

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
IEEE International Committee on Electromagnetic Safety (SCC39)

IEEE
3 Park Avenue
New York, NY 10016-5997, USA

19 April 2006

IEEE Std C95.1™-2005
(Revision of IEEE Std C95.1-1991)

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IEEE Standard for Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3 kHz to 300 GHz

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IEEE International Committee on Electromagnetic Safety (SCC39)

Approved 3 October 2005

IEEE-SA Standards Board

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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)

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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.

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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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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.

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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.

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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.

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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.

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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.

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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.

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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.

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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.

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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,

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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).

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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**.

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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

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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

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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:

$$E_i = E_0 \text{ for } f \le f_e \tag{1}$$

$$E_i = E_0 \, (f/f_e) \text{ for } f \ge f_e$$

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

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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

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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

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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:

$$\sum_{0}^{5\ \mathrm{MHz}} \frac{A_i}{MPE_i} \leq 1 \qquad (2)$$

where A_i is the magnitude of the ith 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.

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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 ith 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-

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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.		

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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 ith 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

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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.

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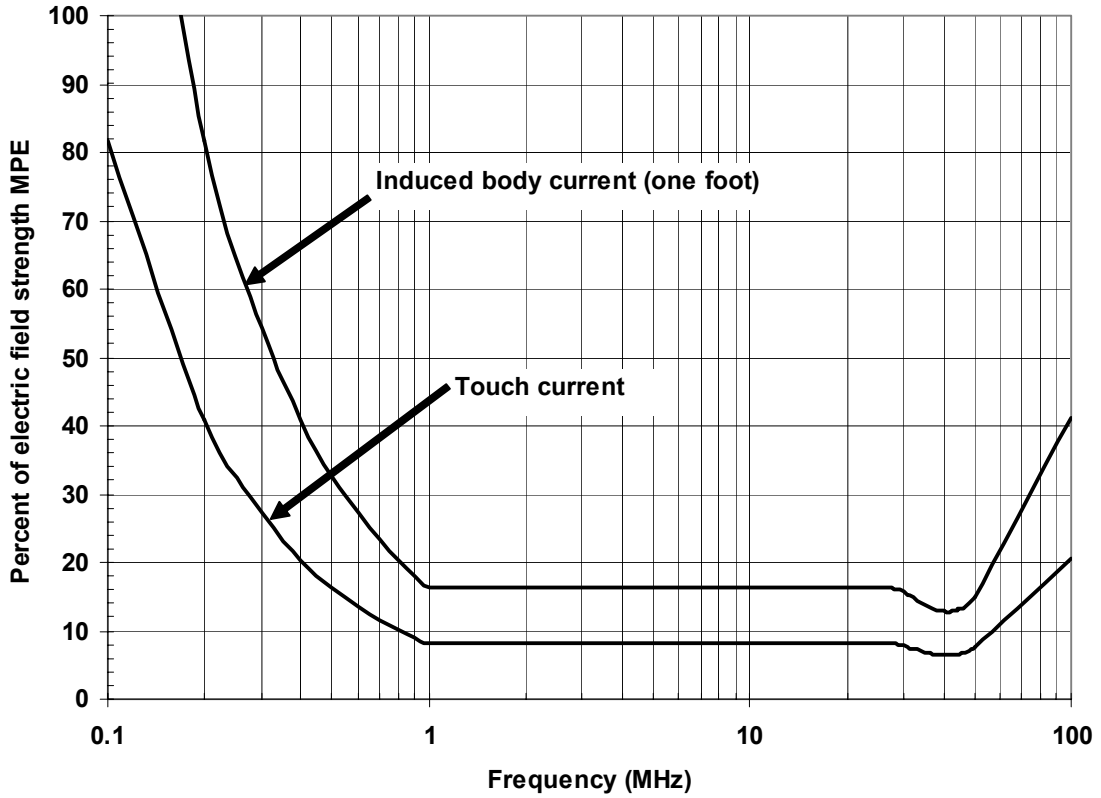

Figure 1—Percent of electric field strength MPE below which the induced current through one foot, or the touch current, will meet the MPEs of Table 7 for the upper tier (exposures in controlled environments); based on a body height of 1.75 m.

