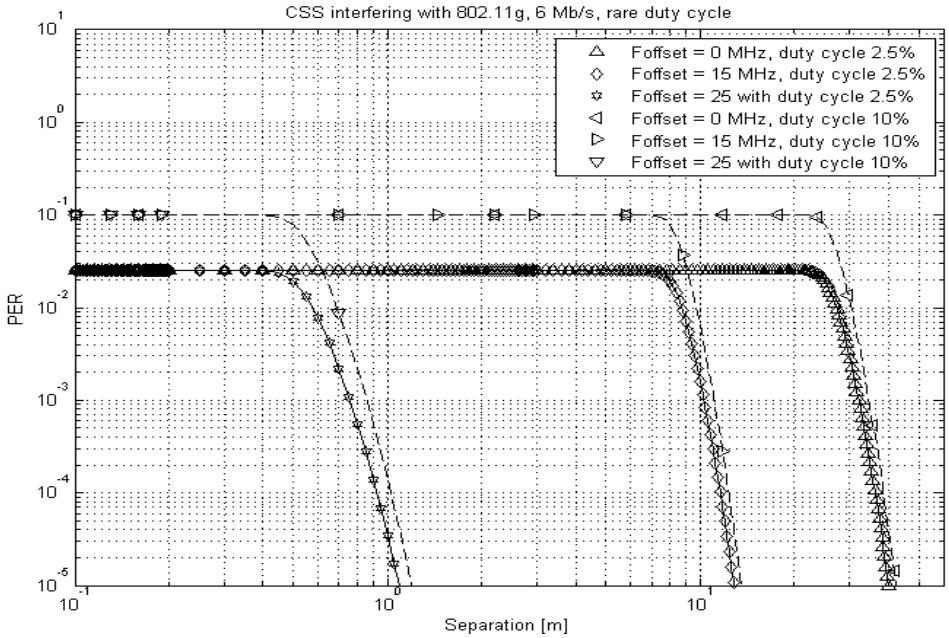


Figure 25—IEEE 802.11 ERP receiver, 6 Mb/s, IEEE 802.15.4 CSS interferer with rare duty cycle

Figure 26 illustrates the effect on an 802.11 ERP receiver, 24 Mb/s, in the presence of a CSS interferer with normal duty cycle.

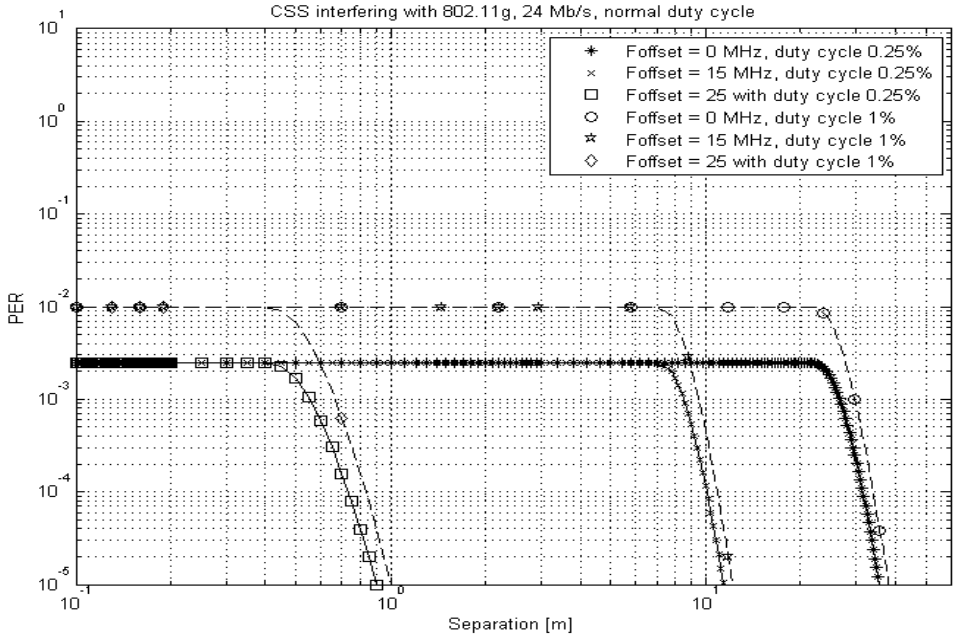


Figure 26—IEEE 802.11 ERP receiver, 24 Mb/s, IEEE 802.15.4a CSS interferer with normal duty cycle

Figure 27 illustrates the effect on an 802.11 ERP receiver, 24 Mb/s, in the presence of a CSS interferer with rare duty cycle.

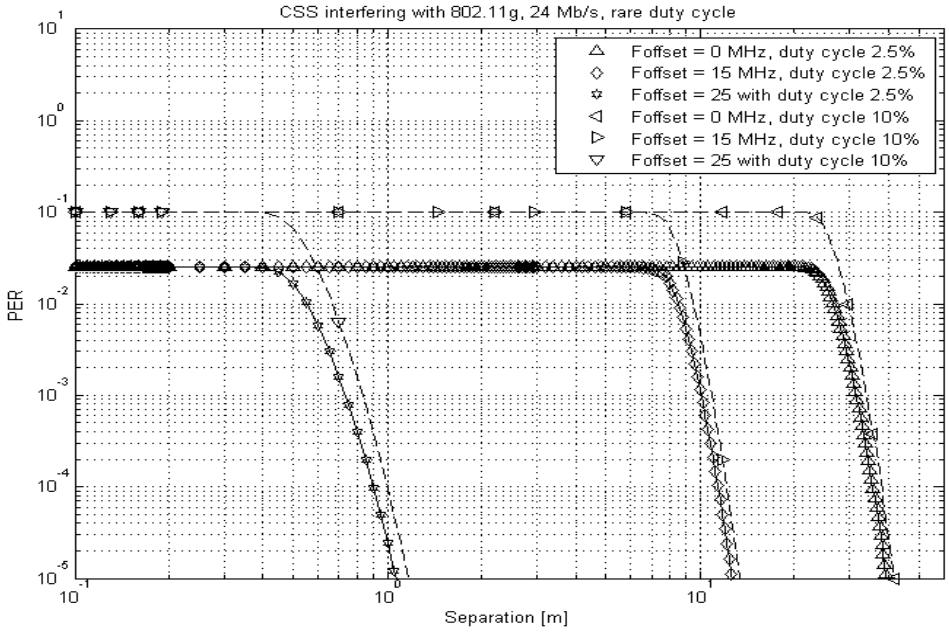


Figure 27—IEEE 802.11 ERP receiver, 24 Mb/s, IEEE 802.15.4 CSS interferer with rare duty cycle

Figure 28 illustrates the effect on an 802.11 ERP receiver, 54 Mb/s, in the presence of a CSS interferer with normal duty cycle.

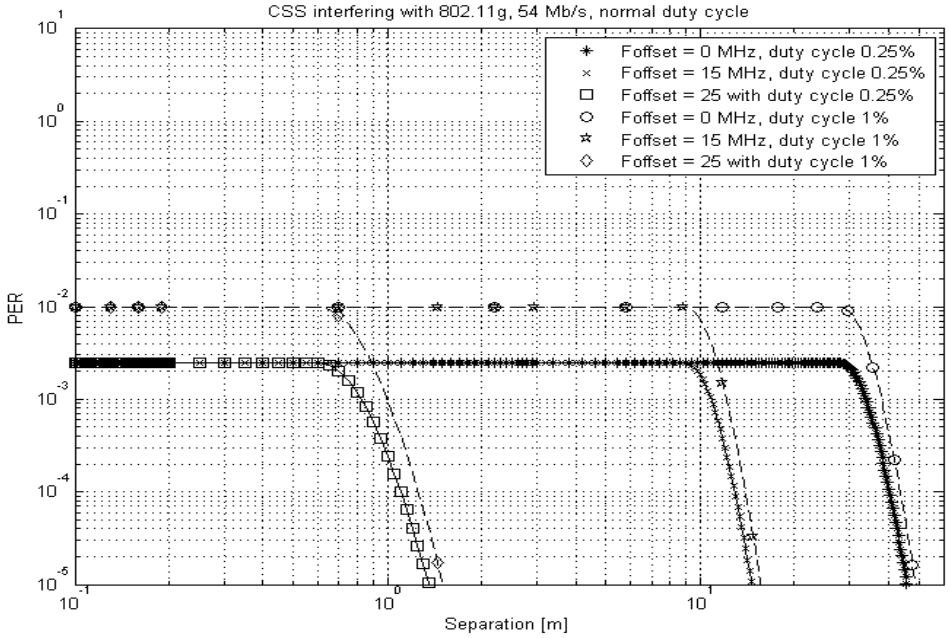


Figure 28—IEEE 802.11 ERP receiver, 54 Mb/s, IEEE 802.15.4a CSS interferer with normal duty cycle

Figure 29 illustrates the effect on an 802.11 ERP receiver, 54 Mb/s, in the presence of a CSS interferer with rare duty cycle.

