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IEEE Standards for Safety Levels with Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz

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Developed by the
IEEE International Committee on Electromagnetic Safety

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IEEE

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(Revision of
IEEE Std C95.1-2005/
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IEEE Standard for Safety Levels with Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz

Developed by the

IEEE International Committee on Electromagnetic Safety

Approved 8 February 2019

IEEE SA Standards Board

Abstract: Safety limits for the protection of persons against the established adverse health effects of exposures to electric, magnetic, and electromagnetic fields in the frequency range 0 Hz to 300 GHz are presented in this standard. These exposure limits are intended to apply generally to persons permitted in restricted environments and to the general public in unrestricted environments. These exposure limits are not intended to apply to the exposure of patients by or under the direction of physicians and medical professionals, as well as to the exposure of informed volunteers in medical or scientific research studies, and might not be protective with respect to the use of medical devices or implants.

Keywords: dosimetric reference limit (DRL), exposure reference level (ERL), induced and contact currents, electric fields, electrical excitation, electromagnetic fields, electrostimulation, general public, IEEE C95.1™, magnetic fields, non-ionizing radiation protection, radio frequency (RF), RF exposure, RF safety, restricted environment, specific absorption rate (SAR), unrestricted environment

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Introduction

This introduction is not part of IEEE Std C95.1-2019, IEEE Standard for Safety Levels with Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz.

In 1960, the American Standards Association approved the initiation of the Radiation Hazards Standards project under the co-development of the Department of the Navy and the Institute of Electrical and Electronics Engineers, Incorporated (IEEE; called the "Institute of Radio Engineers (IRE)" at the time). 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 the "Standards Coordinating Committee 28 (SCC28)" under the development of the IEEE Standards Board. In 2001, the IEEE Standards Association Standards Board approved the name "International Committee on Electromagnetic Safety (ICES)" for SCC28 to better reflect the scope of the committee and its international membership. In accordance with the policies of the IEEE, C95 standards are issued and developed as IEEE standards and submitted to ANSI for recognition.

In June 1995, the IEEE Standards Board approved the establishment of "Standards Coordinating Committee 34 (SCC34), Product Performance Standards Relative to the Safe Use of Electromagnetic Energy." Standards developed by SCC34 do not specify limits for human exposure to electromagnetic fields, but refer to established limits found in science-based standards such as 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 2005, SCC28 and SCC34 became "Technical Committee 95" and "Technical Committee 34," respectively, under a new IEEE Standards Coordinating Committee, SCC39, which is now called "ICES."[1]

The present scope of IEEE ICES Technical Committee 95 (TC95) 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."

There are six TC95 Subcommittees, each of whose area of responsibility is described as follows in correspondence with its designated Subcommittee number:

- SC 1: Techniques, Procedures, Instrumentation, and Computation
- SC 2: Terminology, Units of Measurements, and Hazard Communication
- SC 3: Safety Levels with Respect to Human Exposure, 0 Hz to 3 kHz
- SC 4: Safety Levels with Respect to Human Exposure, 3 kHz to 300 GHz
- SC 5: Safety Levels with Respect to Electro-Explosive Devices
- SC 6: EMF Modeling and Dosimetry

[1] Standards Coordinating Committees are established by the IEEE SA Standards Board and provide a mechanism to oversee the development of standards that are beyond the scopes of individual technical committees within IEEE's societies.

This standard was prepared by Subcommittee 3 and Subcommittee 4 of TC95. TC95 has issued three standards, four recommended practices, and one guide. The present versions are as follows:

— IEEE Std C95.1™-2019, IEEE Standard for Safety Levels with Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz.

— IEEE Std C95.1-2345™-2014, IEEE Standard for Military Workplaces—Force Health Protection Regarding Personnel Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz.

— IEEE Std C95.2™-2018, IEEE Standard for Radio-Frequency Energy and Current-Flow Symbols.

— IEEE Std C95.3™-2002 (R2008), IEEE Recommended Practice for Measurements and Computations of Electric, Magnetic, and Electromagnetic Fields With Respect to Human Exposure to Such Fields, 100 kHz to 300 GHz.

— IEEE Std C95.3.1™-2010, IEEE Recommended Practice for Measurements and Computations of Electric, Magnetic, and Electromagnetic Fields with Respect to Human Exposure to Such Fields, 0 Hz to 100 kHz.

— IEEE Std C95.4™-2002 (R2008), IEEE Recommended Practice for Determining Safe Distances from Radio Frequency Transmitting Antennas When Using Electric Blasting Caps During Explosive Operations.

— IEEE Std C95.7™-2014, IEEE Recommended Practice for Radio Frequency Safety Programs, 3 kHz to 300 GHz.[2]

— IEEE Std 1460™-1996 (R2008), IEEE Guide for the Measurement of Quasi-Static Magnetic and Electric Fields.

Dedication

This edition of IEEE Std C95.1 is dedicated in memory of Ronald C. (Ron) Petersen. Ron served on various IEEE committees beginning in the 1970s, particularly SCC28, SCC34, and SCC39 (ICES). He also served as chair of the International Electrotechnical Commission (IEC) TC 106 (EMF exposure) through its first 15 years. Ron's passing represents a great loss for the IEEE, and especially for ICES. His constant attention to the workings of this committee and the development of standards documents formed the glue that ensured the continued cohesiveness and success of ICES. We will all miss Ron in our meetings—he lives forever in our memories.

[2] The latest revisions of IEEE Std C95.1, IEEE Std C95.1-2345, IEEE Std C95.2, IEEE Std C95.3, IEEE Std C95.3.1, and IEEE Std C95.7 are available at no cost (at the time of publication of this standard) through the IEEE Get Program. (https://ieeexplore.ieee.org/browse/standards/get-program/page/series?id=82).

Contents

IEEE Standard for Safety Levels with Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz

1. Overview

1.1 Scope

This standard specifies exposure criteria and limits to protect against established adverse health effects in humans associated with exposure to electric, magnetic, and electromagnetic fields in the frequency range of 0 Hz to 300 GHz.[1,2] These limits, incorporating safety margins, are expressed in terms of dosimetric reference limits (DRL) and exposure reference levels (ERL). DRLs are expressed in terms of *in situ* electric field strength, specific absorption rate (SAR), and epithelial power density. ERLs, which are more easily determined, are limits on external electric and magnetic fields, incident power density, induced and contact currents, and contact voltages intended to ensure that the DRLs are not exceeded. The limits, which protect against adverse health effects associated with electrostimulation of tissue and local and whole-body heating, are intended to apply to the described human exposure conditions. However, these levels are not intended to address exposures of patients or human research subjects under the care of medical professionals for which other risks and benefits might apply. These exposure limits might not prevent interference with medical and other devices that might exhibit susceptibility to electromagnetic interference (EMI).

1.2 Purpose

The purpose of this standard is to provide science-based exposure criteria to protect against established adverse health effects in humans associated with exposure to electric, magnetic, and electromagnetic fields; induced and contact currents; and contact voltages, over the frequency range of 0 Hz to 300 GHz.[3]

[1] The limits in this standard are intended to protect against adverse health effects except in certain cases of contact current exposures or RF arcing exposures that could result in highly localized burns or transient adverse reactions (e.g., startle or pain).
[2] The limits at 300 GHz for persons permitted in restricted environments are the same as the corresponding values in existing standards for exposure in the infrared frequency range, which begins at 300 GHz (cf. ANSI Z136.1-2014 [B69] and IEC 60825-1 [B653]).
[3] The frequencies and wavelengths covered by this standard are in the "nonionizing radiation" region of the electromagnetic spectrum.

1.3 General introduction

1.3.1 General

This standard is a revision and merging of IEEE Std C95.1™-2005 [B668] and IEEE Std C95.6™-2002 [B671].[4] Updated information is also included from IEEE Std C95.1-2345™-2014 [B669] (military workplaces and personnel protection), which preceded this standard in combining and updating IEEE Std C95.1-2005 and IEEE Std C95.6-2002, introduced expanded frequency-dependent exposure levels for contact currents, and introduced new terminology such as "safety program initiation level" and "unrestricted" and "restricted environments." Recommendations to protect against established adverse health effects to humans from exposures to electric fields, magnetic fields, electromagnetic fields, and contact currents are defined on the basis of a comprehensive review of the scientific literature.

The literature review conducted for IEEE Std C95.1-2005 remains as a strong foundation for the present standard (see C.2 to C.7). As discussed in A.1.7, the International Committee on Electromagnetic Safety (ICES) literature review working group (LRWG) found that many recent health agency and expert group reviews confirm the protectiveness of the existing limits; the major changes in limits in this standard are the DRLs and ERLs above 6 GHz based on recent thermal modeling studies. Detailed reviews of scientific studies dealing with effects at frequencies above 6 GHz are included in C.8. Review of the extensive literature on electromagnetic field (EMF) biological effects revealed that electrostimulation is the dominant effect at low frequencies and that thermal effects dominate at high frequencies. Examination of the radio frequency (RF) exposure literature revealed no reproducible low-level (nonthermal) adverse health effects. Moreover, the scientific consensus is that there are no accepted theoretical mechanisms that would explain the existence of low-level adverse health effects. Since the publication of ANSI C95.1-1982 [B68], advances have been made in the scientific knowledge of the biological effects of exposure to electromagnetic energy. This additional and cumulative knowledge helps strengthen the basis for and confidence in the assertion that the ERLs and DRLs in this standard are protective against established adverse health effects.

Based on the results of the latest ICES LRWG literature review, relevant mechanisms of interaction were found to be stimulation of excitable nervous tissue by electric fields induced within the body due to electric and/or magnetic fields and produced by contact current (frequencies between 0 Hz and approximately 5 MHz),[5] as well as by tissue heating (frequencies above approximately 100 kHz). In the transition region of 100 kHz to 5 MHz, the more restrictive of the two limits apply; generally: 1) ERLs associated with heating are more restrictive for long-term exposures (i.e., greater than the averaging time) to continuous-wave (CW) fields, and 2) ERLs based on electrostimulation are more restrictive for short-term exposure (e.g., short isolated pulses of low duty factor).

The whole-body average (WBA) DRLs and ERLs incorporate conservative safety factors that take into account biological and dosimetry uncertainties in laboratory studies and the variability of response thresholds within the human population. (See B.1.3 for the derivation and detailed aspects of the safety factors.) Because the ERLs are conservatively derived from the DRLs, it is possible to exceed an ERL while still complying with the DRL. While numerous refinements are described in this document, a fundamental aspect of this standard is that it continues to support the findings of earlier editions.

The following short-term reactions associated with electrostimulation at frequencies below 100 kHz for CW exposures have been established: 1) aversive or painful stimulation of sensory or motor neurons; 2) muscle excitation that can lead to injury while performing potentially hazardous activities; 3) excitation of neurons or direct alteration of synaptic activity within the brain; 4) cardiac excitation; and 5) adverse health effects associated with induced potentials or forces on rapidly moving charges within the body, such as in blood flow. These findings are consistent with the conclusions of interdisciplinary panels of scientists that have evaluated the literature on exposures to electric and magnetic fields for scientific and governmental organizations through 2017 (see C.1). Data from studies published between 1901 and 2017 were considered

[4] The numbers in brackets correspond to those of the bibliography in Annex E.
[5] The effects associated with electrostimulation are categorized as short-term effects.

for frequencies below 100 kHz, including those studies that involve exposures below electrostimulation thresholds.

For frequencies between 100 kHz and 300 GHz, tissue heating and whole-body heating predominate. The most sensitive reproducible effect is disruption of food-motivated behavior in animal species, ranging from rodents to primates and over a wide range of frequencies. These findings are consistent with those of interdisciplinary panels of scientists from scientific and governmental organizations that have evaluated the literature, including chronic (long-term) exposure studies (see Annex C). Data from studies published between 1948 and December 2017 were considered for frequencies from 6 GHz to 300 GHz, including those studies that involve low-level exposures where increases in temperature could not be measured or were not expected.[6]

The previous standard IEEE Std C95.1-2005 [B668] contained two exposure tiers: 1) an upper tier for "people in controlled environments," and 2) a lower tier designated an "action level," above which steps should be initiated to protect against exposures that exceed the upper tier (specifically, implementation of an RF safety program). The upper boundary of the lower tier was considered to be an exposure limit for the general public.

Various new definitions are introduced in this standard. The terms unrestricted tier (lower tier) and restricted tier (upper tier) refer to ranges of permissible exposure values, with each tier having an upper limit.[7] The lower tier limit is designated as the "safety program initiation level" (rather than as the "action level") to emphasize that an RF safety program is necessary. It should be noted that in this standard, the upper tier exposure limit is referred to as applicable to "persons permitted in restricted environments," to emphasize that individuals might occupy restricted environments, where the higher ERLs and DRLs are applicable, if those individuals follow the applicable safety program guidance and procedures. This standard specifically avoids the declaration that only individuals who are exposed because of their occupation may enter restricted environments. For portable devices, such as mobile phones and professional two-way radios, the lower tier DRL is applicable to devices available to the general public. The higher tier DRL is applicable to devices for which user awareness information/training is provided.

The literature review also evaluated the possibility of adverse health effects associated with chronic low-level exposure. For exposures to electric, magnetic, and electromagnetic fields at frequencies between 0 Hz and 300 GHz, the following two conclusions were reached:

a) The weight-of-evidence provides no credible indication of adverse effects caused by chronic exposures below levels specified in this standard.

b) No biophysical mechanisms have been scientifically validated that would link chronic exposures below levels specified in this standard to adverse health effects.[8]

[6] Although the literature review cutoff date of IEEE Std C95.1-2005 [B668] was December 2003, several papers published in 2004 and 2005 were included thereunder. While review of the 2003 to 2017 high-frequency literature remains ongoing, the review findings used for this revision cover the literature for frequencies above 6 GHz to address the corresponding changes in the exposure limits between 6 GHz and 300 GHz.

[7] In this standard, the terms "upper tier" and "lower tier" are used interchangeably with "restricted tier" and "unrestricted tier," respectively.

[8] ICES TC95 is aware of epidemiological studies and reviews that have reported positive associations between cancer and electromagnetic fields in both the ELF and RF frequency ranges. The World Health Organization (WHO), in analyzing the 2002 International Agency for Research on Cancer (IARC) evaluation of exposure to ELF magnetic fields came to the following conclusion: "Thus, on balance, the evidence related to childhood leukaemia is not strong enough to be considered causal" (WHO [B1496] and WHO/IARC [B1501]). At higher frequencies, most epidemiologic studies have addressed the reported association of adult brain cancer with inferred RF exposure from the use of mobile telephones. The preponderance of epidemiologic evidence does not provide a sufficient basis for concluding that adult brain cancer is positively associated with mobile telephone use and, by implication, with RF exposures. IARC classified RF electromagnetic fields as a possible human carcinogen (Group 2B) in its classification system (Baan et al. [B93]). After this IARC classification, WHO issued a revised fact sheet 193 (WHO [B1494]) indicating that, "A large number of studies have been performed over the last two decades to assess whether mobile phones pose a potential health risk. To date, no adverse health effects have been established as being caused by mobile phone use." Furthermore, WHO also indicated that, "IARC has classified radiofrequency electromagnetic fields as possibly carcinogenic to humans (Group 2B), a category used when a causal association is considered credible, but when chance, bias or confounding cannot be ruled out with reasonable confidence." The [*Footnote 8 continued from previous page*] International Commission on Non-Ionizing Radiation Protection (ICNIRP) Standing

Based on the collective findings of recent reviews, the weight of the evidence continues to indicate that chronic exposure at levels specified in this standard is unlikely to cause adverse health effects. The ICES Subcommittees will continue to evaluate new research and will, if appropriate, initiate revision of this standard.

1.3.2 Protected population

This standard is intended to apply to all people, with the exception of patients undergoing procedures for medical diagnosis or treatment that requires exposure to fields or current in excess of the DRLs and ERLs of this standard. This exemption is provided under the expectation that the medical staff is appropriately trained in minimizing the risk 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, such as those 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 can exhibit susceptibility to EMI.[9]

Application of this standard is intended to offer protection to all persons in unrestricted exposure environments such as living quarters, public areas, and workplaces (unrestricted tier), as well as to persons permitted in restricted environments (restricted tier). DRLs and ERLs recommended in this standard are intended to protect against established adverse effects. Examples of exposure mitigation include engineering controls (engineering controls are the preferred approach to exposure mitigation in most exposure scenarios), administrative controls, personal protective equipment (PPE) such as insulative gloves and/or protective clothing, awareness programs, and operator training documentation designed to alert personnel to the possibility of effects, or specific work practices that lessen the duration or intensity of exposure (e.g., per IEEE Std C95.7™).[10]

1.3.3 Safety factors

1.3.3.1 General

Safety factors and their rationales are different for frequencies below approximately 100 kHz (but possibly up to 5 MHz for pulsed fields), where the adverse effect being minimized is electrostimulation, and above 100 kHz where the adverse effects being protected against are related to tissue heating.[11] In the transition region of 100 kHz to 5 MHz, both electrostimulation and heating can occur. For frequencies above 6 GHz,

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Committee on Epidemiology also published an epidemiology review to conclude that, "Although there remains some uncertainty, the trend in the accumulating evidence is increasingly against the hypothesis that mobile phone use can cause brain tumours in adults" (ICNIRP [B650]).

In both frequency ranges, cancer bioassays in rodents have been predominantly negative (no persuasive evidence of effects have been found at the exposure levels permitted in the standard). Furthermore, no plausible biophysical mechanisms have been verified that explain hypothesized adverse effects at low-level exposures. Thus, ICES concludes that only the established acute mechanisms related to electrostimulation between 0 Hz and 100 kHz (and up to 5 MHz for exposures to pulses) and tissue heating between 100 kHz and 300 GHz need to be addressed to provide a valid basis for the DRLs and ERLs designated in this standard.

ICES is also aware of the U.S. National Toxicology Program's draft study reports and peer-review outcomes on the effects of Global System for Mobile Communications (GSM) and Code Division Multiple Access (CDMA) RF signals on whole-body exposure of rats and mice at levels that exceed the DRLs in this standard. The NTP studies were conducted for the purpose of hazard identification. As is the continuing practice of ICES, the final NTP reports and subsequent evaluations and risk assessments by other organizations will be addressed in future revisions of the standard. A careful assessment of the NTP study by ICNIRP describes several difficulties in interpreting the results of the study for development of standards, in particular, the use of exposure levels in the NTP study that were considerably above the existing or proposed whole-body exposure limits of IEEE Std C95.1; "ICNIRP NOTE on recent animal carcinogenesis studies," Sep. 4, 2018 (https://www.icnirp.org/cms/upload/publications/ICNIRPnote2018.pdf).

[9] While the issue of RF emissions from wireless transmitters causing electromagnetic interference with medical devices is outside the scope of the current standard, the reader is directed to several relevant standards that recommend immunity levels for external medical devices (e.g., IEC 60601-1 [B651] and IEC 60601-1-2 [B652]), as well as implantable medical devices (e.g., ANSI/AAMI PC69:2007 [B71] and ISO/TR 21730:2007 [B690] offer guidance for the use and operation of mobile wireless transmitters within healthcare facilities).

[10] Information on references can be found in Clause 2.

[11] At low frequencies, safety factors apply to the amplitude of the *in situ* field; at high frequencies, the safety factors apply to the square of amplitudes of the *in situ* or incident fields.

the effect being protected against is tissue surface heating. The three types of effects (i.e., electrostimulation, whole-body heating, and local heating) are protected against through three separate sets of DRLs and ERLs that are applicable within the respective frequency ranges, as described in 1.3.3.2 through 1.3.3.4.

1.3.3.2 Frequencies below 100 kHz (5 MHz for pulsed fields)

Because the predominant interaction mechanisms are different above and below approximately 100 kHz (5 MHz for certain pulsed fields), the nature of and the rationale for the safety factors differ. Below 100 kHz, this standard protects against aversive or painful effects associated with electrostimulation, which has a characteristic response time that is much less than 1 s and exposures are thus assessed in terms of instantaneous fields or currents. DRLs for electrostimulation include upper and lower tier safety factors (divisors, designated as SF_U and SF_L) that are applied to a median adverse-reaction threshold. The value of SF_U, which is 3 for all tissues, protects against adverse reactions for all but a small segment of the population (< 1 %). The value SF_L is 3 for the limbs and 9 for other tissues, thus, providing an additional margin to further reduce the probability of adverse response in exposed individuals not covered by SF_U. For the restricted tier, $SF_U = 3$ is applied to all tissues including brain tissue and neural tissue within the trunk, based on the following observations:

a) An adverse reaction for sensory perception at the upper tier of the DRL would be expected to occur in no more than 1 % of persons permitted in restricted environments and with no credible possibility of cardiac arrhythmias.

b) Electrostimulation that might occur at the upper tier limit has no lasting adverse health effect.

c) A safety program is required when exposures are anticipated above the lower tier ERL.

d) Workers in occupational situations (persons permitted in restricted environments) can be made aware of the potential for adverse reactions and mitigation measures.

1.3.3.3 Frequencies between 100 kHz and 6 GHz

Above 100 kHz, the effect associated with exposure to CW fields transitions from electrostimulation to tissue heating, and exposures are assessed with reference to an averaging time that varies in accordance with whole-body or local exposure. The frequency 100 kHz nominally represents a “thermal crossover” below which electrostimulation effects dominate, and above which thermal effects dominate for CW exposure. However, for pulsed waveforms, especially those of a low duty factor, the upper frequency at which electrostimulation has been demonstrated reaches 5 MHz. This standard contains criteria to protect against adverse electrostimulation effects for pulsed waveforms with fundamental frequencies above 100 kHz.

For short-duration exposures (less than the averaging time) at frequencies above 100 kHz, the DRLs and ERLs associated with tissue heating are related to energy (i.e., specific absorption (SA) or incident energy density). It is possible, however, to continue to use the DRLs and ERLs expressed in power terms (i.e., SAR, power density, or equivalent fields), recognizing their time dependence. In this case the effect to be protected against includes tissue damage that can result from excessive RF heating or from potentially adverse behavioral effects consequent to heating.

For long-duration exposure (equal to or greater than the averaging time), the ERLs are extrapolated from the exposure levels and SARs from laboratory studies of nonhuman species, specifically, exposure levels and SARs that resulted in a behavioral disruption response. Behavioral disruption is considered to be the most sensitive reproducible response to RF exposure indicative of a potential adverse health effect (D’Andrea et al. [B313]). This process results in conservative DRLs and ERLs because of the superior thermoregulatory capacity of humans compared with laboratory animals.

The safety factors for both whole-body and local exposures have been estimated to be approximately 10 and 50 or more in terms of SAR or power density for the upper tier and lower tier, respectively. (See B.4 and B.7.6 for discussions of safety factors.) The safety factors for special exposure measures, such as local exposures, peak (short pulse) limits, and contact and induced currents in the limbs, are also of the order of 5 to 10 above 100 kHz. The margin of safety for the DRLs and ERLs is generally greater for whole-body exposure in the resonance region than for local exposures.

Recently published research results have helped to provide a quantitative perspective on the magnitude of these safety factors (Adibzadeh et al. [B34]). This research in which RF energy absorption rates have been compared to tissue-damaging temperatures has estimated that the arbitrary safety factor of 10 used for the upper tier exposure limits in this standard translates to actual values between 10.9 and 31.2 depending on the target size of the exposed tissue, averaging times, and the degree of perfusion of the tissue of concern.

For local exposure at frequencies where SAR is applicable, the averaging mass for peak spatial-average SAR (psSAR) is 10 g of tissue defined as a tissue volume in the shape of a cube. The averaging volume of 10 g of tissue would be represented as a cube with a volume of approximately 10 cm^3, or about 2.15 cm per side. At 3 GHz, the penetration depth of the field in high-water-content tissues is about 1.6 cm, which is smaller than the depth of the averaging volume. To harmonize with the new ICNIRP RF Guidelines,[12] the frequency range over which the DRL is expressed in terms of SAR has been extended to 6 GHz.

1.3.3.4 Frequencies above 6 GHz

For frequencies above 6 GHz, the effect being protected against is excessive RF heating—primarily surface heating causing a sense of prickling pain on the skin or eyes and possibly leading to tissue damage (burns). Because the heated layer of tissue is thin, the thermal time constant is considered to be short and heating can be rapid. However, heat is conducted into surrounding structures and the temperature rise is limited unless the exposure is prolonged. Also, the smaller the area of exposure, the greater the capacity of tissue to disperse localized heating, thereby reducing the thermal impact of intense exposure.

The body has an efficient thermoregulatory system that is designed to protect the body from sizeable variations in environmental temperature and thermal load due to exercise. Consequently, the DRLs (in terms of epithelial power density) and ERLs (incident power density) are very conservative for whole-body exposures. For whole-body exposures over the entire 6 GHz to 300 GHz range, the safety factor for whole-body exposure (relative to excessive thermal load on the body) in ordinary room environments is more than 10, and for persons in unrestricted areas, it ranges from more than 10 to more than 100. While the safety factors are smaller for individuals in very warm environments, the effect of RF heating of the body at the ERL for the upper tier is far outweighed by effects due to ambient temperature and relative humidity and the type of clothing worn. Consequently, the limits are considered to be sufficiently protective even for individuals in warm environments. In all cases, the safety factors for unrestricted environments are five times greater than those for restricted environments (Moore et al. [B1035]).

1.3.4 Risk assessment and safety programs

A safety program, such as described in IEEE Std C95.7, shall be implemented whenever the lower tier DRLs (or corresponding ERLs) can be exceeded (safety program initiation level). For persons in unrestricted environments, the lower tier DRLs shall not be exceeded. For persons permitted in restricted environments, the lower tier DRLs may be exceeded but the upper tier DRLs shall not be exceeded. The identification of restricted environments is accomplished via an exposure assessment. Any resulting RF safety program shall implement appropriate controls for access to the restricted environment. The purpose of the safety program is to prevent exposures that exceed the upper tier exposure limits. While safety programs are applied to fixed

[12] The draft of the *ICNIRP Guidelines on Limiting Exposure to Time-Varying Electric, Magnetic and Electromagnetic Fields (100 kHz to 300 GHz)* was published for public consultation in July 2018 (https://www.icnirp.org/en/activities/public-consultation/consultation-1.html).

(or stationary) sources of electromagnetic fields, portable devices such as mobile phones or professional two-way radios are subject to separate requirements for limiting psSAR in tissues. Manufacturers shall provide procedures to assure compliance with respect to the DRLs for either lower or upper exposure tiers as appropriate.

1.4 Word usage

The word *shall* indicates mandatory requirements strictly to be followed in order to conform to the standard and from which no deviation is permitted (shall equals is required to).[13,14]

The word *should* indicates that among several possibilities one is recommended as particularly suitable, without mentioning or excluding others; or that a certain course of action is preferred but not necessarily required (should equals is recommended that).

The word *may* is used to indicate a course of action permissible within the limits of the standard (may equals is permitted to).

The word *can* is used for statements of possibility and capability, whether material, physical, or causal (can equals is able to).

2. Normative references

The following referenced documents are indispensable for the application of this document (i.e., they must be understood and used, so each referenced document is cited in text and its relationship to this document is explained). 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™, IEEE Recommended Practice for Measurements and Computations of Radio Frequency Electromagnetic Fields with Respect to Human Exposure to Such Fields, 100 kHz–300 GHz.[15, 16]

IEEE Std C95.3.1™, IEEE Recommended Practice for Measurements and Computations of Electric, Magnetic, and Electromagnetic Fields with Respect to Human Exposure to Such Fields, 0 Hz to 100 kHz.

IEEE Std C95.7™, IEEE Recommended Practice for Radio Frequency Safety Programs, 3 kHz to 300 GHz.

[13] The use of the word *must* is deprecated and shall not be used when stating mandatory requirements, *must* is used only to describe unavoidable situations.
[14] The use of *will* is deprecated and shall not be used when stating mandatory requirements, *will* is only used in statements of fact.
[15] IEEE publications are available from the Institute of Electrical and Electronics Engineers (http://standards.ieee.org/).
[16] The IEEE standards or products referred to in Clause 2 are trademarks owned by the Institute of Electrical and Electronics Engineers, Incorporated.

3. Definitions, acronyms, abbreviations and symbols

3.1 Definitions

For the purposes of this document, the following terms and definitions apply. The *IEEE Standards Dictionary Online* should be consulted for terms not defined in this clause.[17]

action level: Deprecated. *See:* **safety program initiation level**.

action potential: A response of a nerve cell to a stimulus involving a propagating rapid depolarization of the potential across the cell membrane.

adverse health effect: An effect detrimental to an individual's physical well-being due to exposure to an electric, magnetic, or electromagnetic field or to induced or contact currents or voltages. For purposes of this standard, adverse effect is interchangeable with adverse health effect or adverse reaction. *See also:* **established adverse health effect**.

NOTE 1—Adverse effects do not include biological effects without a harmful health effect or changes in subjective feelings of well-being.[18]

NOTE 2—Sensory perceptions *per se* are not necessarily considered adverse health effects. For example, a perception 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.

NOTE 3—Painful or aversive sensations are considered adverse.

adverse health effect exposure level: The magnitude and/or duration of an exposure that can produce an adverse health effect.

adverse reaction factor (F_A): For electrostimulation effects, a multiplier ≥ 1 used to convert a median threshold for a biological effect up to the median level that makes this effect adverse.

arc: A conductive channel of ionized gas formed in the gap between two electrodes when the potential gradient is sufficient to cause dielectric breakdown of the insulating medium (e.g., air) between the electrodes.

average (temporal) power ($\overline{P}$): The time-averaged rate of energy transfer.

NOTE 1—The SI unit of average (temporal) power is watt (W).

NOTE 2—Average temporal power is expressed as

$$\overline{P} = \frac{1}{t_2 - t_1} \int_{t_1}^{t_2} P(t)dt$$

where $P(t)$ is the instantaneous power. The time duration ($t_2 - t_1$) could be source related (e.g., the waveform repetition period or duty factor) or use related [e.g., the averaging time associated with the exposure reference level (ERL)].

[17] *IEEE Standards Dictionary Online* is available at: http://dictionary.ieee.org.
[18] Notes in text, tables, and figures of a standard are given for information only and do not contain requirements needed to implement this standard.

average (temporal) power density: The power density in a propagating wave averaged over a specific time duration.

NOTE 1—The SI unit of average (temporal) power density is watt per square meter (W/m^2).

NOTE 2—The time duration could be source related (e.g., the waveform repetition period) or use related [e.g., the averaging time associated with the exposure reference level (ERL)].

averaging area: The area over which a physical quantity is averaged for assessing compliance.

NOTE—For compliance with the ERLs, the averaging area is the area over which the power density is averaged when the frequency is greater than 100 MHz, and the area over which the field strength squared is averaged when the frequency is between 100 kHz and 100 MHz. For frequencies below 100 kHz, see **averaging distance**.

averaging distance: The distance over which the *in situ* electric field is averaged when determining compliance with a dosimetric reference limit (DRL) for electrostimulation.

averaging mass: The mass over which the specific absorption rate is averaged when determining compliance with a dosimetric reference limit (DRL).

averaging time (T_{avg}): The appropriate time period over which exposure is averaged for purposes of determining compliance with the appropriate exposure reference level (ERL) or dosimetric reference limit (DRL).

averaging volume: The volume over which the specific absorption rate is averaged when determining compliance with a dosimetric reference limit (DRL).

axial cross section: A cross section of the body taken in a plane perpendicular to its long axis.

axial exposure: Exposure by an electric, magnetic or electromagnetic field perpendicular to the axial cross section.

axon: The long extension of a neuron that conducts electrical impulses.

biological effect: A biological effect caused by, or in response to, exposure to a biological, chemical, or physical agent, including electromagnetic energy.

NOTE 1—Biological effects are alterations of the structure, metabolism, or functions of a whole organism, its organs, tissues, and cells.

NOTE 2—Biological effects can occur without adverse health effects or even be beneficial; they can also cause harm in the short term (e.g., heating, without long-term consequences).

NOTE 3—Biological effects also can include sensation phenomena and adaptive responses.

biphasic: A waveform characteristic involving a reversal of polarity.

cardiac excitation: The electrical stimulation of a cardiac contraction.

central nervous system (CNS): The portion of the vertebrate nervous system consisting of the brain and spinal cord but not including the peripheral nerves.

cerebral cortex: The convoluted layer of brain cells (gray matter) forming the outer surface of each cerebral hemisphere.

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 species being considered and its lifespan).

conductivity: A property of materials that determines the magnitude of the electric current density when an electric field is impressed on the material.

NOTE—The SI unit of conductivity is siemens per meter (S/m); the inverse of resistivity.

contact current: The current flowing through a person contacting charged conducting objects.

NOTE—Contact current is specified as either that associated with a grasping contact (area assumed to be 15 cm^2) or a touch contact (area assumed to be 1 cm^2).

contact voltage: For purposes of this standard, an open-circuit voltage between two conducting objects that when bridged by a human would produce contact current. *See also:* **open-circuit voltage**.

continuous exposure: For purposes of this standard, exposure for durations exceeding the corresponding averaging time.

NOTE 1—In this context, exposure for less than the averaging time is considered a short-term exposure.

NOTE 2—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 nonproliferating 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 nonproliferating cells *in vitro* or in tissues, the definition is arbitrary.

controlled environment: Deprecated. *See:* **exposure environment**; **restricted environment**.

core temperature: The temperature of the internal structures of the body.

corona (air): A luminous discharge due to ionization of the air surrounding a conductor caused by a voltage gradient exceeding a certain critical value.

NOTE—Corona by-products can include RF fields, audible noise, and trace amounts of ozone.

coronal cross section: A cross section taken through the long axis of the body in a plane parallel to its front view.

coronal exposure: Exposure by a field perpendicular to the coronal cross section.

current density: The ratio of the flowing current to the cross-sectional area perpendicular to the direction of the current.

NOTE—The SI unit of current density is ampere per square meter (A/m^2).

depolarization (cellular): The reduction of the potential across a cellular membrane relative to its resting value.

direct electrostimulation: Deprecated. *See:* **induced electrostimulation.**

dosimetric reference limit (DRL): The exposure limit based on dosimetric thresholds for established adverse health effects expressed as *in situ* electric field strength (0 Hz to 5 MHz), specific absorption rate (100 kHz to 6 GHz), or epithelial power density (6 GHz to 300 GHz) and which provides an adequate margin of safety.

NOTE 1—DRLs are defined in conjunction with specified spatial and temporal averaging requirements.

NOTE 2—DRLs are equivalent to the quantity previously referred to as basic restrictions.

dosimetry: The discipline that quantifies the relationship between external exposure quantities and internal dose and dose rate quantities. *See also:* **specific absorption; specific absorption rate**.

NOTE—External exposure quantities include electric field, magnetic field, and electromagnetic field (incident power density); internal quantities include *in situ* electric field, dose, dose rate, specific absorption (SA), specific absorption rate (SAR), and epithelial power density, respectively.

duty cycle: *See:* **duty factor**.

duty factor: The ratio of pulse duration to the pulse period of a periodic pulse train.

NOTE—A duty factor of 1.0 corresponds to continuous-wave (CW) operation.

effect threshold: The threshold for a biological effect but not necessarily for an adverse effect.

electric field strength (*E*): Force exerted by an electric field on an electric point charge, divided by the electric charge.

NOTE—The SI unit for electric field strength is newton per coulomb or volt per meter (N/C = V/m).

electric field: A fundamental component of electromagnetic waves, which exists when there is a potential difference between two points in space.

electromagnetic field (EMF): The energy field radiating from a source and containing both electric and magnetic field components.

electromagnetic interference (EMI): Any electromagnetic disturbance, whether intentional or not, which interrupts, obstructs, or otherwise degrades or limits the effective performance or safe operation of an electronic or electrical device or system.

electrostimulation (ES): Induction of a propagating action potential in excitable tissue by an applied electrical stimulus; electrical polarization of presynaptic processes leading to a change in postsynaptic cell activity.

NOTE—Electrostimulation involves excitation of nerve and/or muscle tissue.

environmental field: An electric, magnetic, or electromagnetic field external to the body and measured in the absence of the body (in order not to perturb the field).

epithelial energy density: The energy flow through the epithelium per unit area directly under the body surface (i.e., in stratum corneum of the skin or corneal epithelium of eyes).

NOTE—The SI unit for epithelial energy density is joule per square meter (J/m^2).

epithelial power density: The power flow through the epithelium per unit area directly under the body surface (i.e., in stratum corneum of the skin or corneal epithelium of eyes).

NOTE 1—The SI unit for epithelial power density is watt per square meter (W/m^2).

NOTE 2—In this standard, the epithelial power density just inside the body surface is employed to define local dosimetric reference limits at frequencies greater than 6 GHz.

established adverse health effect: An effect detrimental to the health of an individual due to exposure to an electric, magnetic, or electromagnetic field, or to induced or contact currents, with the following characteristics:

a) It is supported by the weight of the evidence of that effect in studies published in the scientific literature.

b) The effect has been demonstrated by independent laboratories.

c) There is consensus in the scientific community that the effect occurs for the specified exposure conditions.

See also: **adverse health effect**.

established mechanism: For purposes of this standard, a mechanism with the following characteristics:

a) It can be used to explain or describe a biological effect in cells, animals, or humans.

b) An explicit model is proposed using equations or parametric relationships.

c) It has been verified in humans, or in animal data, thus, supporting confidence that the mechanism can be extrapolated to humans.

d) It is supported by scientific evidence.

e) It is widely accepted among experts in the scientific community.

exposure: The state of being in the presence of electric, magnetic, or electromagnetic fields, or in contact with a current or voltage source.

exposure environment: A defined area that is characterized by the maximum potential exposure that could occur within it.

a) **restricted environment:** An environment in which exposure can result in exceeding the unrestricted environment (lower tier) dosimetric reference limit (DRL). *See also:* **restricted tier**.

NOTE 1—Implementation of an effective safety program (see IEEE Std C95.7 for the RF range) is to help ensure that persons are not exposed above the DRL or ERL for the restricted environment.

NOTE 2—In some documents, exposure in restricted environments is referred to as “upper tier” or “controlled environment” or “occupational exposure.”

NOTE 3—Members of the general public are not permitted in restricted environments unless they become subject to the applicable safety program, at which time they are no longer considered members of the “general public.”

b) **unrestricted environment:** An environment in which exposure does not result in exceeding the dosimetric reference limit (DRL) that marks the safety program initiation level, and which serves as an exposure limit for the general public. *See also:* **unrestricted tier**; **general public**.

NOTE 1—The exposures can occur in living quarters or workplaces where there are no expectations that the DRL or ERL for unrestricted environments would be exceeded and where the induced currents or contact currents do not exceed the limits for unrestricted environments.

NOTE 2—In some documents, the unrestricted environment is referred to as a “lower tier” or an “uncontrolled environment” or a “general public exposure.”

exposure reference level (ERL): The maximum exposure level relative to ambient electric and/or magnetic field strength or power density, induced and/or contact current, or contact voltage.

NOTE 1—ERLs provide an adequate margin of safety against established adverse health effects.

NOTE 2—The ERL is expressed as the metric appropriate to the frequency and temporal characteristics of the exposure under consideration.

NOTE 3—The ERL is measured, estimated, or derived from the DRL (*in situ* electric field, SAR, or epithelial power density).

NOTE 4—The ERL may be exceeded if it can be demonstrated that the corresponding DRL is not exceeded.

NOTE 5—The ERLs and DRLs for contact current are the same.

NOTE 6—In some documents, ERLs are called reference levels, derived limits, permissible exposure limits, maximum permissible exposure values, action levels, or investigation levels.

extra systole: A heartbeat occurring before its normal time in the rhythm of the heart and followed by a compensatory pause.

extremities: Deprecated. *See:* **limbs**.

far-field (region): The region where the angular field distribution is essentially independent of distance from the source.

NOTE—In the far-field region, the field has a predominantly plane-wave character (i.e., locally very uniform distributions of electric field strength and magnetic field strength in planes transverse to the direction of propagation). For large antennas especially, the far-field region is also referred to as the "Fraunhofer region."

fluence: A measure of the energy per unit area of exposure contained in a pulse of electromagnetic radiation, that for the purposes of this standard is quantified by the product of the intensity of the pulse (W/m^2) times the pulse duration (s). *See also:* **incident energy density**.

NOTE—The SI unit for fluence is joule per square meter (J/m^2).

general public: All members of the human population who have no knowledge or control of their exposure and are, consequently, not permitted in a restricted environment. The unrestricted tier exposure limit applies to the general public. *See also:* **exposure environment (unrestricted)**.

NOTE—The general public includes, but is not limited to, children, pregnant women, individuals with impaired thermoregulatory systems, and persons using medications that can result in poor thermoregulatory system performance.

grasping contact: An electrical connection with a large energized conductor made by firmly holding the conductor in the hand.

NOTE—For purposes of computation, a contact area of 15 cm^2 is assumed.

grip tetanus: A condition where an electrical stimulus causes the hand to "freeze" its grip on an object. The threshold of grip tetanus is also called the let-go current. *See also:* **let-go current**.

Hall-effect voltage: The voltage developed between two points within a conductive medium due to the redistribution of moving charges in a magnetic field.

hazard: An intrinsic property or condition that has the potential to cause an adverse health effect.

hazard threshold (HT): The level above which an exposure, expressed in terms of the appropriate metric, can result in a hazard.

hertz (Hz): The SI unit of frequency, equal to one cycle per second.

NOTE—Common multiples are kilohertz (kHz; 1000 Hz), megahertz (MHz; 1 000 000 Hz), and gigahertz (GHz; 1 000 000 000 Hz).

incident energy density: For purposes of this standard, the quantity of energy per unit area that impinges on the body surface. *See also:* **fluence**.

NOTE—The SI unit for incident energy density is joule per square meter (J/m^2).

incident power density: For purposes of this standard, the quantity of power per unit area that impinges on the body surface. *See also:* **power density**.

NOTE 1—The SI unit for incident power density is watt per square meter (W/m^2).

NOTE 2—In this standard, the incident power density just outside the body surface is employed to define local exposure reference levels at frequencies greater than 6 GHz.

indirect electrostimulation: Stimulation through contact with a conducting object under the influence of an electric or magnetic field, including spark discharges.

induced current: Electric current flowing in the body of a person in a freestanding condition (no skin contact with conductive objects) due to an electromagnetic field.

NOTE—For compliance with standards, this is normally measured as the current through the feet or ankles of the person to ground with the use of current transformers having a sufficient aperture size to fit around the ankle.

***in silico*:** Performed on computer or via computer simulation.

***in situ*:** For purposes of this standard, *in situ* means within a biological tissue in its normal anatomical position.

***in vitro*:** Refers to studies and/or effects that occur in an artificial environment outside a living organism.

***in vivo*:** Refers to studies and/or effects that occur within the body of living organisms.

induced electrostimulation: Stimulation via the electric field within the biological medium induced by an external electric or magnetic field without direct contact with other conductors or spark discharges.

induction: An electric or a magnetic field in a conducting medium caused by the action of a time-varying external (environmental) electric or magnetic field.

joule (J): A unit of energy.

NOTE—One (1) joule = 1 watt second.

let-go current: The current level above which involuntary muscular contraction prevents release of a grip on an energized conductor. *See also:* **grip tetanus**.

NOTE 1—“Let-go current” is the preferred term but is often used interchangeably with “can’t let go current” or “lock-on current.”

NOTE 2—“Let-go” has occasionally been used for the threshold for withdrawal from an aversive stimulus.

limb: An entire leg or arm.

local exposure: An exposure condition in which a limited portion of the body is subject to most of the incident energy and is usually the result of: 1) the source being located very close to the body, or 2) a highly concentrated region of energy associated with contact with an energized conductor exposed to environmental fields.

lock-on current: *See:* **let-go current**.

lognormal distribution: A statistical distribution in which the logarithm of the statistical variate is normally distributed.

long-term exposure: Exposure for a duration much longer than the corresponding averaging time. *See also:* **chronic exposure**.

Lorentz force: The force on a moving charge within a magnetic field.

lower tier: *See:* **unrestricted tier**. *See also:* **exposure environment (unrestricted)**.

lowest observed adverse effect level (LOAEL): The lowest exposure level at which there is a biologically significant increase in occurrence or severity of an adverse effect in the exposed study population compared with its appropriate control group.

lowest observed effect level (LOEL): Lowest concentration or amount of a substance or physical agent, found by experiment or observation, that causes any alteration in morphology, behavior, functional capacity, growth, development, or lifespan of target organisms distinguishable from normal (control-sham exposed) organisms of the same species and strain.

low-level effects: Biological effects ascribed to exposure to low-level electric, magnetic, or electromagnetic fields, that is, at or below the corresponding dosimetric reference limits, taking exposure duration into account, in the frequency range covered in this standard (0 Hz to 300 GHz).

low-level fields: Electromagnetic fields in the frequency range 0 Hz to 300 GHz that produce induced (*in situ*) electric fields, specific absorption rate (SAR), or epithelial power density at or below the corresponding dosimetric reference limits.

magnetic field: A fundamental component of electromagnetic waves produced by a moving electric charge.

magnetic field strength (*H*): The magnitude of the magnetic field vector.