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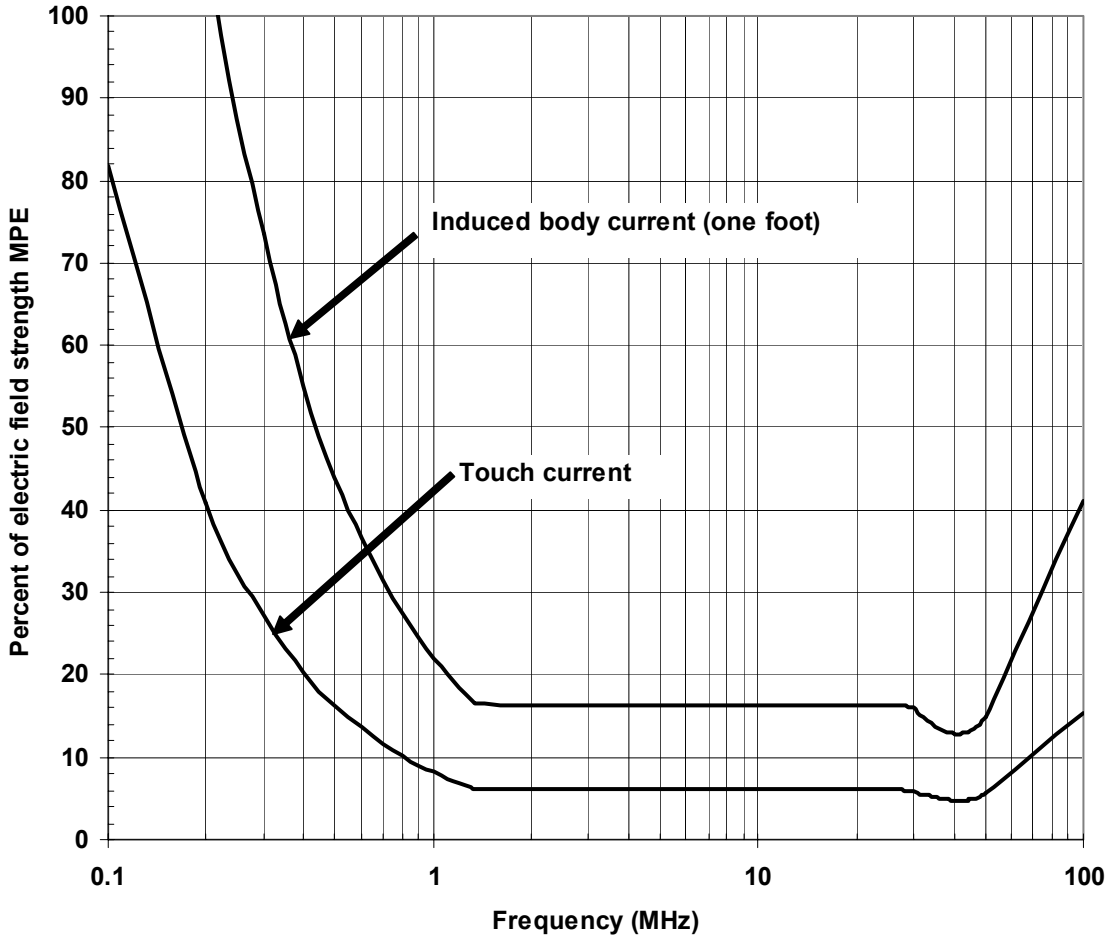

Figure 2—Percent of electric field strength MPE below which the induced current through one foot, or the touch current, will meet the MPEs of Table 7 for the lower tier; based on a body height of 1.75 m.

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-

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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.

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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.

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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.,

$$\sum_{0}^{0.1s} (S_{\text{pk}} \times \tau) \leq \frac{MPE_{\text{avg}} \times T_{\text{avg}}}{5} \leq 28.8 \text{J/kg}$$

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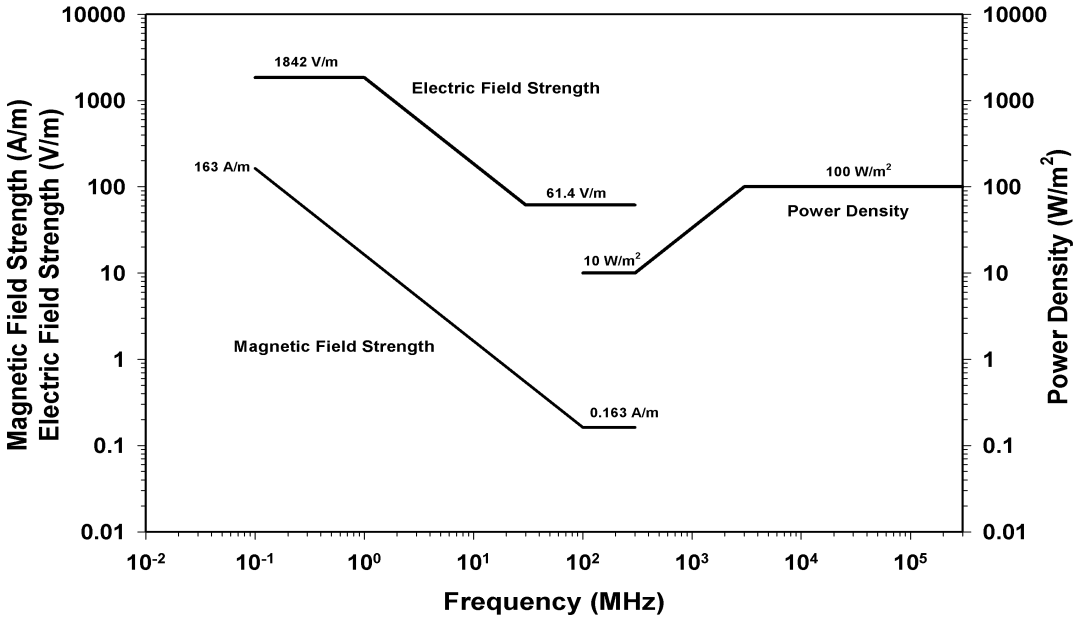