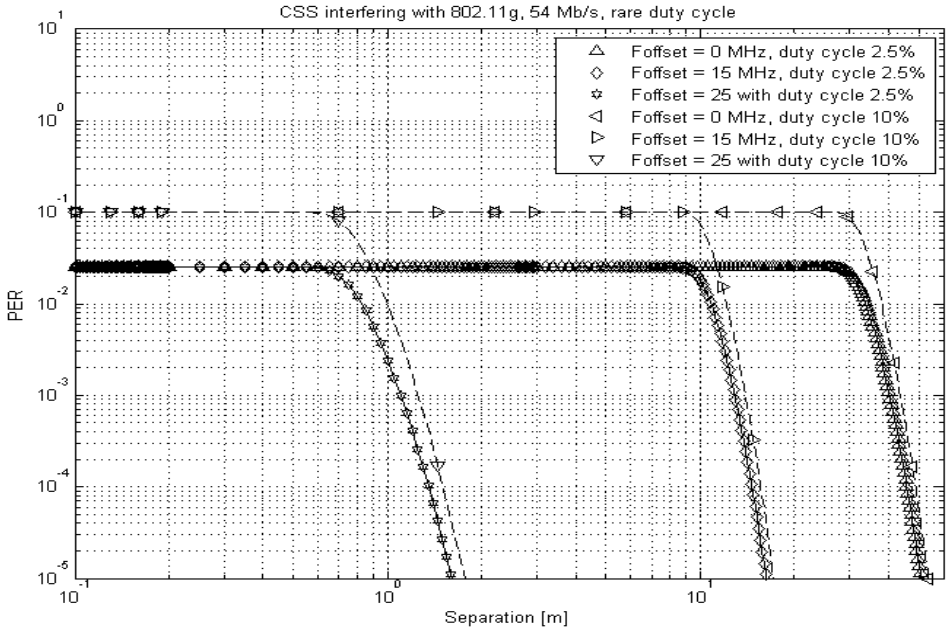


Figure 29—IEEE 802.11 ERP receiver, 54 Mb/s, IEEE 802.15.4 CSS interferer with rare duty cycle

Figure 30 illustrates the effect on a CSS receiver in the presences of an 802.15.1 interferer.

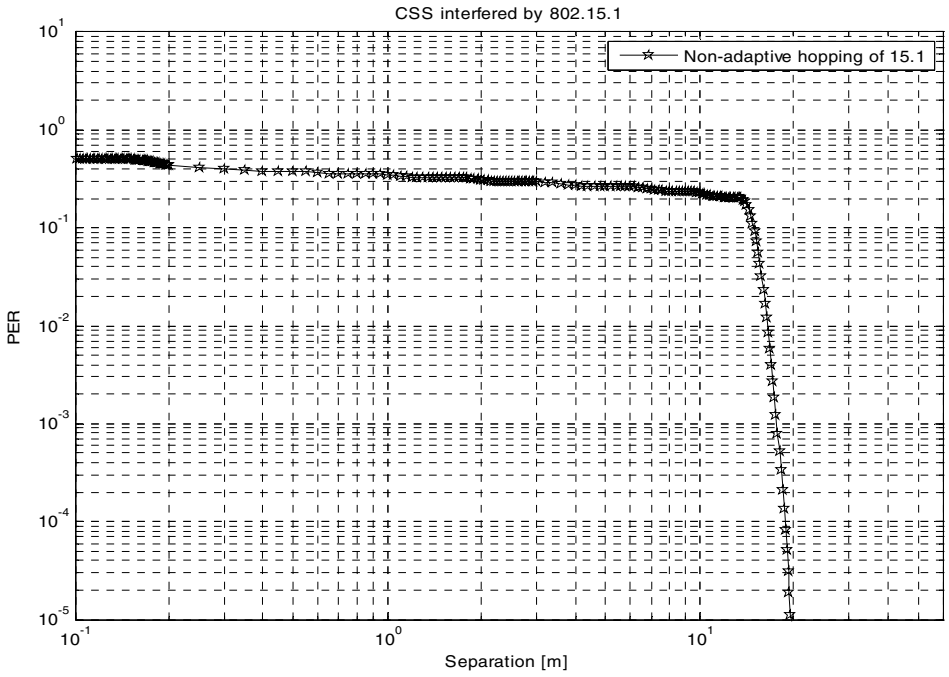


Figure 30—IEEE 802.15.4 CSS receiver, IEEE 802.15.1 interferer

Figure 31 illustrates the effect on an 802.15.1 receiver in the presence of a CSS interferer with normal duty cycle.

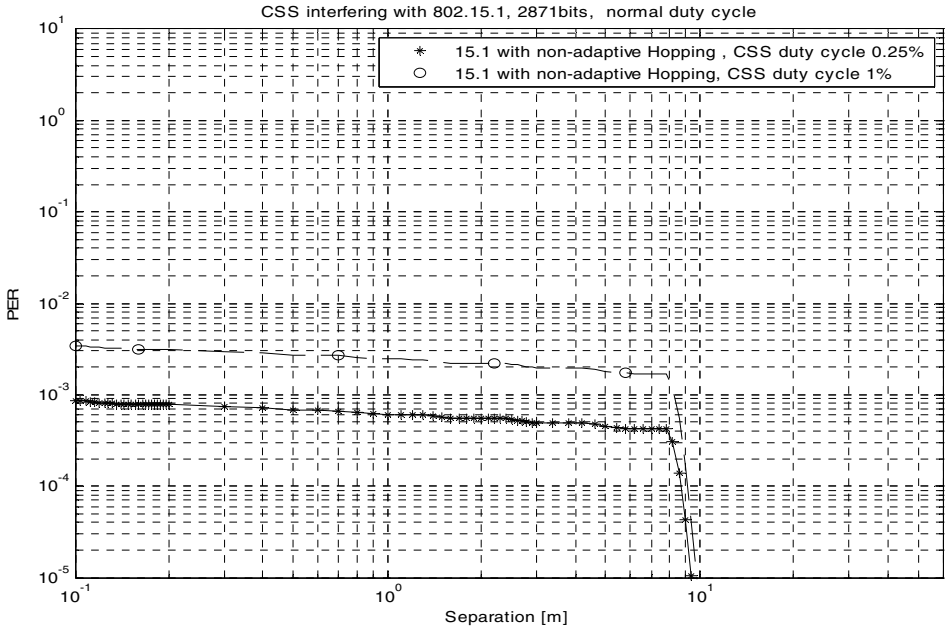


Figure 31—IEEE 802.15.1 receiver, IEEE 802.15.4 CSS interferer with normal duty cycle

Figure 32 illustrates the effect on an 802.15.1 receiver in the presence of a CSS interferer with rare duty cycle.

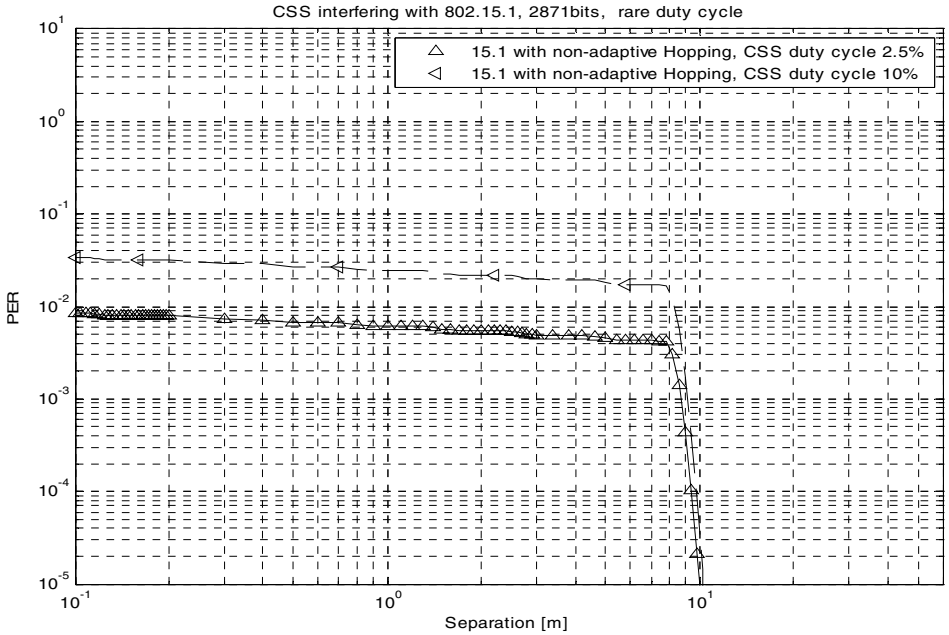


Figure 32—IEEE 802.15.1 receiver, IEEE 802.15.4 CSS interferer with rare duty cycle

Figure 33 illustrates the effect on a CSS receiver in the presences of an 802.15.3 interferer.