NOTE—The SI unit of magnetic field strength is ampere per meter (A/m).

magnetic flux density (*B*): A vector quantity that determines the force on a moving charge or charges (electric current).

NOTE 1—The SI unit of magnetic flux density is tesla (T).

NOTE 2—In air and simple (nonmagnetizable) media, B is related to the magnetic field strength H by $B = \mu \times H$, where μ is the permeability of the medium.

NOTE 3—One gauss (deprecated unit) equals 10^{-4} T.

magnetohydrodynamic effect: A force or potential imparted on a conductive fluid volume arising from its motion in the presence of a magnetic field.

margin of safety: The ratio of the lowest observed adverse effect level (LOAEL) to the dosimetric reference limit, after adjusting for possible measurement uncertainty, in a specific exposure situation.

NOTE 1—This is equivalent to the minimum possible safety factor when uncertainties are accounted for, while the nominal safety factor is derived from the LOAEL and dosimetric reference limit or exposure reference level. 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 the LOAEL and the established adverse health effects level.

NOTE 2—The safety factor incorporates uncertainties and environment safety factor values and is, therefore, somewhat arbitrary and is partially based on the value judgments from all stakeholders.

maximum permissible exposure (MPE) value: Deprecated. *See:* **exposure reference level**.

mean: The arithmetic average of a series of numerical values.

median: The value within a statistical distribution at which 50 % of data are above and 50 % are below.

median threshold: The threshold value within a statistical distribution at which 50 % of subjects have greater thresholds and 50 % have lesser thresholds.

mixed frequency fields: The superposition of two or more electromagnetic fields of differing frequencies.

monophasic: A waveform not reversing in polarity.

motor neuron: (**A**) A central neuron that initiates excitation of a peripheral nerve. (**B**) A peripheral nerve that innervates muscle.

NOTE—Definition (**B**) is generally used in this standard.

myelinated nerve: A nerve fiber containing insulating myelin sheaths that are interrupted by uninsulated segments called “nodes of Ranvier.”

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.

NOTE 1—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 radiating field predominates over the reactive field but lacks substantial plane-wave character and is complicated in structure.

NOTE 2—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 an electrically short antenna (e.g., half-wave dipole, where λ is the wavelength). For electrically large antennas, the radiating near-field region extends out to a distance from the antennas approximately equal to $2D^2/\lambda$, where D is the largest dimension of the antenna.

nerve fiber: A single nerve axon.

nerve: A bundle of axons.

neuron: A single cellular unit usually consisting of an axon, cell body, and dendritic tree.

nonthermal effects: Deprecated. *See:* **low-level effects**.

nonuniform field: A field that is not constant in amplitude, direction, and relative phase over the dimensions of the body or body part under consideration.

NOTE—In the case of electric fields, the definition applies to an environmental field that can be nonuniform when unperturbed by the presence of the body.

normal load conditions: The typical operating voltage and current of an electric power transmission line under conditions that exclude outages or other emergency operating conditions.

open-circuit voltage: The potential difference between two conducting objects without a current load being applied to the objects.

overexposure: For purposes of this standard, exposure that exceeds the applicable dosimetric reference limits defined in this standard.

parasitic reradiator: *See:* **reradiated field**.

partial-body exposure: *See:* **local exposure**.

peak field: The instantaneous value of a time-varying electric or magnetic field when at its maximum value.

peak power density: The maximum spatial and/or temporal power density in a propagating wave.

NOTE—The SI unit of peak power density is watt per square meter (W/m^2).

peak spatial-average SAR (psSAR): *See:* **specific absorption rate—peak spatial average**.

peripheral nerve: Nerve located outside the central nervous system and leading to or from the central nervous system.

phase duration (t_p): The time between zero crossings of a waveform in those cases where zero-crossings can be clearly discerned.

NOTE 1—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.

NOTE 2—For waveforms that have no clear crossings between maxima and minima, and do not correspond to an exponential function, the phase duration is the interval between 90 % of the peak on the rising and falling segments of the waveform.

phosphene: Visual sensation caused by nonphotic stimuli.

NOTE—Electrophosphenes are induced by electric currents; magnetophosphenes are induced magnetically.

pinna: The largely cartilaginous projecting portion of the outer ear consisting of the helix, lobule, and anti-helix. *Syn:* **auricle**.

plane wave: An electromagnetic wave characterized by mutually orthogonal electric and magnetic fields that are related by the wave impedance of a plane wave in a vacuum η_0 ($\approx 377\ \Omega$).[19]

NOTE—For plane waves, power density (S), the electric field strength (E), and the magnetic field strength (H) exhibit the following relationship: $S = E^2/\eta_0$ or $S = \eta_0 H^2$, where S is in W/m^2, E is in V/m, and H is in A/m.

[19] According to the National Institute of Standards and Technology (NIST) (USA), $\eta_0 = 376.730313461\ \Omega$. This exact value of η_0 (derived as the square root of the ratio of free-space permeability μ_0 to free-space permittivity ε_0) $\approx 377\ \Omega$ when rounded to the nearest integer, which is also coincidentally $\approx 120\pi\ \Omega$. Some literature uses rounding to, for example, $\eta_0 \approx 376.73\ \Omega$.

plane-wave-equivalent power density (S_E, S_H): The calculated power density of an electromagnetic wave that is equal in magnitude to the power density of a plane wave having the same electric (E) or magnetic (H) field strength. *Syn:* **equivalent-plane-wave power density**.

NOTE 1—The SI unit of plane-wave-equivalent power density is watt per square meter (W/m^2).

NOTE 2—Plane-wave-equivalent power density is computed as follows:

$$S_E = \frac{|E|^2}{\eta_0} \text{ W/m}^2 \text{ or } S_H = \eta_0 |H|^2 \text{ W/m}^2$$

where $|E|$ and $|H|$ are the root mean square (rms) values of the electric field strengths and magnetic field strengths, respectively, and η_0 is the wave impedance of a plane wave in a vacuum ($\eta_0 \approx 377\ \Omega$).

polarization (cellular): The electric potential formed across a cell membrane.

polarization (electromagnetic): The locus of the tip of the electric field vector observed over time at a fixed point.

postsynaptic cell: The cell receiving excitation in a synaptic junction between two nerve cells.

power density (S): Electromagnetic power per unit area crossing a surface of interest. *See also:* **plane-wave-equivalent power density**.

NOTE 1—The SI unit of power density is watt per square meter (W/m^2).

NOTE 2—The surface of interest is frequently chosen to be orthogonal to the electromagnetic wave direction of propagation.

NOTE 3—For an arbitrary time-dependent signal waveform, the instantaneous power density point value is

$S(t) = \boldsymbol{\mathcal{E}}(t) \times \boldsymbol{\mathcal{H}}(t) \cdot \hat{\mathbf{n}}$ where $\hat{\mathbf{n}}$ is the unit-vector normal to the surface of interest, $\boldsymbol{\mathcal{E}}(t)$ is the instantaneous electric field vector, $\boldsymbol{\mathcal{H}}(t)$ is the instantaneous magnetic field vector, and t is the time variable.

NOTE 4—For a periodic time-dependent signal waveform, the time-averaged power density point value is

$$S_T = \frac{1}{T} \int_{t_0}^{t_0+T} S(t)dt$$

where T is the waveform period and t_0 is any arbitrary time.

NOTE 5—For a time-harmonic signal waveform, the time-averaged power density point value is

$$S = \frac{1}{2} \Re\left(\mathbf{E} \times \mathbf{H}^*\right) \cdot \hat{\mathbf{n}}$$

where $\Re$ is the real-value operator, $\mathbf{E}$ is the complex electric field point value, $\mathbf{H}$ is the complex magnetic field point value, and the superscript * represents complex conjugation.

NOTE 6—The instantaneous electric field and magnetic field vectors depend on their respective complex fields as follows:

$$\boldsymbol{\mathcal{E}}(t) = \Re\left(\mathbf{E}e^{j\omega t}\right),\ \boldsymbol{\mathcal{H}}(t) = \Re\left(\mathbf{H}e^{j\omega t}\right)$$

where $\omega = 2\pi f$ is the waveform radian frequency, having defined f as the waveform frequency and $j = \sqrt{-1}$ as the imaginary unit.

presynaptic cell: The cell that provides or inhibits excitation at a synapse, usually by release of a neurotransmitter to the post synaptic cell.

probability factor: Deprecated. *See:* **safety factor**.

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.

projected area: For purposes of this standard, the maximum cross-sectional area of the exposed portion of the body normal to the direction of propagation.

proposed mechanism: A bioelectric mechanism that is described but lacks experimental or theoretical validation. *See also:* **established mechanism**.

pulse duration: *See:* **pulse width**.

pulse repetition frequency (prf): In pulse-modulated systems using recurrent pulses, the number of pulses per unit of time (s).

pulse repetition period (prp): In a pulse-modulated system using recurrent pulses, the reciprocal of pulse repetition frequency.

pulse width: For purposes of this standard, the time interval between the points on the leading and trailing edges at which the instantaneous value bears a specified relation to the maximum instantaneous value of the pulse, usually the time interval between the half-power points of the pulse. *Syn:* **pulse duration**.

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.

radio frequency (RF): A frequency that is useful for radio transmission, generally considered frequencies between approximately 3 kHz and 300 GHz.

radio frequency burn: An injury that has occurred when contact is made with, or arcing from, an RF-energized conductor resulting in painful heating of tissue commensurate with visible skin damage and is usually accompanied by an involuntary withdrawal.

radio frequency heating: Elevation of tissue temperature that can result from exposure to radio frequency electromagnetic energy.

reduction factor: Deprecated. *See:* **safety factor; margin of safety**.

relative phase: The phase angle of a sinusoidal waveform relative to the phase angle of another waveform measured at a different point within the conductive medium or with respect to a stated reference waveform.

reradiated 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.

NOTE—Reradiated fields are sometimes called "reflected" or more correctly "scattered fields." The scattering object, sometimes called a "reradiator," "secondary radiator," or "parasitic radiator" can be a source of contact currents.

resonance region: The frequency region where absorption of RF energy by an object (e.g., the human body) is enhanced.

NOTE—Absorption of energy by an object in an electromagnetic field depends on the size and orientation of the object with respect to the wavelength and polarization vector of the incident field, respectively.

restricted environment: *See:* **exposure environment**.

restricted tier: A range of potential exposure extending above the limit for the lower tier [dosimetric reference limits (DRL) or, optionally, exposure reference levels (ERL)]; used to define exposure environment (restricted). *Syn:* **upper tier**.

rheobase: For purposes of this standard, the minimum electric field amplitude capable of initiating neural excitation as observed in a strength-frequency (S-F) or strength-duration (S-D) relationship (applicable to a stimulus phase duration that is long in comparison with the strength-duration time constant). *See also:* **phase duration**.

NOTE 1—The unit for rheobase is volt per meter (V/m).

NOTE 2—Also applied to the minimum plateau in an S-F relationship for electrostimulation.

risk: The likelihood or probability that a person will be harmed by a particular hazard.

risk assessment: A process characterized by evaluating the likelihood and consequences of exposure.

root-mean-square (rms) value (of a periodic function): The square root of the arithmetic mean of a sequence of squared values extracted from measurements or other data. For a time-varying function Y with a period T, the rms value of Y is

$$Y_{\text{rms}} = \left[\frac{1}{T} \int_{a}^{a+T} Y^2 dt \right]^{1/2}$$

where a is any value of time t.

safety factor (SF): A divisor (≥ 1) applied to the exposure level that causes an adverse effect, used to establish a dosimetric reference limit (DRL) that includes inter-subject biological variability, uncertainties concerning threshold effects due to pathological conditions or drug treatment, uncertainties in computational models, uncertainties in dosimetry, and variations in temperature and humidity.

NOTE 1—A safety factor is not intended to compensate for uncertainties associated with compliance measurements.

NOTE 2—At low frequencies (generally ≤ 100 kHz for CW exposure), where electrostimulation is the dominant mechanism of interaction, the divisor is applied to the induced *in situ* electric field strength [typically measured in volt per meter (V/m)]; at radio frequencies (generally ≥ 100 kHz for CW exposure), the divisor is applied to the specific absorption rate or epithelial power density, typically measured in watt per kilogram (W/kg) or watt per square meter (W/m^2), respectively.

NOTE 3—When used for frequencies below 100 kHz (where electrostimulation is the effect of major concern), the DRL, the adverse reaction, and probability and safety factors are expressed as first-order quantities, such as *in situ* electric field or current density magnitude.

NOTE 4—When used for frequencies greater than 5 MHz (where heating is the effect of concern), these values are expressed as second-order quantities, such as power and energy. Between 100 kHz and 5 MHz, first-order quantities are used for electrostimulation and second-order quantities are used for thermal effects.

safety margin: *See:* **margin of safety**.

safety program: An organized system of policies, procedures, practices, and plans designed to help ensure compliance with exposure limits associated with electric, magnetic, and electromagnetic fields; contact voltage; and contact and induced currents.

NOTE—A safety program typically includes 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 similar to the elements described in IEEE Std C95.7 for the RF range.

safety program initiation level: The lower tier dosimetric reference limit (DRL) or the corresponding exposure reference level (ERL), above which a safety program shall be implemented.

NOTE—In some documents, the safety program initiation level is referred to as the action level.

sagittal cross section: A cross section along the long axis of the body, parallel to its side view.

sagittal exposure: Exposure by a field perpendicular to the sagittal cross section.

scattered radiation: *See:* **reradiated field**.

short-term exposure: Exposure for a duration less than the corresponding averaging time.

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.

spatial average: A method for evaluating compliance with a recommended exposure value [dosimetric reference limit (DRL) or exposure reference level (ERL)] by averaging field strength, field strength squared, or power density over a specified area or volume. (See Annex D for details.)

spatial maximum: The maximum point value of a spatially distributed parameter.

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 mass density (ρ).

$$SA = \frac{dW}{dm} = \frac{dW}{\rho dV}$$

NOTE—The SI unit of specific absorption is joule per kilogram (J/kg).

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 mass density (ρ).

$$SAR = \frac{d}{dt}\left(\frac{dW}{dm}\right) = \frac{d}{dt}\left(\frac{dW}{\rho dV}\right)$$

NOTE 1—The SI unit of SAR is watt per kilogram (W/kg).

NOTE 2—SAR can be related to the electric field at a point in the body by

$$SAR = \frac{\sigma |E|^2}{\rho}$$

where

σ is the conductivity of the tissue (S/m)
ρ is the density of the tissue (kg/m^3)
E is the root-mean-square (rms) electric field strength in tissue (V/m)

NOTE 3—SAR can be related to the initial rate of rise 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 the specific heat capacity [J/(kg ºC)]

This assumes that measurements are made under "ideal" nonthermodynamic circumstances, that is, no heat loss by thermal diffusion, radiation, or thermoregulation (blood flow, sweating, etc.).

specific absorption rate—peak spatial-average (psSAR): The maximum local SAR averaged over a specified volume or mass (e.g., any 10 g of tissue in the shape of a cube).

NOTE—The SI unit of psSAR is watt per kilogram (W/kg).

specific absorption rate—whole-body-average (WBA SAR): The SAR averaged over the entire mass of the body.

NOTE—The SI unit of WBA SAR is watt per kilogram (W/kg).

specific heat capacity: The amount of heat necessary to raise the temperature of a unit mass of a substance 1 ºC.

NOTE—Specific heat capacity is expressed in units of joule per kilogram kelvin [J/(kg K)] or joule per kilogram degree Celsius [J/(kg ºC)].

strength-duration (S-D) curve: The functional relationship between the threshold of excitation and the duration of an excitatory stimulus.

strength-duration time constant (τ_e): The functional parameter in a strength-duration curve that describes the temporal inflection point between the rheobase and the rising threshold segment.

strength-frequency (S-F) constant (f_e): The functional parameter in an S-F curve that describes the frequency inflection point between the rheobase and the rising threshold segment.

strength-frequency curve: The functional relationship between the threshold of excitation and the frequency of an excitatory stimulus.

synapse: The site of functional apposition between two neurons at which an electrical signal from one neuron is transmitted to another by either electrical or chemical means.

NOTE—In the typical synapse, the impulse is transmitted by a chemical substance called a neurotransmitter.

systole: Contraction of the heart, ejecting blood into the pulmonary artery and the aorta.

thermal effects: Changes associated with heating of the whole body or an affected region sufficient to induce a biological effect.

NOTE—Established adverse health effects are associated with whole-body heating at levels that usually increase core temperature by approximately 1 °C or more.

thermal resistance: The resistance of a material or combination of materials to heat flow across a temperature gradient, usually measured in kelvin per watt (K/W) or degree Celsius per watt (°C/W).

thermal time constant: For purposes of this standard, a measure of the time scale on which a biological system adjusts its temperature in response to added thermal energy.

NOTE 1—In the context of RF exposure, the thermal time constant indicates the time scale over which an exposed region of tissue reaches a new steady-state temperature after initiation of exposure to RF energy.

NOTE 2—The thermal response of tissue is a complex function of its thermal characteristics and RF exposure parameters, and the thermal time constant is to be understood as a measure of the time scale over which the thermal response occurs. A useful operational definition of the thermal time constant τ (in seconds) is

$$\tau = \frac{c\,\Delta T_{ss}}{SAR}$$

where ΔT_{ss} is the steady-state temperature increase [in kelvin (K) or degree Celsius (°C)] in a region of tissue produced by RF exposure at a specific absorption rate [SAR in watt per kilogram (W/kg)], and c is the specific heat capacity of the tissue in joule per kilogram degree Celsius [J/(kg ºC)].

threshold: The level of a stimulus marking the boundary between a response and a nonresponse.

touch contact: A contact of small area made between the human body and an energized conductor.

NOTE—In this standard, a contact area of 1 cm^2 is the assumed touch contact area.

uncertainty factor: Deprecated. *See:* **safety factor**.

uniform field: A field that is constant in amplitude, direction, and relative phase over the dimensions of the body or body part under consideration.

NOTE—In the case of ambient electric fields, the uniformity of an external field is assessed in the presence of any permanent structures and fixtures but without the presence of any human bodies that would disturb the field. In the case of induced *in situ* fields, the definition applies to an electric field that results in a constant potential difference across the axonal nodes of an unbent nerve fiber.

unrestricted environment: *See:* **exposure environment (unrestricted)**.

unrestricted tier: A range of potential exposure extending up to the limit for the lower tier [dosimetric reference limits (DRL) or, optionally, exposure reference levels (ERL)]; used to define exposure environment (unrestricted). *Syn:* **lower tier.** *See also:* **exposure environment (unrestricted)**.

upper tier: *See:* **restricted tier**. *See also:* **exposure environment (restricted)**.

ventricular fibrillation: Arrhythmia of the ventricles of the heart characterized by rapid uncoordinated contractions.

visual evoked potential (VEP): An endogenous potential ensuing in the brain and measured on the scalp in response to a visual stimulus.

voxel: A three-dimensional computational element.

waveform: The variation of an electrical amplitude with time.

NOTE—Unless otherwise stated, in this standard the term waveform refers to values (or measurements) at sites within the biological medium.

weight of scientific evidence: For purposes of this standard, the outcome of assessing the peer-reviewed scientific literature about the biological and health effects from exposure to electric, magnetic, and electromagnetic fields from 0 Hz to 300 GHz. *Syn:* **weight-of-evidence**.

NOTE—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.

whole-body exposure: The case in which the projected area of the entire body is exposed to the incident fields.

3.2 Acronyms and abbreviations

AAMI	Association for the Advancement of Medical Instrumentation
ADP	adenosine diphosphate
AM	amplitude modulated
ANSI	American National Standards Institute
ASHRAE	American Society of Heating, Refrigeration, and Air-Conditioning Engineers
avg	average
BBB	blood-brain barrier
BF	blood flow
BP	blood pressure
CEM	cumulative equivalent minute
CI	confidence interval
CNS	central nervous system
CTM	critical thermal maximum
CW	continuous wave
d	day (as used in days per week, abbreviated to d/wk)
DA	detailed anatomic (model)
DAMPS	Digital Advanced Mobile Phone System
DH	dielectric heater
DRL	dosimetric reference limit
DSB	double strand break
ECT	electroconvulsive therapy
EEG	electroencephalogram
EHC	World Health Organization's "Environmental Health Criteria" document
ELF	extremely low frequency

EM	electromagnetic
EMC	electromagnetic compatibility
EMF	electromagnetic field
EMI	electromagnetic interference
ENU	ethylnitrosourea
ER	endoplasmic reticulum
ERL	exposure reference level
ERP	event-related potential
ES	electrostimulation
FDTD	finite-difference time-domain
GABA	gamma-ami-nobutyric acid
GSM	Global System for Mobile Communications
HACU	high-affinity choline uptake
HP	high power
HR	heart rate
HT	hazard threshold
IARC	International Agency for Research on Cancer
ICES	International Committee on Electromagnetic Safety
ICNIRP	International Commission on Non-Ionizing Radiation Protection
IEC	International Electrotechnical Commission
IEGMP	Independent Expert Group on Mobile Phones
IET	Institution of Engineering and Technology (United Kingdom)
IH	induction heater
ISM	industrial, scientific, and medical
ISO	International Organization for Standardization
LOAEL	lowest observed adverse effect level
LOEL	lowest observed effect level
LP	low power
LPS	lipopolysaccharide
LRWG	Literature Review Working Group
MMW	millimeter wave, mm wave
MN	micronuclei
MPE	maximum permissible exposure
MRI	magnetic resonance imaging
MSC	mesenchymal stem cell
NADH	nicotinamide adenine dinucleotide
NCRP	National Council on Radiation Protection and Measurements
NIEHS	National Institute of Environmental Health Sciences (USA)
NOAEL	no observable adverse effect level
NRC	National Research Council

NRPB	National Radiological Protection Board
ODC	ornithine decarboxylase
OR	odds ratio
PMV	predictive mean vote
PO/AH	preoptic/anterior hypothalamic
PPE	personal protective equipment
prf	pulse repetition frequency
PS	phosphatidylserine
psSAR	peak spatial-average SAR
PW	pulsed wave
RAWG	Risk Assessment Working Group
RBC	red blood cell
RF	radio frequency
rms	root mean square
RT-PCR	reverse transcription polymerase chain reaction
SA	specific absorption
SAR	specific absorption rate
SASB	Standards Association Standards Board
SCE	sister chromatid exchange
S-D	strength-duration (time constant, curve, etc.)
S-F	strength-frequency (constant, curve, etc.)
SI	Système International d'Unités (international system of units)
SkBF	skin blood flow
SD	Sprague Dawley
SSB	single strand break
TG	thapsigargin
UV	ultraviolet
VDT	video display terminal
VEP	visual evoked potential
VF	ventricular fibrillation
VS	vestibular schwannoma
WBA	whole-body average
WHO	World Health Organization
wk	week (as used in days per week, abbreviated to d/wk)

3.3 Frequency bands

ELF	extremely low frequency (0 Hz to 3 kHz)
VLF	very low frequency (3 kHz to 30 kHz)
LF	low frequency (30 kHz to 300 kHz)
MF	medium frequency (0.3 MHz to 3 MHz)
HF	high frequency (3 MHz to 30 MHz)
VHF	very high frequency (30 MHz to 300 MHz)
UHF	ultra high frequency (300 MHz to 3 GHz)
SHF	super high frequency (3 GHz to 30 GHz)
EHF	extremely high frequency (30 GHz to 300 GHz)[20]

3.4 Letter and mathematical symbols for quantities

a, b	semi-major and semi-minor axes of elliptical representation of exposed body part NOTE—The symbol a is also used as the largest dimension of an antenna.
A_i	magnitude of the ith Fourier component of a waveform
B	magnetic flux density, expressed in tesla (T) NOTE—Tesla and gauss (G) are related by 1 G = 10^{-4} T. Similarly, 1 mG = 0.1 μT.
$\dot{B}_0$	rheobase time-rate-of-change of magnetic flux density (dB_0/dt) in a strength-duration or strength-frequency relationship
$\dot{B}$	the time rate of change of magnetic flux density, dB/dt, expressed in tesla per second (T/s)
$\dot{B}_p$	peak allowable limit on the time derivative of magnetic flux density
c	specific heat capacity
CEM_{43}	cumulative equivalent minute at 43 °C
D	the largest electrical dimension of an antenna element or array
d_a	averaging distance used to determine compliance with an *in situ* electric field dosimetric reference limit
d_e	spatial extent of an *in situ* electric field
DRL	dosimetric reference limit
ERL	exposure reference level
E	electric field strength expressed in volt per meter (V/m)
E_0	minimum (rheobase) electric field strength in a strength-duration or strength-frequency relationship (V/m)
E_{0b}	rheobase dosimetric reference limit
E_{0t}	rheobase threshold electric field strength
E_i	*in situ* electric field strength (V/m)
f	frequency expressed in hertz (Hz)
f_e	upper transition frequency in a strength-frequency relation (Hz)

[20] Per ITU Radio Regulations (2016), for the EHF frequency range, the corresponding metric subdivision is known as millimeter waves (abbreviated as MMW in this document; see 3.2).

f_i	frequency of the *i*th Fourier component of a waveform (Hz)
F_A	adverse reaction factor
h	height of a standing person, expressed in meter (m)
H	magnetic field strength, expressed in ampere per meter (A/m); NOTE—Related to magnetic flux density by $B = \mu \times H$.
I	current, expressed in ampere (A)
I_c	contact current, expressed in ampere (A)
J	current density, expressed in ampere per square meter (A/m^2)
ME_i	maximum allowable exposure of either the *in situ* electric field, the environmental field, or the contact current at frequency f_i
P	power, expressed in watt (W)
$P(t)$	instantaneous power, expressed in watt (W)
$\overline{P}$	average (temporal) power, expressed in watt (W)
q	charge, expressed in coulomb (C)
ρ	mass density, expressed in kilogram per cubic meter (kg/m^3)
SA	specific absorption, expressed in joule per kilogram (J/kg)
SAR	specific absorption rate, expressed in watt per kilogram (W/kg)
S	power density, expressed in watt per square meter (W/m^2)
SF	safety factor
S_E	plane-wave-equivalent power density value based on the electric field strength
S_H	plane-wave-equivalent power density value based on the magnetic field strength
SF_U	safety factor, upper tier
SF_L	safety factor, lower tier
T_{avg}	averaging time (s)
t_p	phase duration (s)
V_{oc}	open circuit voltage (V)
W	energy, expressed in joule (J)
σ	conductivity, expressed in siemens per meter (S/m)
ε_0	electric permittivity of free space ($\varepsilon_0 = 8.85 \times 10^{-12}$ F/m)
ε_r	relative permittivity
η	wave impedance, expressed in ohm (Ω)
η_0	wave impedance of a plane wave in a vacuum ($\eta_0 \approx 377$ Ω)[21]
λ	wavelength, expressed in meter (m)
μ	magnetic permeability, expressed in henry per meter (H/m)
μ_0	magnetic permeability in a vacuum or in air ($\mu_0 = 4\pi \times 10^{-7}$ H/m)
τ	pulse width (s)
τ_e	transition duration in a strength-duration relationship, expressed in second (s)
τ_h	time constant of the leakage of charge applied to a human subject (s)

[21] See footnote 19.

3.5 Unit symbols

A	ampere
C	coulomb
ºC	degree Celsius
cm	centimeter
dB	decibel
F	farad
G	gauss
GHz	gigahertz (10^9 Hz)
H	henry
h	hour
Hz	hertz
J	joule
K	kelvin
kg	kilogram
kHz	kilohertz (10^3 Hz)
m	meter
mm	millimeter
m^2	square meter
MHz	megahertz (10^6 Hz)
min	minute
N	newton
Ω	ohm
S	siemens
s	second
T	tesla
V	volt
W	watt

4. Exposure limits

4.1 General

DRLs and ERLs for exposure to electric, magnetic, or electromagnetic fields are defined to protect against painful electrostimulation in the frequency range of 0 Hz 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, while above 5 MHz, only the thermal limits apply, and both sets of limits apply in the transition region (100 kHz to 5 MHz). In the transition region, the limits based on electrostimulation are generally more limiting for low-duty-factor exposures, while the thermal-based limits are more limiting for continuous-wave fields. ERLs also are defined for contact currents, induced currents, and contact voltages for the frequency range of 0 Hz to 110 MHz.

Evaluation of compliance with this standard ideally includes a determination that the DRLs are not exceeded. This determination is difficult in most cases because they can only be carried out using sophisticated analytical or measurement techniques, which are often limited to laboratory-type studies. ERLs are derived from the DRLs to provide a readily assessed quantity via measurements or computations. The value of the ERL is determined such that when the measured exposure complies with the ERL, it is also in compliance with the DRL. The ERL, however, may be exceeded if it can be demonstrated that the corresponding DRL is not exceeded. Assessment of exposure to electric, magnetic, and electromagnetic fields may be accomplished by measurement and/or analysis, using appropriate instrumentation and measurement techniques or numerical/analytical methods, as described in standards such as IEEE Std C95.3, IEEE Std C95.3.1, and IEC 62232 [B656].

4.2 DRLs and ERLs for exposure to electric and magnetic fields—Electrostimulation effects (0 Hz to 5 MHz)

4.2.1 DRLs—*In situ* electric field (0 Hz to 5 MHz)

For human exposure to electromagnetic energy at frequencies from 0 Hz to 5 MHz, the DRLs refer to limits on the *in situ* electric fields that protect against adverse effects associated with electrostimulation. Such limits are derived with consideration of adverse electrical thresholds, their distribution among the population, and safety factors.

Table 1 lists DRLs for particular areas of the body in terms of the *in situ* electric field. Two parameters are listed in the table: the rheobase *in situ* field E_0 and an S-F parameter f_e, which is also referred to as the transition frequency. Limits are determined from Table 1 as in Equation (1).

$$E_i = \begin{cases} E_0 & \text{for } f < f_e \\ E_0(f/f_e) & \text{for } f \geq f_e \end{cases} \quad (1)$$

where E_i is the maximum allowed induced *in situ* electric field.

In Table 1, the *in situ* electric field DRL applies to the rms electric field strength measured in the direction and location providing the maximum *in situ* electric field vector (vector magnitude) over a 5 mm linear distance. The averaging time for an rms measurement is 0.2 s for frequencies above 25 Hz. For lower frequencies, the averaging time is such that at least 5 cycles are included in the average but with a maximum of 10 s. DRLs expressed in Equation (1) apply to frequencies in the range of 0 Hz to 5 MHz.

The DRLs for each specified organ/body area shall all be met. The controlling DRL depends on the exposure situation. With whole-body exposure, the ERLs are specified so as to avoid exceeding a DRL in the most sensitive region of the body. The *in situ* magnetic field DRL below 10 Hz shall be restricted to a peak value

of 167 mT for the unrestricted environment and to a peak value of 500 mT for restricted environments. *In situ* magnetic field DRLs for frequencies above 10 Hz are not necessary because Table 2 (see 4.2.2.1) electrostimulation criteria dominate the biological effect. This is discussed in Annex B (see Table B.7).

Table 1—DRLs for electrostimulation mechanisms (0 Hz to 5 MHz)[22]

Exposed tissue	Frequency f_e (Hz)[b]	Persons in unrestricted environments E_0 (V/m)[a,b,c,d]	Persons permitted in restricted environments E_0 (V/m) [a,b,c,d]
Brain	20	5.89×10^{-3}	1.77×10^{-2}
Heart	167	0.943	0.943
Limb	3350	2.10	2.10
Other tissues	3350	0.701	2.10

[a] Interpretation of Table 1 is as follows: $E_i = E_0$ for $f \le f_e$; $E_i = E_0 (f / f_e)$ for $f \ge f_e$.

[b] Parameters E_0 (induced *in situ* electric field in V/m) and f_e (in Hz) as expressed in Equation (1) apply to DRLs in various regions of the body.

[c] DRLs in Table 1 do not apply to induced fields or current crossing skin-to-skin contact (see B.2.1.2.2.2).

[d] Tabulated values are given as rms quantities.

4.2.2 ERLs for magnetic field (0 Hz to 5 MHz)

4.2.2.1 Exposure of head and torso to sinusoidal magnetic fields

Table 2 lists the ERLs for the magnetic field (magnetic 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 s for frequencies above 25 Hz. For lower frequencies, the averaging time is such that at least 5 cycles are included in the average but with a maximum of 10 s.

Note that the ERLs in Table 2 and Table 4 (see 4.2.3.1) protect against adverse reactions associated with electrostimulation; Table 7 and Table 8 (see 4.3.2) apply to effects associated with tissue heating. All four tables shall be considered and the corresponding values for the appropriate tier satisfied at all applicable frequencies.

Refer to Table 2 to help ensure compliance with the DRLs of Table 1.[23] However, lack of compliance with Table 2 does not necessarily indicate lack of compliance with the DRLs, but that it shall then be necessary to evaluate whether the DRLs have been met to demonstrate compliance. This would typically be done using analytical methods. If the DRLs in Table 1 are not exceeded, then the ERLs in Table 2 may be exceeded. Consequently, it is sufficient to demonstrate compliance with either Table 1 or Table 2. For purposes of demonstrating compliance with this standard, the magnetic field ERL (Table 2) and the electric field ERL (Table 4) shall be considered separately, not additively (see B.2.1.10.3).

[22] Entries in Table 1 and elsewhere in this standard are sometimes given to three significant figures. This degree of precision is provided so that the reader can follow the various derivations and relationships presented in this standard, and it does not imply that the numerical quantities are known to that precision.

[23] The safety limits for electrostimulation are based on conservative assumptions of exposure; however, they cannot address every conceivable assumption.

Table 2—Magnetic field ERLs for exposure of head and torso (0 Hz to 5 MHz) [see Figure 1 and Figure 2 for graphical representation]

Frequency range (Hz)	Persons in unrestricted environments [a,b,c]		Persons permitted in restricted environments [a,b,c]	
	B **(mT)**	***H*** **(A/m)**	***B*** **(mT)**	***H*** **(A/m)**
< 0.153	118	9.39×10^4	353	2.81×10^5
0.153 to 20	$18.1 / f$	$1.44 \times 10^4 / f$	$54.3 / f$	$4.32 \times 10^4 / f$
20 to 751	0.904	719	2.71	2.16×10^3
751 to 3.35×10^3	$687 / f$	$5.47 \times 10^5 / f$	$2060 / f$	$1.64 \times 10^6 / f$
3.35×10^3 to 5×10^6	0.205	163	0.615	490

[a] Tabulated values are given as rms quantities.

[b] f is expressed in Hz.

[c] Tabulated ERLs apply to spatial maximum.

4.2.2.2 Limb exposures

The ERLs for the limbs (entire arms and legs) are listed in Table 3. The averaging time for an rms measurement is 0.2 s. Refer to Table 3 to help ensure compliance with the DRLs of Table 1. However, lack of compliance with Table 3 does not necessarily indicate lack of compliance with the DRLs, but that it shall then be necessary to evaluate whether the DRLs are met to demonstrate compliance.

Table 3—Magnetic field ERLs for the limbs (0 Hz to 5 MHz) [see Figure 1 and Figure 2 for graphical representation]

Frequency range (Hz)	Persons in unrestricted environments [a,b,c]		Persons permitted in restricted environments [a,b,c]	
	B **(mT)**	***H*** **(A/m)**	***B*** **(mT)**	***H*** **(A/m)**
< 10.7	353	2.81×10^5	353	2.81×10^5
10.7 to 3350	$3790 / f$	$3.02 \times 10^6 / f$	$3790 / f$	$3.02 \times 10^6 / f$
3350 to 5×10^6	1.13	900	1.13	900

[a] Tabulated values are given as rms quantities.

[b] f is expressed in Hz.

[c] Tabulated ERLs apply to spatial maximum.

4.2.2.3 Nonuniform 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 values in Table 2 (see 4.2.2.1) shall apply to the maximum field over the head or torso, and Table 3 (see 4.2.2.2) shall apply likewise to the limbs. Alternatively, demonstration of compliance with the DRLs is sufficient to demonstrate compliance with this standard.

4.2.2.4 Pulsed or nonsinusoidal magnetic fields

4.2.2.4.1 General

When the magnetic flux density waveform is nonsinusoidal, the maximum exposure shall conform to either 4.2.2.4.2.2 or 4.2.2.4.2.3. Since both criteria are conservative, adherence to either is sufficient to demonstrate compliance with the ERLs or DRLs.

4.2.2.4.2 Restrictions based on temporal peak field

4.2.2.4.2.1 General

Demonstration of compliance with 4.2.3 is sufficient to demonstrate compliance with restrictions based on the temporal peak field. Subclause 4.2.2.4.2.2 applies to the *in situ* induced electric field; 4.2.2.4.2.3 applies to the external (environmental) field.

4.2.2.4.2.2 Peak *in situ* electric field (DRL)

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 Table 1 for nonsinusoidal waveforms, frequency is defined as $f = 1/(2t_p)$, where t_p is the phase duration of a peak excursion of the *in situ* electric field. Phase duration is defined as the time between zero crossings of a waveform. For an exponential waveform, t_p is interpreted as the duration measured from the waveform peak to a point at which it decays to e^{-1} (≈ 36.8 %) of its peak value. Peak limits apply to instantaneous values measured through a bandwidth from zero to the highest frequency content of the waveform under consideration.

4.2.2.4.2.3 Peak external magnetic field (ERL)

The temporal peak of the time derivative of the external magnetic field B shall be limited according to the following procedure, where B is a time-varying magnetic flux density waveform whose compliance is under evaluation:[24]

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 4.2.2.4.2.2.

c) Determine the allowable peak limit on $\dot{B}$ from Table 2 (see 4.2.2.1) or Table 3 (see 4.2.2.2) as

$$\dot{B}_p = \sqrt{2} ERL \times (2\pi f)$$

where

$\dot{B}_p$ is the maximum permissible value of $\dot{B}$
ERL is the rms magnetic flux density consistent with Table 2 or Table 3
f = $1/(2t_p)$
t_p is the phase duration of a peak excursion of $\dot{B}$

4.2.2.4.3 Restriction based on Fourier components (DRL or ERL)

The requirements of this subclause may be satisfied as an alternative to 4.2.2.4.2.

For an exposure waveform consisting of multiple frequencies, a test for compliance of the exposure waveform shall satisfy the criterion of Equation (2).

[24] Note that $B = \mu \times H$; $\mu = \mu_0 = 4\pi \times 10^{-7}$ H/m when in free space, air, and nonferromagnetic or nonparamagnetic media (that is, in living organisms except very close to deposits of magnetite or other magnetic substances).

$$\sum_{0\ \mathrm{MHz}}^{5\ \mathrm{MHz}} \frac{A_i}{RL_\mathrm{i}} \le 1 \qquad (2)$$

where A_i is the magnitude of the ith Fourier component of the exposure waveform and RL_i represents either the ERL [from Table 2 (in 4.2.2.1) or Table 3 (in 4.2.2.2)] or the basic *in situ* field restriction [DRL from Table 1 (in 4.2.1)] 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 having a meaningful (> 1 %) contribution to the summation (up to 5 MHz). Note that A_i and RL_i each represent the same quantity with the same units. For instance, if A_i is the magnitude of a magnetic flux density waveform, then RL_i shall also be a measure of magnetic flux density. Alternatively, both A_i and RL_i could be measures of the time derivative of the field or the induced *in situ* electric field.

4.2.3 ERL for the external electric field (0 Hz to 100 kHz)

4.2.3.1 Uniform whole-body exposure to sinusoidal electric fields

Table 4 lists ERLs in terms of the unperturbed (absent a person) external electric field. It is assumed that the unperturbed field is constant in magnitude, direction, and relative phase over a spatial dimension that would encompass the human body. The averaging time for an rms measurement shall be 0.2 s for frequencies above 25 Hz. For lower frequencies, the averaging time is such that at least 5 cycles are included in the average but with a maximum of 10 s. For restricted environments in which an exposed individual is not within reach of a grounded conductive object, the ERLs in Table 4 may be exceeded. This standard does not specify limits for situations involving contact with ungrounded conducting objects (e.g., a person touching a conducting object above ground on an elevated, insulated platform). (See Annex B.)

For purposes of demonstrating compliance with this standard, the magnetic field ERLs (Table 2; see 4.2.2.1) and electric field ERLs (Table 4) shall be considered separately, not additively (see B.2.1.10.3).

Table 4—Electric field ERLs (0 Hz to 100 kHz)—Whole-body exposure (see Figure 1 and Figure 2 for graphical representation)

Persons in unrestricted environments		Persons permitted in restricted environments	
Frequency range (Hz)	***E*** [c,d,e] **(V/m)**	**Frequency range (Hz)**	***E*** [c,d,e] **(V/m)**
0 to 368	5 000 [a]	0 to 272	20 000 [b]
368 to 3000	$1.84 \times 10^6 / f$	272 to 2953	$5.44 \times 10^6 / f$
3000 to 100 000	614	2953 to 100 000	1842
NOTE 1—At 5 kV/m, induced spark discharges are painful to approximately 7 % of adults (well-insulated individual touching ground). NOTE 2—Painful discharges are readily encountered at 20 kV/m and are possible at 5 kV/m to 10 kV/m without protective measures.			

[a] Within areas designated as power line rights-of-way (or similarly designated areas, e.g., easement or corridor), the ERL for persons in unrestricted environments is 10 kV/m.

[b] The limit of 20 kV/m may be exceeded in restricted environments when a worker is not within reach of a grounded conducting object. A specific limit is not provided in this standard.

[c] Tabulated values are given as rms quantities.

[d] The assessed value shall be the spatial average of the rms field strength over the projected height of the human body determined in the absence of the body (see D.1.2.1 for a discussion of spatial averaging and field perturbation caused by the observer).

[e] f is expressed in Hz.

4.2.3.2 Exposure to nonuniform 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 when addressing the possibility of electrostimulation as opposed to averaging the squares of fields as at higher frequencies when addressing the rate of the deposition of thermal energy) shall be restricted to the ERL in Table 4 (see 4.2.3.1). For restricted environments in which an exposed individual is not within reach of a grounded conducting object, the ERLs in Table 4 may be exceeded. This standard does not specify limits for such cases. In no case shall the DRLs of Table 1 or the contact current limits of Table 12 and Table 13 (see 4.4.1.1) be exceeded.

4.2.3.3 Pulsed or nonsinusoidal electric fields

When the waveform of the external electric field is nonsinusoidal, such as with pulsed or mixed frequency waveforms, the rms value of the spatially averaged external field shall conform to the ERLs of Table 4 (see 4.2.3.1), as well as to either of the criteria as stated in 4.2.2.4.2 and 4.2.2.4.3 for magnetic fields. For this application, the external magnetic field is replaced by the unperturbed electric field; A_i represents the magnitude of the ith Fourier component of the external electric field waveform, and ERL_i is the maximum permissible electric field magnitude at frequency f_i.

4.2.4 Graphs of the ERLs of 4.2.2 and 4.2.3 for exposure to electric and magnetic fields

Figure 1 (unrestricted environments) and Figure 2 (restricted environments) show graphical representations of the ERLs of Table 2, Table 3, and Table 4 (in 4.2.2.1, 4.2.2.2, and 4.2.3.1, respectively) for electric and magnetic fields.