Figure 3—Graphic representation of the MPEs in Table 8 (exposures in controlled environments)

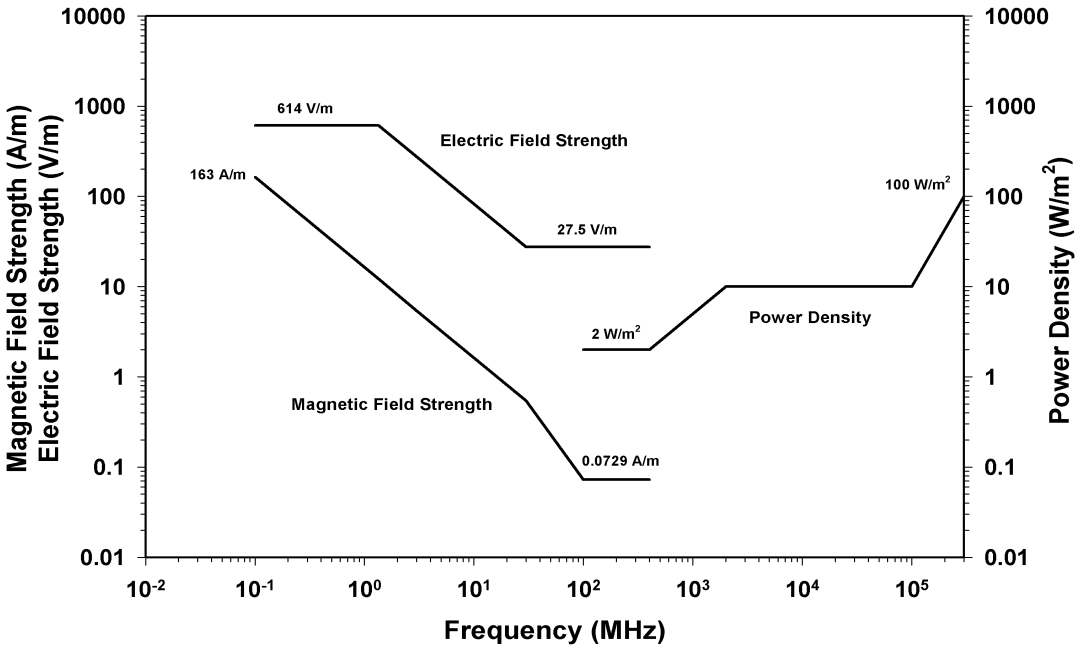

Figure 4—Graphic representation of the MPEs in Table 9 (lower tier—action level)

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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

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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).

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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.

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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

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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,

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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.

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Annex B

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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.

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(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

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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.

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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

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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

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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

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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.

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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

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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],

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[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.

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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

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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-

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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

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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.

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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

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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.

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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

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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

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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

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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]).

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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.

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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

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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]).

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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.

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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.

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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-

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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]).

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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

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(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-

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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.

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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

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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.

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[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.

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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

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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.

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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

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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

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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.

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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

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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

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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

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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

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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

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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.

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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).

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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).

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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]).

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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).

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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

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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.

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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.

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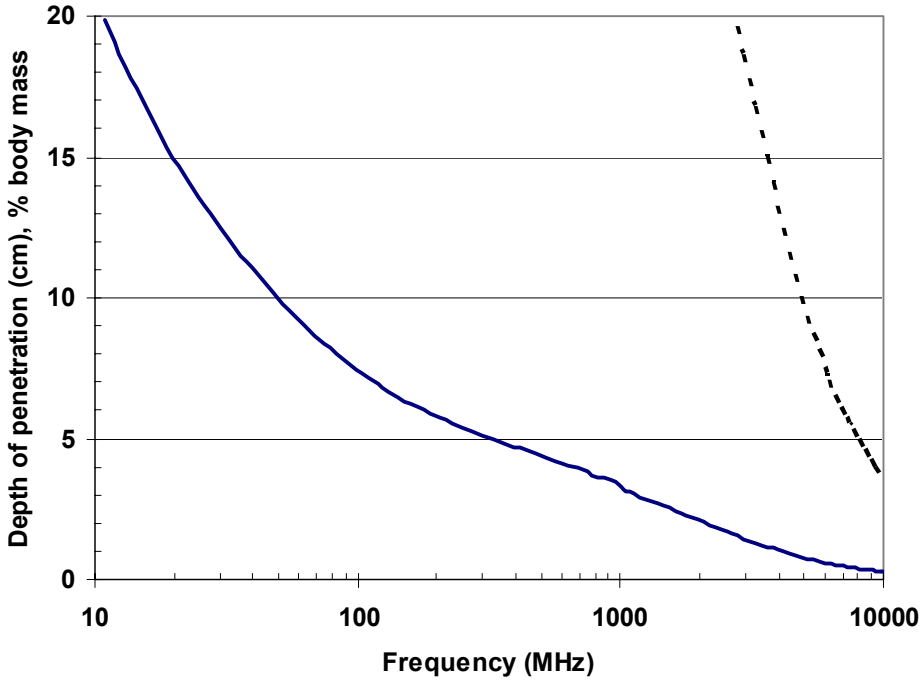

Figure C.1—Calculated depth of penetration (solid line) and percent of body mass (broken line) in which most of the incident RF energy is absorbed as a function of frequency. Calculations are for muscle-equivalent material and are based on a planar slab model.