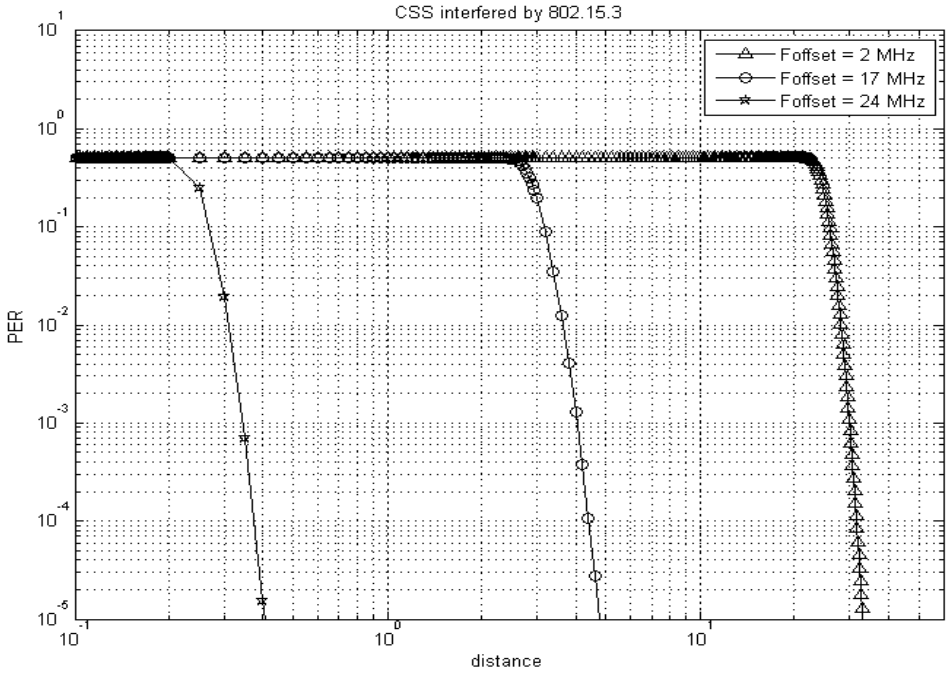


Figure 33—IEEE 802.15.4 CSS receiver, IEEE 802.15.3 interferer

Figure 34 illustrates the effect on an 802.15.3 receiver in the presence of a CSS interferer with normal duty cycle.

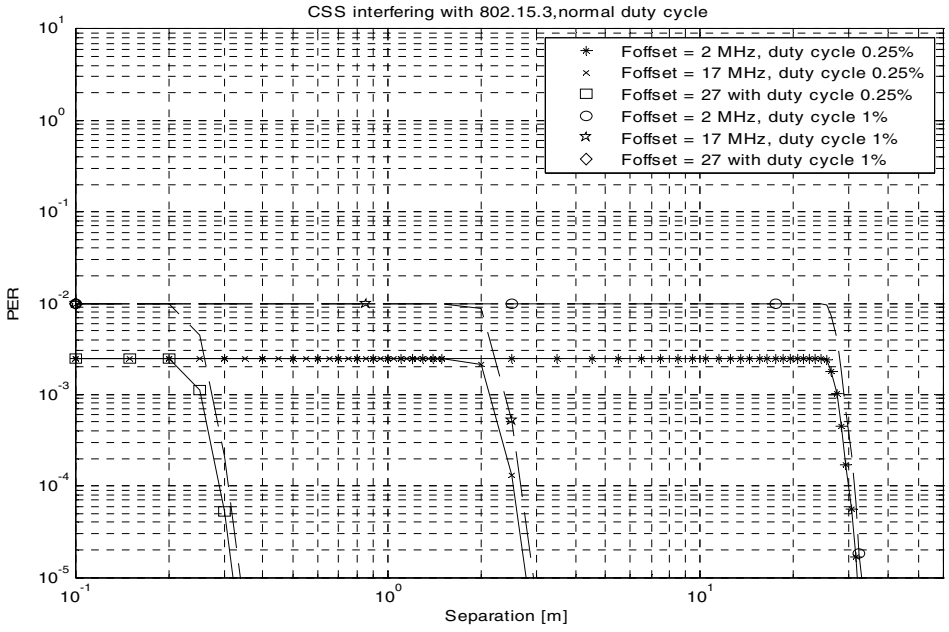


Figure 34—IEEE 802.15.3 receiver, IEEE 802.15.4 CSS interferer with normal duty cycle

Figure 35 illustrates the effect on an 802.15.3 receiver in the presence of a CSS interferer with rare duty cycle.

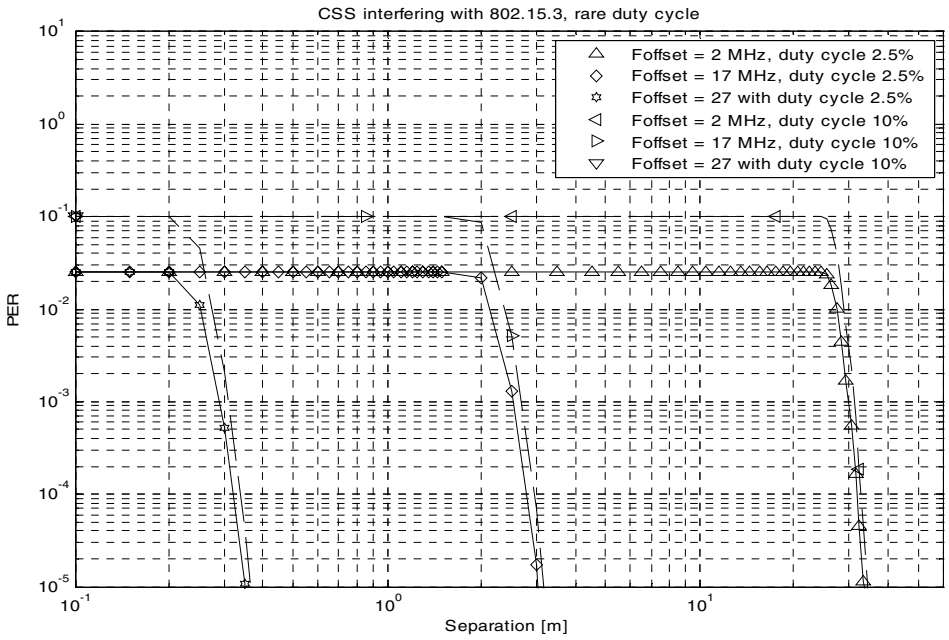


Figure 35—IEEE 802.15.3 receiver, IEEE 802.15.4 CSS interferer with rare duty cycle

Figure 36 illustrates the effect on a CSS receiver in the presence of a O-QPSK interferer with normal duty cycle.