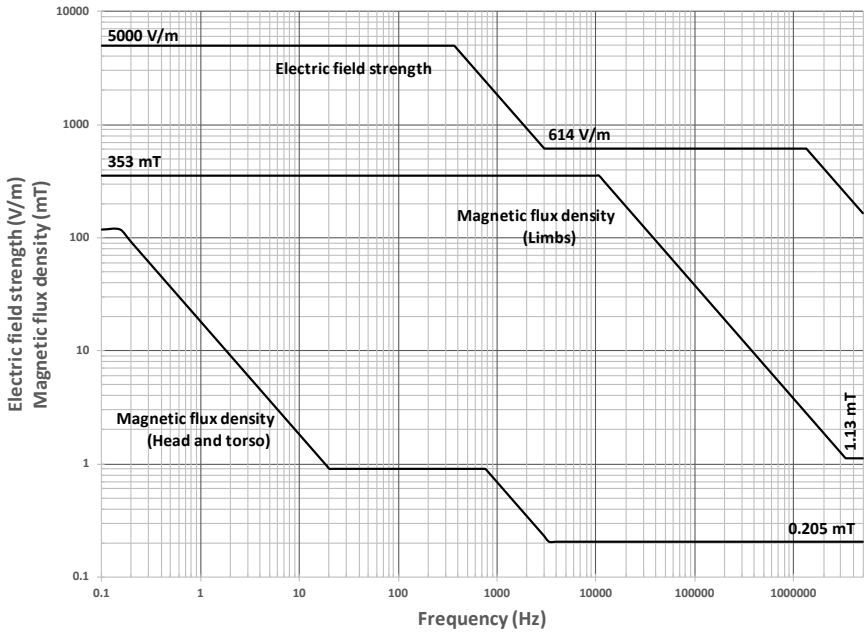


Figure 1—Graphical representations of the ERLs of Table 2, Table 3, and Table 4 for electric and magnetic fields—persons in unrestricted environments

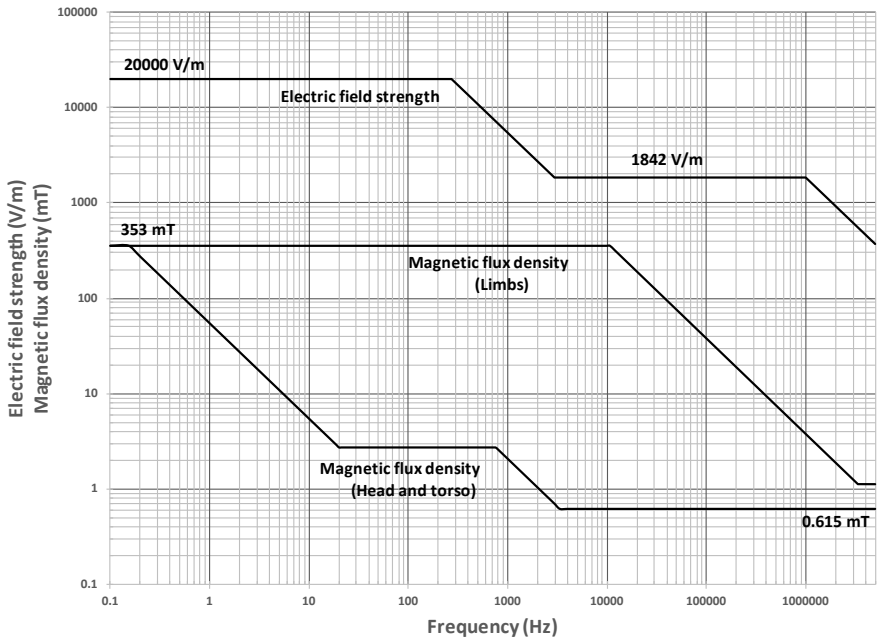


Figure 2—Graphical representations of the ERLs of Table 2, Table 3, and Table 4 for electric and magnetic fields—persons permitted in restricted environments

4.3 DRLs and ERLs for exposure to electromagnetic fields—Thermal effects (100 kHz to 300 GHz)

4.3.1 DRLs (100 kHz to 300 GHz)

Among all the metrics of effects thresholds available in the literature, the exposure response reported for disruption of food-motivated behavior in laboratory animals is the most sensitive, reproducible indicator of an adverse health effect threshold across animal species and across the frequency spectrum. Annex B provides a detailed explanation. The effect threshold of 4 W/kg is associated with a rise in core temperature during whole-body exposure of approximately 1 °C (de Lorge [B334], Ziskin and Morrissey [B1548]). In the RF range, a WBA SAR of 0.4 W/kg is a scientifically based exposure limit deemed to protect against adverse health effects. To minimize the possibility of exceeding this limit, the standard specifies that a safety program shall be established where RF exposures are anticipated to exceed an SAR of 0.08 W/kg.[25] For exposures of persons permitted in restricted environments, in which exposure can exceed the lower tier ERLs, an RF safety program shall be instituted to avoid exposure that exceeds the restricted tier ERLs or DRLs (see IEEE Std C95.7).

In the transition region of 100 kHz to 5 MHz, two sets of DRLs apply [cf. Table 1 (see 4.2.1) and Table 5]. The local exposure DRLs shown in Table 5 are established to protect against an excessive temperature rise in any part of the body that might result from local or nonuniform exposure. When averaging SAR over a 10 g volume of tissue in the limbs and the pinnae, only SAR values for the respective tissue shall be considered. If any cubic volume contains tissue from both the body and the limbs or pinna, each shall be considered separately. Specifically, when determining the average SAR in a 10 g cube of tissue in the body, apply the averaging algorithm using the methodology for calculation and measurements in IEC/IEEE 62704-1™-2017 [B661] and IEEE Std 1528™-2013 [B664], respectively. In addition, the

[25] Safety programs can provide for access to restricted environments by anyone including members of the general public who are subject to a safety program such as IEEE Std C95.7. In such cases, the upper tier DRLs and ERLs apply. However, many restricted environments are unlikely to be conducive to the development of a safety program that would permit the general public to enter.

orientations of the cubes used for SAR averaging shall align with the coordinate axes used in the experimental measurement or numerical procedures.

DRLs to protect against adverse health effects associated with surface heating are established for epithelial power density for local exposures at frequencies between 6 GHz and 300 GHz.[26] These DRLs (Table 6) and associated ERLs (Table 10 in 4.3.3.1 and Table 11 in 4.3.3.2) are defined for local exposures. For whole-body exposure compliance purposes, the limits are defined in terms of ERLs as shown in Table 7 and Table 8 (see 4.3.2).

Table 5—DRLs (100 kHz to 6 GHz)

Conditions	Persons in unrestricted environments SAR (W/kg)[a]	Persons permitted in restricted environments SAR (W/kg)[a]
Whole-body exposure	0.08	0.4
Local exposure[b] (head and torso)	2	10
Local exposure[b] (limbs and pinnae)	4	20

[a] SAR is averaged over 30 min for whole-body exposure and 6 min for local exposure (see B.6 for averaging time).

[b] Averaged over any 10 g of tissue (defined as a tissue volume in the shape of a cube).[27]

Table 6—Local exposure DRLs (6 GHz to 300 GHz)

Conditions	Epithelial power density (W/m^2)[a,b,c]	
	Persons in unrestricted environments	Persons permitted in restricted environments
Body surface	20	100

[a] Epithelial power density through body surface is averaged over 6 min.

[b] Averaged over any 4 cm^2 of body surface at frequencies between 6 GHz and 300 GHz (defined as area in the shape of a square at surface of the body).

[c] Small exposed areas above 30 GHz: If the exposed area on the body surface is small (< 1 cm^2 as defined by −3 dB contours relative to the peak exposure), the epithelial power density is allowed to exceed the DRL values of Table 6 by a factor of 2, with an averaging area of 1 cm^2 (defined as area in the shape of a square at the body surface).

4.3.2 Whole-body exposure ERLs (100 kHz to 300 GHz)

Because of the difficulty in determining whether an exposure complies with the DRLs, ERLs were derived. The ERLs, which protect against adverse health effects associated with heating, are provided in this subclause for convenience in exposure assessments. For human exposure to electromagnetic energy at radio frequencies from 100 kHz to 300 GHz, the ERLs, in terms of rms electric (E) and magnetic (H) field strengths, the power density (S) and plane-wave-equivalent power densities (S_E, S_H) are presented as a function of frequency in Table 7 and Table 8. For uncorrelated (in time) fields, such as multiple field exposure situations (e.g., different frequency field sources), compliance is determined by summing the percentages of the applicable ERLs in terms of E^2, H^2, or power density that each frequency field represents and ensuring that this sum does not exceed 100 % (IEC/TR 62630 [B659]). If exposure levels are determined via theoretical analysis, consideration of possible reflections of fields shall be included.

For frequencies between 100 kHz and 6 GHz, compliance with Table 7 and Table 8 implies compliance with the DRLs for WBA SAR. However, lack of compliance with Table 7 and Table 8 does not necessarily imply

[26] Ongoing research on highly localized exposures (over several mm^2 or less) above 30 GHz will be examined in future revisions.
[27] The averaging volume of 10 g of tissue would be represented as a 10 cm^3 cube (approximately 2.15 cm per side).

lack of compliance with the DRLs, but rather to demonstrate compliance, it shall then be necessary to perform additional evaluations to determine whether the DRLs have been met. If the DRLs given earlier are not exceeded, the ERLs in Table 7 and Table 8 may be exceeded. Consequently, it is sufficient to demonstrate compliance with either the whole-body DRLs in Table 5 (see 4.3.1) or the whole-body ERLs in Table 7 or Table 8. Note that between 6 GHz and 300 GHz, the ERLs in Table 7 and Table 8 are in terms of field strength and power density, and whole-body average SAR does not apply.

Table 7—ERLs for whole-body exposure of persons in unrestricted environments (100 kHz to 300 GHz) [see Figure 3 for graphical representation]

Frequency range (MHz)	Electric field strength (E)[a,b,c] (V/m)	Magnetic field strength (H)[a,b,c] (A/m)	Power density (S)[a,b,c] (W/m^2)		Averaging time (min)
			S_E	S_H	
0.1 to 1.34	614	16.3 / f_M	1000	100 000 / f_M^2	30
1.34 to 30	823.8 / f_M	16.3 / f_M	1800 / f_M^2	100 000 / f_M^2	30
30 to 100	27.5	158.3 / $f_M^{1.668}$	2	9 400 000 / $f_M^{3.336}$	30
100 to 400	27.5	0.0729	2		30
400 to 2000	—	—	f_M / 200		30
2000 to 300 000	—	—	10		30

NOTE—S_E and S_H are plane-wave-equivalent power density values, based on electric or magnetic field strength respectively, and are commonly used as a convenient comparison with ERLs at higher frequencies and are sometimes displayed on commonly used instruments.

[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 ERLs in Table 7. For more typical nonuniform exposures, the mean values of the exposure fields, as obtained by spatially averaging the plane-wave-equivalent power densities or the squares of the field strengths, are compared with the ERLs in Table 7. (See notes to Table 7 through Table 11 in 4.3.5.)

[b] f_M is the frequency in MHz.

[c] The *E, H,* and *S* values are those rms values unperturbed by the presence of the body.

Table 8—ERLs for whole-body exposure of persons permitted in restricted environments (100 kHz to 300 GHz) [see Figure 4 for graphical representation]

Frequency range (MHz)	Electric field strength (E)[a,b,c] (V/m)	Magnetic field strength (H)[a,b,c] (A/m)	Power density (S)[a,b,c] (W/m^2)		Averaging time (min)
			S_E	S_H	
0.1 to 1.0	1842	16.3 / f_M	9000	100 000 / f_M^2	30
1.0 to 30	1842 / f_M	16.3 / f_M	9000 / f_M^2	100 000 / f_M^2	30
30 to 100	61.4	16.3 / f_M	10	100 000 / f_M^2	30
100 to 400	61.4	0.163	10		30
400 to 2000	—	—	f_M / 40		30
2000 to 300 000	—	—	50		30

NOTE—S_E and S_H are plane-wave-equivalent power density values, based on electric or magnetic field strength respectively, and are commonly used as a convenient comparison with ERLs at higher frequencies and are sometimes displayed on commonly used instruments.

[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 ERLs in Table 8. For more typical nonuniform exposures, the mean values of the exposure fields, as obtained by spatially averaging the plane-wave-equivalent power densities or the squares of the field strengths, are compared with the ERLs in Table 8. (See notes to Table 7 through Table 11 in 4.3.5.)

[b] f_M is the frequency in MHz.

[c] The *E, H,* and *S* values are those rms values unperturbed by the presence of the body.

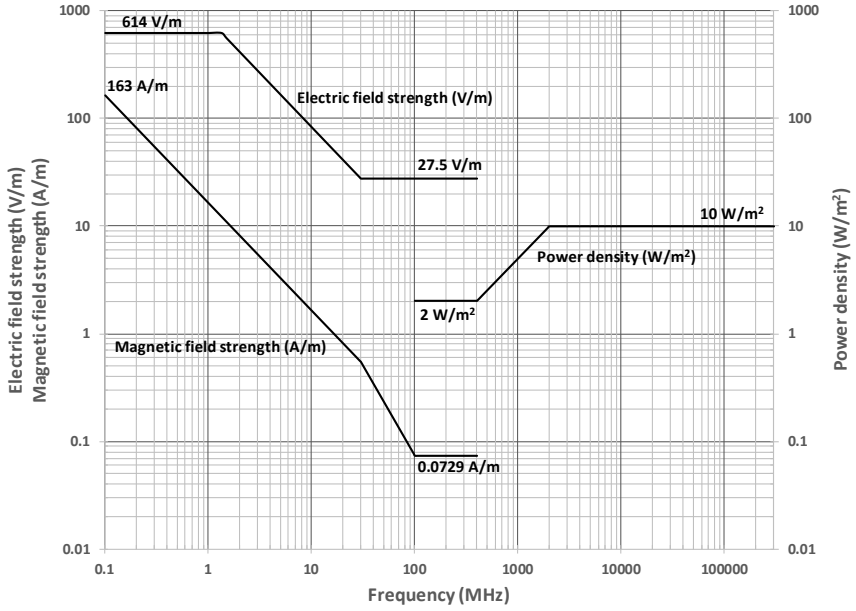


Figure 3—Graphical representations of the ERLs in Table 7 for electric and magnetic fields and plane-wave-equivalent power density—Persons in unrestricted environments

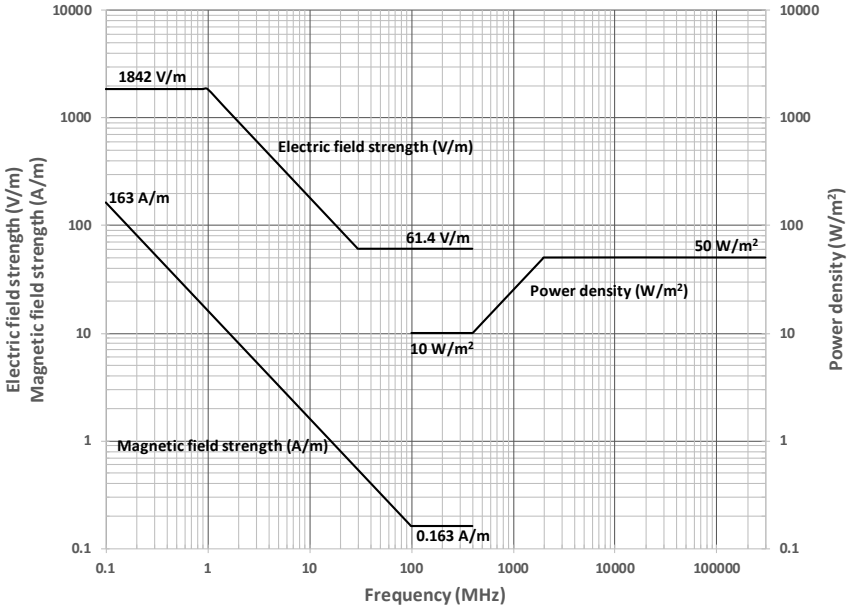


Figure 4—Graphical representations of the ERLs in Table 8 for electric and magnetic fields and plane-wave-equivalent power density—Persons permitted in restricted environments

4.3.3 Local exposure ERLs

4.3.3.1 Frequencies between 100 kHz and 6 GHz

For frequencies below 100 MHz, local-exposure ERLs of Table 9 and Table 10 are set at five times the corresponding whole-body ERL in Table 7 and Table 8, while for frequencies above 2 GHz, local-exposure ERLs are set at four times the corresponding whole-body ERL. Local-exposure ERL values for frequencies between 100 MHz and 2 GHz have been modified to accommodate recent numerical data reported by Findlay and Dimbylow [B416] as well as recommendations by Kühn et al. [B794], suggesting that adjustments in the exposure limits would be necessary to help ensure compliance with the local DRL. In the frequency range 100 MHz to 2 GHz, the formulas for power density in Table 9 and Table 10 result in ratios of the local ERL to whole-body ERL that vary with frequency with a maximum ratio of 9.5 associated with 400 MHz. The ratios are conservative relative to the multiplier values given in Figure B.6; that is, they are always less than the multiplier shown. (Details of the derivation are explained in B.3.3.)

Table 9—Local exposure ERLs (100 kHz to 6 GHz)—Persons in unrestricted environments[e]

Frequency range (MHz)	Electric field strength (E) [a,b,c,d] (V/m)	Magnetic field strength (H) [a,b,c,d] (A/m)	Power density (S) [a,b,c,d] (W/m^2)	
			S_E	S_H
0.1 to 1.34	1373	$36.4/f_M$	5000	$500\,000/f_M^2$
1.34 to 30	$1842/f_M$	$36.4/f_M$	$9000/f_M^2$	$500\,000/f_M^2$
30 to 100	61.4	$353/f_M^{1.668}$	10	$47\,000\,000/f_M^{3.336}$
100 to 400	$21.2 \times f_M^{0.232}$	$0.0562 \times f_M^{0.232}$	$1.19 \times f_M^{0.463}$	
400 to 2000	—	—	$1.19 \times f_M^{0.463}$	
2000 to 6000	—	—	40	

NOTE 1—Below 6 GHz, portable devices are typically tested for DRL compliance (e.g., SAR), for which distinct limits for head and torso, pinnae and limbs are defined.

NOTE 2— S_E and S_H are plane-wave-equivalent power density values, based on electric or magnetic field strength respectively, and are commonly used as a convenient comparison with ERLs at higher frequencies and are sometimes displayed on commonly used instruments.

[a] Determined in air at the location of the body surface.

[b] Spatial and temporal peaks averaged over 6 min.

[c] f_M is the frequency in MHz.

[d] The *E, H* and *S* values are those rms values unperturbed by the presence of the body.

[e] See notes to Table 7 through Table 11 in 4.3.5.

Table 10—Local exposure ERLs (100 kHz to 6 GHz)—Persons permitted in restricted environments[e]

Frequency range (MHz)	Electric field strength (E) [a,b,c,d] (V/m)	Magnetic field strength (H) [a,b,c,d] (A/m)	Power density (S) [a,b,c,d] (W/m^2)	
			S_E	S_H
0.1 to 1.0	4119	36.4/ f_M	45 000	500 000 / f_M^2
1.0 to 30	4119/ f_M	36.4/ f_M	45 000 / f_M^2	500 000 / f_M^2
30 to 100	137.3	36.4 / f_M	50	500 000 / f_M^2
100 to 400	$47.3 \times f_M^{0.232}$	$0.125 \times f_M^{0.232}$	$5.93 \times f_M^{0.463}$	
400 to 2000	—	—	$5.93 \times f_M^{0.463}$	
2000 to 6000	—	—	200	

NOTE 1—Below 6 GHz, portable devices are typically tested for DRL compliance (e.g., SAR), for which distinct limits for head and torso, pinnae and limbs are defined.

NOTE 2— S_E and S_H are plane-wave-equivalent power density values, based on electric or magnetic field strength respectively, and are commonly used as a convenient comparison with ERLs at higher frequencies and are sometimes displayed on commonly used instruments.

[a] Determined in air at the location of the body surface.

[b] Spatial and temporal peaks averaged over 6 min.

[c] f_M is the frequency in MHz.

[d] The *E, H,* and *S* values are those rms values unperturbed by the presence of the body.

[e] See notes to Table 7 through Table 11 in 4.3.5.

4.3.3.2 Frequencies between 6 GHz and 300 GHz

For frequencies between 6 GHz and 300 GHz, the ERLs in Table 11 apply.

Table 11—Local exposure ERLs (6 GHz to 300 GHz)[f]

Frequency	Persons in unrestricted environments Incident Power Density (W/m^2)[a,b,c,d,e]	Persons in restricted environments Incident Power Density (W/m^2)[a,b,c,d,e]
6 GHz	40	200
6 GHz to 300 GHz	$55 f_G^{-0.177}$	$274.8 f_G^{-0.177}$
300 GHz	20	100

[a] Incident power density is averaged over 6 min for local exposure.

[b] Averaged over any 4 cm^2 of body surface for 6 GHz to 300 GHz (area defined as surface of the body in the shape of a square).

[c] Small exposed areas above 30 GHz: If the exposed area on body surface is small (<1 cm^2 as defined by −3 dB contours relative to the peak exposure), the incident power density is allowed to exceed the ERL values of Table 11 by a factor of 2, with an averaging area of 1 cm^2 (defined as area in the shape of a square at surface of the body).

[d] Assessed in air at the location of the body, but the body is absent during assessment.

[e] f_G is the frequency in GHz.

[f] See notes to Table 7 through Table 11 in 4.3.5.

4.3.4 Peak power density limits for local exposure

4.3.4.1 Instantaneous peak power density restrictions

As specified in Table 7 and Table 8 (see 4.3.2), time-averaged whole-body exposure shall meet the whole-body ERL regardless of the waveforms, including pulsed fields. For pulsed RF fields, it was determined that the peak power density limits should be further limited for local exposures for persons in both the restricted and unrestricted exposure environments. For exposures to pulsed RF fields in the range of 100 kHz to

300 GHz, peak power density limits are provided to prevent unintentionally high local exposure and to preclude high SA in the frequency range of 100 kHz to 6 GHz or epithelial energy density above 6 GHz. (The history and rationale for the peak power limits can be found in B.4.3.)

The total incident energy density of a pulse train in any period of 100 ms is

$$\sum_{i=1}^{n}\left(S_{i\text{peak}} \times \tau_i\right) \text{ J/m}^2$$

where

n is the number of pulses within any period of 100 ms. Note that $n = 1$ for a single pulse.
$S_{i\text{peak}}$ is the average (temporal) power density, averaged over τ_i, of the ith pulse (W/m^2).
τ_i is the width (s) of the ith pulse (or a partial pulse width if any period of 100 ms captures only a portion of a pulse).

Compliance is established using Equation (3).

$$\sum_{i=1}^{n}\left(S_{i\text{peak}} \times \tau_i\right) \leq \frac{ERL_{\text{local}} \times t_{\text{avg}}}{5} \text{ J/m}^2 \tag{3}$$

where

t_{avg} is 6 min
ERL_{local} is the ERL for local exposure at the given wavelength shown in Table 9, Table 10, and Table 11

The summation on the left side of the equation applies to any 100 ms of the exposure.

This is also equivalent to limiting the SA (J/kg) below 6 GHz to one-fifth of that which would be permitted for a continuous field as provided in Equation (4), where $SAR_{i\text{peak}}$ is the temporal peak SAR (W/kg) of the ith pulse.

$$\sum_{i=1}^{n}\left(SAR_{i\text{peak}} \times \tau_i\right) \leq \frac{DRL_{\text{local}} \times t_{\text{avg}}}{5} \text{ J/kg} \tag{4}$$

where

$SAR_{i\text{peak}}$ is the temporal peak SAR (W/kg) of the ith pulse
t_{avg} is 6 min
DRL_{local} is the local SAR limit shown in Table 5

The summation on the left side of the equation applies to any 100 ms of the exposure.

4.3.4.2 Incident energy density restrictions for intense pulses

Notwithstanding the limitations on instantaneous peak power density defined earlier, for intense pulses (such as in certain military weapons systems) in the millimeter-wave frequency range (30 GHz to 300 GHz), the maximum local incident energy density per pulse (fluence—incident power density integrated over the pulse duration, J/m^2), shall be limited to

a) **Persons in unrestricted environments:**

$< 0.2\ \tau^{1/2}$ kJ/m^2

b) **Persons permitted in restricted environments:**

$< 1\ \tau^{1/2}$ kJ/m^2

The units for coefficients in these expressions are kJ/(m^2 s$^{1/2}$), where τ is the pulse width in seconds. For these limits, the exposure above 30 GHz shall be averaged over 1 cm^2 areas of body surface.

4.3.5 Notes to Table 7 through Table 11

The following provides explanatory notes for Table 7 through Table 11:

a) The ERLs refer to exposure values obtained by spatially averaging the plane-wave-equivalent power densities, depending on frequency as follows (see Annex D for details):

   1) **Frequencies between 100 kHz and 6 GHz:** The ERLs for fields between 100 kHz and 6 GHz limit the WBA SAR and, therefore, correspond to the spatial average of the incident plane-wave-equivalent power density (or spatial average of the squares of the rms electric field strengths or magnetic field strengths) averaged over the projected area of the body.

   2) **Frequencies between 6 GHz and 300 GHz:** The ERL is expressed in terms of the incident power density spatially averaged over any square area of 4 cm^2. For exposures from millimeter-wave pulses, the fluence limit is averaged over 1 cm^2 square area of the body surface. The ERLs in Table 11 (see 4.3.3.2) provide guidance on limiting local exposure to RF fields above 6 GHz where the region of interest is the body surface.

b) For near-field exposures at frequencies below 400 MHz, the applicable ERL is in terms of rms electric and magnetic field strength, as given in Table 7 and Table 8 (see 4.3.2), columns 2 and 3. For convenience, the ERL may be expressed as plane-wave-equivalent power density, given in Table 7 and Table 8, column 4. For frequencies below 30 MHz, both the rms electric and the magnetic field strength shall be determined; for frequencies between 30 MHz and 400 MHz, either field component is sufficient provided that the point in question is in the far-field of the source. In the near field of a source, both fields shall be determined and compliant with the ERLs. For determining compliance with the ERLs in Table 7 or Table 8 (see 4.3.2) at the higher frequencies (above 400 MHz), either field component may be used when expressed as plane-wave-equivalent power density.

c) Compliance with the WBA ERL is intended to help ensure that the WBA DRL is not exceeded. However, in spatially nonuniform fields, compliance with the WBA ERL might not ensure compliance with the local DRL. For this reason, Table 9 and Table 10 provide guidance on limiting the magnitude of local ERL, which is intended to help ensure that local DRL is not exceeded.

d) For uncorrelated (in time) fields, for instance, mixed or broadband fields at a number of frequencies for which there are different values of the ERL, the percentage of the ERL [in terms of E^2, H^2, or power density (S)] incurred within each frequency interval shall be determined and the sum of all such percentages shall not exceed 100 %. For frequencies between 100 kHz and 5 MHz the ERLs for electrostimulation and for heating apply independently. In this frequency band the fractional summation for each ERL shall be considered separately. See Annex D for examples of ERL summations for each type of ERL.

   In a similar manner, for mixed or broadband induced currents at a number of frequencies for which there are different values of the ERL, the fraction of the induced current limits (in units of A/m^2) incurred

within each frequency interval shall be determined, and the sum of all such fractions shall not exceed unity.

4.3.6 Assessing compliance with the DRLs and ERLs—Thermal effects (100 kHz to 300 GHz)

With the possible exception of the evaluation of devices that produce highly localized exposures at frequencies between 100 kHz and 6 GHz (e.g., mobile telephones), and instances where the body is extremely close to the source, depending on wavelength, direct assessment of the SAR for comparison with the DRLs is generally unnecessary if it is possible to demonstrate by measurement or computational techniques that the various exposure parameters do not exceed the corresponding ERLs of Table 7 or Table 8 (see 4.3.2; e.g., see IEC 62232 [B656] and IEEE Std C95.3).

For frequencies between 100 kHz and 6 GHz, the ERLs are intended to protect against exceeding the DRL for whole-body exposure, but they might not ensure compliance with the local spatial-average SAR DRLs when exposure occurs in fields that are nonuniform over the body (e.g., near-field and reactive near-field exposures wherein there can be substantial coupling between the individual and the source; Findlay and Dimbylow [B416]). In both of these cases, it might be necessary to directly evaluate local SAR through measurement or computational techniques (e.g., IEEE Std 1528-2013 [B664]).

In cases where the measured exposure parameters approach or exceed the ERL for frequencies between 100 kHz and 6 GHz, the more complex evaluation of SAR may be used to make a further determination of compliance with the standard. In many cases, such evaluations can reveal that the DRLs are not exceeded. A practical guideline based on the weight of the subject may eliminate the need to assess compliance with the whole-body-average DRL of 0.4 W/kg for the restricted tier (or 0.08 W/kg for the unrestricted tier). For example, for a 70 kg person, the corresponding RF power would be less than 28 W (restricted tier) or 5.6 W (unrestricted tier). (See IEC 62311:2007 [B657] for additional examples.) If the combined available power of the relevant RF field sources is less than these values, the exposure does not exceed the DRLs on whole-body-average SAR. Such a determination, however, does not necessarily imply that the local peak spatial-average DRLs is not exceeded. SAR measurements or calculations for compliance with local DRLs [Table 5 (see 4.3.1)] are not necessary if the maximum average power of the RF source (measured over any 6-min period) does not exceed 100 mW for the restricted tier and 20 mW for the unrestricted tier. IEC 62479:2010 ([B658]) shows cases of other low-power devices that can be excluded for any evaluation depending on antenna types, separation distances, or frequencies.

For exposures above 6 GHz, the energy is absorbed close to the body surface. DRL is defined as the epithelial power density just under the surface. Numerical simulations or experimental evaluation using thermographic techniques may be used to demonstrate compliance. To comply with ERLs, numerically derived or experimentally determined incident power densities shall be within the ERLs. Proper averaging time and area are needed for whole-body or local exposures. Detailed procedures are currently under development by the IEC TC 106 and IEEE ICES TC34 technical committees.[28]

[28] International Elecrotechnical Commission, Technical Committee 106: Methods for the assessment of electric, magnetic and electromagnetic fields associated with human exposure. IEEE International Committee on Electromagnetic Safety, Technical Committee 34: Product performance standards relative to the safe use of electromagnetic energy.

4.4 ERLs for contact current, induced current, and contact voltage (0 Hz to 110 MHz)

4.4.1 ERLs for contact current and induced current (0 Hz to 100 kHz)

4.4.1.1 ERLs for sinusoidal contact and induced current

Contact and induced current shall be limited as specified in Table 12 and Table 13 for frequencies between 0 Hz and 5 MHz, subject to the following conditions:

a) Contact current limits in Table 12 and Table 13 apply to a freestanding individual who is insulated from ground while touching a grounded conductor. These limits might not protect against aversive sensations from spark discharges just prior to direct contact or upon release from the grounded conductor.

b) Induced current limits in Table 12 and Table 13 apply to an individual with conductive contact to the ground only through the feet. These limits might not protect against aversive sensations from spark discharges just prior to direct contact or upon release from the grounded conductor.

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 restricted environments, limits for grasping contacts apply where personnel are trained to make rapid grasping contact and to avoid touch contacts with conductive objects that present the possibility of painful contact current. A grasping contact area is assumed to be 15 cm^2. The use of appropriate protective gloves, the prohibition of metallic objects, or the training of personnel might be sufficient to assure compliance with the contact current limit in restricted environments. For persons in unrestricted environments, it is assumed that access and contact is unconstrained and protective measures are not employed.

e) A touch contact is assumed to have a contact area of 1 cm^2.

f) Above 100 kHz and long exposure durations ($t >> 1$ s), the values of induced and contact currents protect against heating effects in the RF range and are more restrictive than the corresponding values of currents in Table 12 and Table 13 extrapolated to frequencies greater than 100 kHz. Hence, for long exposure duration, adherence to the limits of Table 14 (see 4.4.2.1) is sufficient to demonstrate compliance with the standard with consideration of both thermal and ES mechanisms.

Table 12—RMS induced and contact current ERLs for continuous sinusoidal waveforms based on ES effects—Frequencies between 0 Hz and 3 kHz

Condition	RMS current (mA)[c]	
	Persons in unrestricted environments	Persons permitted in restricted environments
Induced, single foot[d]	1.35	3.0
Contact, grasp[a,b]	—	3.0
Contact, touch[b]	0.5	1.5

[a] The grasping contact limit pertains to restricted environments where personnel are trained to make grasping contact and to avoid touch contacts with conductive objects that present the possibility of painful contact.

[b] For touch and grasping contact, limits apply to current flowing between the body and a grounded object that can be contacted by the person.

[c] The averaging time for determination of compliance is 0.2 s.

[d] The evaluation of induced current shall be performed with the foot under test grounded and the other foot ungrounded.

Table 13—RMS induced and contact current ERLs for continuous sinusoidal waveforms based on ES effects—Frequencies between 3 kHz and 5 MHz

Condition	RMS current (mA)[c,e,f]	
	Persons in unrestricted environments	Persons permitted in restricted environments
Induced, single foot[d]	$0.45f$	$1.00\,f$
Contact, grasp[a,b]	—	$1.00\,f$
Contact, touch[b]	$0.167f$	$0.50\,f$

[a] The grasping contact limit pertains to restricted environments where personnel are trained to make grasping contact and to avoid touch contacts with conductive objects that present the possibility of painful contact.

[b] For touch and grasping contact, limits apply to current flowing between the body and a grounded object that can be contacted by the person.

[c] The averaging time for determination of compliance is 0.2 s.

[d] The evaluation of induced current shall be performed with the foot under test grounded and the other foot ungrounded.

[e] Above 100 kHz compliance with the ERLs based on thermal effects [shown in Table 14 (see 4.4.2.1)] is also required.

[f] f is the frequency in kHz.

4.4.1.2 ERLs for nonsinusoidal (pulsed or mixed frequency) contact current and induced current

When the current waveform is nonsinusoidal, such as with pulsed or mixed frequency waveforms, exposures shall conform to the rms ERLs of Table 12 and Table 13 (see 4.4.1.1), as well as to either of the criteria as stated for magnetic fields in 4.2.2.4.2 and 4.2.2.4.3. For this application, the external field is replaced by the applied current A_i, which is understood to represent the magnitude of the ith Fourier component of the current waveform, and ERL_i is the maximum permissible current magnitude at frequency f_i.

4.4.2 ERLs for contact current, induced current, and contact voltage (100 kHz to 110 MHz)

4.4.2.1 ERLs for contact current and induced current

Below 5 MHz, compliance with the ERLs on induced and contact currents, which protect against ES effects [shown in Table 12 and Table 13 (see 4.4.1.1)], is also required. Contact and induced current shall both be limited as specified in Table 14, subject to the conditions enumerated in 4.4.1.1, except for a greater averaging time for grasping contact and induced currents. The induced currents specified in this standard in the frequency range 100 kHz to 110 MHz are to be time-averaged over 6 min for local exposures. Note that the touch contact limits are in terms of instantaneous values.[29] The use of frequency appropriate protective gloves (Chatterjee et al. [B244]), or other safe working practices including the avoidance of contact with metal objects that could be energized or to grasp and avoid touch contact may be sufficient to ensure compliance with the limits for persons permitted in restricted environments. (See IEEE Std C95.7-2014 [B672].)

Figure 5 (for persons in unrestricted environments) and Figure 6 (for persons permitted in restricted environments) provide electric field strength values (as percentages of the ERL) below which induced current does not have to be measured. The electric field strength values plotted in Figure 5 and Figure 6 were derived from estimated induced body currents from exposure to uniform electric fields (typically far-field exposures) aligned with the axis of the body of an individual of height 1.75 m standing bare-footed on a conductive ground (Gandhi et al. [B492], Gandhi et al. [B493], and Tofani et al. [B1413]). These assumed exposure conditions are often not applicable to realistic exposures with the result that substantially higher electric field strengths are required to produce the induced body or contact current limits specified in this standard. For example, normal footwear can significantly reduce induced body current. Insulative gloves can also significantly reduce contact currents. In addition, the induced body currents (and grasping contact currents) specified in this standard in the frequency range 100 kHz to 110 MHz are to be time-averaged over 6 min. Touch contact currents are determined and expressed as instantaneous values. Moreover, the percentage of electric field ERL given in Figure 5 and Figure 6 values for induced current is based on the assumption that all current flows through just a single foot. Hence, Figure 5 and Figure 6 provide conservative guidance on when induced and contact current measurements should be performed.

Figure 5 represents the percentage of the electric field ERL below which the induced current through a single foot, or the touch current, meets the limits of Table 14 for persons in unrestricted environments. The indicated percentages of ERL were calculated based on the description in the text and assume an individual of height 1.75 m standing in good conductive contact with the ground.

Figure 6 represents the percentage of the electric field ERL below which the induced current through a single foot, the touch current, or grasping contact current, meets the limits of Table 14 for persons permitted in restricted environments. The indicated percentages of ERL were calculated based on the description in the text and assume an individual of height 1.75 m standing in good conductive contact with the ground.

[29] In evaluating contact currents, special care should be used to ensure that RF voltages associated with the current source are less than 200 V_{peak} ($\cong 140\ V_{rms} \times \sqrt{2}$). Higher voltages can lead to arcing between the current source and the body, resulting in an RF burn.

Table 14—RMS induced and contact current ERLs for continuous sinusoidal waveforms (100 kHz to 110 MHz)

	Persons in unrestricted environments Current (mA)[b,c,d,e,f,g]			Persons permitted in restricted environments Current (mA)[b,c,d,e,f,g]		
Frequency	**100 kHz to 3 MHz**	**3 MHz to 30 MHz**	**30 MHz to 110 MHz**	**100 kHz to 3 MHz**	**3 MHz to 30 MHz**	**30 MHz to 110 MHz**
Induced, each foot	45	45	45	100	100	100
Contact, grasp[a]	NA	NA	NA	100	100 $(f / 3)^{0.3}$	200
Contact, touch	16.7	16.7 $(f / 3)^{0.3}$	33.4	50	50 $(f / 3)^{0.3}$	100

NOTE 1—Calculated values for personnel in both environments are capped at the 30 MHz values since there is insufficient data to extrapolate above 30 MHz.

NOTE 2—Light "brush" contact can result in arcs and shock and burn even at 50 mA and should be avoided especially with long objects such as cranes or cables.

[a] The grasping contact limit pertains to persons in restricted environments where personnel are trained to make rapid grasping contact and to avoid touch contacts with conductive objects that present the possibility of painful contact.

[b] Tabulated values are rms values.

[c] Limits apply to current flowing between the body and a grounded object that can be contacted by the person.

[d] The averaging time for determination of compliance is 6 min for induced currents and grasping contact currents.

[e] Touch contact currents are determined and expressed as the maximum rms current averaged over 1 s.

[f] The ceiling values (temporal peak values as measured with accepted instruments) for all currents are 220 mA for persons in unrestricted environments (for a maximum duration of 93 s) and 500 mA for persons in either environment (for a maximum exposure duration of 14.4 s).

[g] f is the frequency in MHz.

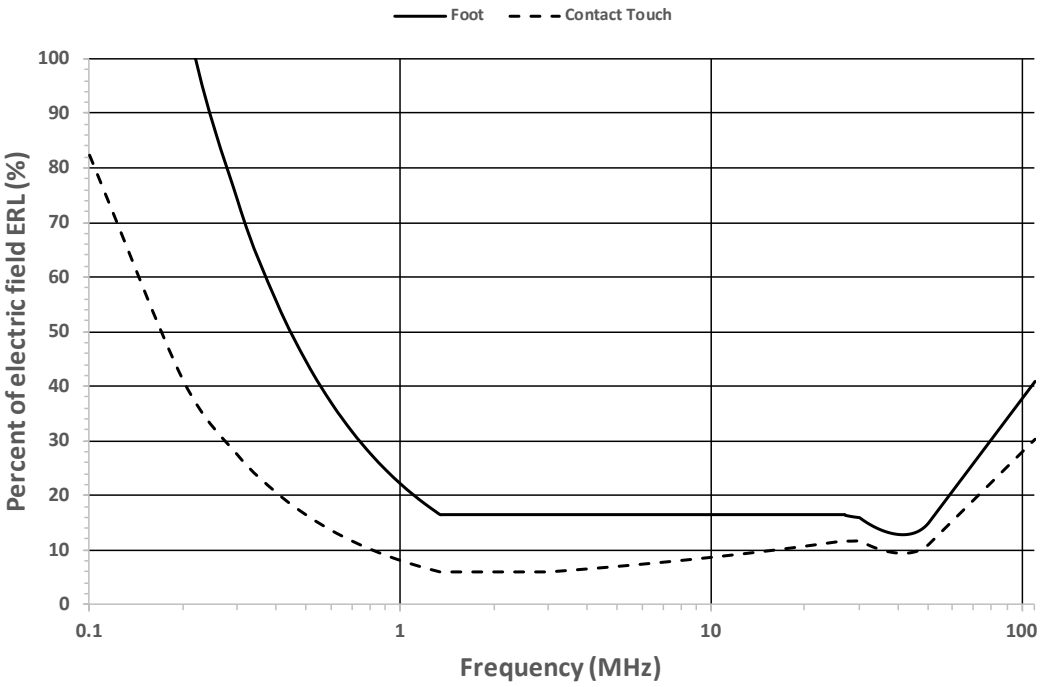


Figure 5—Percentage of electric field ERL resulting in induced and contact current limits for persons in unrestricted environments

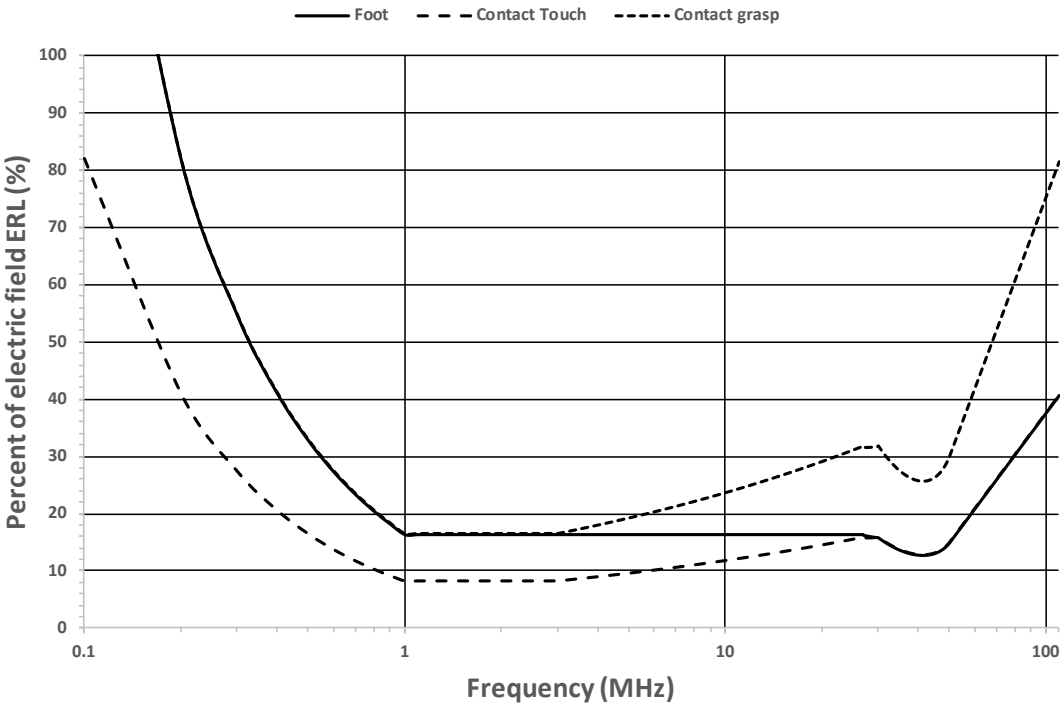


Figure 6—Percentage of electric field ERL resulting in induced and contact current limits for persons permitted in restricted environments

4.4.2.2 ERLs for contact voltage

The empirically determined value of 140 V rms has been used by the U.S. Navy as a protective limit for RF burns that would preclude the possibility of arcing conditions (NAVSEA [B1056]). This suggested interim limit applies to persons in unrestricted environments as well as to persons permitted in restricted environments, and it can be overly conservative in some circumstances; nonetheless, it should be considered to be an optional guideline until such time as a more thorough scientific and technical basis for the limit is developed (NAVSEA [B1056]).

NOTE—Contact voltages above the stated threshold can be induced on large objects even when the RF fields are in compliance with the ERLs for power density.

4.4.2.3 Contact current and voltage-related arcing considerations

Separate exposure limitations are provided for contact current based on contact surface dimensions. Contact with some surfaces that are energized with electromagnetic energy can result in an arc just prior to actual contact that can cause painful burns (see contact voltage in 4.4.2.2). Contact in the context of this standard includes either a touch contact (which assumes a contact surface of 1 cm^2) or a grasping contact (which assumes a larger contact surface of 15 cm^2). A larger contact surface spreads the exposure and reduces the probability of and/or extent of an adverse effect. Pointed contact, however, such as contact with an energized, small diameter wire, can result in greater tissue current density with concomitant greater localized skin heating than that associated with larger contact areas such as those specified for touch.