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.

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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.

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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

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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

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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

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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.

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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.

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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.

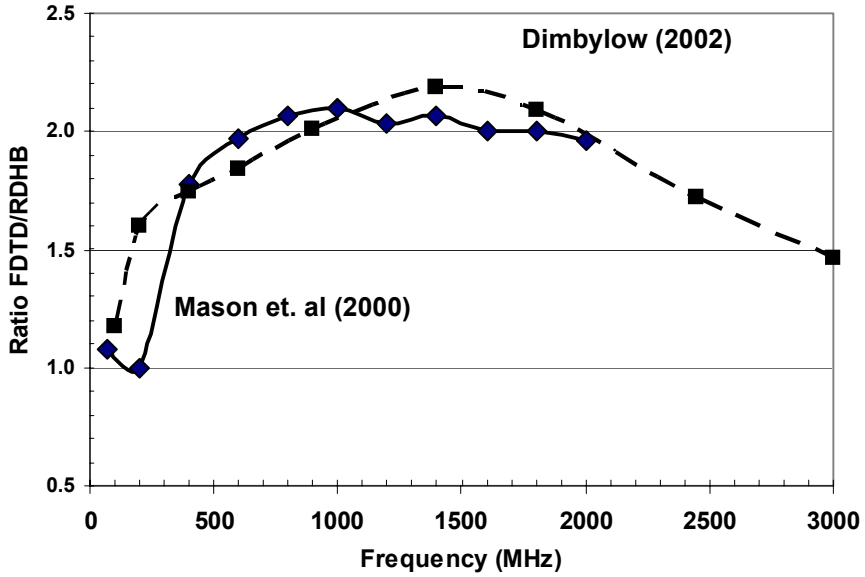

Figure C.2—Comparison of computed adult WBA SAR from two studies (Dimbylow [R1085], Mason et al. [B86]) relative to values from the Radio Frequency Radiation Dosimetry Handbook (RDHB) (Durney et al. [R901]) as a function of frequency

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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.

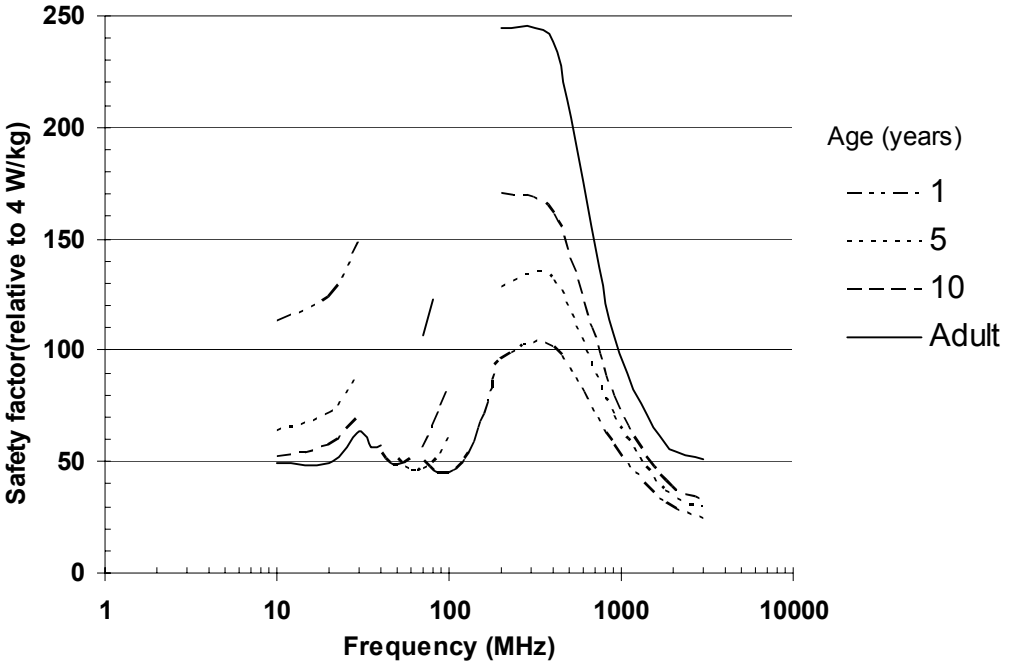

Figure C.3—Calculated ratio (ordinate) of the WBA SAR to 4 W/kg (the threshold for potentially adverse effects) for adults and 1-, 5-, and 10-year-old children, expressed as the SAR based safety factor. The ratio is based on the MPE in IEEE Std C95.1, 1999 Edition [B70] for uncontrolled environments 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.