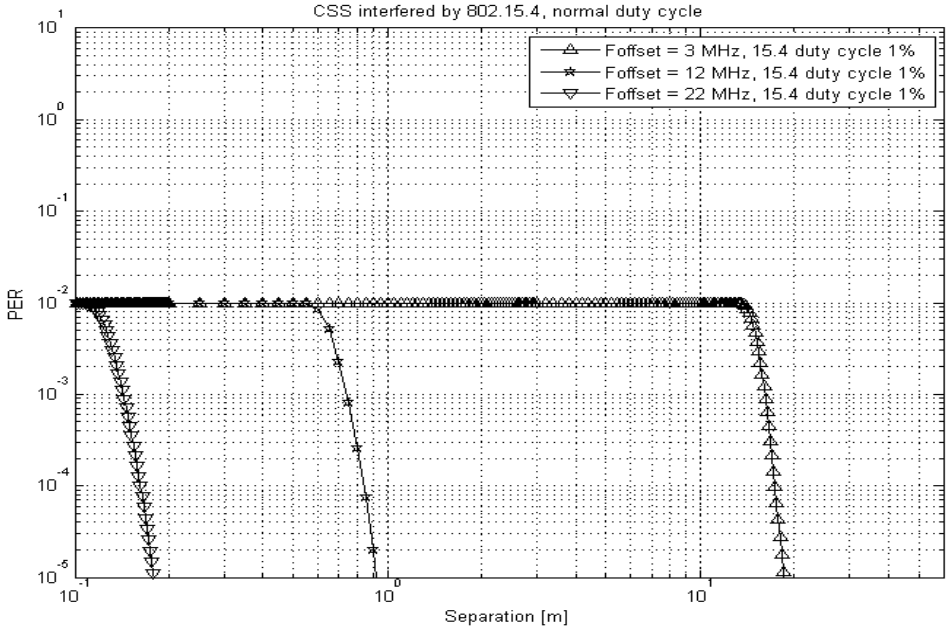


Figure 36—IEEE 802.15.4 CSS receiver, IEEE 802.15.4 O-QPSK interferer with normal duty cycle

Figure 37 illustrates the effect on a CSS receiver in the presence of a O-QPSK interferer with rare duty cycle.

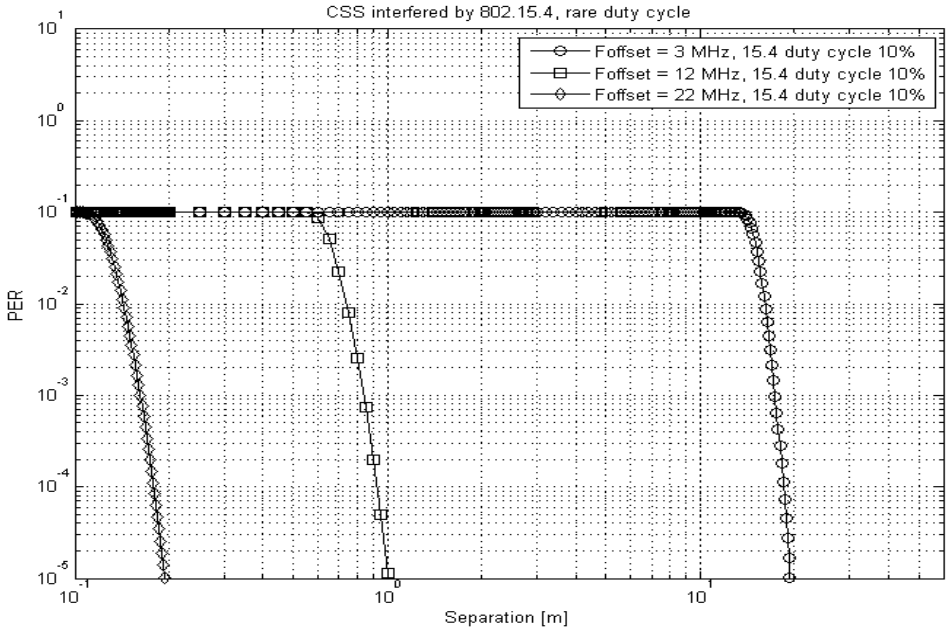


Figure 37—IEEE 802.15.4 CSS receiver, IEEE 802.15.4 O-QPSK interferer with rare duty cycle

Figure 38 illustrates the effect on an O-QPSK receiver in the presence of a CSS interferer with normal duty cycle.

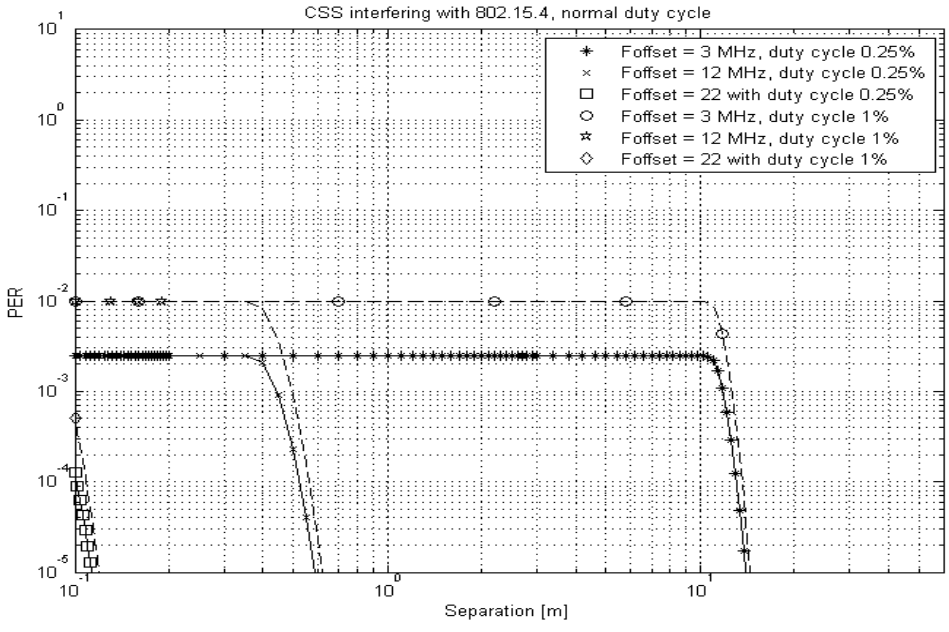


Figure 38—IEEE 802.15.4 O-QPSK receiver, IEEE 802.15.4 CSS interferer with normal duty cycle

Figure 39 illustrates the effect on an O-QPSK receiver in the presence of a CSS interferer with rare duty cycle.

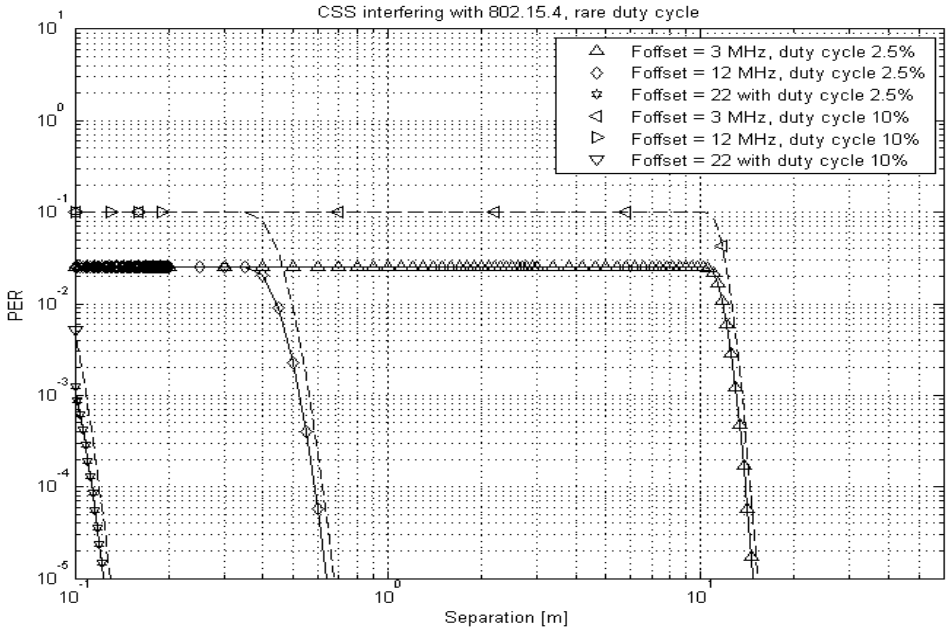


Figure 39—IEEE 802.15.4 O-QPSK receiver, IEEE 802.15.4 CSS interferer with rare duty cycle

5. UWB coexistence performance

5.1 Specific regulatory requirements for UWB coexistence

Surprisingly, despite the wide bandwidth of the UWB PHY, there is only one other IEEE standard waveform that may occupy the same frequency bands, namely, IEEE 802.16 systems below 10 GHz. Cognizant of the potential for coexistence issues, regulators in the parts of the world where IEEE 802.16 systems may be deployed in bands overlaid by UWB spectrum are creating specific regulatory requirements to further reduce the likelihood of any coexistence problems. In both Asia and the European Union, regulators are creating rules for unlicensed UWB operation that will require specific active mitigation mechanisms to ensure peaceful coexistence with IEEE 802.16 systems or other similar systems used for fixed or mobile wireless access.