Skin contact with RF energized (reradiated, parasitic) metallic surfaces can result in instantaneous burning of tissue at the point of contact with the magnitude of contact current that results in a burn depending on frequency. The startle reaction caused by the immediate thermal pain can result in loss of balance, falls, or reflexive withdrawal resulting in unintended contact with machinery producing injury or death.

4.5 Safety programs

Throughout the range of frequencies applicable to this standard, the ERLs apply to exposure of people; that is, compliance with this standard is determined by whether exposures of people to electromagnetic fields, currents, and voltages exceed the applicable ERLs. Where there might be access to RF fields, currents, and/or voltages that exceed the unrestricted environment ERL of this standard, a safety program such as detailed in IEEE Std C95.7 shall be implemented. The primary purpose of the safety program is to help ensure that exposures do not exceed the ERLs or DRLs for persons authorized to operate in a restricted environment. A secondary purpose of the program is to help ensure that those persons accessing the restricted environment are appropriately authorized to enter. Safety programs include various mitigative measures that can be taken to reduce the probability of exceeding the DRL or the ERL for the upper tier. Such programs can include awareness training, implementation of protective measures such as signage and the use of personal protective equipment (PPE) and implementing physical access control, incident response, periodic evaluation of program effectiveness, and assigned responsibilities for implementing the program (see IEEE Std C95.7). Safety programs shall be documented for all restricted areas. While IEEE Std C95.7 only specifically addresses RF fields above 3 kHz, similar exposure mitigation approaches may be used in many cases wherein lower frequency exposures occur.

Annex A

(informative)

Approach to revision of IEEE Std C95.1™-2005 and IEEE Std C95.6™-2002

A.1 Approach to revision process

A.1.1 General

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.1.2 Continuity of the IEEE standards revision process

This standard revises and combines IEEE Std C95.1-2005 [B668] and IEEE Std C95.6-2002 [B671] into a single standard (i.e., the present IEEE Std C95.1-2019). A military-focused standard, IEEE Std C95.1-2345™-2014 [B669], was the first IEEE ICES-developed standard to combine IEEE Std C95.1 and IEEE Std C95.6, which also introduced expanded frequency-dependent exposure levels for contact currents and new terminology including the concept of safety program initiation level and unrestricted and restricted environments. IEEE Std C95.1-2005 was approved by the IEEE Standards Association Standards Board (SASB) in 2005, published in 2006, and accepted for use as an American National Standard by the American National Standards Institute (ANSI) in 2006; IEEE Std C95.6-2002 was approved by the SASB in 2002, published in 2002, reaffirmed in 2007, and approved for use as an American National Standard by ANSI in 2008. The revision process implemented by ICES is described in this Annex.

A.1.3 Open nature of the IEEE ICES standards development process

IEEE ICES and its subcommittees are composed of individuals with a material interest. These committees are composed of individuals affiliated with government, industry, and academia, as well as of independent professionals and the general public in accordance with the membership requirements of all standards committees developed by the IEEE SA. Subcommittee membership is open to all, including experts from a variety of scientific disciplines such as engineering, physics, statistics, epidemiology, life sciences, medicine, risk assessment, and risk management. This wide-ranging participation, including thorough discussions and open decision-making, is the hallmark of the process that led to this standard.

A.1.4 Complete reassessment of the technical rationale

IEEE Std C95.1-2005 was based primarily on research published before 2003; IEEE Std C95.6-2002 was based primarily on research published before 2001. Research has continued since these times, and a reevaluation of the extremely low-frequency (ELF) and radio frequency (RF) biological effects databases was necessary for this revision. Attempts were made to include all of the relevant literature in the databases.

A.1.5 Process clarifications, and appeals

The evaluation of an IEEE standard is a process that is continually ongoing; that is, IEEE standards are "living" documents. Requests for clarification submitted to IEEE ICES are resolved by ad hoc working groups of the ICES TC95 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 into the current revision of the standard by consensus. Appeals of an approved standard are resolved in accordance with the IEEE SASB Policies and Procedures.

A.1.6 The literature surveillance effort

A literature surveillance working group was established to compile a citation list of all relevant published literature. ICES 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. References cited in this standard are listed in Annex E. Literature databases for low frequencies are given in Reilly [B1179] and Reilly and Diamant [B1188] and Annex E.

A.1.7 Literature evaluation process

To begin the literature evaluation process, a literature review working group (LRWG) that included 16 topic groups was established with each topic group assigned one of the 16 topics listed in Annex B of IEEE Std C95.1-2005. Guidelines for the systematic review of the scientific literature on health effects associated with exposure to electric, magnetic, and electromagnetic fields were also developed by the editorial working group. The LRWG decided that as an adjunct to the ICES review, it would be important to take advantage of ongoing literature reviews by other international organizations to augment and enhance the ICES review process and ensure that key papers were identified and reviewed. It was considered important to build on such efforts to the extent feasible to broaden the ICES review process and ensure consistency. Based on the fact that science-based expert reviews of health effects support the exposure limits established in this safety standard (see http://www.ices-emfsafety.org/expert-reviews/), and the availability of new research in the mm-wave range, ICES focused primarily on frequencies between 6 GHz and 300 GHz for this revision. ICES continues to maintain a surveillance of the relevant scientific literature including the upcoming World Health Organization's (WHO) "Environmental Health Criteria" (EHC) document.[30] This information will be used to update Annex C in the subsequent revision of this standard or via an addendum to be issued as soon as feasible.

Consequently, ICES decided to take advantage of many available expert reviews that confirm the protectiveness of the current exposure limits and decided to narrow the extent of this standard revision to frequencies between 6 GHz and 300 GHz. ICES further determined that when the WHO EHC document becomes available, the LRWG will provide a summary of the EHC literature review, provide comments, and review any papers missing in the WHO EHC document that are considered relevant. This will be used to update Annex C in the subsequent revision of this standard or via an amendment to be issued as soon as feasible.

[30] (http://www.who.int/peh-emf/research/rf_ehc_page/en/): "The World Health Organization is undertaking a health risk assessment of radiofrequency electromagnetic fields, to be published as a monograph in the Environmental Health Criteria Series. This publication will complement the monographs on static fields (2006) and extremely low frequency fields (2007), and will update the monograph on radiofrequency fields (1993)." (webpage visited 2018 August 25).

Relative to low frequencies, WHO published two monographs: one published in 2006 addressed static fields (EHC Monograph No. 232 [B1498]), and one published in 2007 addressed extremely low-frequency fields (EHC Monograph No. 238 [B1497]). Both were found useful for summarizing effects at low frequencies, which led to reaffirming the low-frequency limits in IEEE Std C95.6-2002.

A.1.8 Identification of hazards and interaction mechanisms

An international workshop, "Thermal Aspects of Radio Frequency Exposure," co-sponsored by the Mobile Manufacturers Forum (now named the "Mobile & Wireless Forum"), the Groupe Special Mobile (GSM) Association, and the U.S. Food and Drug Administration, was convened in Gaithersburg, Maryland, USA, 11–12 January 2010—several participants were ICES members. The goals of the workshop were to: 1) identify appropriate biological endpoints associated with thermal hazards and their time-dependence thresholds, and 2) outline future directions for research that might lead to an improved understanding of health and safety implications of human exposure to RF energy. The aim of both goals was to ensure the adequacy of contemporary dosimetric reference limits (DRL) and exposure reference levels (ERL). Foster and Morrissey [B431] summarized some of the major conclusions of the speakers and offered comments on the proposed research priorities and implications of the presentations with regard to the DRLs and ERLs of contemporary standards and guidelines for human exposure to RF energy.

Similar to the above workshop, the International Commission on Non-Ionizing Radiation Protection (ICNIRP) also organized a two-day workshop "A closer look at the thresholds of thermal damage," which was held in Istanbul 26–28 May 2015 (Sienkiewicz et al. [B1309]). The purpose of the workshop was to reexamine the thermal basis of the ICNIRP guidelines and provide further information on effects and thresholds of thermally based adverse effects. The participants agreed that established adverse effects that occur at the lowest thresholds are based on a thermal mechanism resulting from RF heating and are consistent with heating from other sources; that is, information derived from such studies can also be applied to RF-induced heating (Sienkiewicz et al. [B1309]).

The conclusions of these workshops indicate that the two major groups that develop RF safety standards and guidelines (ICES and ICNIRP) agree that thermal effects continue to be the appropriate basis for protection against RF exposure at frequencies above 100 kHz. Although the International Agency for Research on Cancer (IARC) classified exposure to RF fields as a possible carcinogen (Group 2B) at the end of May 2011 based on epidemiology studies on mobile phones (http://www.iarc.fr/en/media-centre/pr/2011/pdfs/pr208_E.pdf), the WHO EMF Project stated in Fact Sheet #193 on June 22, 2011 (updated on October 8, 2014) that, "To date, no adverse health effects have been established as being caused by mobile phone use" (Wiktor-Jedrzejczak et al. [B1506]; see also WHO [B1499]).

At frequencies below 100 kHz or a few megahertz for pulse-modulated fields, the established mechanism for adverse effects is electrostimulation (ES; see IEEE Std C95.6-2002 [B671], the ICNIRP guidelines for limiting exposure to time-varying electric and magnetic fields [B644], and WHO Monograph No. 238 [B1497]). The DRLs and ERLs found in contemporary standards and guidelines (e.g., ICNIRP guidelines [B644], IEEE Std C95.6-2002 [B671]) are based on the effects associated with electrostimulation. Compliance with these limits provides adequate protection for acute effects. Although there is no convincing evidence of chronic effects associated with exposures below the established threshold for acute effects, the IARC has classified exposure to ELF magnetic fields as a possible human carcinogen (Group 2B; WHO/IARC [B1501], [B1502]). However, with regard to setting exposure limits, the consensus of the scientific community is that electrostimulation is the appropriate established mechanism.

A.2 Basic concepts for developing the ERLs

A.2.1 General

The process followed by ICES for establishing ERLs with respect to human exposure to electric, magnetic, and electromagnetic fields, 0 Hz 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 [B1080], [B1081], [B1082]; European Commission's Scientific Committee on Emerging and Newly Identified Health Risks, SCENIHR [B407]). The process began with a detailed evaluation of the relevant literature in the scientific database, took advantage of all completed evaluations in the database, and proceeded to a determination of potential hazards to human beings exposed to electric, magnetic, and electromagnetic fields (cf. A.1.7 and A.1.8), from which thresholds of individual responses and dose response functions were determined.

A.2.2 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, for several reasons, they cannot be applied to setting standards for allowable human exposure to electric, magnetic, and electromagnetic fields across the frequency range of this standard. Many of these studies have inherent weaknesses (e.g., 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 exposure to electromagnetic energy. 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 on 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 possible biological effect and that for an established adverse human health effect. The procedures detailed earlier provide the basis for potential hazard identification and the associated risk assessment used in developing this standard.

A.2.3 Assessing thresholds and dose-response relationships

For any given biological response at RF frequencies, thresholds are defined below which the specific response does not occur or is undetectable. For ES effects, this standard defines intersubject median adverse reaction thresholds to which safety factors are applied to arrive at upper and lower tier limits. Above the threshold level, a function that relates dose rate [e.g., specific absorption rate (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.2.4 Selection of safety factors and development of ERLs

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 settings. In practice, the better the hazard

is 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 with respect to the degree of hazard associated with an exposure, the range of thresholds across the relevant population threshold, and is selected to prevent the threshold from being exceeded with a sufficiently wide margin.

A.2.5 Mechanisms of biophysical interaction

A.2.5.1 General

An established human mechanism is one having the following characteristics:

a) It can be used to predict biological effects in humans.

b) An explicit model can be made using equations or parametric relationships.

c) It has been verified in human subjects, or animal data can be confidently extrapolated to humans.

d) It is supported by strong evidence.

e) It is widely accepted among experts in the scientific community.

Mechanisms not having these characteristics are classified as proposed. Progress in research on proposed mechanisms should be monitored and evaluated as to whether any can be included in the list of established mechanisms.

A.2.5.2 Frequencies between 0 Hz and 5 MHz

Established mechanisms have been identified based on these criteria (Reilly [B1179], [B1181], [B1184]; Reilly and Diamant [B1188]). One class of mechanisms relates to membrane polarization (i.e., the alteration of the cellular membrane's natural resting potential by the *in situ* electric field). Depolarization of the membranes of nerve and muscle can lead to their excitation (herein referred to as electrostimulation); these effects are responsible for the minimum thresholds of reaction at frequencies from about 1 Hz to a few megahertz. Magnetohydrodynamic effects, which apply to forces on moving charges in fluids, dominate biological reactions below 1 Hz. These mechanisms are responsible for short-term or acute effects; that is, they result in reactions to electric and magnetic fields that are manifested within seconds (usually a fraction of a second) after the exposure begins. Thermal effects are well understood, but they are not dominant at frequencies below 100 kHz.

The fundamental force responsible for electrostimulation effects is the *in situ* electric field rather than the internal current density (see B.2.1.2). More accurate limits for electrostimulation effects can be derived as a function of the *in situ* electric field rather than of the internal current density as has been a common practice in the past (Bernhardt [B152]; ICNIRP [B646]; IEEE Std C95.1-2005 [B668]). The distribution within the body of *in situ* electric fields differs from the distribution of current density, and the calculation of the *in situ* electric field is less sensitive to assumptions of tissue conductivities compared to those of internal current density.

Mechanisms of interaction that are classified as proposed relate to long-term or chronic exposure effects (Olsen et al. [B1103]; Reilly [B1181]). These mechanisms are typically mentioned in connection with hypotheses concerning chronic exposure to low-level electric and magnetic fields, with outcomes including cancer, reproductive effects, nervous system effects, and so on. While these mechanisms cannot be dismissed as being irrelevant, the body of knowledge concerning them is currently insufficient to establish a confirmed mechanism that would provide a firm basis for deriving human exposure limits.

A.2.5.3 Frequencies between 100 kHz and 300 GHz

As indicated in A.1.8, two workshops have examined the thermal aspects of RF exposures. It has been established that exposures at sufficiently high exposure levels result in heating effects in animal models and humans. Temperature elevation if sufficiently high and prolonged can cause tissue damage. Whole-body and local exposure limits are derived from animal threshold effects data. Safety factors are then applied to establish the DRLs and ERLs for human exposures. Further discussions on the heating mechanism as the rationale for protection in this frequency range are in Annex B.

A.2.5.4 Frequencies between 6 GHz and 300 GHz

Exposures to radiofrequency energy above 6 GHz are characterized by shallow energy penetration, typically limited to the skin. Energy reflection, absorption, and subsequent internal power density distribution is dependent on the physical properties of the skin layers and subcutaneous layers. The main absorber of RF energy in skin is free water. Consequently, the water content in the skin layers is the major determinant of their electrical properties (Ziskin et al. [B1547]).

The energy penetration depth into the skin at 6 GHz is approximately 4 mm, and the penetration decreases monotonically with increasing frequency. At 300 GHz, the energy penetration depth is approximately only 0.12 mm. Although the penetration depth is very shallow, deeper tissues such as fat and muscle can be heated by thermal diffusion from the skin layers. The steady-state temperature elevation is affected by the thickness of the fat layer and the blood perfusion rate: The temperature elevation is greatest with the thickest fat layer and least with the greatest blood flow rate. Also, there can be significant lateral thermal diffusion when skin is exposed to sources with small diameter beams. Thus, for the same incident power density, broad beam exposures produce higher temperature elevations than do narrow beam exposures.

The transmission of energy into the skin can be increased or decreased by the presence of clothing (Gandhi and Riazi [B497]; Wu et al. [B1523]; Zhadobov et al. [B1541]). The presence of water in the clothing reduces the power transmission into the skin. However, the attenuation of millimeter waves by most garment materials is negligible (Wu et al. [B1523]).

Several studies have reviewed electromagnetic absorption of millimeter waves and thermal responses in detailed image-based models of the body or head (Foster et al. [B433]; Ziskin et al. [B1547]). Much of this work has come from A. Hirata and his colleagues. For example, Morimoto et al. [B1038] computed the SAR and temperature increase in the skin and brain for frequencies up to 30 GHz. Hashimoto et al. [B577] determined the area-averaged incident power density and temperature increase in head models exposed to RF energy from 3 GHz to 300 GHz. Sasaki et al. [B1252] conducted a Monte Carlo analysis to estimate skin temperature elevations from RF exposure from 10 GHz to 1000 GHz considering a range of thicknesses of the different tissue layers. The thermal calculations in these studies were based on Pennes' bioheat equation (BHTE; Pennes [B1139]) with fixed values for tissue blood perfusion. While this detailed analysis is valuable in predicting the thermal response of tissue to RF exposure, it does not necessarily encompass all sources of variability that affect an individual's thermal response to RF exposure. A parametric analysis can help one to understand the underlying heat transfer processes and point to sources of variability in a way that complements the numerically intensive computer models.

In addition to the detailed anatomic models described earlier, a greatly simplified model consisting of skin, fat, and muscle that assumes surface heating in only the skin layer has been shown to provide reasonable estimates of temperature elevation for frequencies above 6 GHz (Foster et al. [B432]). This model shows that the increase in surface temperature in the steady state is limited by a combination of dermal blood flow and is determined by the thermal resistance of subcutaneous tissues even though the RF energy can be deposited almost entirely in the skin layer.

A simple thermal model for skin exposed to RF energy with shallow penetration depth was developed by Foster et al. [B432]. The step response of this model (i.e., the increase in surface temperature following sudden imposition of RF exposure) is given by

$$T_{sur}(t) = \frac{I_0 \times T_{tr} \times R_1}{k} \times \mathrm{erf}\left(\sqrt{\frac{t}{\tau_1}}\right)$$

where $T_{sur}(t)$ is the skin surface temperature increase (°C), I_0 is the incident power density (W/m^2), T_{tr} is the energy transmission coefficient, R_1 is an intrinsic distance scale (mm), k is thermal conductivity [W/(m K)], and τ_1 is an intrinsic time (s). For typical tissue values,

$$R_1 = \frac{\sqrt{k}}{\rho\sqrt{m_b c}} \approx 7 \text{ mm}$$

and

$$\tau_1 = \frac{1}{m_b \rho} \approx 500 \text{ s}$$

where ρ is tissue density (1109 kg/m^3), m_b is blood perfusion rate [1.8×10^{-6} m^3/(kg s)], and c is specific heat capacity [3390 J/(kg K)].

Complete details of this derivation are provided in Foster et al. [B432]. A more detailed analysis, including consideration of the finite energy penetration depth into tissue at frequencies above 10 GHz (Sasaki et al. [B1252]) shows that this approximation is excellent above 10 GHz, and it moderately overestimates the rise in surface temperature between 6 GHz and 10 GHz. This approximation does not, however, take into account the thermal resistance of subcutaneous fat, which tends to increase surface temperature somewhat (a factor of 2 or less depending on the thermal characteristics of subcutaneous fat).

For short pulses lasting less than 10 s, the early transient response can be shown to be approximately

$$T_{sur}(t) \approx 10^{-3}\ I_0 \times T_{tr} \times \sqrt{\tau}$$

where τ (s) is the pulse width. The pulses should be far enough apart in time for the transient increases in skin temperature to decay, which implies pulse separation of a few tens of seconds.

To limit the increase in skin temperature to 1 °C from a single pulse of duration τ, this implies that the fluence (integral of the absorbed power density) should be limited to about 1 $\tau^{1/2}$ kJ/m^2. This is valid at millimeter wave (MMW) frequencies and above (30 GHz to 300 GHz). The pulses should be far enough apart in time to allow the skin temperature to relax toward baseline level. This assumes that the average incident power density over 6 min complies with the DRLs and is averaged over 1 cm^2 square of the body surface.

A.3 Adverse health effects

A.3.1 Frequencies between 0 Hz and 5 MHz

The DRLs and ERLs are based on avoidance of the following short-term reactions:

a) Aversive or painful stimulation of sensory or motor neurons

b) Muscle excitation that can lead to injury while performing potentially hazardous activities

c) Excitation of neurons or direct alteration of synaptic activity within the brain

d) Cardiac excitation

e) Adverse effects associated with induced potentials or forces on rapidly moving charges within the body, such as in blood flow

A.3.2 Frequencies between 100 kHz and 300 GHz

A.3.2.1 Frequencies between 100 kHz and 6 GHz

The DRLs and ERLs are based on avoidance of the following adverse reactions:

a) RF shocks and burns

b) Localized RF heating effects

c) Surface heating effects

d) Whole-body heating effects

A.3.2.2 Frequencies between 6 GHz and 300 GHz

Thermal effects occur as a consequence of significant temperature elevations persisting for sufficiently long periods. For any given tissue, the damage resulting from a thermal effect depends on the magnitude of the temperature elevation and on the duration of the exposure, not on the exposure frequency. However, the distribution of the heating depends on the frequency. That is, the higher the frequency, the shorter is the penetration into the skin. After exposures above 6 GHz, the highest temperatures occur at or near the skin surface.

The most serious injury to the skin resulting from exposures to electromagnetic energy above 6 GHz is a burn, which can occur if skin temperature is increased above 45 °C for extended times (tens of minutes or more). The thermal damage at somewhat lower exposure times could cause blisters and skin reddening. The threshold for thermal pain is 44 °C (Walters et al. [B1469]), which is experienced very quickly after the skin temperature exceeds the threshold. Short-term exposures below this would not be expected to cause damage to the skin. However, chronic exposures at temperature slightly below the threshold of pain can result in more subtle injuries such as *erythema ab igne* (Corazza et al. [B298]). A comprehensive review of biological effects of frequencies above 6 GHz can be found in Alekseev and Ziskin [B51].

Annex B

(informative)

Rationale

B.1 Introduction

B.1.1 General

Recent literature reviews by the ICES working groups and the literature review have not revealed reliable evidence that would change the scientific basis for the adverse effect levels. The adverse effect is electrostimulation at low frequency and heating at high frequency. The threshold for WBA SAR of 4 W/kg for established adverse effects remains the same as in the ANSI C95.1-1982 [B68], and the latest IEEE Std C95.1-2005 [B668]. Adoption was based on the decision that the threshold for disruption of ongoing behavior in laboratory animals including nonhuman primates might extrapolate to potentially adverse effect in human beings. The peak spatial-average SAR (psSAR) values were changed in IEEE Std C95.1-2005 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.

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 of IEEE Std C95.1-2005 [B668] (C.2.1 in this standard) and is consistent with the ICNIRP guidelines [B646]. For localized exposure, this standard protects against adverse effects in the tissues most sensitive to thermal effects. Modeling studies report the possibility of a 1 °C or greater rise in tissue temperature at 10 W/kg per 10 g. An increase of 1 °C had been suggested earlier as the upper temperature increase without detrimental health effects (ICNIRP [B646]; UNEP/WHO/IRPA [B1427]; WHO EHC 137 [B1495]). WHO information indicates that a 1 °C rise in temperature, even in the most sensitive tissues and organs, is not adverse (WHO [B1500]).

The upper boundary of the frequency range over which WBA SAR and local SAR are deemed to be the basic restriction has been changed from 3 GHz to 6 GHz to harmonize with the new ICNIRP guidelines [B646]. The tissue-averaging mass for the psSAR has been changed from 1 g to 10 g. This change, which is explained in detail in B.4.1.2.2, B.4.1.2.2.2, and B.7.5, is based on the biologically based rationale of ICNIRP related to exposure of the eyes and extensive theoretical biophysical research quantifying radio frequency (RF) energy penetration in biological tissue. The results of this research show that RF energy, if compliant with this standard, 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. 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, to explain the rationale for adverse effect levels in the frequency range of 100 kHz to 300 GHz (see B.3), several special considerations have been reviewed and explained in detail in B.7 (for example, to cover extreme exposure situations of specific human subpopulations). In summary, this standard incorporates a large margin of safety and an RF safety program is required to provide part of the margin of safety for those exposed above the lower tier level. This standard should also be considered especially conservative because 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 C95.1-2005 [B668] maintains many of the characteristics of the previous standard but also contains several 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 in B.1.2.

B.1.2 Similarities and differences between this standard and IEEE Std C95.6-2002 and IEEE Std C95.1-2005

B.1.2.1 Similarities

Similarities among the standards are as follows:

a) Research on the biological effects of electromagnetic interactions with tissues has not changed the scientific basis of the adverse effect levels (i.e., electrostimulation for low frequencies and heating for high frequencies).

b) Exposure limits for electrostimulation effects are kept the same as in IEEE Std C95.6-2002 [B671] and IEEE Std C95.1-2005 [B668].

c) Exposure limits, termed dosimetric reference limits (DRL), previously called basic restrictions, on WBA SAR and psSAR remain the same to prevent heating effects from exposure over much of the RF spectrum.

d) The exposure reference levels (ERL), previously called maximum permissible exposure (MPE) levels, for the lower tier remain the same as in IEEE Std C95.1-2005.

e) This standard continues to support the position of the earlier editions, (i.e., that upper tier ERLs are protective of public health and safety and that the risk of harm from exposure to fields below the lower tier ERLs has not been confirmed by scientific evidence).

B.1.2.2 Differences

Differences among the standards are as follows:

a) IEEE Std C95.1-2005 [B668] contains two tiers: an upper tier for “people in controlled environments” and a lower tier “action level” for implementing an RF safety program or MPE for the general public when an RF safety program is not available. In this standard, operational definitions are introduced. The terms “lower tier” (“unrestricted tier”) and “upper tier” (“restricted tier”) establish the maximum exposure limits for persons in unrestricted environments and for persons permitted in restricted environments, respectively.

b) A DRL replaces basic restriction, and an ERL replaces MPE, the terms used in the previous standard. The intent is to make the terms more explicit and understandable.

c) The safety program initiation level (previously “action level”) is clarified as the ERL marking the transition point between the lower (unrestricted) tier and the upper (restricted) tier.

d) The upper frequency boundary for WBA SAR has been changed from 3 GHz to 6 GHz because of improved measurement capabilities and to harmonize with the anticipated revised ICNIRP guidelines.

e) The averaging time is 30 min for whole-body RF exposure and 6 min for local exposure. It is scientifically more appropriate to select the averaging time according to the absorbing mass, not according to the exposure tier.

f) The term "extremities" as used in IEEE Std C95.1-2005 is changed to "limbs" involving the whole arms and legs, instead of portions distal to the elbows and knees. This change is to harmonize with IEEE Std C95.6-2002 [B671] and the ICNIRP guidelines [B646].

g) After the publication of novel dosimetry findings, the local exposure ERL is now frequency dependent, instead of being a fixed factor of 20 times the whole-body ERL regardless of frequency.

h) The upper tier whole-body exposure ERLs above 300 MHz are different from those in IEEE Std C95.1-2005 to maintain a consistent 5× factor between tiers and to harmonize with ICNIRP guidelines.

i) The local exposure DRL and ERL for frequencies between 6 GHz and 300 GHz have changed. The DRL is the epithelial power density inside the body surface, and ERL is the incident power density outside the body. Averaging power density area is defined as a 4 cm^2. For smaller areas, relaxed limits are allowed.

j) Peak DRL and ERL limits for local exposures to pulsed RF fields are defined, and new fluence limits for single RF-modulated pulses above 30 GHz are introduced. The averaging area for single pulse fluence is 1 cm^2.

k) The former induced current limit for both feet is considered an unrealistic condition and is removed. The induced current limits for a single foot are retained.

l) Root-mean-square (rms) induced and contact current limits for continuous sinusoidal waveforms (100 kHz to 110 MHz) are changed from those in Table 7 of IEEE Std C95.1-2005 to frequency-dependent values.

B.1.2.3 Three frequency bands

In recognition of the differing biological effects of exposure to particular frequencies, the standard addresses three frequency bands: 0 Hz to 100 kHz, 100 kHz to 6 GHz, and 6 GHz to 300 GHz. The limits in the first band minimize the 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 body surface heating. Differences within each of those bands are provided as follows:

a) **0 Hz to 100 kHz (up to 5 MHz for pulsed fields):** The revision defines DRL in terms of the *in situ* (within biological tissue) electric fields for different regions of the body. Magnetic field ERLs are specified for the arms and legs and for the head and torso. Compliance with the standard may be demonstrated for uniform sinusoidal magnetic fields by showing that either the *in situ* electric field DRL or the magnetic field ERL is satisfied. If the magnetic field is not constant over the head and torso, compliance with the DRLs is satisfied if the spatial peak of the magnetic field ERL is not exceeded. The averaging time for an rms measurement is 0.2 s. Formulas are included to determine the maximum permitted peak electric fields based on evaluations of either *in situ* dose or exposure level. For nonsinusoidal or pulsed sinusoidal fields, DRLs and ERLs are specified based either on instantaneous temporal measurements or on Fourier components of the exposure waveform. The choice between these two compliance tests is left to the discretion of the user. These provisions remain unchanged relative to the existing standard.

b) **100 kHz to 6 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. Local DRLs expressed as the psSAR in 10 g of tissue in the shape of a cube for any body tissue including

the arms, legs, and pinnae are specified. This revision retains the limits for psSAR of 10 W/kg for the restricted tier and 2 W/kg for the unrestricted tier, and those for the limbs and pinnae (20 W/kg and 4 W/kg respectively). The contact current limits for the frequency range of 100 kHz to 110 MHz are frequency dependent and are subdivided into touch and grasping conditions, with the grasping condition constrained to the restricted environment. The permissible touch contact current is reduced for both the restricted environment and the unrestricted environment. The lower portion of this frequency range (i.e., 100 kHz to 5 MHz) is a transition region where both the limits protecting against electrostimulation and the limits protecting against effects associated with heating should be met. The standard identifies factors associated with research on contact currents that need additional study to help ensure that the limits are applicable to practical conditions found in the workplace.

c) **6 GHz to 300 GHz:** The principal changes within this band have been in the local exposure DRLs and ERLs. Since the interactions are mostly at or just within the body surface and are quasi-optical, the DRLs are expressed in terms of a newly introduced metric, the "epithelial power density" and associated exposure averaging interval. The corresponding ERLs are defined as function of frequency to account for the increasing epithelial power entering into the body across the air/body interface. The local exposure DRLs and ERLs are each averaged over 4 cm^2. The availability of the experimental human data upon which to set limits on exposures above 6 GHz is limited; the ICES committee does recognize the need for additional data in this frequency range. Pending the availability of further data, in this standard, the limits above 6 GHz are revised to provide a similar level of protection against thermal hazards as that provided with the existing limits below 6 GHz, using the results of widely accepted thermal modeling and dosimetric studies. At 300 GHz, the ERL of this standard for persons in restricted environments is equal to the reference level (MPE) in the laser standards, the coverage of which begins at 300 GHz (ANSI Z136.1-2014 [B69]; IEC 60285-1 [B653]).

B.1.3 Safety factors

B.1.3.1 General

This standard addresses guidelines for the electromagnetic spectrum for frequencies below 300 GHz. At low frequencies, ERLs and DRLs for electric and magnetic fields are stated in terms of environmental fields and *in situ* dose quantities, respectively. At higher frequencies (up to 6 GHz), the ERLs and DRLs are stated in terms of power density and the rate of thermal energy deposition (or just total energy), respectively. Above 6 GHz, the DRLs and ERLs are stated in terms of power density. These match the terms in ANSI Z136.1-2014 [B69], although in the laser standard, they are named "irradiance" (W/m^2) and "radiant exposure" (J/m^2). ANSI Z136.1-2007 covers the wavelength range of 1 mm (300 GHz) to 180 nm (ultraviolet).

Safety factors and their rationales are different for frequencies below 100 kHz (CW) or 5 MHz (pulsed) where the adverse effect addressed is electrostimulation, as well as for the frequency range above 100 kHz where the adverse effects 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 ERLs and DRLs (although usually only one of the two apply depending on the exposure characteristics).

The term "safety factor" is commonly interpreted to be the ratio of an exposure level causing an adverse effect to the corresponding allowable ERL. Consequently, the development of a safety factor presupposes the selection of a hazard threshold (HT). Comparison of two safety factors of numeric ratios in fields versus power density would be meaningless.

Table B.1 provides an overview of "safety factors" SF_U and SF_L, as used in this standard for upper and lower exposure tiers, respectively. SF_U is a divisor that converts a median adverse reaction threshold into an upper tier exposure limit; SF_L is a divisor that converts a median adverse reaction threshold into a lower tier

exposure limit. As noted in the last column of Table B.1, the divisors apply to different metrics, depending on the frequency regime of interest.

The numerical values of SF_U and SF_L in Table B.1 apply to uniform exposure of the head and torso. For localized exposure, the applicable safety factors can differ. For instance, with low-frequency exposure of the hands, wrists, feet, and ankles, SF_L is reduced in comparison with exposure of other body parts, as seen in Table B.5. Differences in safety factors can also occur when considering differing volumes of tissue being exposed or the location of the tissue within the body. For example, RF exposure at frequencies above 6 GHz is primarily accompanied by surface heating of the skin. In such instances, elevated skin temperatures are more easily accommodated than are increases in the body core temperature. Compared with internal organs and tissues, less uncertainty is associated with surface temperatures that can result in thermal pain or tissue damage and, hence, a smaller safety factor may be appropriate.

Table B.1—Application of "safety factors" to DRLs for whole-body exposure to environmental fields[a]

Frequency range	Adverse effect	Safety factor (Divisor)		Applied metric
		Upper tier SF_U	Lower tier SF_L	
≤100 kHz CW; ≤5 MHz (pulsed)	Pain (PNS)	3	9	*in situ* electric field strength (V/m)
	Synapse Modulation (CNS)	3	9	
100 kHz to 6 GHz	Thermal stress (e.g., as reflected in work stoppage)	10	50	SAR (W/kg) or incident power density (W/m^2)
>6 GHz	Thermal pain in skin	2 to 5	10 to 25	epithelial power density (W/m^2)

NOTE 1—Safety factors in this table apply to DRLs for whole-body exposures to environmental fields. See B.3.2 for special exceptions for localized exposure.
NOTE 2—The safety factors at low frequencies (first row in table) apply to the internal electric field strength. Factors at radio frequencies apply to power, and therefore are applied to the square of the internal or surface electric field strength.

[a] See B.3.2.

B.1.3.2 Minimization of adverse effects associated with electrostimulation (below 5 MHz)

At frequencies below 5 MHz, a relevant hazard is associated with painful or aversive electrostimulation. Because the nature of an 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 (9.5 dB 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 of persons permitted in restricted 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 DRLs or ERLs for fields. It is understood 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 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 understood 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 three 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 permitted in a restricted environment (upper tier) could experience sensation or even the onset of mild pain at the limit. Thus, with regard to electrostimulation, the term minimize adverse effects is used throughout this standard. This small potential for an adverse reaction is considered acceptable for persons permitted in a restricted 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 applicable specifically to exposure of the head and torso, but a substantially greater safety factor applies to exposure of noncritical body parts, such as the hands, feet, wrists, and ankles, collectively referred to as "limbs" in this standard.

The DRLs of Table 1 (see 4.2.1) refer to the electric field induced within various tissues of the body. Table 1 defines DRLs in the frequency range of 0 Hz to 5 MHz based on ES mechanisms. These restrictions have been developed to minimize adverse electrostimulation with an adequate safety factor, as described in 1.3.3.

A lower tier safety factor $SF_L = 9$ applied to establish the DRL for unrestricted environments reduces the probability of an adverse reaction to a negligible value; thus, it protects (possibly) exceptionally sensitive individuals, uncertainties concerning threshold effects due to pathological conditions or drug treatment, uncertainties in reaction thresholds, and uncertainties in the induction models. In the case of the hands, wrists, feet, and ankles, $SF_L = 3$. Because these regions lack critical function when compared with the vital organs, a greater localized electric field is permitted. In the case of the restricted environment, $SF_L = 3$ 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 restricted environment for some mechanisms, but that excitation of the brain or heart is unacceptable for all individuals. $SF_L = 3$ can be justified in some cases in the restricted environment where short-term reactions are immediately apparent to the exposed individuals because they can usually remove themselves from the environment, modify their activities, or take other actions to avoid the exposure entirely.

If $SF_L = 9$ is to be compared with that applied at higher frequencies in this standard, it should be noted that a divisor of 9 applied to the magnitude of the induced field is equivalent to a divisor of 81 (19 dB) against effects associated with heating (100 kHz to 300 GHz).

B.1.3.3 Protection against effects associated with heating (100 kHz to 300 GHz)

Above 100 kHz, exposures are assessed as to potential heating effects and with reference to an averaging time, depending on whether it is whole-body or local exposure. 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 approximately 10 for the upper tier and 50 for the lower tier.

Above 100 kHz, when assessing the heating effects of short-duration exposures (less than the averaging time), the DRLs and ERLs are essentially related to energy (i.e., SA or energy density). However, the DRLs and ERLs expressed in power terms (i.e., SAR or power density or equivalent fields) may continue to be used,

while specifically recognizing their time dependence. The safety factor in this short-duration regime is thought to be at least as large as in the long-duration regime.

In explaining any standard for safe exposure to electromagnetic energy, the basic exposure diagram, such as that in Figure B.1, 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 ERL or a DRL. 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 B.1). Correspondingly the ERL or DRL 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 ERL expressed numerically or in decibels (dB). Cumulative effects such as those occurring with ionizing radiation do not occur at frequencies below 300 GHz.

At the lower laser frequencies and at frequencies below 300 GHz, the HT is generally recognized (Baranski and Czerski [B104]; Minin [B1028]). This is because the biological effects of exposure to electromagnetic fields or energy are known to be deterministic and not intrinsically stochastic (probability based) in nature, as is understood 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 with electrostimulation. The selected threshold of 4 W/kg is based primarily on behavioral disruption data in laboratory animals of several different species exposed to RF fields over durations of minutes to more than 1 h. The hazard from acute (short-term) exposure uses burns or the pain preceding a burn as its basis. For very short exposure times, and 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 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.

Above 100 kHz, the lower ERL curve (or DRL) 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,” is not necessarily exactly below the corresponding thermal time constant (denoted in Figure B.1 by T). If the safety factor is defined as the ratio of the HT curve ordinate in Figure B.1 to the ERL curve ordinate at some time t, then it is clear that the safety factor can be different in the short-term and the long-term range of exposure time. Thus, the classic ERL in the microwave range with a 6-min averaging time exhibits a higher safety factor for short exposure times ($t < T$) than it does for longer exposure times ($t > T$). This is particularly the case when whole-body exposure is considered because the thermal time constant for the whole body is considered to be about 1 h (Tell and Harlen [B1395]).

If the inflection points of both curves A and B in Figure B.1 are the same, which is equivalent to saying that if the averaging time for the lower ERL 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 (that is, as most commonly happens, the averaging time is considerably smaller than the corresponding thermal time constant), then the safety factor drops from a larger value at time below the averaging time to a smaller value for times greater than the thermal time constant. Most of the time safety factor is addressed in the long-term exposure regime, and these considerations are moot. In practice, most cases of overexposure and injury (burns) occur as a result of short-term exposures. This is to be expected because 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 ERL 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-duration exposures, the ratio of the HT and ERL abscissas in time is also 10.

In the ordinate is a power entity so that the short-duration branch of the ERL curve and (presumably) the HT curve is one of constant energy and is a straight line on a log–log plot expressing the fact that $\mathrm{SAR} \sim 1/t$.

If the curves are replotted in terms of the electric field strength 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 has been determined that the upper tier ERL of this standard for prolonged whole-body exposure incorporates a safety factor of at least 10, and possibly more (Adair et al. [B19]). The lower tier ERLs incorporate a larger safety factor.

The ERLs in the restricted environment are well matched, albeit conservatively, to the laser limits at 300 GHz (USAFRL [B1433]). 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 (Riu and Foster [B1201]). Because this standard does not provide a relaxation of the ERL at 300 GHz as the beam area decreases, but ANSI Z136.1-2014 [B69] allows a tenfold relaxation, this standard is more conservative at 300 GHz than is ANSI Z136.1-2014; that is, the RF standard incorporates an extra safety factor at 300 GHz.

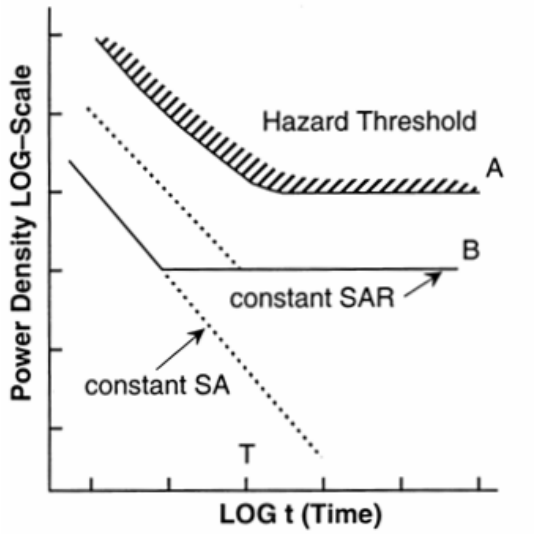


Figure B.1—Thresholds for various effects and hazards expressed as a function of time

B.1.3.4 Equivalence between RF exposure and metabolic rate

The notion of finding an equivalence between RF energy absorption and metabolic activity of humans was addressed approximately 40 years ago (Tell and Harlen [B1395]) but is still of interest today in exploring practical thermal impacts of RF exposures. An informative insight into 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 [B516]) at different work rates expressed in W/kg. Figure B.2 presents the results of applying empirical formulas derived from human data representing the equilibrium value of rectal temperature, typically obtained within ~1 h of work activity, depending on the ambient temperature. Rectal temperature rises as work begins and eventually reaches a nominal plateau value shown in Figure B.2 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 workload and increased ambient temperature. At very high air temperatures, the core temperature 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 B.2 and likely do not accurately represent core temperatures when an equilibrium value could never be achieved due to extraordinary environmental circumstances.

It is of interest to note that when the added workload is equivalent to a whole-body-average RF energy absorption rate of 0.4 W/kg, the DRL 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 ~40 °C (104 °F). This observation is consistent with human RF exposure studies wherein exposures substantially exceeding the upper tier ERL resulted in only minimal increases in core body temperature (Adair et al. [B9], [B20], [B24], [B25]; Adair and Black [B18]) and adds support to the contention that the upper tier DRLs (100 kHz to 6 GHz) in this standard should be protective for all humans.

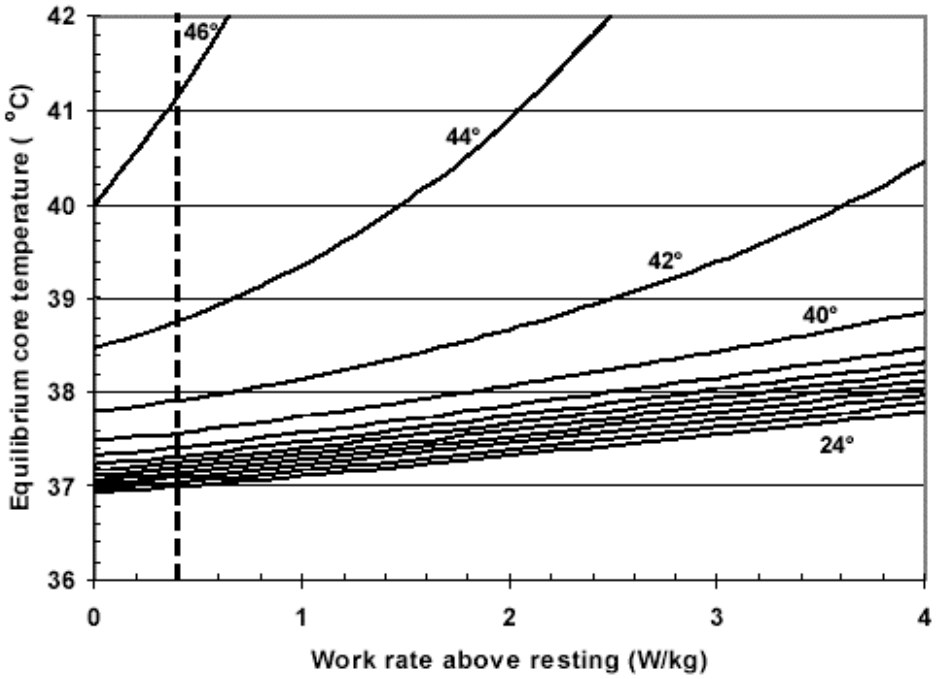


Figure B.2—Projected equilibrium core temperature as a function of additional workload above resting metabolic rate for a range of ambient air temperatures (24 °C to 46 °C) derived from empirical data obtained on U.S. Army soldiers

B.1.4 Uncertainty parameters

In the preceding discussion, except when considering the statistical spread for electrostimulation, it has been assumed that both the HT and the corresponding ERL are clear entities with definite values. In reality, many uncertainties modify the meaning of safety factor and its dependability in practice.