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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 $\propto I^2$).

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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

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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.

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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

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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])

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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

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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.

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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-

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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

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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

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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-

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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*.

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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.

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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.

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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 ≈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.

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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.

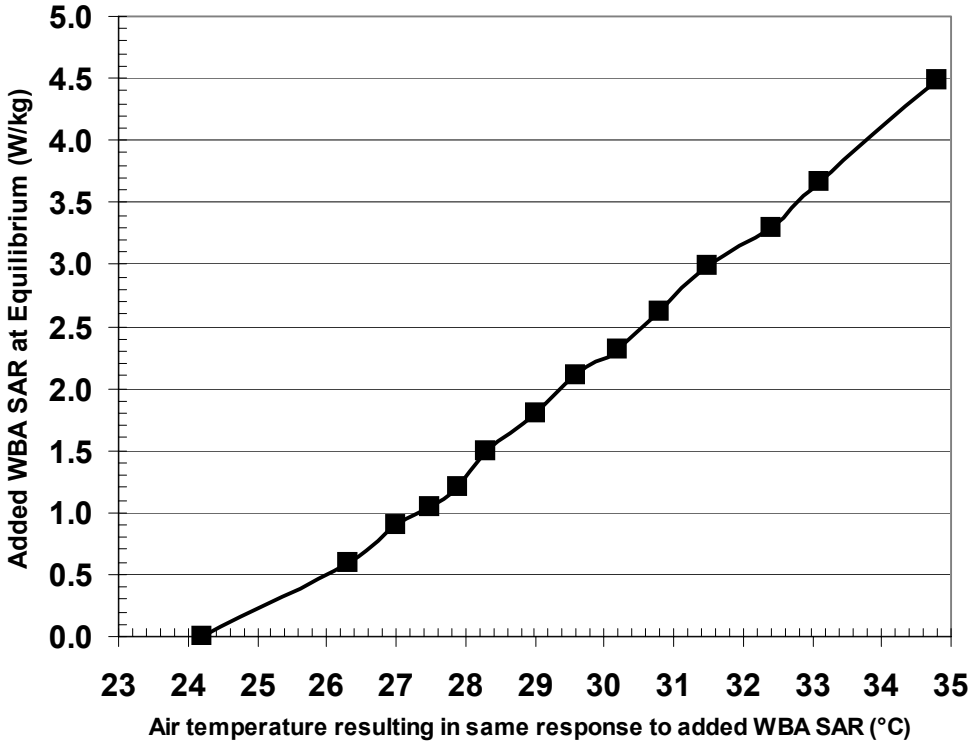

Figure C.4—Estimated added RF thermal load (W/kg) at rest and in a thermally comfortable environment to produce an equivalent discomfort response at rest to elevated ambient air temperature

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

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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

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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

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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., $t < T_{avg}$.

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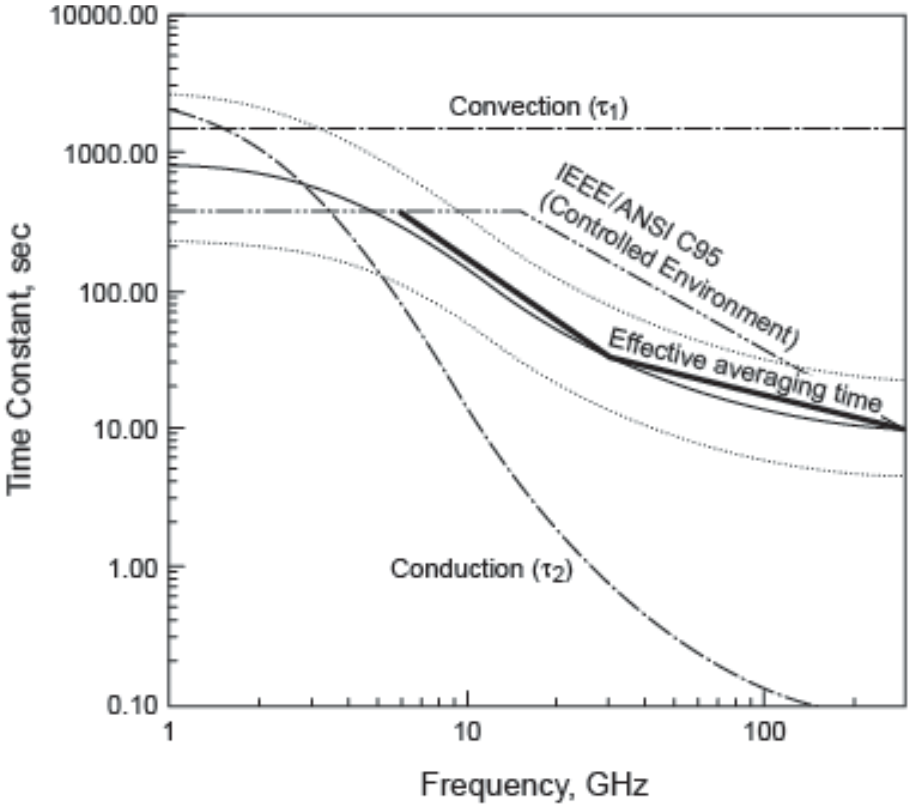

Figure C.5—The new averaging times included in this standard (a two-step variation) provide for shorter values at the upper end of the spectrum and are consistent with other standards and guidelines at 300 GHz

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).