Additionally, a proposed IEEE standard, P802.22, proposes to occupy parts of the bandwidth in the UWB PHY 150–650 MHz band. In the regulatory domains where this is presently allowed (FCC), the maximum transmit power is specified an additional (approximately) 35 dB lower compared the limits for the 3.1–10 GHz bands. Some regulatory domains (including FCC) have suggested that certain applications, specifically those involving personnel location in emergency response situations, would be allowed at higher PSD levels under specific conditions, where other factors such as operating limitations would provide required protection of incumbent services. Clearly it is beyond the scope of this standard to anticipate specific future regulatory actions. However, in considering the application scenarios presented in the call for applications and responding to specific guidance from regulators in the United States, it can be observed that coexistence with the IEEE P802.22 systems and other known incumbent systems is assured through operating conditions. As a primary mitigation factor, it is unlikely such systems will be operating in near physical proximity at the same time as emergency response teams. Such conditions are the scope of

regulatory agencies to define, and it is the responsibility of implementers of this standard to conform with applicable regulations and conditions.

In considering other personnel location scenarios, the mitigations factors described for other UWB applications apply equally to all UWB bands.

5.2 Mitigation of interference from UWB PHY devices using low duty cycle PANs

One proposal is to use a lower duty cycle within a UWB WPAN to reduce potential interference effects. Low-duty-cycle WPAN scenarios could be used in the following situations:

— UWB PHY devices are deployed in high density in a limited area, e.g., hot-spot deployment scenarios.
— UWB victim systems cover much larger area than the coverage of a typical LR-WPAN.

In these cases, transmissions from every device in the WPAN can affect the victim receiver. For reasons of less complexity, lower power consumption, as well as physical limitations, it is difficult for simple UWB PHY devices to detect victim system reliably. The aggregate interference from the WPAN increases with increment in number of WPAN members. The interference to victim systems could be limited by controlling duty cycle of the WPAN through general active/inactive periods. The UWB traffic can occur only in the active period. Victim systems would then be free of interference in the inactive period. The interference level could be controlled by the ratio of active period to the total period.

5.3 Coexistence assurance: methodology and assumptions

In order to quantify the coexistence performance of the IUWB PHY, the techniques described by Shellhammer [B6] have been adapted.

The coexistence assurance methodology predicts the PER of an affected wireless network (AWN, or victim) in the presence of an interfering wireless network (IWN, or assailant). It its simplest form, the methodology assumes an AWN and an IWN, each composed of a single transmitter and a receiver. The methodology takes as input a path loss model, a quantitative model for the BER of the AWN, and predicted temporal models for packets generated by the AWN and for “pulses,” i.e., packets generated by the IWN. Based on these inputs, the methodology predicts the PER of the AWN as a function of the physical spacing between the IWN transmitter and the AWN receiver.

The appeal of the coexistence assurance methodology is that multiple networking standards can be characterized and compared with just a few parameters, notably,

— Bandwidth of AWN and IWN devices
— Path loss model for the networks
— BER as a function of SIR of AWN devices
— Temporal model for AWN packets and IWN “pulses” (interfering packets)

The following subclauses describe the general assumptions made across all of the PHYs covered under this standard.

5.3.1 Victims and assailants

At present, this is the only standard for UWB systems in the UWB bands covered under IEEE Std 802®. The only other IEEE wireless standard waveforms that overlap this same spectrum are IEEE 802.16 systems

occupying 3.4–3.8 GHz licensed frequency bands in some regions (parts of Europe and Asia). In addition, the proposed standard IEEE P802.22 would occupy parts of the band between 150 MHz to 650 MHz.

In addition to IEEE standardized wireless systems, another UWB standard produced by ECMA is specified in ECMA 368. A limited analysis of the coexistence between this system and UWB PHY waveform is given here.

In this analysis, the assumption is made that the PHYs will serve as both victims (i.e., participants in AWNs) and as assailants (i.e., participants in IWNs).

5.3.2 Bandwidth for UWB systems

The UWB PHYs in this standard that operate in any of the three UWB bands have one or more channels, approximately 500 MHz wide or, optionally, 1300 MHz wide. The ECMA 368 PHY has a nominal bandwidth of 1500 MHz. In contrast to these UWB systems, the narrowband IEEE 802.16 PHYs that operate in the 2–10 GHz band have multiple defined channels, each 20 MHz wide or less. IEEE P802.22 would have multiple defined channels, each 6 MHz to 8 MHz wide. The coexistence methodology assumes that any UWB device in an AWN or IWN will have a much greater bandwidth than a narrowband device in a corresponding AWN or IWN.

5.3.3 Path loss model

The coexistence methodology uses a variant of the path loss model described by Shellhammer [B7] which stipulates a two-segment function with a path loss exponent of 2.0 for the first 8 meters and then a path loss model of 3.3 thereafter. The generalized form developed in 3.3 is used in this analysis:

Using f = 3400 MHz, then $pl(1)$ = 43.03 and $pl(8)$ = 61.09. The path loss function modified for 3400 MHz is, therefore,

$$pl(d) = \begin{cases} 43.08 + 20\log_{10}(d) & d \le 8 \text{ m} \\ 61.09 + 33\log_{10}\left(\frac{d}{8}\right) & d > 8 \text{ m} \end{cases}$$

Using f = 400 MHz for the sub-gigahertz UWB band, then $pl(1)$ = 24.49 and $pl(8)$ = 42.55. The path loss function for 400 MHz center frequency is then

$$pl(d) = \begin{cases} 24.49 + 20\log_{10}(d) & d \le 8 \text{ m} \\ 42.55 + 33\log_{10}\left(\frac{d}{8}\right) & d > 8 \text{ m} \end{cases}$$

A plot of the path loss as a function of device separation distance is shown in Table 40.

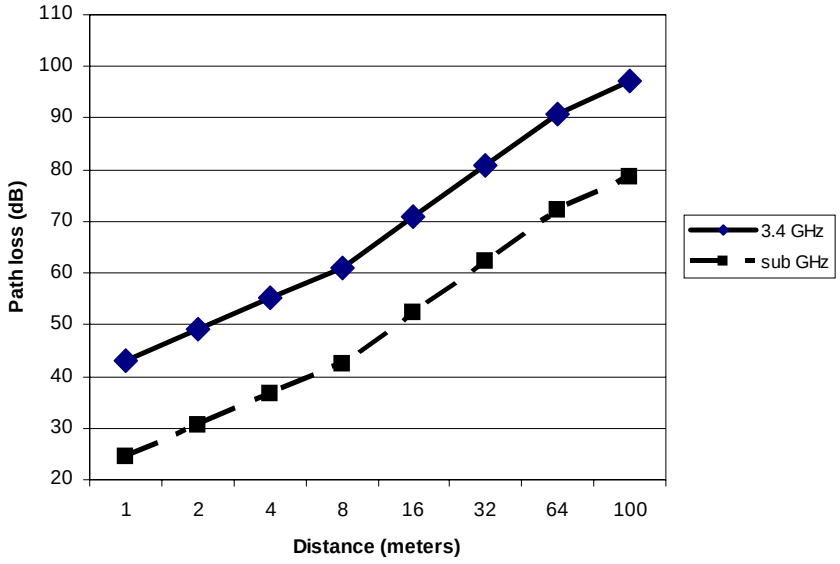


Figure 40—Path loss function

5.3.4 BER as a function of SIR

For the PHY specifications analyzed in this standard, there are no analytic expressions for the BER or symbol error rate (SER) of the signal due to the use of FEC methods to improve reliability.

In this analysis, a method is used that is equivalent to using interpolation of table values. In order to simplify the calculations and still provide meaningful results, the relationship is approximated between the changes in BER (on a logarithmic scale) and varying SNR as a linear with a slope of 0.6 dB per order of magnitude (10×) change in BER over the range of BER that is relevant to this analysis (about 1e–8 to 1e–5 BER). This approximation is reasonable for the FEC methods used for IEEE Std 802.16-2004 (Reed-Solomon block code), ECMA 368, IEEE P802.22, and the UWB PHY in this standard (convolutional coding).