Figure B.3 shows a representation of the range of uncertainty above and below both the HT and ERL curves. In the most general view, these uncertainties, ΔU_r, could be different below and above a curve and could be different as a function of time. The uncertainties in the HT curve include the following:

a) Dosimetry 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 nonuniform fields.

d) Effects of environmental factors like temperature, humidity, air flow, insulation, and so on.

e) Statistical variation among people as to thresholds for tolerance of electrostimulation or heating under various conditions.

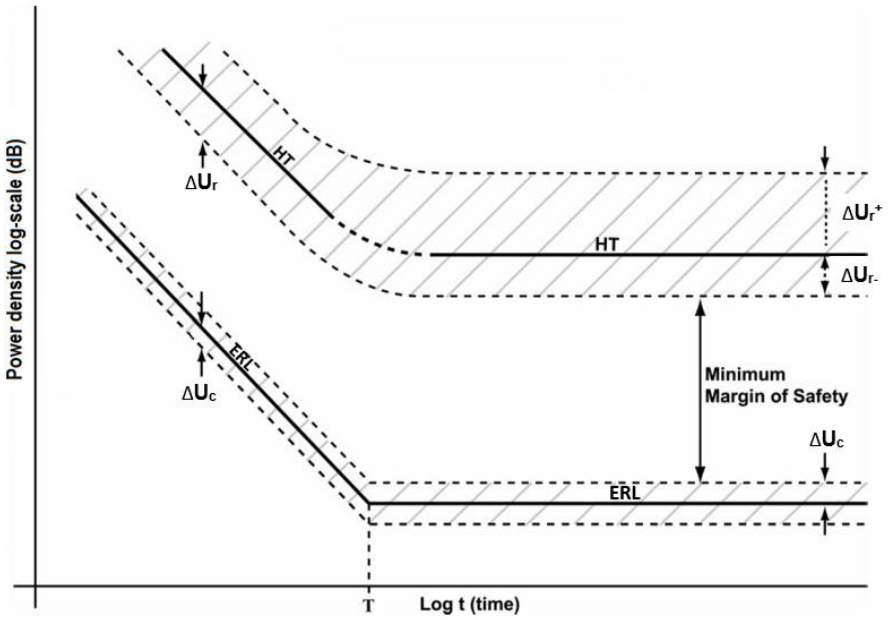
f) Extrapolation of experimental data from animals to humans. This uncertainty could be small when dealing with electrostimulation or local heating but could be large when dealing with complicated higher-level phenomena in animals, such as behavioral effects. It can be encouraging, however, to note that humans have generally a far superior system of thermoregulation than do most animals.

g) It has been long recognized (NRC [B1080]) 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, errors in determining exposure contribute to a compliance error ΔU_c.

i) Finally, based on 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 ERL, for which there is recognition of some margin of safety, beyond allowance for uncertainties.

Figure B.3 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.



[a] This diagram is based on power. A similar diagram could be based on internal electric field.

Figure B.3—Hypothetical exposure diagram illustrating uncertainty factors and resulting minimum margin of safety[a]

B.1.5 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 compliance with this standard protects against this hazard. Transient electrical sensations, even those that are painful, are sometimes experienced by electrical workers; these are made improbable by compliance with this standard. Overall, the standard incorporates reasonably large margins of safety. An RF safety program shall be implemented whenever individuals could be

exposed above the lower tier limits (see 4.5). Indeed, the standard should be considered especially conservative because the safety factors are applied against perception phenomena (e.g., electrostimulation and 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.

B.2 Rationale for limits based on electrostimulation (0 Hz to 5 MHz)

B.2.1 Introduction

B.2.1.1 General

The exposure limits in the lowest frequency regime of this standard are based on electrostimulation, which is manifested by excitation of nerve and muscle, as well as by alteration of neural synapse activity. An upper frequency limit on electrostimulation occurs with continuous sinusoidal waveforms at 100 kHz. Below 100 kHz, electrostimulation thresholds are typically lower than thermal perception thresholds, and above that, the opposite is true; that is, thermal effects typically exhibit a lower threshold than do electrostimulation effects (Chatterjee et al. [B244]; Dalziel and Mansfield [B311]). However, for pulsed waveforms of low duty factor, electrostimulation thresholds may remain below thermal thresholds to significantly higher frequencies (Reilly [B1179], [B1186]). Electrostimulation thresholds have been experimentally demonstrated with sinusoidal waveforms up to frequencies of several megahertz (Reilly [B1179], pp. 140–141: Rogers et al. [B1208]). Criteria for determining compliance with this standard for pulsed or nonsinusoidal waveforms are specified to include frequencies up to 5 MHz.

The low-frequency regime consists of the following subdivisions:

a) Quasi-static regime (0 Hz $\leq f \leq$10 Hz) where magnetohydrodynamic effects can be dominant. The DRLs are expressed here in terms of the *in situ* magnetic field.

b) Electrostimulation regime (0.153 Hz $\leq f \leq$ 100 kHz), where electrostimulation effects prevail with continuous sinusoidal waveforms. DRLs are expressed in terms of the *in situ* electric field.

c) Electrostimulation/thermal transition regime (100 kHz $\leq f \leq$ 5 MHz), where with pulsed waveforms, either electrostimulation or thermal effects, can be dominant, depending on the duty factor of the pulses. DRLs are expressed in terms of the *in situ* electric field and the SAR.

If a frequency falls in an overlap region where two mechanisms could apply, the more stringent criterion defines the acceptable exposure limit such that the DRL criterion for each mechanism is satisfied. For example, at 1 Hz, the exposure limit is such that the more stringent of the magnetohydrodynamic magnetic field limit and the electrostimulation limit is satisfied. At 1 MHz, the exposure limit is such that the more stringent of the electrostimulation and the SAR thermal limit is satisfied.

B.2.1.2 Excitation thresholds—S-D and S-F laws

B.2.1.2.1 General

Electrostimulation is initiated by depolarization of the excitable cellular membrane (nerve or muscle; Reilly [B1179]). This modification of the cellular resting potential is determined by the electric field in the medium surrounding the excitable tissue (the component of the field parallel to the long axis of the cell). Knowledge of the spatial and temporal properties of the electric field is required to assess electrostimulation. Of course, the electric field can be derived from the current density by taking the ratio J/σ, where J is current density

and σ is the conductivity of the medium. But basing a standard on current density rather than on the *in situ* electric field introduces an additional parameter. Thus, the *in situ* electric field is used as the fundamental metric for electrostimulation limits in this standard.

An *in situ* electric field, strength-duration (S-D) curve, which defines thresholds for monophasic stimulus waveforms, can be defined by two parameters: the minimum (rheobase) excitation threshold E_0 and the S-D time constant τ_e.[31] Values of E_0 and τ_e differ considerably for nerve excitation, muscle excitation, and synaptic activity alteration. Table B.2 lists median threshold values of E_0 and τ_e that are used in this standard to limit electrostimulation effects. Peak electric field rheobase thresholds are listed in Table B.2 along with τ_e values. The derivation of these values is explained in the following sections. Equation (B.1) and Equation (B.2) are used to determine the median excitation thresholds at a given frequency as follows:

$$E_i = E_0 \qquad \text{for } t_p \geq \tau_e \tag{B.1}$$

$$E_i = E_0 \left(\tau_e / t_p\right) \qquad \text{for } t_p \leq \tau_e \tag{B.2}$$

where t_p is the phase duration of the E_i waveform.

Alternatively, the limits can be determined in terms of sinusoidal frequency as shown in Equation (B.3) through Equation (B.5):

$$E_i = E_0 \qquad \text{for } f \leq f_e \tag{B.3}$$

$$E_i = E_0 \left(f / f_e\right) \qquad \text{for } f \geq f_e \tag{B.4}$$

$$f_e = 1/(2\tau_e) \tag{B.5}$$

Equation (B.5) has been determined using a theoretical model of myelinated nerve (Reilly [B1179]; Reilly and Diamant [B1188]). Because of the nonlinear electrodynamics of excitable tissue, Equation (B.5) differs from linear systems for which a relationship $\tau = 1/(2\pi f)$ would be anticipated.

Nerve excitation thresholds follow a U-shaped S-F curve, with a low-frequency asymptotic upturn below about 10 Hz and a high-frequency asymptotic upturn above f_e. The plateau between the upper and lower transition frequencies is the rheobase. Theoretical models suggest that the S-D time constant and upper transition frequency are related by $f_e = (2\,\tau_e)^{-1}$ (Reilly [B1179], [B1184]; Reilly and Diamant [B1189]). A low-frequency upturn occurs for *in situ* sinusoidal waveforms initiated at a zero crossing because the slow rate of rise of the sinusoid allows the nerve to accommodate to the stimulus—a feature absent in the square-wave stimulus or a sinewave initiated at a peak. To allow for stressful-case conditions, the induced field waveform is assumed to be initiated at a peak. Because the induced field is proportional to the derivative of the environmental field at frequencies covered by this standard, this assumption corresponds to a sinusoidal environmental field initiated at a zero crossing. Above f_e, thresholds converge to a slope that is proportional to frequency for continued sinusoidal stimulation.

[31] The S-D curve discussed here refers to a plot of excitation threshold versus duration of a monophasic stimulus; τ_e is the duration at which the stimulus magnitude versus duration and the charge versus duration curves intersect. τ_e is a more robust parameter than is "chronaxie," which is often used to designate the temporal response of excitable tissue, and it is a more suitable for expressing DRLs in exposure standards, as in Equation (B.1) and Equation (B.2) (Reilly [B1185]).

For a given stimulus duration, a monophasic square-wave current provides the lowest threshold of electrostimulation. Brief biphasic current wave shapes in general have higher thresholds of excitation. The increase in threshold due to a biphasic current reversal becomes greater as the phase duration becomes shorter (i.e., as the frequency content of the event becomes higher). However, for repeated biphasic waves (e.g., a repeated sinusoid), thresholds converge to a value that is approximately that for a single monophasic square wave having the same phase duration (Reilly and Diamant [B1188], Figure 3.6 and Figure 7.2). Consequently, thresholds pertaining to monophasic square-wave stimuli, which establish a lower limit, have been applied to biphasic waves with the same phase duration. For a single biphasic event of brief duration, the excitation threshold can be higher than that for a monophasic stimulus, and therefore, this approach is conservative.

Table B.2—Models for established thresholds of reaction—Median *in situ* electric field strength thresholds[a]

Reaction	E_0 peak (V/m)[b]	τ_e (ms)	f_e (Hz)
Synapse activity alteration, brain	0.075	25.0	20
10 μm nerve excitation, brain	12.3	0.149	3350
20 μm nerve excitation, body	6.15	0.149	3350
Cardiac excitation	12.0	3.00	167

[a] Adapted from Reilly [B1185].

[b] E_0 peak refers to the temporal peak of the electric field.

B.2.1.2.2 Nerve excitation

B.2.1.2.2.1 General

Excitation of nerve and muscle requires adequate depolarization of the membrane resting potential (e.g., by approximately 15 mV to 20 mV for pulse durations > 100 μs) at some point along the axon or muscle fiber—the exact amount depends on the stimulus waveshape and other factors. In the region of a locally constant electric field, excitation is initiated where a nerve is terminated, or undergoes a rapid bend, such as occurs at a motor neuron end plate or at sensory receptors (Reilly [B1179], [B1182]). Under these conditions, the threshold of excitation of a nerve cell is inversely proportional to the diameter of the nerve axon. The nerve cell can also be excited at a point on the axon where the spatial gradient of the external field is sufficient, although such criteria have not been applied to this standard.

Fibers that are most sensitive to electrostimulation have a diameter at the outer limit of the distribution of fiber sizes found in humans. Accordingly, a maximum diameter of 20 μm is assumed for a peripheral nerve and 10 μm for a central nervous system (CNS) neuron. The theoretical model[32] used in this standard predicts E_0 = 6.15 V/m and 12.3 V/m for stimulation of 20 μm and 10 μm nerve fibers, respectively, and τ_e in the range 100 μs to 170 μs for either fiber size, depending on the distribution of the excitatory field along the linear dimension on the affected fiber (Reilly and Diamant [B1188]).

These values correspond well to experimental data. Median experimental values of τ_e with magnetic stimulation are reported in the range 146 μs to 152 μs (Barker et al. [B109]; Bourland et al. [B184]; Mansfield and Harvey [B965]), although larger values have also been reported (Bourland et al. [B184]; Havel et al. [B580]; Nyenhuis et al. [B1089]). Experimental values of τ_e with contact current or magnetic stimulation encompass a fairly wide range (Reilly [B1183], Table 7.1), most of which fall within the range of values obtained with the SENN model (Reilly and Diamant [B1188], Figure 4.4 and Figure 8.15). Alternatives to the SENN model are compared in Reilly [B1187].

[32] The electrostimulation model used in this standard is known as the "spatially extended nonlinear node" (SENN) model. It is available free of copyright restrictions and free of charge from a publisher's website: http://us.artechhouse.com/Assets/reslib/reilly/reilly.html

Rheobase values obtained with the SENN model are also in reasonable correspondence with experimental data. The threshold electric field with a coil encircling the forearm was found to be 5.9 V/m (Havel et al. [B580]), which is quite close to the SENN rheobase value of 6.15 V/m for a 20 μm fiber. In addition, an underlying neural excitation assumption of 6.15 V/m correctly reproduces the distribution of let-go current thresholds in adults (Sweeney [B1372]). One research group (So et al. [B1328]) evaluated human perception data collected with magnetic field stimulation by computing the induced electric field strength at the periphery of the body based on a detailed anatomical induction model. This group reported peripheral nerve rheobase thresholds in the torso of 3.8 V/m to 4.6 V/m. Another study using a detailed anatomical model (Den Boer et al. [B340]) suggested similarly low rheobase values, although several prior studies by the same group supported larger values in line with the SENN model and ellipsoidal induction model (Reilly [B1179], pp. 379–381). Using a similar detailed anatomical model, another group found that human perception thresholds with magnetic stimulation were representative of previously published results with the SENN model (Liu et al. [B905]). Experimental bases for model parameters require further examination.

Table B.2 adopts a value of $\tau_e = 149$ μs as suggested by an average of the lower experimental values mentioned earlier. The theoretical value of $E_0 = 6.15$ V/m is considered a median within a distribution of thresholds in healthy adults. The most sensitive means of exciting skeletal muscle is via electrostimulation of the motor neurons that innervate it. Consequently, thresholds for muscle stimulation follow those for nerve excitation. An exception to this occurs with cardiac stimulation.

Numerous experiments with perception of sinusoidal current reveal an S-F law with a minimum plateau below a critical frequency f_e, above which thresholds converge to a frequency-proportional law when the current is of a continuous nature (Reilly [B1182]). With continuous sinusoidal stimulation, a frequency-proportional asymptote has been demonstrated in humans to a frequency of 100 kHz, above which thermal perception thresholds dominate (Chatterjee et al. [B244]; Dalziel and Mansfield [B311]). However, for pulsed sinusoidal waveforms, the frequency-proportional relationship can be extended into the megahertz region as suggested by neurostimulation experiments in rats (LaCourse et al. [B804]), and in human experiments using brief ($\cong$ 0.1 μs) pulses (Reilly [B1179]). For largely monophasic waveforms, S-D laws have been verified to pulse widths of a few nanoseconds (Rogers et al. [B1208]).

B.2.1.2.2.2 DRLs not applied with current crossing skin layers

DRLs in Table 1 (see 4.2.1) do not apply to currents that cross skin layers. A high-resolution detailed anatomic (DA) model could potentially show exceptionally high induced fields in low-conductivity layers of the skin (e.g., stratum, corneum, and subdermal adipose tissue) through which current (or the induced electric field) passes radially across layers of skin. One example could include touch contact. Another scenario could include electromagnetic field (EMF) induction in which the induced field takes a path across skin-to-skin contact at the armpit or crotch or, in a posterior view, from left to right buttocks. Such scenarios have been studied recently by others (e.g., De Santis et al. [B347]). In such cases, the specification of a DRL should properly be stated in terms of the spatial derivative dE/dx of the induced *in situ* electric field, rather than in terms of its magnitude, as applied to other tissues (cf. Reilly and Diamant [B1188], pp. 48–49). Such criteria have not yet been developed for application to ES-based standards.

B.2.1.2.3 Cardiac excitation

Cardiac excitation, which refers to electrical stimulation of a contraction (systole), follows S-D and S-F laws like those for nerve excitation, except with much greater values of τ_e (smaller values of f_e). Experimental data demonstrate that τ_e depends on the focality of the stimulus. For focal stimuli, as with a small electrode near the excitable tissue, time constants can be much smaller than when the stimulus is more diffuse, as it would be for magnetically induced *in situ* electric fields. An S-D time constant $\tau_e = 3$ ms has been assumed to apply to large contact electrodes or diffuse stimulation of cardiac tissue; $E_0 = 12$ V/m has been assumed as a median rheobase for excitation based on experimental data (Reilly [B1179], [B1186]).

Cardiac excitation is not necessarily hazardous, although ventricular fibrillation (VF) is a life-threatening condition. Minimum thresholds for VF typically exceed those for excitation by a factor of 50 or more. However, if the heart is repeatedly excited, the VF threshold drops such that the margin between VF and excitation thresholds can be reduced to a factor as little as two if the stimulus is applied during the vulnerable period within the cardiac cycle. For prolonged exposures exceeding ~90 s, recent experimental data show that the VF thresholds can almost equal thresholds where cardiac arrhythmias are observed (Kroll et al. [B786]). The cardiac DRL in Table 1 (see 4.2.1) applies to the apex of the heart, which has been shown to be the most sensitive region of the heart for inducing VF (Roy et al. [B1220]).

Cardiac excitation is not an exposure issue under most circumstances since the exposure limits for peripheral nerve excitation with exposure of the torso would preclude over-exposure of the heart.

B.2.1.2.4 Synaptic activity alteration

When stimulated by a long pulse (> 0.1 ms), the nerve cell requires membrane depolarization of approximately 15 mV to 20 mV to initiate an action potential. Synaptic processes can be affected by altering the presynaptic membrane potential by less than 1 mV, and possibly by as little as 60 μV, as with electrical stimulation of synapses in the retina (Knighton [B763], [B764])—a factor 250 times below minimum neural excitation thresholds. Consequently, the synapse is a potentially sensitive site for neural interaction with applied electrical stimuli. A small change in presynaptic potential can have a much larger percentage change in postsynaptic potentials (Katz and Miledi [B738]). Since the postsynaptic cell sums the inputs from many presynaptic cells, a small change in presynaptic potential can have a significant postsynaptic effect and can be either inhibitory or excitatory (i.e., can result in the excitation of a neuron that would otherwise not have been excited) or can inhibit excitation of a neuron that would otherwise have been excited.

An example of a synaptic polarization effect is attributed to the phenomenon of electrophosphenes and magnetophosphenes, which are visual effects resulting from electric currents or magnetic fields applied to the head (Adrian [B35]; Barlow et al. [B110], [B111]; Baumgardt [B116]; Bergeron et al. [B134]; Budinger et al. [B212]; Carstensen [B237]; Clausen [B273]; Lövsund et al. [B924], [B925]; Silny [B1311]). Experimental evidence suggests that phosphenes result from modification of synaptic potentials in the receptors and neurons of the retina (Knighton [B763], [B764]; Lövsund et al. [B925]), rather than excitation of the optic nerve or the visual cortex, although visual sensations with stimulation of the visual cortex have been demonstrated with much stronger stimuli (Brindley and Lewin [B196]; Brindley and Rushton [B197]; Ronner [B1212]).

Using data from magnetophosphenes (Lövsund et al. [B924], [B925]), the corresponding induced electric field strength in the head at the most sensitive frequency tested (20 Hz) is 0.075 V/m peak as calculated with an ellipsoidal model of the head (see Annex B of IEEE Std C95.6-2002 [B671]). At the retina, where the electrical interaction is thought to take place, the calculated field is 0.053 V/m rms, which is consistent with the current density threshold of 0.008 A/m^2 at the retina determined for electrophosphenes (Lövsund et al. [B924]) assuming the conductivity of the brain is 0.15 S/m. The internal electric field strength corresponding to phosphene perception at the optimum frequency is a factor of ~100 below rheobase thresholds for neural stimulation.

In the large majority of magnetophosphene studies, the known stimulation metric is the external magnetic field rather than the *in situ* electric field. In these cases, the DRL is derived from the external field by use of an induction model—in this standard, the ellipsoidal model described in IEEE Std C95.6-2002 [B671] is used. Consequently, if one wishes to calculate an *in situ* field resulting from a particular exposure scenario for comparison with the standard's DRL, one should determine compliance with the same model. If one wishes to use a detailed anatomical model (see B.2.1.4.2), for instance, one should first derive a DRL based on that same model.

Experimental S-D data show that τ_e for phosphenes using electrodes on the temples is approximately 14 ms (Baumgardt [B116]; Bergeron et al. [B133]), and for electrically evoked potentials in the frog's eye, τ_e is in

the range 14 ms to 36 ms (Knighton [B763], [B764]). These values are consistent with the phosphene data described earlier, but they are ~100 times greater than corresponding values for peripheral nerves.

Few data exist on synaptic polarization effects by applied electric fields. Considering this dearth of data, reasonable assumptions are made based on the available synaptic effects from experimental data and on assumed parallels with nerve excitation properties. One class of these properties concerns S-D and S-F characteristics. An average S-D time constant for synapse effects is $\tau_e = 25$ ms. Using the relationships noted for nerve excitation, an S-F constant of $f_e = 20$ Hz is expected above which *in situ* electric field thresholds should increase. This increase is indeed observed in the case of electrophosphene thresholds, although the rate of increase is greater than that observed with nerve excitation (Adrian [B35], Clausen [B273]). Magnetophosphene S-F curves reported by Lövsund et al. [B924], [B925]) show a minimum at 20 Hz, as well as rising thresholds at lower frequencies, in accord with electrophosphene data. Thresholds above 20 Hz vary somewhat with the experimental parameters (background illumination and wavelength, subject visual acuity). Considering electrophosphene and magnetophosphene S-F and S-D curves in total, it is conservative to adopt a threshold curve similar to that found in electrostimulation of nerve and muscle but with a much lower S-F constant (or, equivalently, with a larger S-D time constant) and with a lower rheobase. Additional study of CNS synaptic interaction effects is needed to clarify these assumptions.

Frequency sensitive thresholds for phosphenes have been experimentally tested only to a maximum frequency of about 75 Hz. The conservative assumption is made in this standard that synaptic polarization thresholds follow a frequency-proportional law above 20 Hz to a frequency of at least 760 Hz (above which peripheral nerve excitation limits dominate the magnetic field ERLs).

In connection with phosphene threshold experiments, Lövsund et al. ([B925], p. 330) state: “Virtually all the volunteers noted tiredness and some reported headaches after the experiment. Some experienced after-images which were generally of only short duration following exposure to the magnetic field. In one case, however, they persisted up to 10 min after the experiment. Individual volunteers reported spasms of the eye muscles, probably arising from stimulation by the field.” These findings were similar to those of Silny [B1311], who reported headaches, indisposition, and persistent visual evoked potential (VEP) alterations at magnetic flux density levels above phosphene thresholds but still well below nerve excitation thresholds (by a factor of 23).

This standard does not consider the production of phosphenes *per se* as adverse. Near the threshold of perception, these visual effects are subtle and require carefully controlled lighting conditions for detection. However, it remains to be demonstrated that the mechanisms that produce phosphenes also occur at synapses within the brain. That possibility should be taken seriously, especially considering that clearly adverse reactions that can be attributable to CNS reactions (tiredness, headaches, muscle spasms, persistent after images) are reported in connection with phosphene threshold experiments. It is unlikely that the phosphenes themselves were causing the reported adverse reactions. A plausible explanation is that the adverse effects were due to electrostimulation of brain neurons in accord with the synapse mechanism discussed previously.

The ability of subexcitatory fields to alter neuronal response has also been reported after exposure of hippocampal slices from the rat brain to magnetic fields (Bawin et al. [B124], [B125]) in which induced electric field strengths were as low as 0.75 V/m peak—a factor of 16 below the threshold of 12.3 V/m for excitation of a 10 μm neuron. The rate of maze learning in living mice was significantly reduced by exposure to flux densities at and below 0.75 mT at 50 Hz (Sienkiewicz et al. [B1306], [B1307]). Although the cited studies did not establish a synaptic mechanism, they do support the view that CNS effects, including adverse ones, might be possible well below thresholds of excitation of brain neurons. In light of the existing data, this standard conservatively considers alteration of synaptic activity in the brain from electrostimulation to be an adverse effect if not applied in a medical setting.

The synaptic activity restrictions of this standard do not, however, apply to the spinal cord, even though it also contains synapses. Spinal functions are important to the organism (e.g., control of posture and reflex activity). Tests have been conducted with human subjects whose torsos were subjected to the strong switched gradient fields of experimental MRI systems (see B.2.1.2.2 and B.2.1.4.3). Perception was sometimes preferentially reported in the small of the back at stimulus levels corresponding to nerve stimulation thresholds in accord with expectations from an elliptical induction model (see B.2.1.4.3 of

this standard and Annex B of IEEE Std C95.6-2002 [B671]). These tests showed no observable effects below the neural threshold for perception. The lack of an observable effect below electrical perception thresholds suggests one of three possible explanations. One is that spinal synapse interactions did occur, but they were imperceptible to the subject. Another is that the induced field in the spinal column was below synapse interaction thresholds, even though the levels just outside of the spinal column were approximately two orders of magnitude above synapse thresholds. A third is that stimulation thresholds are significantly greater than what has been assumed for synaptic effects in brain neurons (Table B.2).

Considering that we could find no data to suggest any observable effects from stimulation of the spinal cord at the levels attributed to synapse thresholds, protection in this standard for synaptic activity alteration is focused on the brain rather than on the spinal cord.

B.2.1.2.5 Averaging time

The rms metrics specified in Table 1 (see 4.2.1) through Table 4 (see 4.2.3.1) and Table 12 and Table 13 (see 4.4.1.1) require the specification of an averaging time. For sinusoidal stimulus waveforms, thresholds of nerve excitation evaluated at half-cycle increments oscillate between gradually falling maxima at odd numbers of half cycles, and minima at even number of half cycles, and converge to a single minimum threshold at ~1.3 ms of stimulus duration (Reilly [B1179]). The time constants of excitation threshold versus duration for muscle and nerve synapse stimulation exceed that for nerve stimulation by factors of 20 and 168, respectively (Table B.3). Consequently, a measurement averaging duration of 200 ms ($\cong 168 \times 1.3$) would encompass the maximum integration duration needed to characterize minimum nerve, muscle, and synapse excitation thresholds. For sufficiently low frequencies, the variation of threshold with the number of cycles above one is trivial, and a measurement averaging time of a few cycles appears adequate. For frequencies below 0.1 Hz, a maximum averaging time of 10 s (one cycle) is considered adequate.

Criteria for nonsinusoidal or pulsed field require instantaneous values of the *in situ* stimulus waveform (for evaluation of DRLs, or ERLs). Refer to B.2.1.6 for additional discussion.

B.2.1.2.6 Spatial averaging of induced *in situ* fields

When determining compliance with the DRLs (Table 1 see 4.2.1), an important parameter is the averaging distance d_a over which the *in situ* electric field should be quantified. A related question is the required distance over which the electric field needs to exist for efficient electrostimulation. For cases of practical interest involving unintended electrical exposure, the most sensitive means of exciting a nerve fiber is via an *in situ* electric field oriented with the long axis of the nerve fiber and acting at its terminus (Reilly [B1182]). An exception to this statement might occur when a small stimulus electrode is situated near the nerve, but such cases would normally be found only in medical applications rather than in chance electrical encounters or electromagnetic induction.

The relationship between the threshold of excitation and the distance over which the field exists (d_e) has been determined using a nonlinear model of a myelinated nerve (Reilly and Diamant [B1191]). With this model, a minimum threshold was obtained with d_e of seven or more internodal spaces. With d_e of one internodal space, the threshold was twice the minimum value. With $d_e = 2$, 3, 4, and 5 internodal spaces, the threshold exceeded the minimum value by 34 %, 14 %, 7 %, and 3 %, respectively. For a nerve axon diameter of 20 μm (the size assumed in this standard for peripheral nerves), the internodal distance is 2 mm. If an averaging distance (d_a) of 5 mm is used, and assuming a field just at the threshold of excitation corresponding to d_e, the measured average field with $d_e \leq 2$ internodal spaces would be within 19 % of the basic restriction value (Table 1). For larger d_e and with a corresponding threshold field, the measured average field over 5 mm approaches the basic restriction value within a few percent. It appears that 5 mm represents a reasonable averaging distance, which is neither overly conservative nor permissive. Consequently, this standard specifies that the *in situ* electric field be determined as the average over a distance $d_a = 5$ mm, which can be determined from the potential difference at a spacing of 5 mm.

The specification of an averaging distance suggests a simple measurement that might be applied to *in vivo*, *in silico*, or phantom measurements (or calculations). To evaluate compliance, simply determine the voltage difference, ΔV, across a probe with two contacts spaced $d = 5$ mm, and oriented arbitrarily within the tissue of interest. The ratio $\Delta V/d$ provides the field averaged over the specified distance.

B.2.1.3 Adverse reaction criteria

The purpose of DRLs and ERLs is to avoid adverse reactions, not just perceptible ones. Aversive or painful electrostimulation is considered adverse. Potentially hazardous startle reflex reactions are also considered adverse; such reactions have been shown to occur at levels consistent with the threshold of pain (Reilly, [B1179]). Consequently, protection against painful electrostimulation would also protect against potentially hazardous startle reflexes.

Painful sensations from magnetic stimulation of peripheral nerves are reported at multiples above perception thresholds of 1.3 (Budinger et al. [B212]), 1.6 (Bourland et al. [B184]), and 1.48 (Nyenhuis et al. [B1089]; Schaefer et al. [B1256])—an average multiple of 1.45. The mean threshold for intolerable pain was observed at a perception multiple of 2.05 (Schaefer et al. [B1256]). The median rheobase threshold for painful stimulation by a square-wave pulse or a continued sinewave is taken as $E_0 = 6.15 \times 1.45 = 8.92$ V/m (peak). Based on a lognormal probability model of human perception thresholds of electrical stimuli (see B.2.1.9), a conservative estimate of a one-percentile pain reaction threshold for healthy adults is a factor of 3 below the median, resulting in a rheobase of 2.97 V/m (peak).

In the case of contact current stimulation, unpleasant and painful sensations are elicited at greater multiples above perception than with magnetic stimulation. Based on experimental data from several sources (Reilly, [B1179], Table 7.3), painful stimulation is estimated to occur at a multiple of 2.4 above the perception threshold; unpleasant sensations are estimated to occur at a multiple of 1.7; the ratio of pain to unpleasantness thresholds is about 1.4.

That smaller pain-to-perception ratios are found with magnetic stimulation than with contact current stimulation can be explained by the fact that in magnetic stimulation, the distribution of induced current varies only gradually with respect to body dimensions. Consequently, at a field level where some neurons first begin to be excited, a small increase in the field can excite neurons over a large area. If pain is magnetically induced in some area of the body, it is likely to be in an extended area. In contrast, cutaneous stimulation is usually more focal. Suprathreshold stimulation in a large area can be more painful than in a small area, and that might account for the differences in pain-to-perception ratios between magnetic induction and small-area contact current.

Cardiac excitation is considered adverse. Although not necessarily life threatening in itself, it is potentially dangerous if it is repeated in close succession, such as can be the case with sinusoidal or repeated pulse stimulation of the heart (see B.2.1.2.3).

With synaptic effects, this standard treats any alteration of brain activity as a result of electrical stimulation of brain neurons via the induced *in situ* electric field as a potentially adverse outcome. Such conservatism was motivated by reports of adverse reactions (tiredness, headaches, muscle spasms, persistent after-images) in laboratory experiments with human subjects using magnetic field exposures near the magnetophosphene threshold (see B.2.1.2.4). However, these findings have yet to be confirmed.

With magnetohydrodynamic effects and forces on charges due to rapid body motion in strong static and quasi-static fields, a variety of biological effects have been observed (see B.2.1.5). In light of these observations, adverse reactions are assumed at 1.06 T rms (1.5 T peak) in 50 % of human subjects at frequencies below 1 Hz, which can include nausea, vertigo, and taste sensations associated with head movement.

B.2.1.4 Threshold limits for magnetic field exposure

B.2.1.4.1 General

To derive an environmental magnetic field (ERL) from allowable *in situ* electric field strength (DRL), it is necessary to apply an induction model. Traditional methods used to predict induced *in situ* electric fields during magnetic field exposure include the use of ellipsoid shapes arranged to mimic an animal or a human (Reilly [B1182]). More recently, high-resolution anatomical models have been developed to enhance the capability to predict localized electric field, such as within a single organ or part of an organ. Such models are discussed in B.2.1.4.2.

B.2.1.4.2 Detailed anatomical induction models

IEEE Std C95.6-2002 [B671] relied on a homogeneous, isotropic ellipsoidal model of the human body to derive ERLs (previously referred to as "MPEs") based on both rheobase and tissue time constant data available at that time, as well as on data describing the exposure and frequency response for magnetophosphenes. Although detailed, anatomically correct computer modeling of dose induced in tissue by external magnetic fields had been reported, IEEE Std C95.6-2002 nevertheless opted for the simpler ellipsoidal induction model, rather than the detailed anatomical model for which large variations in calculated maximum induced fields were reported among various investigators. Such deviations, thought to be due to computational artifacts, are especially important in projecting exposure levels that correspond to electrostimulation thresholds, where the maximum induced field, in particular, classes of tissue, are relevant. This stands in contrast to thermal effects at higher frequencies, where organ averages of absorbed energy are relevant, and where a few large outliers have little importance. Also, consistency among laboratories, even reporting mean or median values within organs of detailed anatomical models, had not yet been narrowed to an acceptable quality.

More recently, consistency among laboratories using detailed anatomical models has been narrowed to a typically acceptable range when the 99th percentile induced in particular classes of tissue is used rather than the maximum value, although some large variations of calculated values among laboratories are still encountered. Hirata et al. [B617] suggested that the 99th percentile could be viewed as an acceptable surrogate of the tissue maximum dose for electrostimulation predictions, although this assertion has not yet been verified with laboratory measurements of electrostimulation dose and response. The evidence for improved consistency using the 99th percentile calculated values is a positive development (Caputa et al. [B228]; Hirata et al. [B624]; Stuchly et al. [B1359]). Such improvement in consistency has led some agencies to calculate ERLs from DRLs using detailed anatomical models (ICNIRP [B644]).

For modeling purposes, the 99th percentile electric field within each tissue type has been applied as the dosimetric estimate of the tissue maximum (Caputa et al. [B228]; Hirata et al. [B617]; So et al. [B1328]; Dimbylow, [B359], [B360]; Kavet et al. [B741]; Den Boer et al. [B340]; Kavet et al. [B740]). For the heart and a 10 μm CNS neuron, the rheobase and tissue time constants would remain substantially unchanged from those in IEEE Std C95.6-2002 if a detailed anatomical rather than an ellipsoidal model were used. For DRLs based on peripheral nerve stimulation, thresholds of adverse reaction might require revision based on a reevaluation of laboratory data, as well as on anatomical modeling in which fat and/or skin are used as a surrogate for peripheral nerve tissue (see B.2.1.2.2).

At the present time, ICES does not see the need to revise the DRLs solely on the basis of existing detailed anatomical models.

B.2.1.4.3 Ellipsoidal induction model

Limits on environmental magnetic fields in this standard are based on an ellipsoidal model of the head and torso of a large individual, with uniform conductivity, and a constant magnitude and relative phase of the field over the body dimensions as described in Annex B of IEEE Std C95.6-2002 [B671]. In all calculations, a worst-case assumption has been made for the direction of the field relative to the body.

Using this model, an *in situ* field of 6.15 V/m (the presumed median nerve excitation threshold among subjects) has been calculated to be induced in the periphery of the torso with whole-body exposure to $dB/dt = 37.5$ T/s (see Annex B of IEEE Std C95.6-2002 [B671] and Table B.2). That theoretical value applies to conditions of exposure that result in a conservative estimate of the excitation threshold, namely, a very large adult; constant magnitude, direction, and relative phase of the incident field over the dimensions of the body; and a monophasic square-wave shape of the *in situ* electric field. In most cases, experimental conditions deviate from the optimal parameters resulting in greater thresholds than the conservative estimates in this standard.

One of the cited conservative assumptions was a monophasic square-wave shape for the induced electric field. Note that the *in situ* field follows the waveform of the time derivative of magnetic flux density, dB/dt, which is necessarily biphasic for a magnetic pulse; the mean induced field is zero if the rise and fall magnitudes of magnetic flux density are equal, although the rise and fall times need not be equal. If the induced waveform is such that the phase reversal is either delayed or is gradual, then the threshold can be effectively the same as would apply to a monophasic waveform.

The conservatively derived theoretical value of 37.5 T/s mentioned earlier can be compared with experimental thresholds conducted with pulsed magnetic field exposure of the human torso in magnetic resonance imaging (MRI) studies (Bourland et al. [B184], [B185], [B186], [B187]; Budinger et al. [B212]; Cohen et al. [B289]; Mouchawar et al. [B1044]; Nyenhuis et al. [B1089]; Schaefer et al. [B1257], [B1258]; Yamagata et al. [B1529]), as previously reviewed (Reilly [B1179], Sect. 9.7). Mean perception thresholds of 60 T/s were reported by two investigators (Budinger et al. [B212]; Cohen et al. [B289]), and a minimum threshold of 45 T/s was reported by another (Bourland et al. [B184]). Higher thresholds were reported by others, but, like the cited studies, these involved suboptimum waveforms or conditions not conducive to minimum rheobase values. (See also B.2.1.4.)

Simulated MRI fields used in the experiments discussed previously varied considerably in amplitude and relative phase over the dimensions of the human torso. The optimum field metric for electrostimulation is not clear when such nonuniformity exists. Recent studies report perception thresholds in terms of the spatially averaged exposure, rather than the spatial peak, as in most of the studies mentioned in the previous paragraph. Using a spatial average metric, an average rheobase value of the mean perception threshold was reported at 25 T/s in one study involving 65 subjects (Hebrank [B592]) and at 28.8 T/s in another study involving 84 subjects (Nyenhuis et al. [B1089]).

Cardiac excitation thresholds using magnetic stimulation have been determined in dogs. Early results (Mouchawar et al. [B1045]; Yamaguchi et al. [B1529]) indicated dB/dt thresholds in excess of what would be predicted from the models used here (Table B.2 and Table B.3), although this could be explained by the use of suboptimum exposure conditions in the cited studies (Reilly [B1186]). More recent test results with dogs (Schaefer et al. [B1256]) conformed well with the models used in this standard when scaled from animal to human dimensions. It was also established that the addition of a 1.5 T static field to the time-varying excitatory field does not alter cardiac excitation thresholds (Bourland et al. [B188]).

With consideration of theoretical and experimental data, this standard adopts as median thresholds the peak dB/dt (denoted as $\dot{B}$) values listed in Table B.3 and Annex B of IEEE Std C95.6-2002 [B671] describes the methods whereby the external field $\dot{B}$ thresholds of Table B.3 are derived from the *in situ* parameters of Table B.1.

Thresholds are computed from the parameters of Table B.3, and as shown in Equation (B.6) and Equation (B.7) as follows:

$$\dot{B} = \dot{B}_0 \quad \text{for } t_p \geq \tau_e \tag{B.6}$$

$$\dot{B} = \dot{B}_0 \left(\tau_e / t_p\right) \quad \text{for } t_p \leq \tau_e \tag{B.7}$$

where t_p is the phase duration of the $\dot{B}_0$ waveform.

Alternatively, the limits can be determined as shown in Equation (B.8) and Equation (B.9).

$$\dot{B} = \dot{B}_0 \quad \text{for } f \leq f_e \tag{B.8}$$

$$\dot{B} = \dot{B}_0 \left(f / f_e\right) \quad \text{for } f \geq f_e \tag{B.9}$$

Magnetic flux density B listed in Table B.4 can be computed from the Table B.3 criteria using the relationships for sinusoidal fields shown in Equation (B.10) and Equation (B.11).

$$\dot{B} = \dot{B}_0 \Big/ \left(2\pi f_e\right) \tag{B.10}$$

$$B_0(\text{rms}) = B_0\left(\text{peak}\right) \Big/ \left(\sqrt{2}\right) \tag{B.11}$$

where

$\dot{B}_0$ is the minimum (rheobase) threshold value of dB/dt

B_0 is the minimum threshold value of B

Median magnetic flux density thresholds are computed from Table B.4 and from Equation (B.12) and Equation (B.13) as

$$B = B_0 \quad \text{for } f \geq f_e \tag{B.12}$$

$$B = B_0 \left(f_e / f\right) \quad \text{for } f \leq f_e \tag{B.13}$$

Considering the procedures discussed, it is apparent that the magnetic flux density limits in Table B.4 are based on the assumed *in situ* limits of Table B.2 (see B.2.1.2.1) evaluated at the site of interaction. For instance, the brain exposure limits are based on the estimated field induced in the outer perimeter of the cerebral cortex; cardiac excitation applies to the field induced in the apex of the heart; and peripheral nerve limits are based on the maximum induced field in the periphery of the torso.

Table B.3—Models for established magnetic *dB*/*dt* thresholds of reaction—Whole-body exposure; median thresholds

Reaction	$\dot{B}_0$ peak (T/s)[a]	τ_e (ms)	f_e (Hz)
Synapse activity alteration,	1.45	25.0	20
10 μm nerve excitation, brain	237	0.149	3350
20 μm nerve excitation, body	37.5	0.149	3350
Cardiac excitation	93.0	3.00	167

[a] $\dot{B}_0$ peak refers to the temporal peak of the time rate of change of magnetic flux density.

Table B.4—Median magnetic flux density thresholds—Whole-body exposure[a]

Reaction	B_0 rms (mT)	H_0 rms (A/m)	f_e (Hz)
Synapse activity alteration, brain	8.14	6.48×10^3	20
10 μm nerve excitation, brain	7.97	6.34×10^3	3350
20 μm nerve excitation, body	1.27	1.00×10^3	3350
Cardiac excitation	62.7	4.99×10^4	167

[a] Interpretation of table as follows: $B = B_0$ for $f \geq f_e$; $B = B_0 (f_e / f)$ for $f \leq f_e$.

B.2.1.5 Static or quasi-static magnetic field exposure

Whereas Equation (B.13) indicates that magnetic flux density thresholds would increase to infinity as the frequency approaches zero, an upper limit on magnetic flux density is required to avoid adverse effects from magnetohydrodynamic forces on moving charges within a magnetic field. Such movement is typically associated with the vascular system, although observable effects can also result from the rapid movement of the head, body, or eyes within a strong static field. The physical effects are Hall Effect voltages or Lorentz forces.

With static magnetic fields, reactions under laboratory conditions include a 17 % increase in human cardiac cycle length at 2 T (Jehesen et al. [B708]). The authors opined that the observed effect is probably harmless in healthy subjects, but that its safety in dysrhythmic patients was not certain. Other observations included a 0.2 % to 3 % change in blood velocity between 1 T and 10 T (Dorfman [B368]; Keltner et al. [B745]). A host of effects, some adverse, were noted at 1.5 T, including: vertigo, difficulty with balance, nausea, headaches, numbness and tingling, phosphenes, and unusual taste sensations. Much more marked reactions were noted at 4 T (Schenck et al. [B1259]). Other effects include benign enhancement of the cardiac T wave in rats at 4 T (Gaffey and Tenforde [B474]; Tenforde et al. [B1398]).

The studies of Schenck and colleagues report adverse effects in a significant number of subjects at 1.5 T, which this standard adopts as a median threshold for adverse effects. A peak value of 1.5 T is associated with a slowly varying sinusoidal field of 1.06 T rms. A statistical model has been assumed for the distribution of thresholds that follows the same lognormal distribution found in other electrical thresholds (see B.2.1.9). Consequently, at a factor of 3 below the median, namely, 353 mT [the value listed in Table 2 (see 4.2.2.1) for the lowest frequencies], the adversely affected population of sensitive individuals is estimated to be less than 1 % of exposed individuals. For the individuals in unrestricted environments, this standard applies an additional safety factor of 3, which leads to the value of 118 mT (as listed in Table 2).