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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

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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.

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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.

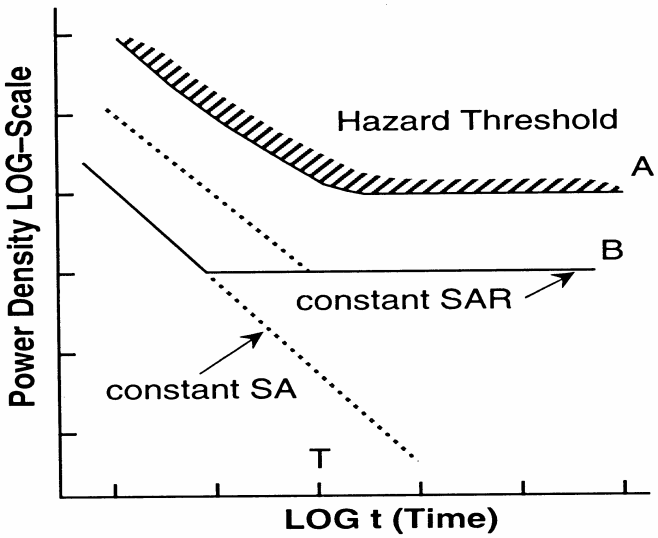

Figure C.6—Thresholds for various effects and hazards expressed as a function of time

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

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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-

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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.

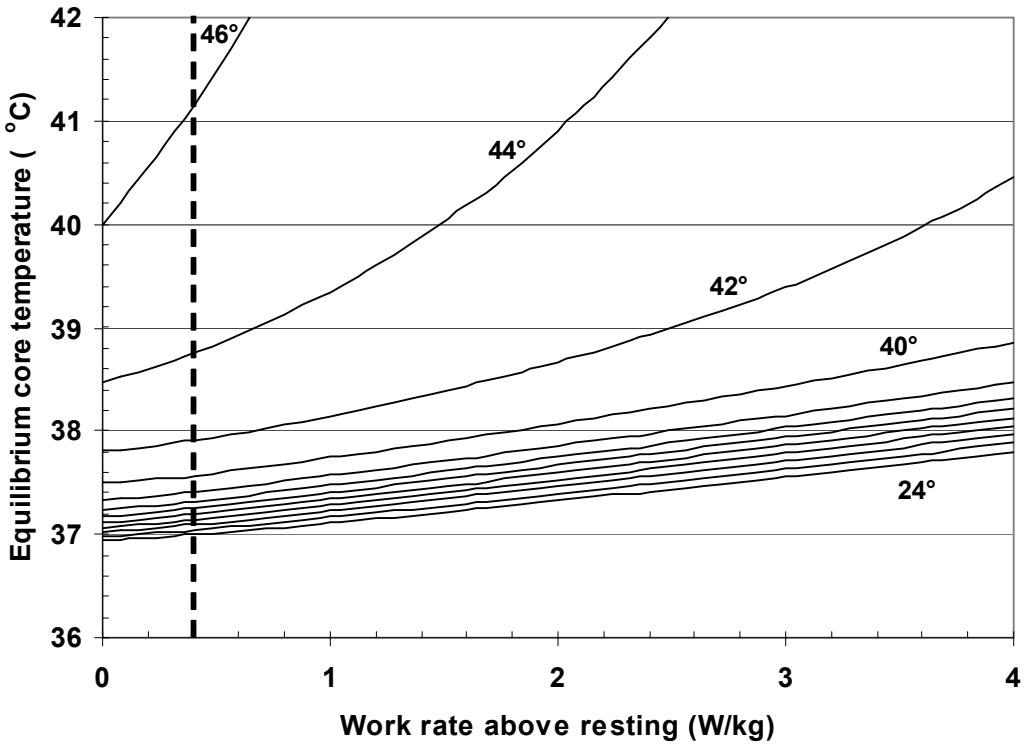

Figure C.7—Projected equilibrium core temperature as a function of additional workload above resting metabolic rate for a range of ambient air temperatures (24 °C to 46 °C) derived from empirical data obtained on army soldiers

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.

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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.