For each of the systems, the effect of the IWN on the AWN is characterized by computing the rise in the effective operating noise floor of the AWN by the interference of the IWN (modeled as uncorrelated wideband noise). The analysis will assume a baseline operating effective noise floor (including effects of thermal noise floor, noise figure, and operating margin to account for other real-world effects such as multipath propagation effects and co-channel or adjacent channel interference). This approach allows the characterization of the effect of the IWN on the AWN as the IWN is moved from a large separation distance (when the AWN has a baseline nominal PER) to a very close distance where the interference effect of the IWN dominates the PER during periods of operation (subject to duty cycle assumptions).

Although this analysis approach is perhaps not as elegant as the use of an analytic expression (not possible in these cases), it will provide a good characterization of the coexistence of these systems under real-world conditions and can be used to estimate a range of effects for an equivalent range of assumptions about operating margin.

5.3.5 Temporal model

For the UWB PHY, packet overhead is kept to minimum. The maximum PSDU size is 128 octets, and a typical packet is only 32 octets, including PSDU and synchronization octets. For this coexistence methodology, all packets, whether belonging to the AWN or IWN, are assumed to be 32 octets.

Although there is no duty-cycle limitation in the authorized UWB bands at this point, many IEEE 802.15.4 UWB PHY networks are expected to operate at well under 5% duty cycle, particularly devices that are battery-powered. This 5% duty cycle level has also been used by regulators as a high value for expected UWB communications device operating levels on various coexistence studies. In addition, the UWB PHYs in this standard use an ALOHA contention-based access mechanism that is intended to support only lower duty cycle applications. Based on these factors, it is reasonable to expect that UWB PHY piconets used for many applications will operate at duty cycles as high as 10%. For purposes of modeling coexistence, the assumption is made that all UWB PHY devices operating in piconets will have a shared duty cycle of 10% and that such piconets will operate within a range of a few tens of meters. Based on this and a typical active device population of five devices per piconet, an average operating duty cycle of 2% is assumed for any particular device within a piconet.

For the other wireless systems considered in this analysis (IEEE 802.16, IEEE P802.22, and ECMA 368), anticipated applications are focused on higher bandwidth connectivity over wide areas for IEEE 802.16 and IEEE P802.22 systems and over short WPAN ranges for ECMA 368 systems. Because these systems are not deployed in great numbers, it is not possible to qualify typical operating duty cycle. For this analysis, therefore, the initial assumption is a very conservative continuous operation as a baseline worst-case scenario.

5.4 Coexistence analysis

This subclause details the assumptions for the coexistence analysis and presents the results for each of the cases analyzed.

5.4.1 Impact of UWB PHY devices on IEEE 802.16 networks

The assumptions for this scenario are:

— The IEEE 802.16 receiver is the victim (AWN) and is an indoor fixed or nomadic client node of the network. The base station node will not be susceptible to IEEE 802.15.4a UWB interference due to site positioning. The AWN operates in 3.4–3.8 GHz licensed bands (available in most of the world except the United States).

— The IEEE 802.16 receiver is operating in a real-world environment in the presence of multipath fading and interference, and a 3–10 dB margin above sensitivity functions well. The baseline PER is 1e–6 at 3 dB above sensitivity in the absence of any UWB device effects, and the receiver noise floor is 6 dB.

— UWB interference is wideband uncorrelated noise since the bandwidth is much wider than victim receiver. The difference in antenna gains is 10 dB since the indoor or outdoor IEEE 802.16 antenna will have gain in the direction of the desired base station downlink signal. The UWB device will not directly block the LOS.

Table 7 shows the calculation of the allowable path loss that would result in an IEEE 802.15.4a UWB emission level at the AWN equal to the effective operating noise floor.

Table 7—Computation of acceptable levels of UWB PHY device emissions for an operating IEEE 802.16 client node

Quantity	Symbol	Value	Units	Notes
UWB transmit PSD limit	P_{lim}	–41.3	dBm/MHz	Set by regulatory authority.
Average margin to limit	M_{B0}	1.7	dB	Transmit power back-off due to spectral ripple (0.5+ dB) and ~1 dB margin for manufacturing tolerance, etc.
Average UWB antenna gain	G_{UWB}	–2	dBi	Average gain from small, low-cost UWB antenna to arbitrary victim receiver over 360°.
Average emissions PSD seen by IEEE 802.16 device receiver	–	–45	dBm/MHz	Average PSD seen in direction of arbitrary victim receiver. ($P_{lim} - M_{B0} + G_{UWB}$)
IEEE 802.16 thermal noise floor	kTB	–114	dBm/MHz	Thermal noise floor (room temperature).
IEEE 802.16 noise figure	NF_{16}	6	dB	Noise figure for indoor IEEE 802.16 terminal.
Average IEEE 802.16 antenna gain in direction of interfering UWB	G_{16}	–4	dBi	Gain of IEEE 802.16 antenna in main beam (to desired IEEE 802.16 base station) is 6–7 dBi and to nearby UWB interferer (not blocking antenna main beam)
IEEE 802.16 operating margin	M_{16}	3–10	dB	Operating margin for acceptable performance in presence of multipath fading and adjacent cell/channel interference.
IEEE 802.16 effective operating noise floor for UWB interference susceptibility	–	–101 to –94	dBm/MHz	The effective operating noise floor level for the IEEE 802.16 operating receiver. ($kTB + NF_{16} - G_{16} + M_{16}$)
Level of wideband UWB PHY interference that result in a 3 dB rise in IEEE 802.16 effective operating noise floor	–	–101 to –94	dBm/MHz	For 3 dB rise, wideband UWB emissions in-band can be at the same level as effective operating noise floor for indoor IEEE 802.16 node receiver.
Path loss (range) from UWB to IEEE 802.16 receiver (average case) for 3 dB rise in effective operating noise floor	–	49 to 56 (2 to 4.5)	dB (m)	For 3 dB rise, wideband UWB emissions in-band can be at the same level as effective operating noise floor for indoor IEEE 802.16 node receiver.
Path loss (range) from UWB to IEEE 802.16 receiver (average case) for 1 dB rise in effective operating noise floor	–	55 to 61 (4 to 8)	dB (m)	For 1 dB rise, wideband UWB emissions in-band must be 6 dB below effective operating noise floor for indoor IEEE 802.16 node receiver.

Based on this path loss, the effect on AWN PER is computed as a function of separation distance, shown in Figure 41.

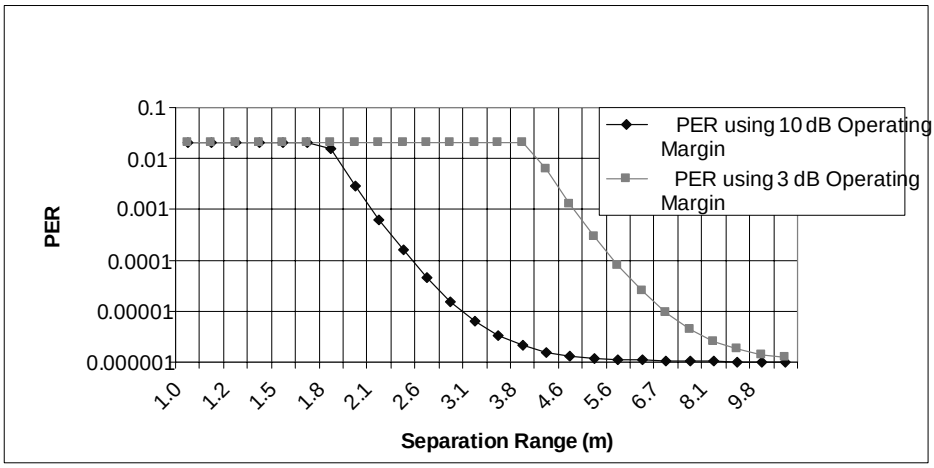


Figure 41—Effect on IEEE 802.16 AWN as a function of separation distance from a UWB PHY device

5.4.2 Impact of an IEEE 802.16 device on IEEE 802.15.4a UWB networks

The assumptions used in this analysis are:

— The IEEE 802.15.4a UWB device is the affected device (AWN). The IEEE 802.16 device is the interferer (IWN) and is an indoor fixed or nomadic client node of the network. The base station node will have less interference effects on IEEE 802.15.4a UWB devices due to UWB device deployment much closer to subscriber or mobile IEEE 802.16 devices. The IWN operates in 3.4–3.8 GHz licensed bands (available in most of the world except the United States). For this analysis, the IWB operates at a conservative 50% duty cycle (IEEE 802.16 subscriber uplink).