B.2.1.6 Nonsinusoidal or pulsed fields

The DRLs and ERLs levels in Table 1 (see 4.2.1) through Table 4 (see 4.2.3.1) are expressed as a function of frequency assuming a sinusoidal exposure waveform. In many practical situations, however, the applicable waveform might not be sinusoidal, such as with a waveform having harmonic distortion or with pulsed waveforms. Subclause 4.2.2.4 expresses tests for determining the compliance of a nonsinusoidal

waveform (pulsed or mixed frequency) based on previous studies (Reilly [B1183]; Reilly and Diamant [B1189]). One of these tests is required to be met in addition to satisfying the rms limits of Table 1 or Table 2 (see 4.2.2.1). Consequently, instantaneous, rather than time-averaged values are required to demonstrate compliance with acceptance criteria for pulsed or nonsinusoidal waveforms.

The criteria in 4.2.2.4.2 are based on the temporal peak and phase duration of either the *in situ* field (or contact current) or the derivative of the environmental field. Alternatively, Equation (2) in 4.2.2.4.3 uses Fourier components of the test waveform. Since criteria in either subclause are conservative, either may be used to test for compliance. The choice could be dictated by the relative ease of obtaining the requisite data to implement the test (Fourier components versus temporal peak and phase duration). The method based on Fourier components does not account for the phases of the individual components. Essentially, the method assumes a worst case in which the individual components are phased such that the peaks align. This assumption ensures a conservative test, regardless of waveform. Note that linear-systems analysis methods are not reliable when applied to the highly nonlinear electrodynamics of the excitable membrane.

In some cases, the compliance tests can be overly conservative. Such cases can occur when the waveform appears as a low-frequency wave on which is superimposed a short-duration impulse. The degree of conservatism would increase as the impulse becomes shorter in duration and greater in amplitude. A more precise test would require evaluation of the threshold of a specific waveform with a neural excitation model, such as the one used in the cited study (Reilly and Diamant [B1189], [B1190]). This standard permits such analysis for compliance testing of nonsinusoidal waveforms.

B.2.1.7 Exposure to environmental fields

Because environmental electric fields induce *in situ* electric fields and body currents, it might seem logical to conclude that the induced field should be limited so as to preclude direct electrostimulation effects. In practice, however, contact current and spark discharge criteria (indirect electrostimulation) limit environmental electric fields to values significantly lower than what is required to directly induce *in situ* electric fields at the levels in Table 1 (see 4.2.1) and Table B.2 (see B.2.1.2.1). For example, the DRL for the *in situ* electric field in the brain is 17.7 mV/m at 60 Hz for persons in unrestricted environments (Table 1). To induce this field in a grounded, standing person would require an environmental field of ~59 kV/m (Carstensen [B237]). Considering that the unperturbed field is enhanced at body surfaces—18 times, for example, on the head of a standing person (Kaune [B739]), and even greater enhancements are possible on extended fingertips—parts of the body could be in a state of corona at environmental field levels necessary to induce the cited electric field strength within the brain.

Indirect stimulation effects occur through charge transfer between a person and a conducting object within the field. With sufficiently strong fields, an individual can perceive spark discharges just prior to the moment of direct contact and just after breaking contact with conducting objects that are well insulated from ground. It is also possible to perceive current through direct contact with such objects.

The contact current component I_c for a standing person touching a grounded conductor in a vertically polarized electric field is (Reilly [B1179]) as in Equation (B.14).

$$I_c = \left(9.0 \times 10^{-11}\right) \times h^2 \times f \times E \tag{B.14}$$

where

h is the height of the person
f is the frequency of the field
E is the environmental field strength

For fields at frequencies below 5 MHz, in which the environmental field magnitude varies over the volume that would be occupied by the body, the field strength in Equation (B.14) may be replaced with the average environmental field over the volume in which the body is placed (Deno and Zaffanella [B341]; Kaune [B739]).

Exposure limits on environmental electric fields in Table 4 (see 4.2.3.1) are intended to avoid aversive or painful contact currents or spark discharges when a standing person touches a conductive path to ground. In this instance, the individual is the induction object if that person is insulated from ground (e.g., rubber sole shoes or standing on an insulated surface). The limits in Table 4 do not apply to grounded individuals from adverse electrostimulation when touching large conductive objects that are insulated from ground.

The field limitations in Table 4 that provide protection against adverse contact current vary in inverse proportion to frequency. If this law were extended to zero frequency, the electric field limit would approach infinity. An upper limit is placed on the maximum permissible electric field strength to limit the probability of an adverse reaction to a spark discharge.

The maximum permissible field in Table 4 is 5 kV/m for persons in unrestricted environments. It is estimated that spark discharges would be painful to approximately 7 % of adults who are well insulated and who touch a grounded object within a 5 kV/m field. This statement was justified by (Reilly [B1179]) per the following parameters:

— Person's capacitance, $C_0 = 120$ pF (Table 9.1 in Reilly [B1179]).

— Median perception voltage (V_0) in ac field at 120 pF = 1.2 kV rms (Fig. 9.9 in Reilly [B1179]).

— Median pain multiplier = 2.9 (Table 9.5 in Reilly [B1179]), giving median pain threshold = $2.9 \times 1.2 = 3.5$ kV.

— Voltage on a 1.8 m-tall person in a field of 5 kV/m = 1.7 kV rms [apply Equation (B.9) and Equation (B.13)].

— The percentile of subjects feeling pain ≈ 5 % (lognormal distribution with $X_{50}/X_i = 3$ for fingertip perception (Reilly and Diamant [B1188]), where X_i is the statistical variate at the ith probability rank.

Unpleasant spark discharges can also occur when a grounded person touches a large conductive object that is well insulated from ground situated within a strong field. It is not possible to protect against all possibility of adverse stimulation without mitigating the induced charge on the object when very large (or long) objects are situated near sources that produce electric fields that are very extended spatially, such as is the case with high-voltage power transmission lines. For instance, one might postulate a long fence wire on insulated posts running parallel to a high-voltage transmission line. In such cases, it is preferable to restrict electrostimulation by properly grounding the conducting object (as stated in other safety codes), rather than by limiting the electric field to an impractically small level.

In restricted environments where the ERL is limited to 20 kV/m, painful spark discharges, but not contact currents, can be readily encountered at the stated limit for an insulated person at ground level touching a grounded conductive object. In such strong fields, workers should limit the probability of painful spark discharges by appropriate use of protective clothing, grounding measures, contacting techniques, or other work practices that consider these environmental electric field effects. In the restricted environments, conductive suits can be worn that shield the body from high environmental electric fields, thereby greatly reducing indirect electrostimulation. Currents conducted to the body of individuals wearing protective clothing should not exceed those in Table 1, Table 12, and Table 13 (see 4.4.1.1). Currents conducted to the body of individuals wearing protective clothing shall not exceed the ERLs specified in Table 12, Table 13, and Table 14 (see 4.4.2.1).

Power line rights-of-way fall somewhere between the definitions of "unrestricted" and "restricted" environments in that activity of the general public can be circumscribed by the utility but that access available to the general public is often allowed for recreational (biking, jogging) or commercial (beekeeping) activities.

Consequently, this standard specifies a limit of 5 kV/m for the general public (equivalent to persons in unrestricted environments) in regions off the right-of-way, but it allows an intermediate field of 10 kV/m within the right-of-way when the line is operating under normal conditions. (If the powerline right-of-way conforms to the requirements of a restricted environment, then the restricted environment limits apply.) Experimental data using spark discharge stimuli on human subjects (Reilly [B1179]; Reilly and Larkin [B1193]) can be applied to this exposure. In a field of 10 kV/m, ~50 % of adult subjects (1.8 m tall) who are well insulated from ground would experience painful discharges when contacting a grounded conductor. The stated probability would increase with taller subjects and decrease with shorter ones. It is also decreased by imperfect insulation of the person with respect to ground. Also, see Bracken et al. [B191] for an analysis of spark discharge exposures of workers engaged in tasks in transmission towers.

Maximum electric fields permitted within and off power transmission line rights-of-way are subject to limitation from other agencies or requirements, such as the U.S. National Electrical Safety Code and other electric utility regulations. The National Electrical Safety Code® (NESC®) [B665] specifies a safety limit of 5 mA short circuit current (i.e., the current into a low-impedance connection to earth) from objects within the electric field of a high-voltage transmission line. The intent of this provision is to limit contact currents to the "let-go" level of a few percent of sensitive children under worst-case conditions, rather than to avoid aversive or painful perception of contact current or spark discharges.

In the absence of indirect stimulation, environmental electric fields can sometimes be perceived through vibration of body hair caused by the interaction of the field and charged hair follicles. With a sufficiently strong field, the sensation can be annoying to some people. For instance, at 20 kV/m in an outdoor environment, 50 % of standing adults can perceive a 60 Hz field, and an estimated 5 % consider the sensation annoying (Deno and Zaffanella [B341]; Reilly [B1182], pp. 353–358). Although 20 % of subjects perceived a 60 Hz electric field at 9 kV/m, less than 5 % could detect electric fields of 2 kV/m or 3 kV/m (Reilly [B1182]). With hands raised above the body, the median perception threshold is 7 kV/m.

When an exposed individual is not within reach of a grounded conducting object, such as with a live power line worker in an insulated bucket, the maximum exposure limits in Table 4 might not apply. In such cases, the magnitude of contact current and spark discharges is determined by the potential difference between the individual and the touched object and their capacitances. This standard recommends adherence to the limits of Table 4 for the general public; however, the limits of Table 4 may be exceeded for persons permitted in restricted environments in which workers are not within reach of grounded conducting objects. This standard does not have a specific recommendation at this time for this situation. Regardless of the size and proximity of conducting objects that could be touched by the exposed individual, an absolute upper limit on acceptable exposure is determined by the need to prevent corona on body surfaces. It is unlikely that exposures in excess of 30 kV/m (unperturbed field) would be acceptable on any exposed body part.

B.2.1.8 Static or quasi-static electric fields

The maximum permissible environmental electric field has been capped to limit the probability of painful spark discharges. This limit could, in principle, be extended to arbitrarily low frequencies since even a single discharge can be painful. However, at a sufficiently low frequency, the time constant τ_e at which a human can maintain a charge begins to limit the magnitude of the induced charge. The time constant is given by the product of the person's capacitance and resistance to ground. For example, a resistance of 1000 MΩ (exceeded by 10 % of people with normal footwear on dry ground as assumed by Reilly [B1178], [B1179]), and a capacitance of 150 pF—a typical value of a person standing on a ground plane—result in a time constant of 150 ms, which is equivalent to a frequency of 1 Hz below which the induced voltage in a given field would fall, and the permissible exposure could rise. However, for people on well-insulated surfaces, longer time constants would be possible. The validity of this observation is apparent considering that one can experience an unpleasant carpet spark 1 s or more after the charge has been acquired.

These observations are applicable to the standards of Table 4 (see 4.2.3.1) as follows. For leakage resistance of 1000 MΩ, the allowable maximum limits below 1 Hz could be increased approximately in inverse proportion to frequency; for greater resistances, the applicable frequency would become lower.

B.2.1.9 Statistical variations in thresholds of reaction

Large variations in electrical thresholds are observed from one person to another. The statistical distribution of electrical reaction thresholds is typically represented by a lognormal distribution (i.e., one in which the logarithm of a statistical variate has a normal distribution). The mean of a lognormal distribution always exceeds the median. The mean-to-median ratio, ρ, is expressed as (Hastings and Peacock [B578]) in Equation (B.15).

$$\rho = \exp\left(\sigma^2/2\right) \tag{B.15}$$

where σ is the variance of the natural logarithm of the statistical variate.

The lognormal distribution provides a very good fit to statistical electrostimulation thresholds from a broad range of human and animal experiments, although sometimes original data needs to be re-plotted to demonstrate that fact. The lognormal distribution is found in human perception of contact current (Larkin et al. [B834]); bovine perception of contact current (Reinemann et al. [B1194]); human "let-go" thresholds (Dalziel [B309]); human perception of electric fields (Larkin et al. [B834]); human perception of pain from time-varying magnetic fields (Nyenhuis et al. [B1089]); human electroconvulsive therapy (ECT) seizure thresholds (Weaver and Williams [B1485]) and cardiac VF thresholds in dogs (Reilly [B1179]).

A lognormal slope can be expressed as the ratio of the median to the one-percentile thresholds (X_{50}/X_i). Lognormal slope parameters are summarized in Table B.5. It can be seen that a slope parameter of $X_{50}/X_i = 2$ represents a typical condition, although a more conservative value of 3 (applicable to forearm current perception) is used in this standard. For a distribution in which the ratio $X_{50}/X_i = 3$, the mean-to-median ratio is 1.12 (i.e., the mean exceeds the median by 12 %). This relationship is useful in cases where an experimental mean is given rather than a median.

Exposure limits are set such that the DRLs for each specified organ/body-area are all met. The controlling DRL depends on the exposure situation. With whole-body exposure, the ERLs are specified to avoid exceeding a DRL in the most sensitive region of the body. As seen in Figure B.5, the above would be satisfied by the ERLs for both upper and lower tiers. This would include the synapse effects at frequencies below about 750 Hz, and the PNS (20 μm nerve pain in Figure B.3) above that. If, however, the exposure excludes the head, then the DRL based on PNS would dominate at frequencies both above and below 750 Hz, thereby allowing greater exposure levels at frequencies below 750 Hz compared with the scenario with head included.

Investigators Leitgeb and Schröttner ([B856], [B857] also found good fits to the lognormal distribution of perception thresholds among a large sample of adult human subjects.[33] Some were visitors to product fairs; others were tested in their homes. Subjects received 50 Hz stimulation on the forearm. These data exhibited exceptionally large variance as compared with the other studies listed in Table B.5. The size of the experimental variance is especially striking considering that the authors separately reported data for males and females, which should reduce the variance as compared with a combined population. ICES suggests that such atypical data should prompt a critical examination of the experimental process used to derive them.

The SENN model returns a threshold of 6.15 V/m peak for a 20 μm fiber excited by a uniform electric field. That threshold appears to be consistent with a median of experimental thresholds. Based on experimental lognormal slopes in Table B.5, ICES makes the conservative assumption that a one-percentile pain reaction threshold for healthy adults would be a factor of 3 below the median, resulting in a rheobase of 2.97 ($6.15 \times 1.45/3$) V/m.

Column F of Table B.7 lists upper tier safety factors SF_U based on the assumption $R_{50,1} = 3$ for the listed responses. ICES reasoned that this approach was conservative: if $R_{50,1}$ were less than 3, Figure B.4 suggests

[33] This section has been adapted from pp.111–114 of Reilly and Diamant [B1188].

that the fraction of the population predicted to experience an adverse reaction would drop precipitously below 1 %.

Table B.6 provides examples of lognormal models (medians normalized to 1.0) applicable to sensory stimulation of the forearm of healthy adult humans, and to VF in healthy dogs (Reilly [B1179]). Experimental data for fingertip perception more closely follow the VF values. Compared with data from healthy animals, a much broader distribution of VF thresholds has been reported for direct electrode contact to the hearts of human patients undergoing open-heart surgery for valve replacement (Watson et al. [B1481]). Thresholds for persons in a pathological state or under drug treatment have not been otherwise tested.

It is tempting to extrapolate the distribution model of Figure B.4 to arbitrarily small percentile ranks. However, experimental evidence is insufficient to support extrapolation much below the rank of about 1 % due to limitations in the numbers of subjects represented in available experimental data. This standard adopts a factor of three to convert median thresholds into a sensitive individual. This would typically encompass 1 % of most sensitive individuals, but generally a much smaller percentile would be affected for most reactions treated in this standard. With a lower tier safety factor $SF_L = 9$ applied to the putative median, the percentile of people affected would be negligible, but a numerical probability level cannot be confidently assigned due to limitations in numbers of test subjects in reported literature.

Variations in thresholds from one individual to another are not well understood. The only significant physiological parameter that has been correlated with electrical thresholds is body size and related parameters, such as gender and age (Dalziel [B309]; Larkin et al. [B834]; Reilly [B1179], [B1193]). The correlation is such that small individuals tend to have lower thresholds. A body size relationship is found in sensory reactions, let-go thresholds, and ventricular fibrillation.

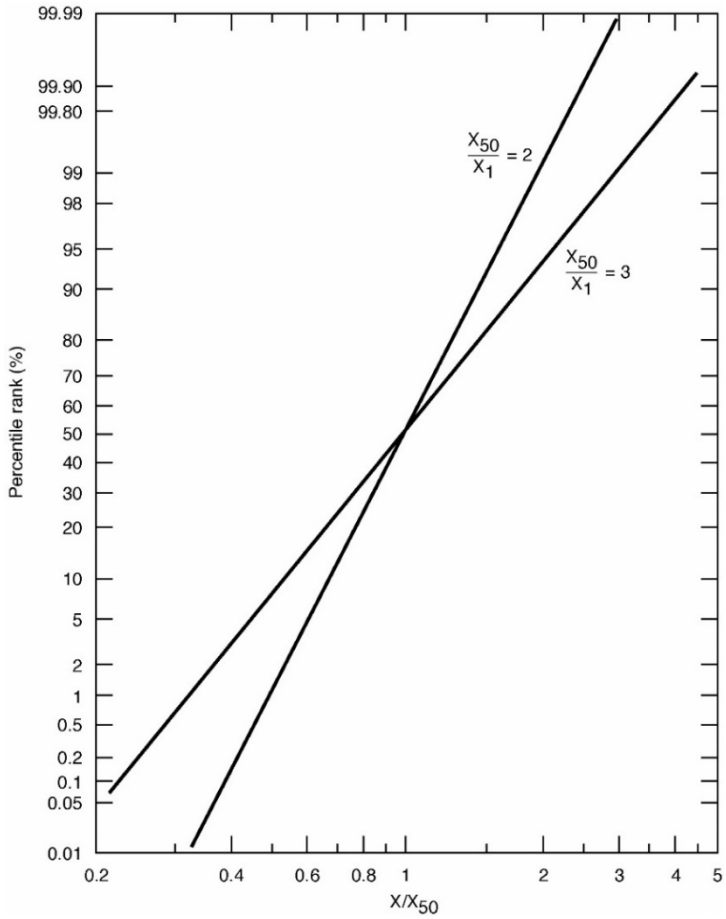
Experimental evidence indicates that thresholds of pain in humans and VF thresholds in animals vary approximately with the square-root of body weight, although other relationships have been proposed (Reilly [B1179]). Let-go thresholds in humans vary approximately in proportion to body weight. Consequently, small individuals, especially children, would be most susceptible to electrical stimulation effects. On the other hand, the magnitude of current induced by electric or magnetic fields diminishes with decreasing subject size. And with contact current, the small individual typically has a greater inter-limb resistance than does a larger person. Because of these compensating factors, the effect of body size on perception thresholds is not expected to be great. Indeed, a study of the relationship between magnetic field perception thresholds and morphological factors (subject gender, girth, weight, and age) demonstrated a lack of significant correlation with any of these factors (Nyenhuis et al. [B1089]).

Subclause B.2.2.2.3 provides an example of the application of the lognormal statistical model.

Table B.5—Experimental lognormal parameters for electrostimulation

Electrical reaction	Species	$R_{50,1}$ (X_{50}/X_1)	Citation
Forearm perception, contact current	human	3.0	(a)
Fingertip perception, contact current	human	2.0	(a)
Perception & pain, time-varying magnetic fields	human	1.9	(b)
Electroconvulsive therapy seizure	human	2.0	(c)
Contact current perception	bovine	2.3	(d)
Ventricular fibrillation	canine	2.0	(e)
Forearm perception (see text)	human	5.0 (M) 7.7 (F)	(f)
NOTE—$R_{50,1}$ applies to curve fit between 50 % and 1 % ranks.			

Citations: (a) Larkin et al. [B834]; (b) Nyenhuis et al. [B1089]; (c) Weaver and Williams [B1485]; (d) Reinemann et al. [B1194]; (e) Dalziel [B310]; (f) Leitgeb and Schröttner [B856], [B857]. (Table adapted from Reilly and Diamant [B1188].)



[a] The vertical axis follows a standard normal model; the horizontal axis applies to the statistical variate normalized by its median. Lognormal distributions are represented as straight lines on this plotting format (from Reilly and Diamant [B1188]).

Figure B.4—Lognormal distributions[a]

Table B.6—Normalized distribution of electrical reaction thresholds using lognormal model for healthy adult population (male and female)[a,b]

Percentile rank (%)	Threshold multiplier perception and pain	Threshold multiplier ventricular fibrillation
99.5	3.45	2.33
99.0	3.11	2.14
95.0	2.24	1.67
90.0	1.85	1.51
75.0	1.40	1.24
50.0	1.00	1.00
25.0	0.72	0.80
10.0	0.54	0.66
5.0	0.45	0.60
1.0	0.32	0.47
0.5	0.29	0.43

[a] Perception distribution based on human experimental data for arm contact. Ventricular fibrillation distributions from healthy dog hearts.

[b] *Source:* Reilly [B1179].

B.2.1.10 Acceptance criteria

B.2.1.10.1 Exposure reference levels

B.2.1.10.1.1 General

The DRLs listed in Table 1 (see 4.2.1) were derived from the median thresholds of Table B.2 by applying multipliers that convert from a median threshold of excitation into an adverse reaction threshold with small probability in healthy adults and with an adequate safety factor. Table B.7 summarizes factors used to derive the DRLs. The factors in each column are further described as follows:

a) Reaction: Adverse reaction used as a basis for deriving a DRL.

b) Locus: Location on the body where the reaction is manifest.

c) Median reaction threshold: The estimated level of exposure that produces a detectable response in 50 % of the population.

d) Adverse effect multiplier (F_A): Multiplier that converts a just detectable response (e.g., perception) to an adverse reaction threshold (e.g., pain or aversion).

e) Median Adverse Effect Threshold = (Column C) × (F_A).

f) Upper Tier Safety Factor *(SF_U)*: To be applied as a divisor to the median adverse reaction threshold to obtain the upper tier DRL.

g) DRL upper tier (V/m) = (Column E)/(SF_U): Understood to be an exposure level at which at most 1 % of exposed healthy subjects would experience adverse reaction (pain). The rheobase threshold of a strength-duration curve applicable to persons qualified to enter restricted environments (Table 1).

h) Lower Tier Safety Factor (SF_L): To be applied as a divisor to the median adverse reaction threshold to obtain the lower tier DRL.

i) DRL Lower Tier (V/m) = (Column E)/SF_L): The rheobase threshold of a strength-duration curve applicable to all persons in unrestricted environments.

DRLs listed in Table 1 (see 4.2.1) are in terms of *in situ* induced electric fields; the mode of induction, however, can be through the action of the environmental magnetic or electric field. In addition to induced electric field specifications, it is also necessary to restrict the *in situ* magnetic field to avoid adverse reactions due to magnetohydrodynamic effects from very low-frequency magnetic fields (see B.2.1.5). Table 1 specifies such restrictions below 10 Hz. It is not necessary to specify magnetic DRLs at greater frequencies because potential adverse effects would be related to the induced electric field, rather than the *in situ* magnetic field itself. The following paragraphs summarize the rationale for the multipliers appearing in Table B.7.

Table B.7—Factors converting median rheobase perception thresholds to DRLs at low frequencies (0 Hz to 100 kHz for CW exposures)

A Reaction	B Locus	C Median reaction threshold (V/m)	D Adverse effect factor multiplier (F_A)	E Median adverse effect threshold (V/m)	F Upper tier safety factor (divisor) (SF_U)	G DRL upper tier (V/m)	H Lower tier safety factor (divisor) (SF_L)	I DRL Lower tier (V/m)
Synapse modulation	Brain	0.053	1.0	0.053	3.00	1.77×10^{-2}	9.00	5.89×10^{-3}
10 μm neuron excite	Brain	8.70	1.0	8.70	3.00	2.90	9.00	0.970
20 μm neuron pain	Body	4.35 (percept.)	1.45 (pain)	6.31	3.00	2.10	9.00	0.700
20 μm neuron pain	Hands, feet, wrists, ankles	4.35 (percept.)	1.45 (pain)	6.31	3.00	2.10	3.00	2.10
Cardiac excitation	Heart apex	8.49	1.0	8.49	9.0	0.943	9.0	0.943

NOTE 1—The tabulated electric field strengths (columns C, E, G, and I) are rms rheobase values.

NOTE 2—Incorporation of rheobase DRLs into frequency relationships is given by formulas in footnote a of Table 1 (see 4.2.1).

NOTE 3—F_A is a multiplier. SF_U, and SF_L are divisors.

NOTE 4—Upper tier applies to persons qualified to enter restricted environments.

NOTE 5—Lower tier applies to all persons in unrestricted environments.

NOTE 6—With whole-body exposure, compliance with DRL as determined by ERL limits based on avoidance of 20 μm neuron pain would preclude reaching the DRL listed here for cardiac excitation by at least an additional factor of 2 [see Figure B.5 (in B.2.2.1)].

B.2.1.10.1.2 Adverse reaction factor

Pain is considered an adverse response with peripheral nerve excitation. An adverse reaction multiplier of $F_A = 1.45$ is applied to the nerve excitation threshold to derive a pain threshold (see B.2.1.3). With synaptic effects, brain stimulation, and cardiac excitation, excitation itself is considered adverse as noted in B.2.1.2.3 and B.2.1.2.4; hence, the adverse reaction multiplier of $F_A = 1.0$ is applied to the excitation threshold for these reactions.

B.2.1.10.1.3 Upper tier safety factor

The upper tier safety factor SF_U (divisor) is applied to convert from a median threshold into a small-probability one. For a lognormal distribution in which the slope parameter (X_{50}/X_1 = median-to-one-percentile ratio) is 3, the divisor of 3 applied to the median threshold corresponds to a one-percentile most sensitive subject. Whereas a slope parameter of 3 is observed in some cases (e.g., contact current perception on the forearm), with other reactions of critical application to this standard (magnetic field perception, cardiac VF, brain ECT thresholds), the slope parameter is very close to 2.0 (see B.2.1.9). With a slope parameter of 2, a divisor of 9 applied to the median threshold would result in a 0.01 % probability rank. Thus, the assumption that $(X_{50}/X_1) = 3$ is conservative for most cases for which we have data.

B.2.1.10.1.4 Lower tier safety factor

A lower tier safety factor (divisor) $SF_L = 9$ allows for protection of exceptionally sensitive individuals, uncertainties concerning threshold effects due to pathological conditions or drug treatment, uncertainties in the reaction thresholds, and uncertainties in the induction models. In the case of the hands, wrists, feet, and ankles, $SF_L = 3$ applies to persons permitted in restricted environments. Because these regions lack critical function when compared with the vital organs, a greater localized electric field is permitted. In the case of the restricted environment, $SF_L = 3$ for all of the reaction types except for cardiac excitation under the assumption that a small probability of discomfort is acceptable for persons permitted in restricted environments for some mechanisms, but that cardiac excitation is unacceptable for all individuals. The safety

factor $SF_L = 3$ can be justified for the indicated exposures because this standard is based on avoidance of short-term reactions that are immediately apparent to the exposed individual. It is assumed that because the short-term reactions are apparent to exposed individuals, they can remove themselves from the environment, modify their activities, or can take other action to avoid the exposure entirely.

B.2.1.10.2 Exposure reference levels

Sophisticated computational capabilities can sometimes be required to assess whether DRLs are met. Consequently, it is desirable to define ERLs that are reference levels in terms of the environmental field, rather than the induced *in situ* field. The ERLs listed in Table 2 (see 4.2.2.1) incorporate conservative assumptions such that adherence to them ensures that the basic restrictions are not exceeded. However, since the ERLs are conservatively derived, it is possible that one can exceed them and still be within the DRLs.

Figure B.5 illustrates the derivation of low-frequency ERLs levels for magnetic fields. The figure shows median thresholds of adverse reaction (broken lines) and ERLs (solid lines) with whole-body exposure. The ERLs are derived from the minimum adverse thresholds at each frequency, decremented by the appropriate safety factors in Table B.7. The curve for synapse alteration has been extended to 1000 Hz. The ERL curves have been derived from the lowest adverse reaction threshold across the frequency spectrum as follows: 0 Hz to 0.153 Hz, magnetohydrodynamic effects; 0.153 Hz to 759 Hz, synapse alteration above 759 Hz, peripheral nerve pain. Note that the ERLs in the restricted environment correspond to small-probability reaction thresholds (≤ 1 %). The limits applicable to persons in unrestricted environments are lower by a factor of three. Table 2 expresses the ERLs.

B.2.1.10.3 Nonadditive effects of environmental magnetic field and electric field exposures

For purposes of demonstrating compliance with this standard, Table 2 (see 4.2.2.1) and Table 4 (see 4.2.3.1) should be considered separately, and not additively. This is because the *in situ* electric field induced by environmental electric fields and magnetic fields are maximized in disparate regions of the body under the conditions represented in Table 2 and Table 4. With whole-body magnetic field exposure, the location in the body of the maximum induced *in situ* electric field strength is at the periphery of the torso and with whole-body electric field exposure is at the ankles. Induced fields at these locations do not coincide, and are therefore not additive.

B.2.2 Exposure

B.2.2.1 Whole-body and localized or nonuniform exposure

The limits of Table 2 (see 4.2.2.1) are designed to avoid adverse reactions with whole-body exposure to magnetic fields that are constant in magnitude and in relative phase over the entire body. Because the contribution of the *in situ* electric field within the head and torso due to exposure of the arms and legs is not great, the limits also apply to a constant field over only the head and torso. However, when a magnetic field is not constant over the head and torso, a conservative approach for magnetic fields would be to limit the spatial peak of the actual field in accordance with Table 2. It is possible that such an approach might be unduly restrictive. An acceptable alternative would be to limit the external magnetic field such that the *in situ* electric field strengths do not exceed the DRLs of Table 1 (see 4.2.1). To determine compliance with Table 1, it would be necessary to model the induction process using the actual field values (direction, magnitude, and relative phase), and an appropriate physiological model (computational or physical), along with the orientation of the model with respect to the direction of the field (e.g., see Bracken [B189], [B190], [B191]; Kavet et al. [B740]; Stuchly and Gandhi [B1358]). Note that separate limits are provided in Table 3 (see 4.2.2.2) for primary exposure of the limbs, absent significant exposure of the torso.

For situations where there is a significant disparity in magnetic field exposure of the head and torso, the magnetic field ERLs needed to meet the DRLs (Table 1) can change considerably. To illustrate this point, consider a 60 Hz field where only the torso is exposed versus one where both the head and the torso are exposed. If only the torso were exposed, the ERL would be limited by peripheral nerve stimulation rather than by brain synapse effects. For torso exposure, the upper tier ERL at 60 Hz would be 34.8 mT—approximately 13 times the limit of 2.71 mT for both head and torso exposure (Table 2).

The electric field ERLs in Table 4 (see 4.2.3.1) are not based on the *in situ* electric field limits of Table 1; rather, these limits are based on indirect electrostimulation. Spark discharge and contact currents are within levels this standard finds acceptable if the environmental electric field averaged over the dimensions of the body does not exceed the limits shown in Table 4. These limits are based on the assumptions that the exposed person is insulated from ground, is much closer to the ground than the field source, and is within reach of a grounded conducting object.

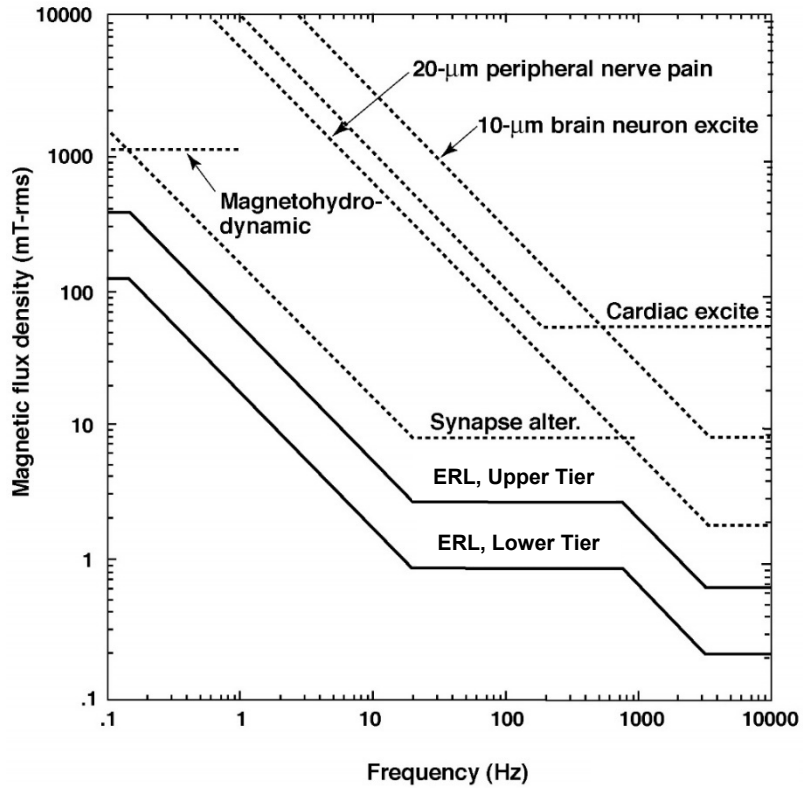


Figure B.5—Median thresholds for adverse stimulation from magnetic field exposure (broken lines) and ERLs (solid lines)—Whole-body exposure to spatially constant field

B.2.2.2 Induced and contact current

B.2.2.2.1 General relationships

S-D and S-F curves characterize thresholds of nerve stimulation for contact currents. The rheobase threshold value of current from a contact electrode varies inversely with the contact area. A touch contact area of 1 cm^2 is assumed for the area of a light fingertip contact, whereas a much larger contact area ($\cong$ 15 cm^2) can apply to a grasped contact. Consequently, separate values are cited in Table 12 and Table 13 (see 4.4.1.1) for grip and touch contacts. The grasping contact limit for persons permitted in restricted environments pertains where

personnel are trained to effect grasping contact and to avoid touch contacts with potentially energized conductors or grounded conductors when the person is the induction object. It is assumed that persons in unrestricted environments are not aware of the possibility of conducted current from energized objects, and the method of contact is unconstrained. Specified limits reduce the probability that inadvertent contact with energized objects could lead to tiny localized burns of the outer layer of skin (with spark discharges), painful sensations, or startle reactions that, while not hazardous *per se*, could conceivably lead to an accident.

Based on nonlinear nerve excitation models, S-D and S-F constants are related by $f_e = 1/(2\,\tau_e)$. Consequently, factors leading to small values of τ_e would increase f_e. Experimental values of f_e vary significantly, and although some factors accounting for this variation are explainable from theoretical models [B1188], not all are well understood. ICES assumes that f_e for contact current is 3 kHz, allowing extrapolation to lower frequencies from thresholds determined at higher frequencies using a slope of f with a minimum threshold at and below 3 kHz. Further research is needed to understand the variation of experimental constants observed in S-D and S-F curves.

B.2.2.2.2 DRLs for contact current

Footnote c of Table 1 states that DRLs do not apply to contact current or to induced current crossing skin-to-skin contacts with magnetic field exposure scenarios. The reasons for this exclusion are as follows:

a) In general, DRLs apply to *in situ* electric fields and ERLs to environmental fields. Instead, contact limits are a hybrid between these two types of measures. The contact current limits apply to current entering the skin and in this respect is analogous to a DRL. However, the contact current specification makes irrelevant the environmental field that is the source of that current. One might argue that the current limit itself could be either an ERL or a DRL.

b) In principle, one could use a detailed anatomical model to derive *in situ* electric field strengths surrounding cutaneous neurons affected by contact current. However, evaluation of the electric field strengths so derived are complicated by two factors that would apply both to contact current exposure or to induced current crossing skin-to-skin contacts under magnetic field exposure, such as might occur at the crotch, armpits, buttocks, or touching finger tips with extended arms (De Santis et al. [B347]).

    1) Conductivity of the different skin strata and subdermal adipose tissue differs considerably and depends significantly on hydration. Furthermore, the thickness of the skin layers can be well below the resolution typically used in DA models. These details can result in large values of *in situ* dE/dx, which might affect the excitability of the cutaneous receptors that most like are stimulated by current flowing across skin strata. Note that the DRLs in the standard are derived by assuming that dE/dx along the neural axon is negligible. (See Reilly and Diamant [B1188], ch. 4.)

    2) DA model methods for addressing high-resolution dE/dx effects in the dermis have not been established, nor have general criteria for neural excitation of cutaneous receptors been established.

c) Perception-to-pain multipliers (F_P) differ considerably for *in situ* electric fields induced via magnetic field exposure (applicable to Table 1 DRLs) versus multipliers for contact current exposure (see B.1.2.2).

B.2.2.2.3 Example of statistical relationships—Contact currents

Pain levels with touch contact can be extrapolated downward in frequency from Chatterjee et al. [B244] to a frequency of 3.0 kHz, which is the postulated corner frequency (above which there is a frequency-

proportional slope). At 10 kHz (the lowest frequency tested by Chatterjee et al.), the mean pain level is 8.0 mA for adults (males and females mixed) and 6.0 mA for 10-year-old children. Those values can be converted into median thresholds by dividing by the factor 1.12 as noted in B.2.1.9. The 10 kHz thresholds are extrapolated to a 3 kHz rheobase by applying the multiplier 0.3 (the ratio 3 kHz/10 kHz). The result is a median pain threshold of 2.14 mA for adults and of 1.6 mA for 10-year-old children. Using a discomfort-to-pain ratio of 0.7 for contact current (see B.2.1.3), the median discomfort rheobase level is estimated to be 1.5 mA for adults and to be 1.12 mA for children. Applying these median values to the lognormal model with a median-to-one-percentile ratio of 3.0, the following reaction probabilities are determined. At a touch contact level of 0.5 mA (the ERL for the general public) in children, the fraction of individuals predicted to experience discomfort is 5 %, and the fraction of individuals predicted to experience pain is 1 %. In adults: the fraction of individuals predicted to experience discomfort is 1 %, and the fraction of individuals predicted to experience pain is 0.1 %. At a touch contact current level of 1.5 mA in adults, the probability of pain is 23 % and the probability of discomfort is 50 %.

Current thresholds for perception and pain are considerably greater if contact is made with a grasping contact rather than a touch. A mean perception level for a grasping contact at 10 kHz is 13 mA for adults (Chatterjee et al. [B244]). Extrapolating to a frequency of 3 kHz, a median perception threshold of 3.48 mA is determined. The median discomfort or pain threshold is determined by applying the multipliers 2.4 and 1.7, respectively (see B.2.1.3), resulting in a median rheobase discomfort level at 5.92 mA and a pain level at 8.35 mA. At a grasping contact current of 3 mA [specified in Table 12 and Table 13 (see 4.4.1.1) for grasping contact ERLs for persons permitted in restricted environments], the probability of discomfort in adults is estimated at 8 % and the probability of pain at 1.6 %.

The upper tier contact current levels in Table 12 and Table 13 do not contain safety factors and the lower tier safety factor SF_L is 3. The reduced safety factors are justified by noting that the reaction levels for contact current are better understood and are more easily mitigated or avoided than are the other reaction thresholds addressed in this standard.

B.2.2.3 Additive effects of environmental magnetic field and electric field exposure

With whole-body magnetic field exposure, the location in the body of the maximum induced *in situ* electric field is at the periphery of the torso and with whole-body electric field exposure is at the ankles. These locations do not coincide, and therefore, any additive effect is inconsequential.

B.2.2.4 Medical devices and medical implants

Medical devices and metallic implants can involve special health and safety problems when the individual using them is exposed to electric and magnetic fields. This standard does not necessarily provide protection against interference with such devices or hardware. The recipient or provider of these devices should be aware of the potential for hazards and precautions that might be necessary with such devices.

Electronic medical devices can be susceptible to interference from many different sources of electrical energy. Interference with medical devices can occur in fields below those cited as thresholds for electrostimulation effects. While several types of medical devices have been designed for immunity to electrical interference (e.g., cardiac pacemakers), many devices in use have not been designed or tested for immunity. Even with reasonable immunity to interference, serious patient consequences can occur if the immune threshold is exceeded. Concerns for device interference extend to many electronic medical devices. Examples of such devices where there are concerns for interactions include, but are not limited to, pacemakers, defibrillators, drug delivery pumps, neurostimulators, hearing aids, apnea monitors, hospital beds, and powered wheelchairs. When deemed necessary, advice should be sought from the manufacturer of the device and/or from the patient’s medical practitioner.

A few standards address the electromagnetic compatibility (EMC) of medical devices and the device performance during exposure. The most widely recognized medical device standard published by the International Electrotechnical Commission (IEC 60601-1-2 [B652]) covers many, but not all, medical devices. There are also general standards for active implantable medical devices that contain EMC requirements (EN 45502-1 [B396]; IEC 60601-1-2 [B652]; International Organization for Standardization (ISO) 14708-1 [B689]). In addition, work is underway to update the IEC medical equipment EMC standard and to develop more consistent standards for pacemakers and implantable defibrillators that include EMC requirements, such as in the United States [Association for the Advancement of Medical Instrumentation (AAMI) TIR18:2010 [B1] and Europe (EN 45502-2-1 [B397]; EN 45502-2-2 [B398]).

Metallic implants comprise another class of medical implants, such as metallic stents, staples, and orthopedic rods and plates. In some cases, metallic implants can contact sensitive tissue, as with cardiac staples. Unlike electronic medical devices, such implants might not have a failure mode due to electrical interference. Nevertheless, metallic hardware implanted in the body can enhance induced electric fields either by providing a magnetic induction loop or a high-conductivity region that can locally enhance the induced electric field, and thereby enhance the possibility of electrical stimulation in localized regions near the implant (Reilly and Diamant [B1192]).

B.3 Rationale for limits based on heating (100 kHz to 300 GHz)

B.3.1 Risk profile for adverse effects

While developing IEEE Std C95.1-2005 [B668], the Risk Assessment Working Group (RAWG) had been particularly concerned about the lack of rigor in defining the safety factors used to derive the ERLs (called “MPEs” and “action levels” in IEEE Std C95.1-2005). 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 (nonthermal) exposures received extensive attention, discussion, and evaluation.

The risk profile in the following list 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 as follows. 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 are ranked as follows:

a) **RF shocks and burns:** These 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 amplitude-modulated (AM) radio broadcast antennas.

b) **Localized RF heating effects:** These are undeniably realistic hazards, but they occur much less commonly than do RF shocks and burns.[34]

c) **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 nonlethal 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.

d) **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 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."

e) **Microwave hearing effects:** These effects, while possible over a range of frequencies, are even rarer than items a) to d). The perception of a barely audible click, buzz, or hiss, from pulsed radar type signals in a very quiet environment, based on real-world exposures, is not adverse to health.

f) **Low-level effects:** Despite about 70 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.

B.3.2 Change in limits (6 GHz to 300 GHz)

B.3.2.1 General

The goal in updating the existing standards in this frequency range was to provide a consistent level of protection against thermal hazards below and above the transition frequency of 6 GHz. Below 6 GHz, the local exposure DRL is expressed in terms of psSAR in the body averaged over 10 g of tissue, while at frequencies above 6 GHz, the DRL is expressed in terms of epithelial power density at the tissue surface, averaged over a specified area. Several recent papers (e.g., Colombi et al. [B292]) have noted that current standards entail a large discrepancy in the levels of protection against thermal hazards as this transition is crossed.

There are only limited experimental human data on the heating of tissue by RF energy in the frequency range 6 GHz to 300 GHz that are useful for setting exposure guidelines. The current revisions are based on available data as well as on widely accepted modeling studies using a standard theoretical model for heating of tissue (the so-called "bioheat equation") as developed by several recent authors (Foster et al. [B432], [B433]; Morimoto et al. [B1038]; Sasaki et al. [B1251]).

The change in transition frequency from 3 GHz (IEEE Std C95.1-2005 [B668]; IEEE Std C95.1a-2010 [B668]; IEEE Std C95.1-2345-2014 [B669]) to 6 GHz (as in this standard) was based on the conclusion of

[34] 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).

ICES that SAR measurements or computations become progressively more difficult to perform at higher frequencies but are still practical up to 6 GHz. The choice of the transition frequency of 6 GHz was done for this reason and to harmonize with the new ICNIRP limits.