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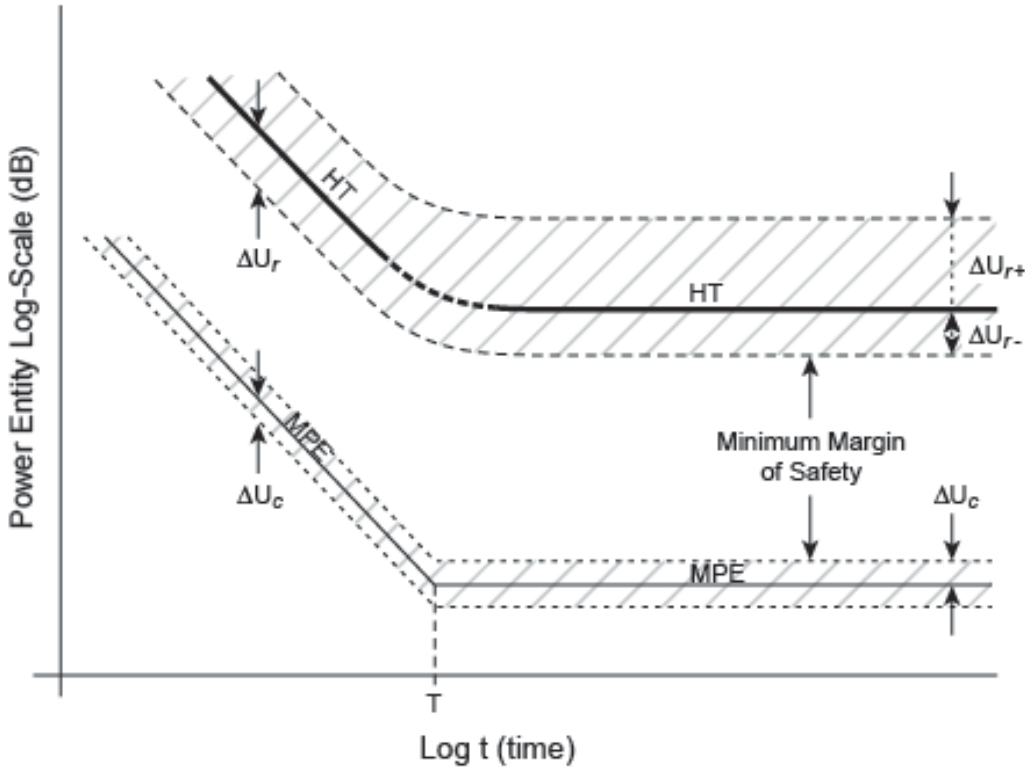

Figure C.8—A hypothetical exposure diagram illustrating uncertainty factors and resulting minimum margin of safety. This diagram is based on power. A similar diagram could be based on current density or internal electric field.

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

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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-

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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

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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

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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.

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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.

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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

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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.

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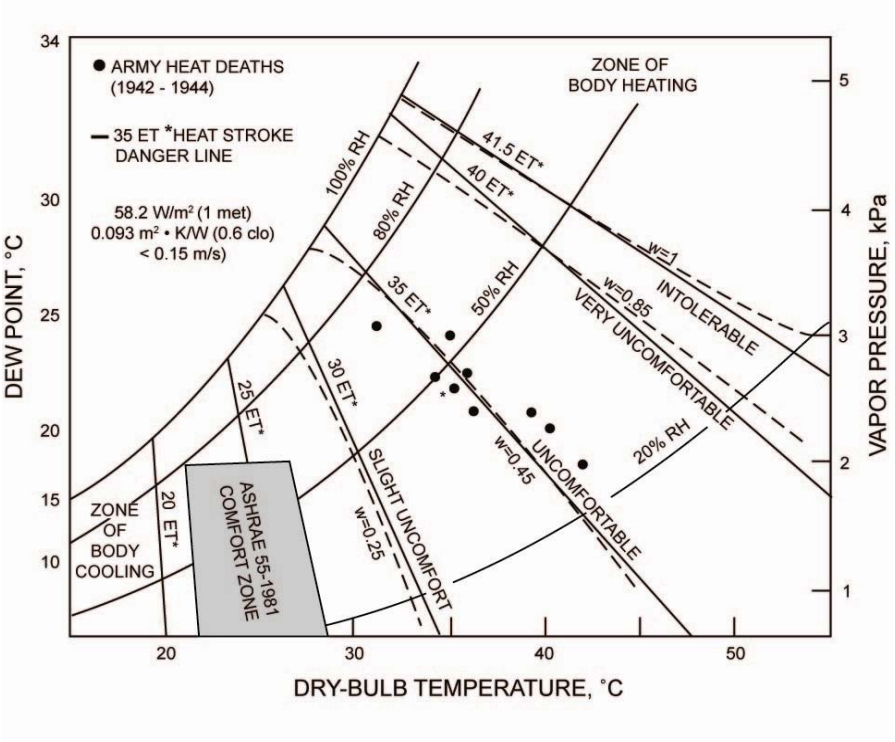

Figure C.9—A standard psychrometric chart that delineates the zone of thermal comfort (shaded), zones of body heating and cooling in terms of dew point, ambient vapor pressure and dry-bulb temperature. ET* defines the limits of thermal comfort (ASHRAE [B13]).

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

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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.

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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.