— The IEEE 802.15.4a UWB receiver is operating in a real-world environment in the presence of multipath fading and interference, and the margin above sensitivity is 3 dB during operation. The baseline PER is 1e–7 at 3 dB above sensitivity in the absence of any UWB device effects, and the receiver noise floor is 10 dB.

— UWB interference is wideband uncorrelated noise since the bandwidth is much wider than victim receiver. The difference in antenna gains is 10 dB since the indoor or outdoor IEEE 802.16 antenna will have gain in the direction of the desired base station downlink signal. The UWB device will not directly block the LOS.

Table E.7 shows the calculation of the allowable path loss that would result in a IEEE 802.15.4a UWB emission level at the AWN equal to the effective operating noise floor.

Table 8—Computation of acceptable levels of IEEE 802.15.4a device emissions for an operating IEEE 802.16 client node

Quantity	Symbol	Value	Units	Notes
IEEE 802.16 client device transmit power	P_{16}	17	dBm	Assumes subscriber station in small cell.
IEEE 802.16 client device bandwidth	B_{16}	5	MHz	
UWB device bandwidth	B_{UWB}	500	MHz	
Average IEEE 802.16 antenna gain	G_{16}	–2	dBi	Average gain from antenna to arbitrary victim receiver over 360° (IWN typically not in main beam).
Average emissions PSD seen by UWB PHY device receiver	–	–12	dBm/MHz	Average PSD seen in direction of arbitrary victim receiver (assumes that UWB receiver can spread interference power into receiver bandwidth). $P_{16} + G_{16} - 10\log(B_{UWB})$
UWB PHY thermal noise floor	kTB	–114	dBm/MHz	Thermal noise floor (room temperature).
UWB PHY noise figure	NF_{UWB}	10	dB	Noise figure for low-cost UWB PHY device.
UWB PHY operating margin	M_{UWB}	3	dB	Operating margin for acceptable performance in presence of multipath fading (assumes no interference other than IWN).
UWB PHY effective operating noise floor for UWB interference susceptibility.	–	–101	dBm/MHz	The effective operating noise floor level for the IEEE 802.15.4a operating receiver. $kTB + NF_{UWB} + M_{UWB}$
Level of interference power density to achieve a 3 dB rise in UWB PHY effective operating noise floor	–	–101	dBm/MHz	For 3 dB rise, IEEE 802.16 power emissions in-band can be at the same level as effective operating noise floor for UWB receiver.
Path loss (range) from IEEE 802.16 to UWB receiver (average case) for 3 dB rise in effective operating noise floor	–	89 (48)	dB (m)	For 3 dB rise, IEEE 802.16 power emissions in-band can be at the same level as effective operating noise floor for UWB receiver.
Path loss (range) from IEEE 802.16 to UWB receiver (average case) for 1 dB rise in effective operating noise floor	–	95 (75)	dB (m)	For 1 dB rise, wideband UWB emissions in-band must be 6 dB below effective operating noise floor for indoor IEEE 802.16 node receiver.

Base on this path loss, the effect on AWN PER is computed as a function of separation distance, shown in Figure 42.

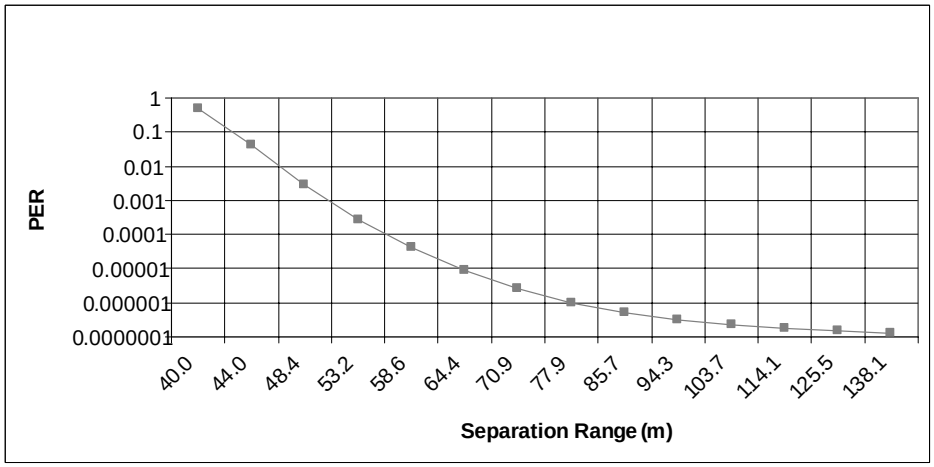


Figure 42—Effect on a UWB PHY device as a function of separation distance from an IEEE 802.16 IWN device

5.4.3 Low-duty-cycle UWB PHY interferring with a WiMAX link

These results are an extract from a French contribution to Electronic Communications Committee (ECC) Task Group 3 meeting #15.

The impact of UWB on a fixed broadband wireless access system is measured on video streaming is listed in Table 9. Video streaming is considered a relevant service in term of vulnerability, bandwidth use, and timing constraint.

Table 9—Impact of UWB on fixed broadband wireless access system measured on video streaming

Degradation (dB)		Distance (m)			
AF (T_{on}/T_{off} ms)		0.5	1	2	4
2%	075/38	1	N/A	1	N/A
	5/245	0	N/A	0	N/A
	10/490	1	N/A	1	N/A
5%	2/38	2	1	0	N/A
	5/95	1	N/A	1	N/A
	10/190	0	N/A	1	N/A
10%	2/18	3	N/A	0	0
	5/45	3	2	0	0
	10/90	2	N/A	1	0.5

The methodology used is the following:

— Set the WiMAX received signal strength at equipment at minimum sensitivity level (–98 dBm).
— Get a reference measure without UWB (depending on each test case).
— Measure the degradation with low-duty-cycle UWB emission [for any considered activity factor (AF) and distances]. Degradation is, in decibels, the increase of power needed by the WiMAX receiver to reestablish the reference link quality.

Table E.9 shows the evolution of the lowest needed receive signal strength indicator (RSSI) to achieve a reliable 1 Mb/s throughput with respect to UWB activity. The reference level is –98 dBm (i.e., without UWB activity).

Table 10—Lowest RSSI to achieve reliable 1 Mb/s throughput

RSSI needed to achieve 1 Mb/s data rate (dBm)		**Distance (m)**		
AF (T_{on}/T_{off} ms)		0.5	2	4
2%	075/38	–98 (–98)	–98 (–98)	N/A
	5/245	–98 (–98)	–98 (–98)	N/A
	10/490	–97 (–98)	–97 (–98)	N/A
5%	2/38	–98 (–98)	–98 (–98)	N/A
	5/95	–98 (–98)	–98 (–98)	N/A
	10/190	–97 (–98)	–98 (–98)	N/A
10%	2/18	–97 (–98)	–98 (–98)	N/A
	5/45	–98 (–98)	–98 (–98)	N/A
	10/90	–97 (–98)	–98 (–98)	N/A

5.4.4 Impact of UWB PHY devices on ECMA 368 networks

The assumptions in this analysis are:

— The ECMA 368 receiver is the victim (AWN). The AWN operates using frequency hopping in bands across the 3.1–4.8 GHz unlicensed UWB bands (available only in the United States at this time), but the IEEE 802.15.4a device operates only in band 3 (mandatory).
— The ECMA 368 receiver is operating in a real-world environment in the presence of multipath fading and interference, and a 5 dB margin above sensitivity functions well. The baseline PER is 8e–2 at sensitivity (8e–7 at 3 dB above sensitivity) in the absence of any UWB device effects, and the receiver noise floor is 6 dB.

Table 11 shows the calculation of the allowable path loss that would result in an IEEE 802.15.4a UWB emission level at the AWN equal to the effective operating noise floor.