The ERL for whole-body exposure as well as ERL and DRL for local exposure in the frequency range 6 GHz to 300 GHz were chosen based on two considerations: (a) to maintain a similar total absorbed power in the body from whole-body exposures at the ERL below and above the transition frequency; and (b) to maintain a similar peak temperature increase in tissue produced by RF exposure across the transition. The fivefold ratio in exposure between unrestricted and restricted exposure conditions (i.e., a safety factor of 5) is maintained across the entire frequency range up to 300 GHz.

a) **Whole-body exposure [(**Table 7 and Table 8 (see 4.3.2)**].** The ERL from 6 GHz to 300 GHz is calculated to maintain a total absorbed power in the body approximately constant across the transition. The calculations assume a 100 kg individual exposed over a body surface area of 1 m^2, which would represent whole-body exposure from a RF source impinging on the body from one direction. No DRL is defined for whole-body exposure in this frequency range because it can be assumed that the radiating element producing such exposure is sufficiently distant from the body that the free-space power density can be used for the ERL to assess compliance.

b) **Local exposure [**Table 6 (see 4.3.1)**].** Definition of DRL from 6 GHz to 300 GHz is determined to maintain the same peak temperature increase at any location in the body that would be allowed by the DRL in current limits below 6 GHz. This is estimated based on thermal modeling by Hirata's group (e.g., Kodera et al. [B765]; Morimoto et al. [B1038]) in terms of the heating factor (which is the ratio of peak temperature increase in the body to spatially peak 10 g average SAR in the body).

Several computational modeling studies reviewed in Foster et al. [B432] show that in the frequency range 100 kHz to 6 GHz, the heating factor is approximately 0.25 °C/(W/kg), corresponding to a peak temperature increase of approximately 2.5 °C at the DRL below 6 GHz for restricted exposure conditions. To provide consistency, the same theory (based on Pennes' bioheat equation, [B1139]) was used to determine the ERL and DRL maximum increase in skin temperature at approximately the 2 °C to 3 °C over the entire frequency range (restricted exposure conditions). The analysis for exposures from 6 GHz to 300 GHz relies on modeling studies, such as Foster et al. [B432], Hashimoto et al. [B577], Laakso et al. [B801], and Sasaki et al. [B1252]. An additional safety factor of 5 was applied to obtain the ERL and DRL for unrestricted exposure conditions.

B.3.2.2 Short, high-intensity, millimeter-wave pulses (30 GHz to 300 GHz)

These limits are based on a thermal analysis that shows that exposure to brief (≤ 10 s) high-intensity pulses in the currently considered frequency range (30 GHz to 300 GHz) can produce thermal hazards (Foster et al. [B432]). Currently such exposures might be produced by some military nonlethal weapons (which are not in the purview of the present standard) but future sources of high-powered millimeter-wave (MMW) or terahertz energy for industrial applications might pose similar hazards. Fluence is the power density of the energy integrated over pulse time (i.e., the energy density of a pulse). It is anticipated that most such sources are sufficiently distant from the body that free-space power density can be used to assess compliance with these limits. The fluence should be averaged over a 1 cm^2 square.

B.3.2.3 Interface with ANSI Z136.1 (American National Standard for Safe Use of Lasers)

ANSI Z136.1 [B69] provides that for large area exposures to the skin (at wavelengths > 1400 nm), the exposure limit is as follows:

— 1000 W/m^2 for beam area of 100 cm^2

— 100 W/m^2 for beam area 1000 cm^2 or more

For exposure areas of 1000 cm^2 or higher, the ERL for the restricted environment of this standard agrees with ANSI Z136.1 [B69] at the frequency (300 GHz) where the two standards meet. For small beam areas (< 100 cm^2), the limits in ANSI Z136.1 are considerably higher than the present RF exposure standard, and the concept of averaging area is implemented differently in the present (IEEE Std C95.1) and laser (ANSI Z136.1) standards. This difference reflects in part the technological differences in the exposure sources (e.g., low-gain RF transmitters versus lasers with narrow beamwidth) and in part a lower degree of conservatism in ANSI Z136.1 versus this standard.

This standard also coincides at 300 GHz with ICNIRP limits for far infrared energy (ICNIRP [B648]). The rationale for that standard, which is principally designed to protect against thermal hazards in the far infrared band of the spectrum, is extensively discussed in ICNIRP [B647]. ICES could find no later scientific reports that would materially affect the major conclusions of the ICNIRP [B647] scientific review.

B.3.2.4 Thermal basis of limits

The goal of providing consistency in level of protection against thermal hazards above and below the transition frequency (6 GHz) was achieved based on thermal modeling using the same underlying theory (Pennes' bioheat equation, [B1139]). The limiting thermal hazard from RF energy with short penetration depth (which is the case between 6 GHz and 300 GHz) is thermal pain from skin. As described in ICNIRP [B647], exposure at the threshold for thermal pain from extended (> 500 s) exposure to far infrared radiation is approximately 800 W/m^2. ICNIRP [B647] concludes that "a very conservative irradiance guideline for continuous exposure is provided by the whole-body IR-C laser irradiation limit of 100 W/m^2, and this limit, while overly conservative for cooler environments, would preclude delayed effects such as *erythema ab igne*" (i.e., "hot water bottle rash").

Based on thermal modeling (Sasaki et al. [B1251]), the DRL for local exposure would result in a steady-state temperature increase at the surface of the skin of approximately 2 °C to 3 °C in restricted environments. While it is not possible to calculate the steady-state temperature increase precisely because of the many biological and environmental variables, it is anticipated that continuous (10 min or more) exposure to the body at limits for restricted exposures results in peak temperature increases of this order. This is approximately a factor of 3 to 5 below the threshold for thermal pain (approximately 44 °C) assuming skin temperature of 34 °C under ordinary room ambient conditions. The limits (ERL and DRL) increase as the frequency is reduced to 6 GHz, in part reflecting the smaller transmittance into tissue at lower frequencies, and in part the deeper penetration of energy into tissue.

Consequently, it is concluded that, based on current data and analysis, as well as on previous analysis of far infrared safety limits, the present limits are conservative against thermal hazards including thermal pain from RF heating of skin.

It is noted by ICES that there is scant experimental data for heating of skin by RF energy in the frequency range 6 GHz to 300 GHz, and most available studies involve exposure circumstances (small exposure areas or brief pulses of high intensity) that might not be representative of all realistic exposure scenarios (Foster et al. [B432], [B433]). Consequently, the present standard has been designed using a combination of modeling studies with experimental evidence. In view of the foreseeable increase in use of the RF spectrum between 6 GHz and 300 GHz, more experimental studies are clearly needed.

While there have been reports of "nonthermal" (not heating related) effects of RF energy in the frequency range 6 GHz to 300 GHz, ICES does not conclude that sufficient evidence exists to develop exposure limits based on such effects. In 2018, the World Health Organization reported that it was undertaking a comprehensive review of the biological effects of RF energy in this frequency range.[35] In addition, the scientific literature and expert reviews by health agencies around the world are being monitored by

[35] See footnote 30.

IEEE ICES. The scientific bases for the recommended limits are evolutionary and subject to revision as new data and analyses become available.

B.3.3 Spatial peak power density ERLs for local exposure of any part of the body

The previous standard, for local exposure in unrestricted environments, stated that the spatial maximum value of the power density or mean squared field strength should not exceed 20 times the square of the allowed spatially averaged values in Table 7 and Table 8 (see 4.3.2) at frequencies below 400 MHz; should not exceed the equivalent power density of 40 W/m^2 at frequencies between 400 MHz and 6 GHz; should not exceed 20 times the square of the allowed spatially averaged values in Table 8 at frequencies below 400 MHz, and should not exceed the equivalent power density of 200 W/m^2 at frequencies between 400 MHz and 6 GHz.

Analysis of Findlay and Dimbylow [B416] data shows that the previously specified local exposure ERLs for both upper and lower tiers do not necessarily protect against exceeding the underlying DRL in all cases of exposure. An important observation from this analysis is that a multiplier of 20 is excessive if the local DRL is to be maintained during exposure. This multiplier could be as low as about 5 at 2000 MHz or as high as 19 at 200 MHz, both values being smaller than the multiplier of 20 defined for frequencies below 300 MHz for the restricted environment in IEEE Std C95.1-2005 [B668].

The multipliers shown in Figure B.6 are derived from an analysis of data contained in Findlay and Dimbylow [B416], which gave calculated values for maximum SARs associated with near-field exposures to half-wave dipole antennas at 65 MHz, 100 MHz, 200 MHz, 600 MHz, 1000 MHz, and 2000 MHz positioned at three distances from the body and at three different heights. For frequencies above 2 GHz, the multiplier is set at 4 so that the ERLs in Table 9 and Table 10 (see 4.3.3.1) match the 6 GHz ERLs in Table 11 (see 4.3.3.2).

These results should be put in context in that they are for a limited number of static exposure scenarios with varying degrees of near-field exposure and only over six frequencies. Nonetheless, the results suggest caution in setting local exposure values that are simple multiples of the WBA ERLs for limiting psSARs to comply with specified local DRLs.

Users of the ERLs for local exposure should be aware of these limitations. Furthermore, pending further analysis of these results and future published research relevant to this topic, the more conservative ERL values specified in Table 9 and Table 10 are recommended to make it likely that compliance with the ERL supports compliance with the corresponding DRL.

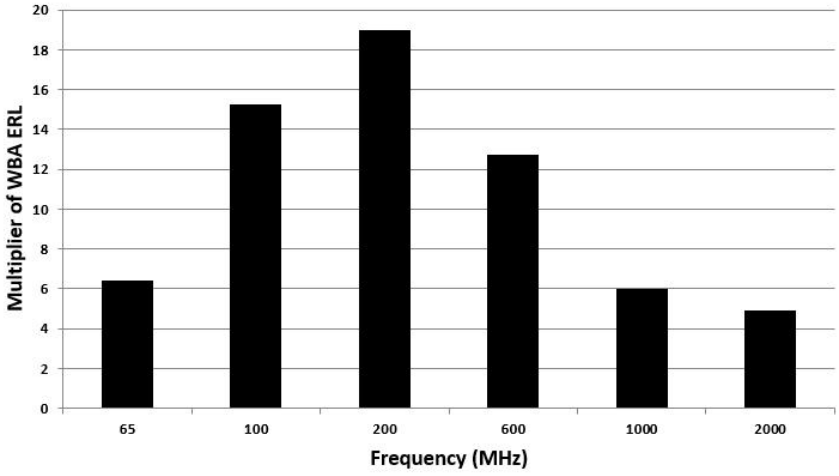


Figure B.6—Factor by which the WBA ERL should be multiplied to provide local SAR equivalent to the local DRL

B.4 DRL and ERL

B.4.1 DRLs

B.4.1.1 DRLs for whole-body exposure—100 kHz to 6 GHz

DRLs for frequencies between 100 kHz and 6 GHz are expressed in terms of the SAR (see Table 5 in 4.3.1). 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 DRLs of both Table 1 (see 4.2.1) and Table 5 should be applied.

The weight of the scientific evidence continues to support the determination made in ANSI C95.1-1982 [B68] and IEEE Std C95.1-2005 [B668] 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 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 after long-term exposure up to two years (lifetime exposure). The weight of the scientific evidence is based on the literature described in Annex B of IEEE Std C95.1-2005 (C.2 to C.7 in this standard); reviews of literature above 6 GHz are summarized in C.8.

The lower tier DRL of this standard (0.08 W/kg) is accepted for all persons in unrestricted environments, and 0.4 W/kg is established as the DRL for persons permitted in restricted environments, where access is limited to persons who are informed about procedures to prevent overexposure. Since publication of ANSI C95.1-1982 [B68], 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 ERLs 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 database, these ERLs are presented as upper limits of exposure.

Arguments supporting the lower tier include:

a) Two tiers of protection have been traditionally established in the major health and safety standards for RF exposure.

b) A lower tier DRL, applicable to all persons, serves as a safety program initiation level.

c) Exposure standards such as this one 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.

In IEEE Std C95.1-2005 [B668], there was a change in the frequency range over which WBA SAR is deemed to be the DRL for frequencies less than 3 GHz, and 3 GHz to 6 GHz as a transition for compliance purposes to either use SAR or power density. In this standard, WBA SAR is specified as a DRL up to a frequency of 6 GHz. The transition frequency of 3 GHz is changed to 6 GHz to harmonize with the new ICNIRP guidelines, where ICNIRP is changing from 10 GHz to 6 GHz.

B.4.1.2 DRLs for local exposure—100 kHz to 6 GHz

B.4.1.2.1 General

These restrictions are established to protect against an excessive temperature rise in any part of the body that might result from local or nonuniform exposure. To meet the DRLs, exposure should not result in a psSAR that exceeds 10 W/kg as averaged over any 10 g of tissue (defined as a tissue volume in the shape of a cube),[36] except for the limbs and the pinnae where the psSAR should 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 psSAR 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 limbs and the pinnae where the psSAR should 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 revised limits are 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 B.4.1.2.2.2 and B.7.5). Consequently, the revised limits prevent adverse local temperature rises in various tissues in humans.

B.4.1.2.2 Rationale for changing the values and averaging mass of the psSAR

B.4.1.2.2.1 General

The 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 local 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 local areas of the body such as the lens of the eye. As explained elsewhere, exposure at the WBA SAR limit (0.4 W/kg) is protective against core body temperature increases of more than a small fraction of 1 °C because this SAR limit is 10 times less than that needed to increase core temperature by 1 °C in rats and monkeys (cf. C.5.3). 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. The local 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 (see B.4.1.2.2.2). The threshold temperature increase for adverse effects in the fetus and testes is ~2 °C (see C.6.1.1 and C.6.1.2). Potentially adverse effects in the brain apparently require higher temperature increases than do those known to cause adverse effects in the testes and developing organism (Sharma and Hoopes [B1285]).

B.4.1.2.2.2 Change from dosimetry to biologically based rationale

As described in more detail in B.7.6, the psSAR limits in ANSI C95.1-1982 [B68] and IEEE Std C95.1-1999 [B667] were based on dosimetry considerations. The 8.0 W/kg and 1.6 W/kg limits were determined from the 20:1 ratio between the psSAR and the WBA SAR in experimental data available in the late 1970s (ANSI C95.1-1982 [B68]; IEEE Std C95.1-1999 [B667]). 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 psSAR to WBA SAR for a 1 g averaging mass can be much higher, with reported values of more than 100:1 (Bernardi et al. [B149]). ICES, however, considered it inappropriate to relax the psSAR 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 psSAR limit. In an ICNIRP statement [B646], a 10 g 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

[36] The volume of the cube is approximately 10 cm^3.

equivalent mass." ICES agrees that the biologically based ICNIRP rationale is more appropriate than the purely dosimetry based rationale in ANSI C95.1-1982 [B68] and IEEE Std C95.1-1999 [B667]. Furthermore, the limit of 10 W/kg averaged over 10 g is supported by results from animal experiments (Elder, [B388]; Guy et al. [B550]; Kramar et al. [B778]), showing that this limit is 10 times below the SAR threshold for cataracts in rabbits, which is estimated to be 100 W/kg deposited in the eyeball, which is a mass of about 10 g.

B.4.1.2.2.3 Temperature increase in the eye and brain due to localized RF exposure

This subclause addresses concerns about temperature increases in the eye and brain from an exposure of 10 W/kg averaged over 10 g and the potential for such temperature increases to cause adverse health effects.

On 1 May 2003, the National Radiological Protection Board (NRPB) of the United Kingdom released a consultation document titled "Proposals for limiting exposure to electromagnetic fields (0 Hz to 300 GHz)" [B1087]. 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 can exceed 1 °C. ICES TC95 Subcommittee 4 formed a task group to analyze the modeling papers cited in the NRPB 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 as follows.

Eye temperature: In the NRPB document [B1084], the data for the human eye were taken from two theoretical papers (Hirata et al. [B617], [B621]). 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 B.8). Table B.8 includes results on the effect of thermal modeling on the eye temperature increases from Hirata's laboratory, which shows the strength of including blood flow in the retina, choroid, and sclera and the weakness of conventional isolated eye-modeling temperature increases in the eye ranging from 0.94 °C at 900 MHz to 2.4 °C at 6 GHz.

Later, computations on the lens temperatures were conducted by different groups using realistic conditions (Buccella et al. [B210]; Flycket et al. [B426]; Wainwright [B1467]) pointed out that the dominant factors for eye temperature elevations are eye dimensions, blood flow in the skin and eye tissues, and so on, even though different factors affecting the temperature increases exist. If the parameters are adjusted to standard values, the lens temperature elevation at eye-averaged SAR of 10 W/kg was consistent at 1.1 °C to 1.5 °C. In most studies, the SAR averaged over the eye was considered instead of SAR in cubic shape, which is prescribed in the IEEE standard. If this is adjusted to the current averaging volume in the IEEE standard, the temperature increase can be lowered somewhat in most cases because the SAR in the skin is larger than that in the eye; for example, peak SAR appeared around the nose for plane-wave–like exposures.

It is noted that the maximum eye temperature increase in Table B.8 is below the temperature threshold (41 °C) for cataracts. The models in Table B.8 did not take into account thermoregulatory mechanisms; therefore, the results estimated temperature rise conservatively. Physiological observation and its interpretation are summarized as follows:

a) The statement that the eye cannot effectively dissipate heat due to limited blood vascular systems is frequently mentioned, but Carpenter et al. [B231] 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-min exposure. If a lethal dose of anesthetic is then injected intravenously, the heart will stop beating, whereupon the intraocular temperature will rapidly rise another 10 °C, thus

indicating that the vascular system is capable of handling at least two-thirds of the thermal stress which radiation imposes on the eye" (Carpenter et al. [B231], p. 354).

b) In the thermal analysis paper by Emery et al. [B395], 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 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. [B770] showed intraocular temperatures in rabbits were significantly higher (2 °C to 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 as a result of anesthesia. These results imply that the results of RF exposure experiments describing lens opacities in animals under anesthesia should be interpreted with care.

d) Hirata et al. [B622] replicated Kojima's study computationally, demonstrating that the vasodilatation in the nonanesthetized rabbit eye is significant, lowering the eye temperature increase. Later, the same authors computationally presented that the temperature increase in the skin is more significant rather than in the eye for the human [B1097]. This finding is consistent with the facial burning in the monkeys reported earlier [B777].

In summary, based on numerical modeling, an exposure of 10 W/kg averaged over 10 g produces 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 psSAR 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 (Bernardi et al. [B149]; Gandhi and Kang [B495]; Van Leeuwen et al. [B1442]; Wang and Fujiwara [B1474]; Wainwright [B1468]), describing thermal models of the brain. Among them, the NRPB study (Wainwright [B1468]) gave the highest temperature rise. Therefore, the NRPB stated that, "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 four additional new papers were analyzed (Bernardi et al. [B148]; Hirata and Shiozawa [B619]; Hirata et al. [B617]; Yioultsis et al. [B1537]). Wainwright [B1468] 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. [B1442]. Gandhi and Kang [B495] and Wang and Fujiwara [B1474] showed 0.5 °C to 0.6 °C increase with the same exposure. Bernardi et al. [B150] reported a 1.2 °C increase. NRPB recognized some of the uncertainties indicated by the range of the modeling data relating temperature rise with local 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 B.9 summarizes the analysis of nine papers. The values in the rightmost column were calculated from the model data at either 835 MHz/900 MHz or 1500 MHz/1800 MHz, whichever gave the greater temperature increase. When the psSAR is 10 W/kg averaged over 10 g of head tissue, four papers show that the brain temperature increase is greater than 1 °C (Bernardi et al. [B150]; Van Leeuwen et al. [B1442]; Wainwright [B1468]; Yioultsis et al. [B1537]). The highest temperature rise of 1.64 °C was reported by Wainwright [B1468], 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 °C 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. [B148] calculated smaller temperature changes for a model that incorporated antenna patterns of modern mobile phones. As shown in Table B.9, the temperature changes in the new results were less than half of the earlier values obtained with other

antennas (Bernardi et al. [B150]). Two other papers (Van Leeuwen et al. [B1442]; Yioultsis et al. [B1537]) showed that brain temperature rise can be higher than 1 °C. However, the majority of the papers (Bernardi et al. [B148]; Gandhi and Kang [B495]; Hirata and Shiozawa [B619]; Hirata et al. [B617]; Wang and Fujiwara [B1474]) reported temperature increases usually below 1 °C in the brain. As shown in Wang and Fujiwara [B1474], Hirata et al. [B617], and Hirata and Shiozawa [B619], the peak temperature rise in the brain due to 10 W/kg per 10 g exposure ranges from 0.567 °C to 1.25 °C. However, the SAR outside the brain tissue is 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, after a thorough review of current scientific knowledge, including the recently published modeling studies described earlier, 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 [B1128]). The Board, therefore, recommended the adoption in the United Kingdom of the ICNIRP guidelines for limiting exposure to electromagnetic fields between 0 Hz 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 [B1084]).

In summary, interpretations of the temperature data from modeling studies of the brain and eye should include consideration of the following limitations of the models: 1) the adequacy of physiological blood flow in many of the numerical model studies has not been verified, 2) none of the results for brain and eye have been validated in live animals and humans, and 3) the results from independent laboratories varied over a wide range. Until these limitations can be resolved, thermal models are useful but in and of themselves are not sufficient for safety standard development. Animal studies have shown that temperature elevations of less than 2 °C produce no adverse effect on the embryo or testes, the two most thermally sensitive organs (Edwards et al. [B385]); even higher temperatures are required to produce adverse effects in the brain (Sharma and Hoopes [B1285]). Although modeling data do not exist for all cases, local SAR of 10 W/kg averaged over 10 g of tissue gave calculated temperature elevations ranging from about 0.5 °C to 1.6 °C in the brain (see Table B.9), 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 developmental effects in embryos because the required threshold is a temperature increase of 2 °C to 2.5 °C (Edwards et al. [B385]) or 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 C.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 [B388]) and is protective against potentially adverse effects in the central nervous system as shown by the following information. Several 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. [B1312] and in Sneed and Stea [B1326]. A review of these studies indicates that the lowest brain temperature associated with contrast enhancement on computer tomography images [an indicator of blood-brain barrier (BBB) breakdown] was 40.3 °C for 30 min (Fike et al. [B415]). 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 BBB (Sharma and Hoopes [B1285]). A study of human cancer patients given whole-body hyperthermia treatment showed that the critical thermal maximum temperature was 41.6 °C to 42 °C for 45 min to 8 h (Sharma and Hoopes [B1285]; see also Bull et al. [B214]). 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 [B1285]). No adverse effects were observed in other physiological systems (cardiac, hepatic, and renal systems) after whole-body hyperthermia treatment (39 °C to 39.5 °C for 3 h or 6 h and 39.5 °C to 40 °C for 6 h; see Kraybill et al. [B784]).

IEEE standards are based on currently available knowledge; there may be substantive new information developed, and if new adverse effect information is established, the standard can be revised by amendments. ICES continues its efforts to monitor RF bioeffects research for the next standard revision.

Table B.8—Maximum increase in eye temperature calculated from thermal models for RF exposures (0.9 GHz to 6 GHz) of 10 W/kg averaged over 10 g

(Author(s) [Reference]): Comment	Frequency (GHz)	ΔT (°C)
(Hirata et al. [B621]): isolated eyeball	1.9	4
(Hirata et al. [B617]): human eye model thermally isolated from head	1	1.1
	1.9	1.2
	6	2.2
(Hirata et al. [B623]): human eye model thermally isolated from head	0.9	0.94
	1.9	1.3
	6	2.4
(Hirata [B614]): blood flow in retina, choroid, and sclera included	0.9	1.7
	1.5	1.7
	1.9	1.7
(Buccella et al. [B210]) blood flow in retina, choroid, and sclera included	0.9 to 2.5	1.5 to 1.7 (lens)
(Flycket et al. [B426]) Blood flow in retina, choroid, and sclera included and more detailed vasculature modeling	0.9 to 1.8	0.9 to 1.3 (lens)
(Wainwright [B1468])	0.9 to 1.8	1.8 to 1.9 (lens)
(Laakso [B800]) blood flow in retina, choroid, and sclera included	1 to 5	1.1 to 1.6 (eye) 1.0 to 1.4 (lens)

Table B.9—Correlation of the SAR_{max} (10 g) in the whole head with the maximum temperature rise in the brain (SAR head—ΔT brain)

Reference	835 MHz/900 MHz		1500 MHz/1800 MHz		
	SAR_{max} (W/kg)–10 g	ΔT (°C)	SAR_{max} (W/kg)–10 g	ΔT (°C)	ΔT_{max} (°C) @ 10 W/kg SAR_{max} (10 g)
(Wang and Fujiwara [B1474])	0.92	0.053	0.59	0.045	0.763
(Van Leeuwen et al. [B1442])	0.91	0.117	—	—	1.286
(Wainwright [B1468])	1.43	0.201	2.43	0.398	1.22[a]
(Bernardi et al. [B150])	1.08	0.13	—	—	1.204[b]
(Gandhi and Kang [B495])	2.00	0.103	2.00	0.068	0.515
(Bernardi et al. [B148])	1.19	0.061	0.87	0.036	0.513
(Yioultsis et al. [B1537])	2.072	0.331	0.591	0.079	1.597
(Hirata et al. [B617])	1.31	0.154	2.41	0.166	0.836[c] (avg)
(Hirata and Shiozawa [B619])	1.62	0.132	1.42	0.108	0.721[c] (avg)

[a] Due to an error in tissue classification in Wainwright [B1468], the temperature increase of 1.64 °C did not occur in the brain; the revised brain temperature increase is 1.22 °C (see earlier text).

[b] The same authors published a paper one year later showing that the brain temperature increase is less than 1 °C (see Bernardi et al. [B148] in the table).

[c] Averaged values provided by Akimasa Hirata.

B.4.1.2.3 Rationale for changing from “extremities” to limbs

B.4.1.2.3.1 General

IEEE Std C95.1-2005 [B668] relaxed the exposure limits for the hands, wrist, feet, and ankles. These higher local SARs were permitted because of: 1) the high surface-to-volume ratios of these parts of the body, 2) the common experience of 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. IEEE Std C95.6-2002 [B671] sets limits for arms and legs. ICNIRP guidelines have separate limits for limbs. To remove the inconsistencies, ICES decided to change the limits for “extremities” to

"limbs" because the three justifications listed also apply to these limbs and this change removes the ambiguity of establishing compliance.

B.4.1.2.3.2 Rationale for applying the psSAR values for the limbs to the pinnae

The rationale for applying the same psSAR values to the limbs and the pinna is provided in this subclause. For purposes of regulating exposure to RF energy, the pinna (auricle of the external ear) is subjected to the same SAR limits as the limbs of the human body. 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, which is a composition similar to that of the limbs. 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 °C to 35.0 °C. However, being a thin appendage, the pinnae normally has a somewhat cooler surface temperature (e.g., ~30 °C; see Guyton and Hall [B551]).

During use of a handheld mobile phone, a pinna can be pressed against the head and an increase in its surface temperature can occur largely because surface heat loss by convective cooling is impeded. In addition, thermal conduction of heat generated within the device can 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. [B722] reported that cheek temperature near an active mobile phone might increase by 1.7 °C to 4.5 °C relative to the opposite cheek. Bernardi et al. [B150] calculated a maximum pinna temperature increase from RF energy absorption of 0.23 °C after 80 min and an additional increase of ~1.0 °C after 15 min 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. [B214]; Sharma and Hoopes [B1285]). Also, during lengthy telephone use, convective heat transfer by the blood stabilizes pinna temperature. Even in hot environments or after exercise, an additional increase of 1 °C to 2 °C from use of a mobile phone would result in pinna temperatures that are well below the level (~42 °C to 45 °C) at which cellular injury or pain occurs.

B.4.1.3 DRLs—6 GHz to 30 GHz

DRLs are established for the epithelial power density of RF fields at frequencies between 6 GHz and 300 GHz. The DRLs are derived with consideration of adverse effects thresholds, population groups (i.e., workers and the general public), and safety factors. They were established after the thorough review and consideration of the literature. The derivation of the resulting values and their rationale are described in this annex.

For purposes of assessing compliance with the local exposure DRL at frequencies between 6 GHz and 300 GHz, the epithelial power density at body surface is spatially averaged over any square area with a size of 4 cm^2. For smaller exposed areas, higher exposure limits are allowed.

B.4.2 Whole-body ERLs—100 kHz to 300 GHz

Inspection of Table 7 and Table 8 (see 4.3.2) illustrates another change in this standard compared with IEEE Std C95.1-1999 [B667] and IEEE Std C95.1-2005 [B668]. Specifically, the ERL for the lower tier and upper tier now both ramp up to 2 GHz from 400 MHz instead of from 300 MHz to 3 GHz as in IEEE Std C95.1-2005 (upper tier). The change in the lower ERL in IEEE Std C95.1-2005 was based on a single 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 DRL in the 1 GHz to 3 GHz frequency range (Dimbylow [B361]). The change in the upper ERL in this standard is to keep a consistent ratio of 5 between the upper tier and the lower tier ERLs between 100 kHz and 300 GHz.

In the Dimbylow [B361] study, using an improved human model and finite-difference time-domain (FDTD) methods, the WBA SAR was computed for several different sized children as well as for an adult from approximately 70 MHz to 3 GHz. Similar data for the adult only using an alternative human model but also using the FDTD modeling method (Mason et al. [B976]) can be used for comparing nominal consistency between the two studies. Figure B.7 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. Second, and importantly, both studies reveal a WBA SAR up to more than two times the *Radio Frequency Radiation Dosimetry Handbook* (Durney et al. [B376]) value upon which the previous ERLs were derived. When the newly calculated WBA SAR values for small children are examined (Dimbylow [B361]), it becomes apparent that when exposed at the previous ERL, 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 ERL was likely inconsistent with the stated objective of the standard to limit WBA SAR to no more than 0.08 W/kg.

Several later studies with small children models that used different postures (sitting, arms up, etc.) also show similar results wherein, in some frequency ranges, the WBA SAR can exceed the lower tier DRL of 0.08 W/kg by up to 45 % (Bakker et al. [B96]; Conil et al. [B293]; Findlay et al. [B417]; Kühn et al. [B794]; Lee and Choi [B853]; Nagaoka et al. [B1051]; Wang et al. [B1476]). While the change in the lower tier ERLs was made in the standard, it should be noted that due to the inherent conservatism in the previous standard, the difference in ERL magnitudes is likely inconsequential when compared with the large safety factors embedded in the limits.

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 ERL. 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 B.8 illustrates this analysis for the grounded adult as well as for 1-year-old, 5-year-old, and 10-year-old children for unrestricted environments. For restricted environments, the curves are the same, except a factor of 5 smaller. Also, the same applies for Figure B.9, which is for the ungrounded case.

Figure B.8 shows that the SAR-based safety factor is generally greater than 50 at most frequencies, but in the 1 GHz to 3 GHz frequency range, it can be less than 50; the smallest safety factor is approximately 42 for a 1-year-old infant. At other frequencies, the safety factor can be as great as 316 for adults and much greater than 100 for a 1-year-old infant. There was considerable debate within ICES Subcommittee 4 as to whether such a finding was of sufficient biological significance to require modifying the ERL 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 ERLs rather than to change the stated safety factor from 50 to 25. No change in the MPE (ERL) for the upper tier 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. However, in this revision, ICES decided to keep the ratio between the upper and the lower tier

ERL at a factor of 5, the upper tier ERLs ramp up from 10 W/m^2 at 400 MHz to 50 W/m^2 at 2 GHz, and remain at the same value to 300 GHz.

The safety factor in Figure B.8 and Figure B.9 is presented for 1-year-old, 5-year-old, and 10-year-old children and for adults. The relationship between exposure to the ERL and WBA SAR was determined using data from Dimbylow [B361].

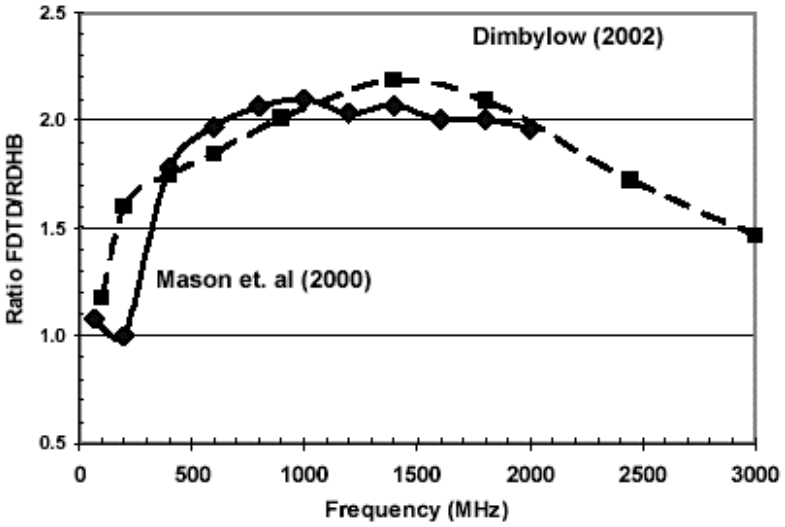


Figure B.7—Comparison of computed adult WBA SAR from two studies (Dimbylow [B361]; Mason et al. [B976]) relative to values from the *Radio Frequency Radiation Dosimetry Handbook* (RDHB) (Durney et al. [B376]) as a function of frequency

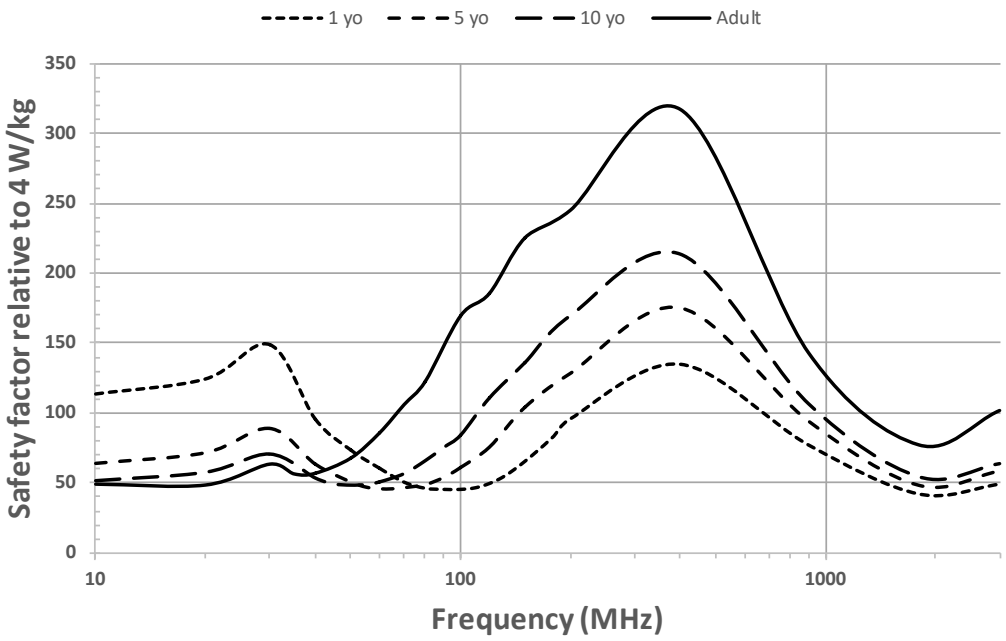


Figure B.8—Safety factor for the unrestricted environment for the condition of conductive contact between the body and the ground (grounded), determined by the ratio of 4 W/kg (the hazard exposure level) to the calculated WBA SAR resulting from exposure to the ERLs of this standard

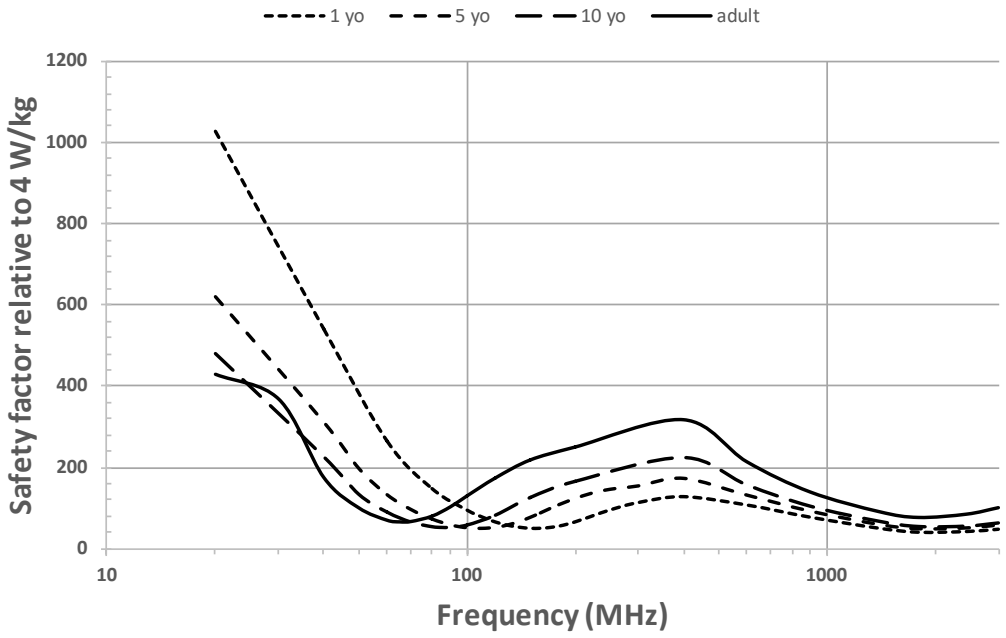


Figure B.9—Safety factor for the unrestricted environment for the condition of no conductive contact between the body and the ground (ungrounded), determined by the ratio of 4 W/kg (the hazard exposure level) to the calculated WBA SAR resulting from exposure to the ERLs of this standard

B.4.3 Rationale for pulsed RF field limits

ANSI C95.1-1982 [B68] specified a single set of limits as Radio Frequency Protection Guides that applied to all persons regardless of the nature of the exposure environment. It was stated that, "For situations involving unrestricted exposure of the body, the radio frequency protection guides are understood to result in energy deposition averaged over the entire body mass for any 0.1-h period of about 144 J/kg or less. This is equivalent to a specific absorption rate (SAR) of about 0.40 W/kg or less, as spatially and temporally averaged over the entire body mass."

Two exposure environments (controlled and uncontrolled) with averaging times of 6 min and 30 min, respectively, were introduced in IEEE C95.1-1991 [B666]. A special provision was made relative to exposure to pulsed RF fields. For pulsed RF fields, the whole-body SA was limited to 1/5 of SA that would be permitted for a continuous, nonpulsed field. The SA limit became 28.8 J/kg for both exposure environments reflecting a factor of five difference in the MPE (uncontrolled environment MPE being lower) and a factor of five difference in the averaging time (the averaging time for uncontrolled environments being greater) for any 100 ms period in either exposure environment. The incorrect placement of the SA limit in with the equation for peak power density of pulsed RF fields is corrected in this present standard.

This standard no longer considers that the nature of the exposure environment (restricted versus unrestricted) should dictate the averaging time for exposure. (See B.1.2.2 d).) Rather, considering that the thermal time constants are proportional to the volume of tissue heated, a 30 min averaging time is chosen for whole-body exposure and 6 min for local exposure. As specified in Table 7 and Table 8 (see 4.3.2), time-averaged, whole-body exposure shall meet the whole-body ERL regardless of the waveforms, including pulsed fields. For pulsed RF fields (see 4.3.4), it was determined that the peak power density limits should be further limited for local exposures for persons in both the restricted and unrestricted exposure environments.

For frequencies below 30 GHz, the limits on peak pulsed RF fields are based on two effects of high peak energy deposition: microwave hearing (Chou et al. [B262]; Elder and Cahill [B390]) and the stun reaction (unconsciousness) due to high temperature in the brain. The stun reaction is reported by Guy and Chou [B545]. Briefly, rat heads were exposed to a single 915 MHz pulse at SAR as high as 4×10^5 W/kg. When the peak absorbed energy density in the brain exceeded 28 kJ/kg, which was associated with a maximum temperature rise of 8 °C in the brain (> 46 °C), rats displayed petit mal or grand mal seizures, followed by an unconscious state until the brain temperature returned to within 1 °C of their normal value. Both auditory and stun effects are presumably related to high peak energy deposition in the head and no other part of the body, although the exposure levels associated with the two effects differ by many orders of magnitude. The limits on peak power density (ERL) are the values obtained by consideration of a well-established scientific base of data that includes the auditory effect in humans and RF energy-induced unconsciousness in rats (see Chou et al. [B262]; Guy and Chou [B545]; Guy et al. [B547]; Lin [B882], [B884], [B886]).

However, as a conservative approach, limits on peak exposure apply to any part of the body, not just to the head. For frequencies above 30 GHz, the biological effects from exposures compliant with the standard are limited to the skin. The concerns for effects limited to the skin are the same whether they occur on the head or on any other part of the body. Millimeter-wave exposures are typically small in extent and produce localized exposures. Therefore, the limits on peak pulsed millimeter waves apply only to local exposures for both restricted and unrestricted environments.

For exposures to pulsed RF fields in the range of 100 kHz to 300 GHz, peak power density limits are provided to prevent unintentionally high local exposure and to preclude high SA in the frequency range of 100 kHz to 6 GHz or epithelial energy density above 6 GHz. Historically, it has been recognized that for decreasingly short RF pulse widths, the lack of such consideration in the standard allows the peak power density to rise arbitrarily, as long as average power density complies with the standard.

Furthermore, under exposure to high-intensity pulsed fields, it is advisable to be conservative in view of some uncertainty about the magnitude of spatial peak SAR or epithelial power density. In pulsed conditions (pulse widths less than 100 ms), the ERL, as averaged over any 100 ms, is reduced by a factor of five times.

The 100 ms averaging time and the factor of 5 were chosen by the committee that developed IEEE C95.1-1991 [B666] as reasonable and conservative.[37]

For a single pulse, this is equivalent to reducing the ERL by a factor of five times below the value that normal time averaging would permit. A maximum of five such pulses are permitted during any period equal to the averaging time. If there are more than five pulses in any period equal to the averaging time, normal time-averaging further reduces the permissible peak power density. The intent of this limitation for pulse widths of 100 ms or less is to reduce the local specific absorption SA (J/kg) below 6 GHz or epithelial energy density (J/m^2) in tissue above 6 GHz by a factor of five times below that which would be associated with the local DRL.

B.4.4 Contact current

Skin contact with RF-energized metallic surfaces, when directly driven by an RF source via reradiation, can result in burning of tissue at the point of contact and arises at certain values or thresholds in a frequency-dependent manner. Although the RF burn in itself can be insignificant, the startle reaction can result in loss of balance, falls, or reflex reaction resulting in unintended contact with machinery possibly producing injury or death. Above 100 kHz, the effect of contact with RF-energized surfaces transitions from electrostimulation to a thermal or heating sensation. From 100 kHz to 3 MHz, human tests reveal painful sensations at about 50 mA in 50 % of subjects (Chatterjee et al. [B244]; Reilly [B1179]). The reporting of pain followed approximately 10 s to 20 s of electrode contact (electrode with an area of 25 mm^2) with a contact current that produced a threshold response to warmth. Additional tests of human perception of RF current suggest that thresholds rise with stimulation frequency from 3 MHz to 20 MHz (the highest frequency tested) in proportion to $f^{0.37}$, where f is the sinusoidal frequency (Rogers [B1207]).[38]

In other research, a somewhat similar increase in threshold for sensation of pain has been found across the high-frequency (HF) band from 1.9 MHz to 28 MHz where a 100 mA RF current delivered via a round, 100 mm^2 (1 cm^2) electrode resulted in an increase of 5 ^oC at the surface of a synthetic tissue in 3 s to 250 s, depending on frequency.[39] A key finding was that the surface heating was related almost entirely to the electrode contact resistance at the tissue surface. For example, with the application of conductive gel on the electrode, substantially greater currents are required to achieve the same surface temperature within a given duration of current flow.

Several variables associated with the determination of thresholds related to RF contact currents are potential sources of disparity among the scant data on observed effects of contact currents. These variables include frequency, contact resistance, shape of the contact electrode used in tests, uniformity of skin heating beneath the electrode, and the force exerted by the electrode on the skin (Tell and Tell [B1397]). Because of the potential impact of these factors, the specified contact current limits in this standard should be understood to represent reasonable estimates based on available data. However, the subject of contact currents in the RF spectrum would seem to be ripe for additional research.