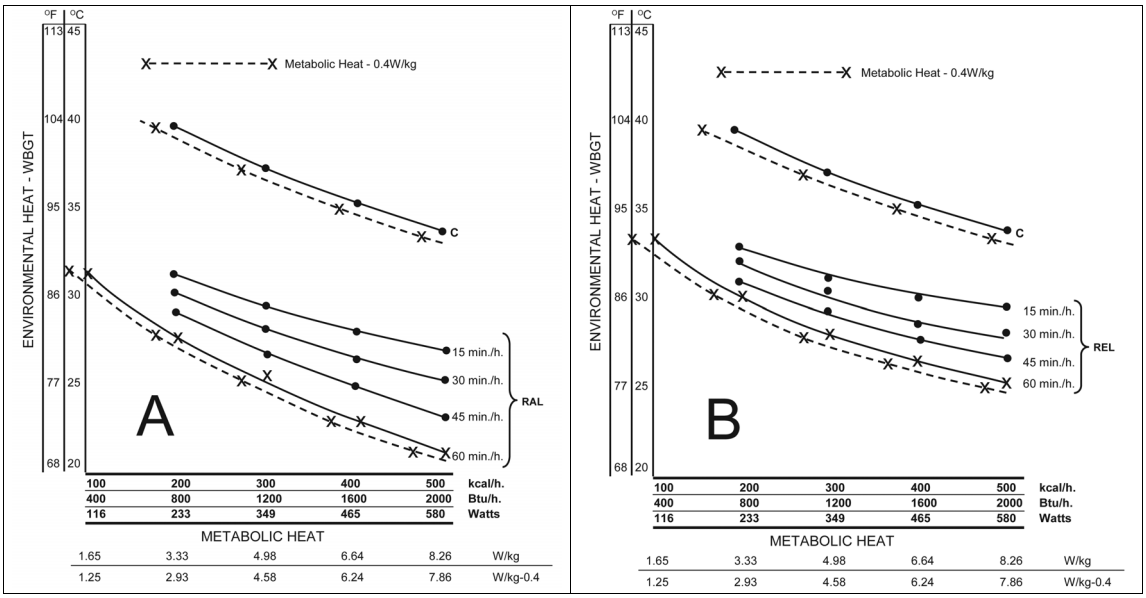

Figure C.10—NIOSH recommended heat-stress alert limits for unacclimatized workers (Panel A) and exposure limits for heat-acclimatized workers (Panel B) as modified to include the SAR criterion of 0.4 W/kg adopted for many RF exposure guidelines for humans (dashed lines). C = ceiling limit, RAL = recommended 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].

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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

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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

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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.

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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.

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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

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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

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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

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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-

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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.

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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.

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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.,

$$\sum_{0}^{0.1s} (S_{\mathrm{pk}} \times \tau) \leq \frac{MPE_{\mathrm{avg}} \times T_{avg}}{5} \leq 28.8 \mathrm{J/kg}$$

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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

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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.

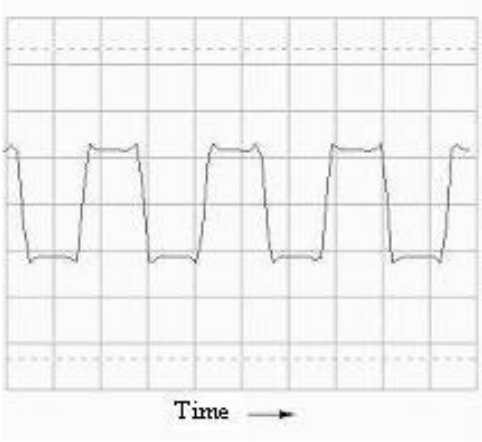

Figure D.1—Square wave in the time domain

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

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$$\dot{E}_p = \sqrt{2} MPE_E(2\pi f)$$

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.

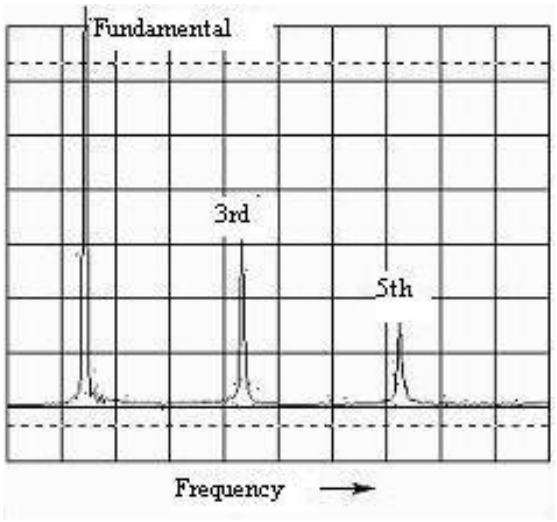

Figure D.2—Square wave in the frequency domain (Fourier spectrum)

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):

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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

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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:

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$$\sum_{i=1}^{i=6} \frac{S_{E_i}(duty\ factor)}{MPE_{E_i}} < 1$$

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 ith 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.

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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.

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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.

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Annex E

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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.

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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).

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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

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