Table 11—Computation of acceptable levels of a UWB PHY device emissions for an operating ECMA 368 device

Quantity	Symbol	Value	Units	Notes
UWB Transmit PSD Limit	P_{LIM}	–41.3	dBm/MHz	Set by regulatory authority
Average margin to limit	M_{BO}	1.7	dB	Due to spectral ripple (0.5+ dB) and ~1 dB margin for manufacturing tolerance, etc.
Average UWB antenna gain	G_{UWB}	–2	dBi	Average gain from small, low-cost UWB antenna to arbitrary victim receiver over 360°
Average emissions PSD	–	–45	dBm/MHz	Average PSD seen in direction of arbitrary victim receiver. $P_{LIM} - M_{BO} + G_{\mathrm{UWB}}$
ECMA 368 victim thermal noise floor	kTB	–114	dBm/MHz	Thermal noise floor (room temperature)
ECMA 368 victim noise figure	NF_{ECMA}	6	dB	Noise figure for the ECMA 368 receiver
ECMA victim frequency diversity	D_{FD}	3	dB	ECMA UWB system uses 2x band frequency diversity for then encoding of each bit as part of its frequency hopping scheme
UWB victim operating margin	M_{ECMA}	5	dB	Operating margin for acceptable performance in presence of multipath fading and RF interference
ECMA 368 effective operating noise floor for UWB interference susceptibility:	–	–100	dBm/MHz	The effective allowable interference power level for the ECMA 368 operating receiver ($kTB + NF_{\mathrm{ECMA}} + D_{FD} + M_{\mathrm{ECMA}}$)
Level of wideband UWB emissions that result in 3 dB rise in ECMA 368 effective operating noise floor	–	–100	dBm/MHz	For 3 dB rise, IEEE 802.15.4a UWB emissions in-band can be at the same level as effective operating noise floor for AWN device receiver
Path loss (range) from UWB to ECMA 368 receiver (average case) for 3 dB rise in effective operating noise floor	–	55 (3)	dB (m)	For 3 dB rise, wideband UWB emissions in-band can be at the same level as effective operating noise floor for AWN device receiver
Path loss (range) from UWB to ECMA 368 receiver (average case) for 1 dB rise in effective operating noise floor	–	61 (6)	dB (m)	For 1 dB rise, wideband UWB emissions in-band must be 6 dB below effective operating noise floor for indoor IEEE 802.16 node receiver

Base on this path loss, the effect on AWN PER is computed as a function of separation distance, shown in Figure 43.

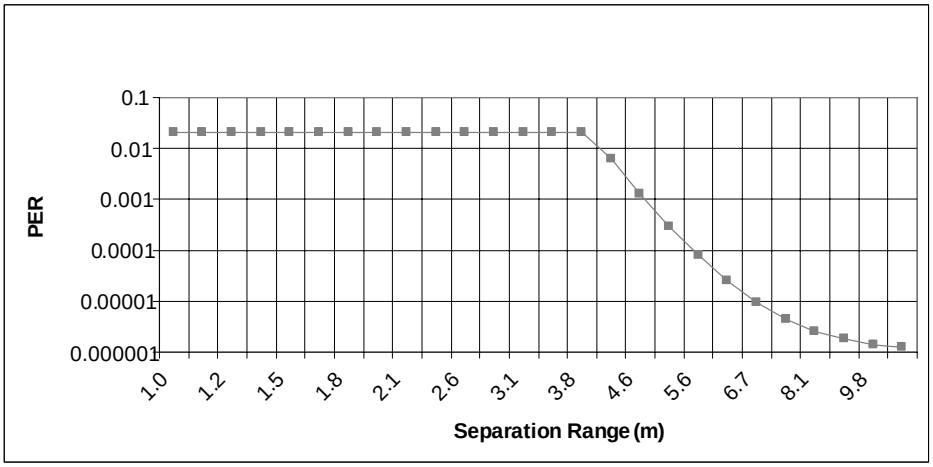


Figure 43—Effect on an ECMA 368 AWN device as a function of separation distance from a UWB PHY device

5.4.5 Impact of IEEE 802.15.4a devices on IEEE P802.22 networks

Based on the currently available draft of IEEE P802.22, the operating conditions are generally similar to IEEE Std 802.16-2004. The primary operating considerations include the following:

— The IEEE P802.22 network is a fixed-point-to-multipoint network, operating in a narrow band (6–8 MHz) widely spaced between 54 MHz and 862 MHz; the fixed node will not be susceptible to IEEE 802.15.4a interference due to positioning.

— The UWB PHY channel at 150 MHz to 650 MHz is operating, on average, at least –75 dBm (set by regulation, using current FCC limits), which is at approximately 34 dB lower power than the higher band UWB PHY (–41.3 dBm).

— UWB interference is wideband uncorrelated noise since the bandwidth is much wider than the victim receiver. A 10 dB difference in antenna gain is assumed in anticipation that the IEEE P802.22 antenna will require gain in the direction of the desired fixed node (base station) downlink signal, and it is also assumed that the UWB device will not directly block the LOS.

At the time of this analysis, the characteristics of the IEEE P802.22 AWN were not completely defined. Assuming similar characteristics to an IEEE 802.16 device with the operating frequencies specified above, note that the 150–650 MHz UWB PHY has a similar path loss curve to the 3100–4800 MHz UWB PHY with the noted 6–8 dB difference along the curve. Note further that the maximum radiated power is 34 dB lower and the effective interference seen by the AWN will be lower than shown for the IEEE 802.16 case.

5.5 Conclusions

These analyses characterize the expected coexistence behavior between UWB PHY devices and IEEE 802.16 devices. Also described are the expected effects of a UWB PHY device on an ECMA 368 receiver and the proposed IEEE P802.22 devices. One conclusion that can be drawn is that the relative effects of the UWB PHY device and IEEE 802.16 device to each other are quite different. The UWB PHY device is impacted by the IEEE 802.16 device at much longer range than vice versa. The implication is that the UWB PHY device would not be able to operate at all at ranges where its emissions would impact the IEEE 802.16 device because of the large asymmetry in the transmit power levels (+17 dB for the IEEE 802.16 device

versus –15 dBm for the UWB PHY device). In such a case, either the UWB PHY device would accept the much higher PER, or else it could simply use a different channel or some other form of interference mitigation.

A similar conclusion can be reached regarding proposed IEEE P802.22 devices; there is an even greater asymmetry in power levels, as the sub-gigahertz band is operated at a substantially lower level than the higher UWB bands. One form of mitigation (in both directions) is to observe that when considering the application environment in which the sub-gigahertz UWB band has greatest advantage and is, therefore, most likely to be used, the operation of IEEE P802.22 devices in near proximity is unlikely. In application scenarios where it is expected that UWB PHY sub-gigahertz devices may operate in proximity to IEEE P802.22 devices, the UWB PHY devices may need to employ some other forms of interference mitigation. Additional mitigation is available to the IEEE P802.22 device. Note that a great number of potential channels are available above 650 MHz and provide the option to the IEEE P802.22 device to change to a channel outside the operating range of the UWB PHY sub-gigahertz devices.

6. Notes on the calculations

The calculations for this annex were based on the formulas and descriptions from IEEE Std 802.15.2-2003.

7. Bibliography

[B1] ERC Recommendation 70-03, Relating to the Use of Short Range Devices (SRDs), April 2002.[3]

[B2] ETSI EN 300 220-1, Electromagnetic Compatibility and Radio Spectrum Matters (ERM); Short Range Devices (SRDs); Radio equipment to be used in the 25 MHz to 1 000 MHz frequency range with power levels ranging up to 500 mW; Part 1: Technical characteristics and test methods.[4]

[B3] ETSI EN 300 328-1, Electromagnetic Compatibility and Radio Spectrum Matters (ERM); Wideband Transmission Systems; Data transmission equipment operating in the 2,4 GHz ISM band and using spread spectrum modulation techniques; Part 1: Technical characteristics and test conditions.

[B4] ETSI EN 300 328-2, Electromagnetic Compatibility and Radio Spectrum Matters (ERM); Wideband Transmission Systems; Data transmission equipment operating in the 2,4 GHz ISM band and using spread spectrum modulation techniques; Part 2: Harmonized EN covering essential requirements under article 3.2 of the R&TTE Directive.

[B5] FCC Code of Federal Register (CFR), Part 47, Section 15.35, Section 15.205, Section 15.209, Section 15.231, Section 15.247, and Section 15.249. United States.[5]

[B6] Shellhammer, S. J., “Estimating Packet Error Rate Caused by Interference—A Coexistence Assurance Methodology,” IEEE 802.19-05/0029r0, September 14, 2005.

[B7] Shellhammer, S. J., “Estimation of Packet Error Rate Caused by Interference using Analytic Techniques—A Coexistence Assurance Methodology,” IEEE 802.19-05/0028r2, September 14, 2005.

[B8] Sklar, Bernard, Digital Communications: Fundamentals and Applications. New Jersey: Prentice Hall, 1988.

[3]ERC publications are available from the European Communications Office (http://www.erodocdb.dk/).

[4]ETSI publications are available from the European Telecommunications Standards Institute (http://www.etsi.org).

[5]FCC publications are available from http://www.fcc.gov