[37] The 100 kV/m peak (temporal) value of the electric field ERL for exposure to high-peak-power, ultrashort pulsed electromagnetic fields (100 kHz to 300 GHz) found in earlier IEEE C95.1 standards was established in 1971 as a "provisional" limit by the U.S. Air Force [B1429]. The NATO Research Task Group (RTG HFM-189) conducted a seven-nation, three-year review and found this ultraconservative limit had neither an underlying biological mechanism nor was it based on any adverse health effect to support its existence. A consensus statement recommended eliminating the high-peak-power, ultrashort, pulsed electric field exposure limitation. The recommendation was adopted in IEEE Std C95.1-2345-2014 [B669], which is covered by the NATO Standardization Agreement 2345 Edition 4, 2015 [B1054]. ICES agreed to remove the 100 kV/m limit in this standard.

[38] The report by Rogers [B1207] lacks peer review to qualify as a definitive reference. Responses to contact current have been reported in the peer-reviewed literature at frequencies up to 3 MHz (reviewed in Kavet et al. [B742]). Further research is needed to clarify this subject.

[39] Technical report prepared by Richard Tell Associates, Inc. for the Electric Power Research Institute (EPRI), Technical Update, 3002009501, January 2017. Report available to the public at:
https://www.epri.com/#/pages/product/000000003002009501/?lang=en-US

B.5 Adverse effect levels

B.5.1 Specific absorption rate and temperature

The time rate of temperature change (dT/dt in °C/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
c	is the specific heat capacity expressed in J/(kg °C)

The preceding equation can be simplified by assuming that a steady-state condition exists in the tissue prior to exposure; that is,

$$M = K + C$$

SAR, in perfect adiabatic conditions, 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 ~1 °C/min.

The preceding 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 [B1060]). 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 can be complicated.

The studies that provided the evidence for a threshold SAR of about 4 W/kg for behavioral effects (work stoppage) in rodents and nonhuman primates show that exposures for ~1 h at the threshold SAR are associated with an increase in body temperature of ~1 °C (see C.5.3.1.1). These experiments also demonstrated that SAR is a better predictor of biological effect compared with power density (NCRP [B1060]).

In addition to the complicated dependence of SAR on frequency, polarization of applied field, and the dielectric properties, size, and shape of the exposed object, the relation between SAR and temperature increase is further complicated by the heat transfer characteristics of the exposed object to its environment.

The environmental factors include air flow, ambient temperature, humidity, insulation, and so on. At resonance, for example, SAR and temperature elevation are maximal, but at frequencies below and above resonance, SAR and temperature elevation decrease. Due to their much larger surface-area-to-volume ratio, mice dissipate heat much more readily than do 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 affects the temperature of objects and thereby affects 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 lessens 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. [B777]). 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 [B255]). These experiments show that the causative factor for the biological responses was due to the rise in temperature and not to the RF energy *per se*. Therefore, although the DRLs in the frequency range of 100 kHz to 300 GHz are SAR or epithelial power density, the basis for the DRLs is temperature rise to protect against adverse health effects due to heating. The possibility of using temperature directly for exposure limits can be discussed in the future revision of IEEE Std C95.1.

B.5.2 Levels at which increased temperature causes adverse effects

B.5.2.1 General

Exposures equivalent to the ERL in the body resonance range result in energy deposition, averaged over the entire body mass for any 30 min period of approximately 720 J/kg. This SA corresponds to an SAR of ~0.4 W/kg, as spatially averaged over the entire body mass. This WBA SAR is equivalent to approximately one third of the resting metabolic heat production of an average human adult. This level is completely benign; it does 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 the heat dissipation capability, which in turn is affected by clothing, humidity, air flow, and so on.

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. A 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.

For thermal damage to occur, human skin would have to be heated at 43 °C for 10 h to 12 h (Moritz and Henriques [B1039]). On the other hand, for brief (3 s to 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 °C to 60 °C (Hardy et al. [B576]). 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 [B1039]).

Dewhirst et al. [B355] 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 °C to 44 °C for the initiation of significant damage. Murine data (Hume et al. [B642]) suggest that thresholds for thermal injury can differ for different tissue types. The testes and brain can be more sensitive to heat than can other tissues (e.g., the intestines and skin), although specific end points (necrosis versus function versus appearance) can 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 unlikely that human tissues are more thermally sensitive than those of other species (Dewhirst et al. [B355]).

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 [B82], ANSI C95.1-1982 [B68], IEEE Std C95.1-1999 [B667], and IEEE Std C95.1-2005 [B668]. Table B.10 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 B.10—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 (°C) and SAR (W/kg)	Exposure duration	Reference number
Heat stroke	Human (core temperature) Human (brain temperature)	> 42 °C ≥ 40.5 °C	varies with T	Bynum et al. [B220] Cabanac [B223]
CNS deterioration	Human (CNS)	42 °C to 43 °C	varies with T	Bynum [B220]
Skin necrosis Skin necrosis Full thickness burn Full thickness burn Pricking pain	Human Human Human Human Human	43 °C 55 °C to 50 °C 45 °C 60 °C 45 °C	10 h to 12 h 3 s to 10 s 100 min 5 s 3 s to 10 s	Dewhirst et al. [B355]
Thermal injury	Rat, mouse, dog, cat (spinal cord, brain)	43 °C to 44 °C	1 min to 80 min	Dewhirst et al. [B355]
Fetal abnormalities	Rat (whole body)	2 °C to 2.5 °C increase	tens of minutes up to 1 h	Edwards et al. [B385]
Behavioral disruption	Rat (whole body) Monkey (whole body)	1 °C increase, 4 W/kg	40 min to 60 min	de Lorge [B337], [B334] D'Andrea et al. [B322]
Cataract	Rabbit (eye)	> 41 °C (> 150 W/kg)	> 30 min	Kramar et al. [B779] Guy et al. [B550] Carpenter. [B229]
Convulsions	Mouse	T_{rectal} = 44 °C		Wright [B1519]
Increase in BBB permeability	Rat	> 40 °C brain temperature (> 4 W/kg WBA SAR)	4 h	Merritt et al. [B1001] Finnie et al. [B420], [B419] Sharma and Hoopes [B1285]

B.5.2.2 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 [B18]). The core temperature in humans is generally stable within the range of 36.5 °C to 37.5 °C at most environmental temperatures encountered; however, the 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 it can occur at any core temperature within the range of 39 °C to 47 °C (see B.5.3.1.2).

Studies of human beings deliberately exposed to RF energy are rare, and most of those reported involve local RF exposure. In volunteers undergoing MRI, when the SARs = 2.7 W/kg to ~6.0 W/kg for 30 min, the core body temperature (tympanic) could rise as much as 0.4 °C. 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. [B1293], [B1294], [B1296]). As the frequency of localized RF exposure increases, the 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 W/kg to 15.4 W/kg in controlled thermal environments, the 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 [B7]; Adair et al. [B13], [B19], [B21], [B22], [B23], [B24]). Individual physiological responses (skin temperatures, sweating rate, skin blood flow) were a function of ambient temperature (T_a = 24 °C, 28 °C, 31 °C), frequency (450 MHz, 2450 MHz), and field strength (when the subjects were exposed at 180 W/m^2 and 240 W/m^2 at 450 MHz, or 270 W/m^2, 350 W/m^2, 500 W/m^2, and 700 W/m^2 at 2450 MHz). Corresponding normalized peak SARs at 2450 MHz were 6.0 W/kg, 7.7 W/kg, 11.2 W/kg, and 15.4 W/kg, the highest being well outside the guidelines of IEEE Std C95.1-2005 [B668].

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 times the free-space wavelength (resonant frequency) (Durney et al. [B376]). RF exposure of nonhuman primates at resonance yields somewhat less efficient thermoregulation than does exposure to subresonant or supraresonant frequencies (Adair et al. [B13]; Krupp [B788]; Lotz [B917]; Lotz and Saxton [B921]). Although the threshold for a reduction in metabolic heat production (*M*) can be lower at resonance, the magnitude of the response change can be lower for a given SAR than at nonresonance and the body temperature can rise. However, the hyperthermia is modest and well regulated. The situation is similar to that of humans during exercise (Adair [B7]). Some have expressed concern that human exposure at resonance might 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 eight times the limits of the IEEE Std C95.1-1999 [B667] at 100 MHz (Adair et al. [B22]).

B.5.2.3 Local exposure

If a nonhuman 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 [B4]). If an animal is exposed to RF energy at SARs greater than those that reduce *M* to the resting level, thermoregulation is accomplished by mobilization of the next response in the thermoregulatory hierarchy, that is, changes in vasomotor state or conductance, including blood flow (ACGIH [B3]; Adair [B4]; Candas et al. [B225]; Lotz [B917]). Experimental partial-body, far-field exposures of human volunteers have been conducted at 450 MHz and 2450 MHz for several field strengths in controlled environments (ACGIH [B3]; Adair et al. [B19], [B23], [B24]). Even though the exposures covered only the dorsal aspects of the head, trunk, and upper arms, increased local skin temperatures provoked strong heat loss responses of increased skin blood flow and sweating, thereby ensuring a stable core temperature. Complementary whole-body exposures at these frequencies have not been conducted. The necessity for very large anechoic chambers and extremely high-powered 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 [B1287] investigated the possibility that high RF “hot spots” might generate thermal “hot spots.” During MRI, RF energy is mainly absorbed by peripheral tissues, allowing for 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 [B1287]; Shellock and Crues [B1290]; Shellock et al. [B1291], [B1292], [B1293], [B1294], [B1295]). In general, localized temperature increases, including that of the cornea, were modest and not deleterious.

Since the 1930s, 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 1960s 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 initiates sweating in an exercising person or a person exposed to a warm environment, and 2) an abrupt increase in regional blood flow occurs when the local tissue temperature reaches 42 °C to 43 °C.

Cunningham [B304] 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 mL/(100 cm^2 min) to 20 mL/(100 cm^2 min)], where T_{sk} is calculated by this equation across a range of 15 mL 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 [B1355]).

It is more difficult to measure blood flow (BF) changes in deep tissues, such as muscle. Lehmann et al. [B855] pioneered the use of localized diathermy applicators (900 MHz and 2456 MHz) to heat subsurface tissues. Sekins et al. [B1277] 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 five tissue depths with nonperturbing 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. [B1277]) confirmed the threshold for a rapid increase in muscle BF at 42 °C to 43 °C. Other findings included the occurrence of gradients of local BF in fat and muscle at specific skin depths, accumulation of sufficient physiological data for accurate modeling, and establishment of appropriate conditions for efficient treatment of restricted tumors located well below the skin surface. A high incidence of heat intolerance occurs in multiple sclerosis, where it is particularly noticeable (at some level in up to 85 % of the patients).

Multiple sclerosis is a disease of the nervous system characterized by a patchy loss of the myelin surrounding nerve fibers. This loss affects the transmission of nerve impulses and produces the symptoms of the disease. The demyelinated nerves are heat sensitive, and small increases in temperature lead to a worsening of clinical symptoms such as muscle weakness and visual blurring. The magnitude of temperature elevation sufficient to induce this unfavorable reaction can be very small, perhaps as small as a few tenths of a degree. The exacerbation of symptoms is temporary, producing no actual tissue damage, and generally is rapidly reversed when the source of the increased temperature is removed. Home air conditioning is frequently prescribed for patients with multiple sclerosis (if they do not have it already).

After an extensive series of animal experiments in which histopathology of many organs has been performed, there have been no reports that chronic RF exposure causes demyelination. There is no evidence that chronic exposure to RF fields causes multiple sclerosis or any of the clinical conditions listed earlier.

B.5.2.4 Sensitive tissues and organs

Although some information on this topic appears in B.2.2.2, 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, that is, 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 can 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 into 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 [B353]). The equation for converting one time-temperature combination to another is

$$CEM_{43} = t \times R^{(43-T)}$$

where

CEM_{43} is the cumulative equivalent minutes at 43 °C
t is the time (min)
T is the average temperature (°C) during the time interval t
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 [B353]) 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. [B355] 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 [B213]; Hardy et al. [B576]; Moritz and Henriques [B1039]). 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} at various durations from < 1 min to > 80 min, reveals the thermal sensitivity of many animal tissues (Dewhirst et al. [B355]). Based on histopathological analysis, testicular and brain tissues appear to be the most sensitive to thermal insult for exposures of short duration. Changes in 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 can damage the prostate and the 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 significant damage to most of the tissues in the body in rabbits, dogs, and pigs. Evidently, rodents do not survive CEM_{43} exposures of durations much longer than 40 min.

B.5.3 Relevance of information from classical heat stress studies

B.5.3.1 Levels at which health or a physiological function are adversely affected

B.5.3.1.1 General

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 °C to 43 °C and convulsions can occur. At this temperature, protein denaturation can begin and cells can 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 can rise 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, which is a true hazard to human beings.

Any factor that either reduces heat loss or increases heat gain predisposes one to heat stroke. Three main factors have been identified that predispose to the breakdown of heat loss mechanisms. These include: 1) dehydration, which perturbs the cutaneous circulation and sweat secretion; 2) poor acclimatization to heat;

and 3) 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 B.5.3.1.2, B.5.3.1.3, and B.5.3.1.4.

B.5.3.1.2 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. [B220] 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 can be adjusted by the several factors known to influence heat tolerance. For example, the CTM can be raised by heat acclimation, fitness, or high motivation; it can 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, in terms of either 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 [B1519]).

In heat stroke, disturbances of the CNS are always present and the level of consciousness is often depressed. The symptoms include coma, sleep, or delirium. Pathology after heat-induced death shows edema in the brain tissue and meninges with a flattening of the brain convolutions, facts that infer that the temperature of the CNS tissue is critical to the occurrence of heat stroke. Thus, the defense (maintenance) of brain temperature seems to be of paramount importance. Whether the brain temperature decreases to a lower temperature than core temperature during heat stroke is unknown, especially under prolonged steady-state conditions when the thermoregulatory system fails. Those patients who have survived heat stroke with core temperatures of 45 °C to 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 [B223] that such a mechanism exists.

B.5.3.1.3 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 PCO_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), are prone to hypokalemia as a first step in the pathogenic process that leads to heat stroke.

B.5.3.1.4 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 et al. [B1299]). 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 PO_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. [B1050]).

B.5.3.2 Additional factors in heat stroke

B.5.3.2.1 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. [B218]). This result implies that endotoxemia of intestinal origin was sufficiently severe to contribute to the fatal outcome.

Butkow et al. [B217] 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 should be mentioned that no verified reports exist of injury to human beings, or of adverse effects on the health of human beings, who have been exposed to electromagnetic fields within the limits of frequency and SAR specified by previous standards, including ASA C95.1-1966 [B82], ANSI C95.1-1982 [B68], IEEE Std C95.1-1999 [B667], and IEEE Std C95.1-2005 [B668].

B.5.3.2.2 Effects on evoked potentials

Britt et al. [B198] 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 °C to 45 °C) on changes in brain function of anesthetized cats. They measured evoked potentials (brain stem auditory, somatosensory, and visual), core and brain temperatures, heart rate, arterial pressure, hematocrit, blood gases, and 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 °C to 44.0 °C resulted in complete loss of the waveform without recovery after cooling.

Other studies (Lyons et al. [B941]) using ultrasound- or microwave-induced heating of normal brain tissue in cats showed cytological evidence of damage after heating at 42.2 °C 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.

B.5.3.3 Levels at which behavior is adversely affected

Research conducted during the past four 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 [B336] 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 °C 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 W/kg 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-field 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 W/m^2 to 1400 W/m^2. 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 disruption 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 W/kg to 4 W/kg (D'Andrea et al. [B322], [B325]; de Lorge [B335], [B336]; de Lorge and Ezell [B338]), 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 6 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. [B322]; Schrot and Hawkins [B1265]). 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 [B1344] and others have pointed out that the reduction of response of a learned

task might not necessarily imply a hazardous effect but could simply reflect the animal's attempts to engage in other behaviors (e.g., escape and cooling off). These responses 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 can cause behavioral and other effects, some of which can be adverse. Justesen [B724] 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 [B667] and IEEE Std C95.1-2005 [B668] 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. [B322], [B325]; de Lorge [B335], [B336]; de Lorge and Ezell [B338]). Because of the paucity of reliable behavioral data from chronic exposures, ICES 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 after low-level chronic microwave exposure. For example, D'Andrea et al. [B318] 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. [B356]). Neither of these experiments replicated earlier findings reported by Rudnev et al. [B1224] and by Shandala et al. [B1282]. One can only conclude that these experiments were below the threshold for reliable effects to be observed and, therefore, that 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. [B317]). The few biological effects reported subsequent to chronic microwave exposure (Lovely et al. [B922]), 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 previously reported biological effects during or subsequent to chronic, low-level exposure have 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, needs to be superseded eventually by reliable data for the species in question, *homo sapiens.*

A consensus of ICES 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 6 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.

B.5.3.4 Levels at which other effects are adverse

As indicated, 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, and so on would be too lengthy to be discussed here. A few examples are described in this subclause (see Table B.11). Death (50 % mortality) of mice and rats was observed, respectively, after exposures at 42 W/kg 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.[B138]). For comparison, another paper (Petin et al. [B1142]) reported survival times of ~1 h or so at ~14 W/kg for rats and at ~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. [B203]; Lary et al. [B837], [B838], [B839]), while the threshold for memory deficits is 10 W/kg at 600 MHz (Mickley and Cobb [B1012]; Mickley et al. [B1013]). Multiple effects including bradycardia were reported after exposures at 2450 MHz and 6.5 W/kg (Phillips et al. [B1148]). At 2450 MHz, an SAR of 5.6 W/kg produced temporary sterility in rats (Berman and Carter [B143]). 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 [B710]). 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. [B146]). All of these effects can be attributed to the thermalizing effects of sustained whole-body RF exposure.

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. [B777]) after exposures at 2450 MHz.

Table B.11—Adverse biological effects produced by RF exposure greater than 4 W/kg

Endpoint	Species	Frequency (MHz)	Threshold (W/kg)	Exposure duration	Reference number
Death	Mouse	2450	42	4 h	Berman and Carter [B138]
	Rat		18	4 h	Petin et al. [B1142]
	Mouse	7000	30	50 min to 70 min	
	Rat		14	60 min to 100 min	
Birth defects	Rat	27.12	~11	3 min (42.2 °C)	Brown-Woodman et al. [B203]
				10 min to 40 min (41.5 °C)	Lary et al. [B839]
				26 min to 32 min (43 °C)	Lary et al. [B837]
				120 min (41.5 °C)	Lary et al. [B838]
Memory deficit	Rat	600	10	20 min	Mickley et al. [B1013] Mickley and Cobb [B1012]
Reduced fetal weight	Rat	970	4.8	22 h/d, days 1 to 19 of gestation	Berman et al. [B146]
	Rat	6000	7.3	6140 min during 12 d to 14 d of gestation	Jensh [B710]
Fertility (temporary sterility in male rats)	Rat	2450	5.6	4 h/d, 5 d/wk, 4 weeks	Berman et al. [B143]
Bradycardia	Rat	2450	6.5	30 min	Phillips et al. [B1148]
Reduced weight gain in pregnant rats (heat stress)	Rat	970	4.8	22 h/d, days 1 to 19 of pregnancy	Berman et al. [B146]

B.5.3.5 Levels associated with uncomfortable or painful sensations

B.5.3.5.1 Thermal stimulation

RF energy of millimeter wavelength (30 GHz 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. [B171]). To evoke pain, RF exposure must raise the surface temperature of the skin by 10 °C to 13 °C, depending on the duration of exposure. Very rapid heating evokes pain at lower temperatures of approximately 43 °C to 44 °C, compared with slower heating at higher temperatures of approximately 44 °C to 46 °C. At frequencies above 100 GHz, power densities greater than 5000 W/m^2 produce such surface temperatures. At lower frequencies, RF energy is less efficient in raising skin temperature, as the absorption is spread over greater depths (volumes) of tissue. Below 6 GHz, it takes

approximately 20 times as much incident power density as at 100 GHz to produce equivalent heating. Over most of the frequency range in which protection against adverse effects is associated with heating (100 kHz to 300 GHz), exposure under normal circumstances at the ERL for the lower tier cannot even be perceived. Even in the millimeter wavelength range, extended exposures at the ERLs are unlikely to elevate skin temperature more than 2 °C to 3 °C. RF exposures at lower frequencies (< 30 GHz) are much less effective in heating the skin.

B.5.3.5.2 Human response to thermal environments and equivalent RF exposure

Another interesting insight into 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. [B411]; Fanger [B412]; Gonzales and Gagge [B519]; Meese et al. [B995]; Tham [B1400]; Wyon [B1525], [B1526]; Wyon et al. [B1527], [B1528]). 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, and so on. A widely recognized American Society of Heating, Refrigeration, and Air-Conditioning Engineers (ASHRAE) standard (ASHRAE-55 [B85]), 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 [B85], a standard used primarily outside the United States has been developed by the International Organization for Standardization (ISO 7730 [B688]). 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 [B436]) and that are employed widely for designing heating and air conditioning systems for the workplace. The ASHRAE *Thermal Comfort Tool* [B85] 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 met (equivalent to 105 W)[40] and dressed in summer attire with a rating of 0.5 clo.[41] The model can 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 examine 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

[40] The met is the unit used to express the metabolic rate per unit DuBois skin surface area. The met is defined as the metabolic rate of a sedentary person (seated, quiet), 1 met = 58.2 W/m^2 = 50 k cal/(h m^2). A normal healthy man has a maximum energy capacity of $\approx M_{act}$ = 12 met at age 20. Typical metabolic heat generation for various activities ranges from 0.7 met to 8.7 met.

[41] 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 clo to 0.48 clo, or greater.

loading on the body from metabolic activity would be similar to that imposed by whole-body RF energy absorption.

Figure B.10 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 ~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 ~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 [B135]) wherein the RF energy dissipated in the body is compared with 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 earlier. For example, data have been obtained (Adair and Adams [B11]) that demonstrate how squirrel monkeys preferentially select a particular lower ambient temperature when subjected to differing levels of RF exposure. In one case (Adair and Adams [B11]), an incident power density of 20 mW/cm^2 at 2.45 GHz resulted in a preferred decrease in environmental temperature of 3 °C, compared with the ambient temperature with no exposure. This exposure is comparable to a WBA SAR of about 2 W/kg in the squirrel monkey (Durney et al. [B376]). From Figure B.10, 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 °C and 5 °C, which is 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 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 the values for the upper tier exposure limits are protective.

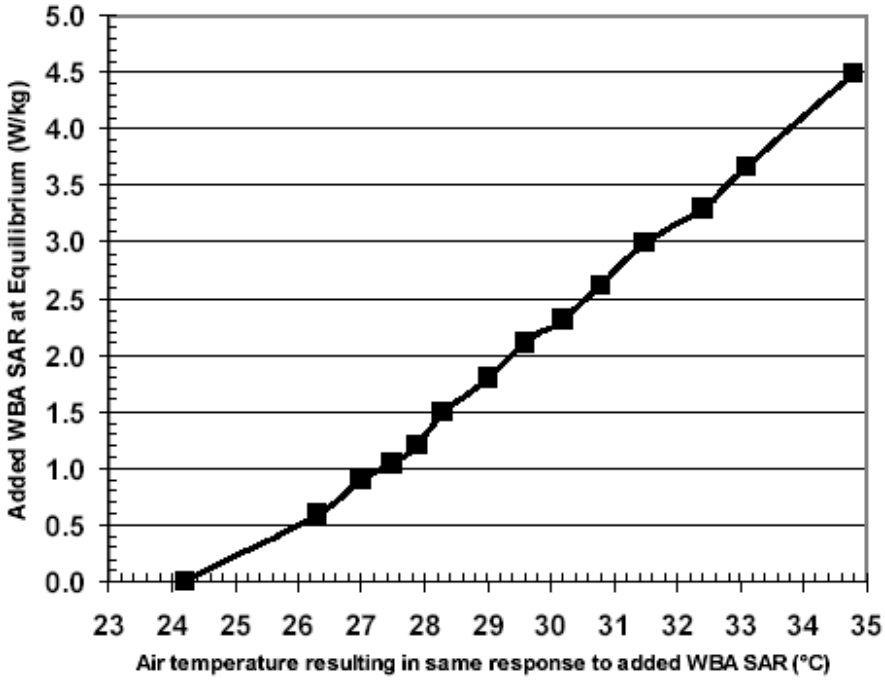


Figure B.10—Estimated added RF thermal load (W/kg) at rest and in a thermally comfortable environment to produce an equivalent discomfort response at rest to elevated ambient air temperature

B.6 Averaging time

The IEEE C95.1-series standards have historically used a one-tenth of an hour (6 min) averaging time for exposures in restricted environments and 30 min for exposures in unrestricted environments. This reflected the factor of 5 difference in the limits themselves.

In the current version, these averaging times have been modified to more explicitly reflect the characteristic time scales for clearance of heat from the body. For whole-body exposure, the relevant time scale is governed by the rate of exchange of heat between the body core and the external environment, and is of the order of tens of minutes (e.g., Adair and Black [B18]).

For local exposures, the time scale is determined by transfer of heat from the exposed region of tissue to the body core. The time scale for this process depends on several factors. One is the rate of convective heat exchange by blood flow, which for purposes of this standard is modeled by Pennes' bioheat equation [B1139]. This introduces an intrinsic time scale that is inversely proportional to the local rate of blood perfusion and is of the order of 5 min to 10 min (e.g., Foster et al. [B432]). A second factor is conduction of heat from the exposed area. According to the theory of heat conduction, this introduces a time scale that is proportional to the square of the size of the exposed area. These two time scales combine in a way that depends on the extent of the exposed region of the body. Detailed numerical analysis (e.g., Morimoto et al. [B1037]) as well as simplified analytical models (Foster et al. [B432]) show that for typical exposure scenarios, the overall rate of heat clearance from an exposed region of tissue in most cases is governed by thermal convection due to blood flow. This is the case also for exposures above 6 GHz, where the incident energy is absorbed close to the body surface but the heat is removed in part by conduction into deeper layers of tissue. Exceptions occur if the exposed area of body surface is small (less than 1 cm in diameter) or for brief but intense millimeter-wave pulses. In both of these cases, the increase in skin temperature is limited by heat conduction over small distances in tissue and the effective time constant is reduced. The case of intense brief pulses at millimeter-wave frequencies is considered in a separate provision limiting fluence. There is no special provision for small exposed areas in the present standard.

In IEEE Std C95.1-2005 [B668], the thermal averaging time for exposures above > 6 GHz was progressively reduced with frequency to meet up with the limits of the ANSI laser standard (ANSI Z136.1-2014 [B69]) and the ICNIRP IR guidelines ([B647], [B648]) at 300 GHz. At infrared wavelengths, a typical hazard scenario involves exposure to small-diameter infrared laser beams. In this standard, the averaging times have been changed at high frequencies from the previous edition, based on the judgment that potentially hazardous exposures at MMW frequencies usually involve somewhat larger areas of skin than is the case with infrared laser exposures. These revised averaging times are supported by more detailed thermal models than in the previous edition of this standard (e.g., Hashimoto et al. [B577]).

It is noted that the "averaging time" in this standard was derived from a judgment about the approximate time scales required for heat clearance from the body in different exposure situations; it is not a precisely determined quantity. In realistic exposure circumstances, the time required to clear heat from the exposed region of tissue, and consequently the increase in tissue temperature, depends on the details of the exposure as well as on the environmental and physiological variables. The judgment of ICES is that the specified averaging times are appropriate for foreseeable exposure conditions but may be revised in the future, as a result of either more detailed thermal analysis or the emergence of new exposure sources.

B.7 Special considerations

B.7.1 Recognition of whole-body resonance

As is true of ANSI C95.1-1982 [B68], IEEE Std C95.1-1999 [B667], and IEEE Std C95.1-2005 [B668], the ERLs in this standard are based on recommendations of field strength or of plane-wave-equivalent power densities of incident fields. These limits are based on well-established findings that the body, as a whole, exhibits frequency-dependent rates of absorbing electromagnetic energy (Barber [B108]; Durney [B374]; Durney et al. [B375], [B376]; Gandhi [B489]). Whole-body-averaged SARs approach maximal values when the long axis of a body is parallel to the electric 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 (Durney et al. [B376]; Gandhi and Chatterjee [B491]). In consideration of this dependency, recommended ERLs 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 flat for frequencies in the range of about 1 GHz to 3 GHz. The recommended ERLs 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 ~15 GHz to 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 local ERL at 300 GHz in a restricted environment is consistent with the limits at a wavelength of 1 mm as specified in ANSI Z136.1-2014 [B69] and IEC 60825-1 [B653].

B.7.2 Nonuniform exposure fields

From a dosimetry viewpoint, an important description of an exposure field is whether it is uniform or nonuniform. Uniform fields are those having a locally plane-wave character; that is, 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 wave impedance. Uniform fields exist in the far-field region of a radiating source (antenna) that is free from reflections from objects and the ground. At locations close to the source, exposure fields are usually nonuniform, their electric field and magnetic field polarizations are not well defined, and

the field strengths can vary in an oscillatory fashion with distance. In addition, the ratio of electric field strengths 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 can result in enhanced electric and/or magnetic fields close to the object surface. The enhanced fields are nonuniform 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 nonuniform exposure fields by theoretical methods. The reliable way to determine actual levels of these fields is by measurement.

B.7.3 Near-field versus far-field exposures and SAR

Depending on the distance from an RF source, a person can be exposed to RF energy in the near field or far field. Even in the far field, RF energy absorption in tissues is a complex function of many variables (Chou et al. [B255]). The absorption generally produces a nonuniform 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 configuration (there is no interaction or "coupling" between the source and the object). However, in the near field (closer than $2D^2/\lambda$), the body can 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 can 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 [B1360]). Kuster and Balzano [B798] 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 the 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 that is typically taken to be a distance equivalent to $\lambda/2\pi$, or approximately one sixth of the incident field wavelength (CENELEC [B399]). Table B.12 summarizes these distances within which the resulting whole-body SAR might not follow a direct relationship with the incident plane-wave-equivalent power density or the square of the electric field strength 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 field and magnetic field components do not exceed the corresponding ERLs. In some cases, however, alternative measures, such as induced body current, can 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 can be necessary.

Because of the 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 ERL as specified in Table 7 and Table 8 (see 4.3.2). 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, might be needed to determine compliance accurately; while WBA SAR should be conservatively estimated in the reactive near field by determining the electric and magnetic fields, local SAR might not be needed. If one can show compliance with field or power density measurements, no further SAR measurement is needed.

However, if SAR measurements show that the DRL is met, the ERL may be exceeded. For example, McCoy et al. [B982] 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 strength in the back seat exceeds the ERL. This situation complies with the standard because the DRLs have been met.

Table B.12—Estimated reactive near-field region of RF field sources within which SAR assessment might 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

B.7.4 Spatial considerations (peak versus whole-body average values)

Under conditions of nonuniform illumination, it is possible that the average field exposure over the whole body does not exceed the ERL, but it still results in excessive localized heating. To accommodate these circumstances, the ERLs 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 7 and Table 8 (see 4.3.2).

The choice of 4 cm^2 (for ERL between 6 GHz and 300 GHz) and 1 cm^2 (for high-power pulsed RF exposures) for the spatial peak averaging area was influenced by several factors. First, there is general agreement with other guidelines and standards including ICNIRP [B647] and ANSI Z136.1-2014 [B69]. Second, the smaller averaging area (1 cm^2) provides an additional level of conservatism for brief, high-power pulses, where the transient temperature increase at the skin shows a pattern similar to that of the epithelial power density.

B.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 local 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.

Nonuniform 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. Nonuniform exposures generally occur for sources that are close to the body, but they also can occur when the sources are at a distance for frequencies where the penetration depth is small.

A localized high SAR value produces localized heating and a localized increase in temperature. The magnitude of the temperature rise in a small region of body tissue is determined by the local 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. Studies using biophysical models for temperature distribution in a tissue heated by a localized source of RF energy have reported 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 [B1201]; Van Leeuwen et al. [B1442]; Wang and Fujiwara [B1474]) 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 °C 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. [B1442]).[42] This maximum temperature occurred superficially in the skin of the skull; temperature increases elsewhere in the head were lower. In comparison with 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. [B1442]). 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); and 2) the temperature in a small volume resulting from an RF exposure with a high SAR cannot be increased significantly compared with 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 B.4.1.2), the 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 nonuniform 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 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} (1 g 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. In IEEE Std C95.1-2005 [B668], from 3 GHz to 6 GHz, SAR is allowed to demonstrate compliance of local exposure. For this standard, ICES decided to set 6 GHz as the ending frequency for using SAR as the DRL. This is also harmonized with the new ICNIRP guidelines[43] in which 10 GHz used in the 1998 guidelines is lowered to 6 GHz.

B.7.6 Historical perspective on the evolution of the lower tier

This subclause provides a perspective on the development of the lower tier DRLs for whole-body and local exposure. The 1982 and earlier ANSI RF exposure standards had a single tier of exposure limits. The DRLs in ANSI C95.1-1982 [B68] were based on research demonstrating a 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 SAR of 0.4 W/kg. This DRL was considered to be a conservative limit, given the far greater thermoregulatory capacity of humans versus the laboratory animal species studied. For local exposure, the magnitude of the

[42] 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 a 10 g averaging volume are adequate to limit excessive local SAR.
[43] See footnote 12.

psSAR limit was derived from the WBA SAR limit based on studies showing the ratio of psSAR to WBA SAR to be ~20:1 (ANSI C95.1-1982 [B68]). A tissue mass of 1 g 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 on these decisions, the spatial peak SAR in ANSI C95.1-1982 [B68] was set at 8 W/kg (a value 20 times the WBA SAR limit of 0.4 W/kg) as averaged over any 1 g of tissue. Thus, the 1982 standard specified a single tier of exposure limits incorporating a safety factor of 10 that was deemed to be protective of all persons in the population.

The measurement of WBA SAR in the studies supporting the 1982 standard was accurate. Furthermore, WBA SAR represented the dosimetric quantity most meaningfully related to behavioral disruption, the effect still considered today to be the most sensitive biological indicator of potentially adverse health effects. Behavioral disruption in rats and nonhuman primates exposed to RF energy was often associated with a core body temperature increase of ~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 low-level effects. Even so, it was recognized that humans have a greater thermoregulatory capacity compared with laboratory animals, and for this reason, the limits were judged conservative and protective despite other uncertainties in the extrapolation of animal data to human beings.

By applying an additional safety factor of 5 to the original DRLs, a lower tier for people in “uncontrolled environments” was included in IEEE C95.1-1991 [B666], 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. During the development of the 1991 standard and the subsequent 1999 edition, the drafting committee 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 [B1059] 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). The rationale did not recognize that the worker is exposed as a member of the public 16 h/d and then possibly exposed at a higher level when working. IEEE Std C95.1-1999 [B667] maintained the original safety factor of 10 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. [B149]). This insight suggests that existing psSAR 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 psSAR limits.

Furthermore, contemporary dosimetric and state-of-the-art thermophysiological modeling that incorporates FDTD and realistic human and animal models (Bernardi et al. [B149]; Mason et al. [B976]) has shown that earlier experiments, from which SAR was derived from simplistic simulations using prolate spheroids, could have underestimated values by two times or more (Durney et al. [B375]). Such findings could imply that safety factors assumed for DRLs based on these data might have been half of what was initially thought (ANSI C95.1-1982 [B68]; NCRP [B1059]). 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 GHz to 3 GHz range (Dimbylow [B361]). These data, based on computational modeling,

indicate that the DRLs for children could be exceeded (i.e., the safety factor would be less than 50 in the 2 GHz to 3 GHz range). Discussions within ICES focused on the already inherently conservative DRLs 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 [B1084]). Despite similar arguments, this discussion resulted in a consensus within ICES to preserve the approximate 50-fold safety factor for the lower tier.

Finally, ICES understands that while safety factors have historically been defined in terms of SAR safety 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. [B149]). In summary, the ERLs in this revised standard are derived from prolate spheroidal models as in ANSI C95.1-1982 [B68] with adjustments based on more recent anatomical dosimetric models. Within the scientific uncertainties associated with the complex subject of RF dosimetry, the ERLs represent reasonable estimates of exposure values that yield SARs that do not exceed the DRLs 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 on ratios of peak to WBA SARs or other similar dosimetric constructs.

B.7.7 Adverse environmental conditions and workloads

RF exposure that results in energy absorption equivalent to the DRLs (0.08 W/kg or 0.4 W/kg whole-body average) imposes an additional thermal burden on the body. However, such a thermal load is below the resting metabolic rate of individuals (which is approximately 125 W for an average adult or the equivalent of about 65 W/m^2 of body area; Parsons [B1125]). The equivalent heat load represented by the upper tier DRL is comparable to approximately 40 % of the resting metabolic rate, which would be characteristic of very slight activity, for example, sedentary activity such as working at a desk (https://epi.grants.cancer.gov/atus-met/met.php). Consequently, the maximum thermal burden from RF exposure, even at the upper tier level, represents a small perturbation compared with those resulting from variations in activity level of an individual.

A recent study by Moore et al. [B1034] concluded that, "different environmental conditions had minimal impact on the magnitude of the thermal response due to RF-EMF exposure, and that the current safety factor of 10 applied in international RF-EMF safety guidelines and standards for RF-EMF workers is generally conservative, though it is only narrowly so when workers are subjected to the most adverse environmental conditions."

Consequently, ICES concluded that the DRLs are sufficiently protective, even at the upper tier for workers, in extreme environmental conditions. The larger issue of worker protection against heat strain under extreme environmental or work conditions is beyond the scope of this standard.

B.7.8 Presence of medical devices or metallic implants

Whenever an RF field impinges on a metallic object, reradiated 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 modest and would not be expected to cause any harmful tissue temperature rise for RF exposures at the ERL. 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 guidance should be considered:

a) The frequency of exposure, the shape of the implant, and its orientation with respect to the polarization of the *in situ* field all affect SAR distributions around the implant.

b) Linear implants that are oriented parallel to the *in situ* electric field produce resonant field enhancements around their tips when their length is around one third of the field wavelength in the tissue.

c) A loop-shaped metal implant that is oriented normal to the *in situ* magnetic field strength (H) can produce enhanced SAR in any gap in the loop.

d) Metal plates screwed on to bones that lie directly beneath the skin can enhance SAR in the skin at microwave frequencies due to constructive interference.

e) The reradiated fields around an implant tend to decay rapidly in a lossy dielectric tissue environment.

f) Field enhancements can occur around any sharp point in the implant, although these are often so localized that their influence is not noticeable in a 10 g averaging mass.

g) An implant is a passive reradiator and by itself cannot create additional RF energy absorption but can concentrate it. Thus, at some locations (e.g., near sharp metal points), can cause tissue damage. Thus, the overall RF heating in the vicinity of the implant generally remains about the same. One possible exception to this rule is the case of a large implant in one leg (e.g., a metal rod in the tibia), which by providing a lower impedance conductance path diverts additional current flow to that leg for exposure frequencies around and below whole-body resonance frequencies.

h) The potential for excessive local SARs around an implant is only realistic in parts of the body where *in situ* fields are already high. Thus, implants located in parts of the body that are deep seated are not problematic.

i) From a thermal perspective, the implant can act as a heat sink. Temperature variations around the localized parts of the implant due to SAR enhancements tend to be equalized by heat conduction through the implant. Furthermore, by displacing blood perfused tissue, the metallic implant can reduce the surrounding tissue temperature.

j) Some implants are located in a thermal environment where efficient heat transfer mechanisms 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.

k) Metal dental fillings are not normally regarded as problematic since any localized heating associated with exposures below the ERL 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 can 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 United States (FDA CDRH [B1430], ACGIH [B3]); Canada (Health Canada [B582]); United Kingdom (MHRA [B1426]); and Japan (Ministry of Public Management, Home Affairs, Posts and Telecommunications [B694]) suggest keeping a minimal separation distance between mobile phones and implantable cardiac devices of between 15 cm and 22 cm, but they 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.

B.7.9 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, cause an increase in body temperature, resulting in a feverish condition. This would add to the overall heat load on a person being overexposed 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 it 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 should be noted that no adverse effect of RF exposure to individuals under the influence of any of the drugs listed earlier would be possible unless the RF exposure was significantly above the ERL. At levels at or below the ERL, RF fields would not adversely affect any individual whether or not he or she used such drugs.

B.7.10 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 restricted environment limit of 0.4 W/kg). Consideration of this question should be related to the frequency at which the individual is exposed, with secondary considerations related to exposure characteristics. The depth of penetration, and whether or not RF energy even reaches the fetus or embryo, is directly related to the frequency. The exposure characteristics include field strength, near versus far field, potential for regions of peak SAR (electrical hot spots), 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 is absorbed in a highly nonuniform fashion, even for the ideal case of exposure to a uniform plane wave. Whole-body exposures in uniform far-field conditions 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 receive measurable levels of RF energy. At the restricted environment level, even at millimeter-wave frequencies, surface heating is 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 upper tier level (0.4 W/kg) at frequencies above 2 GHz, insufficient RF energy is 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, can be found in the differences between Figure 6.6 and Figure 6.7 in the 4th edition of the *RF Radiation Dosimetry Handbook* [B376] (figures not reproduced here). These figures represent calculated plane-wave 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 [B424]; see C.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 ERLs.

The results of a study (Adair et al. [B22]) 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 eight times the upper tier ERL 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 local and WBA SAR.

Each subject served in 12 test sessions during which the ambient temperature T_a was controlled at one of three levels (24 °C, 28 °C, and 31 °C). Three power densities (40 W/m^2, 60 W/m^2, and 80 W/m^2) 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 six sites, metabolic heat production, local sweat rate at two sites, and local skin blood flow at three or four sites. Because theoretical dosimetry indicated high RF energy absorption in the legs, the 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 four 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/m^2, 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 °C to 0.15 °C) due to the increased sweating and skin blood flow that were T_a dependent. Since individual skin temperatures (except for the ankle) changed hardly at all, it is clear that the physiological heat loss responses of increased blood flow and sweating must have been stimulated by thermoreceptors deep in the body, not by those located in the skin. These results indicate that thermoregulation proceeds normally when humans are exposed to RF energy at close to the resonant frequency even though the individual might not sense the presence of the RF field. This observation and the fact that core body temperature, even at levels eight times the ERL for the restricted environment, changed less than the typical diurnal body temperature fluctuation of humans suggests that an RF-induced thermal hazard to either a pregnant woman or to the embryo/fetus when the mother is exposed within allowed limits would be unlikely. Other studies involving localized RF exposure of human volunteers to 450 MHz and 2450 MHz at or above the upper tier ERLs, also support this finding (Adair et al. [B11], [B19], [B21], [B24]).

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 IEEE Std C95.1 limit for unrestricted environments (Adair et al. [B22]). 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. Furthermore, the data show that exposure at a level eight times the limit for restricted environments is essentially benign in terms of impaired thermoregulation. Thus, for women in the workplace, the IEEE Std 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. [B385]) supports the conclusion of absence of risk to the embryo/fetus upon exposure of the parent to RF energy at the ERLs and DRLs of this standard.

As mentioned earlier, 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 (six times and eight times the controlled limit) in the warmest environment (31 °C) (Adair et al. [B22]). The results

from this short-term exposure study indicate that the IEEE Std C95.1 RF exposure limits imply thermal comfort even when there is unusual heat loading from ambient thermal conditions and workload.

Regarding dosimetry, SARs have been calculated, using simple models of a pregnant woman, for exposures in the 80 MHz to 1500 MHz frequency range. Exposure of the model at the upper tier DRL (0.4 W/kg) resulted in SARs in the fetus greater than three times higher (0.27 W/kg versus 0.08 W/kg) than the DRL for the lower tier (Fleming and Joyner [B424]). 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. [B731]).

There are 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 there would need to be unhealthy ambient thermal conditions (i.e., high temperature, high humidity, and low air flow) that a lesser RF exposure might exacerbate, combining to produce a rise in body temperature of ~2 °C above normal. In addition to the adequacy of the ERL in this RF exposure standard to protect against a 2 °C 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 °C (ACGIH [B3]). Also, the results of animal studies are in good agreement with the human threshold (~2 °C) for fetal defects. A maternal temperature increase of ~2 °C to 2.5 °C was associated with abnormalities in the offspring of laboratory animals (Edwards et al. [B385]).

Studies in the medical literature related to tissue temperature elevations associated with hyperthermia applications also support the view that exposures that result in local energy absorption rates equivalent to the upper tier DRL of this standard should be safe for all humans including the fetus in pregnant women. Adibzadeh et al. [B34], using thermal modeling methods, examined the magnitude of the safety factor of 10 inherent to the IEEE standard relative to tissue damage. In their research, they compared temperature elevations in different tissues for given SAR values with the CEM_{43} thermal dose definition. Using different databases of thermal tissue properties that included thermoregulated perfusion in tissues, the authors found that the heating factors determined were dependent on the size of the targeted tissue and the length of the exposure and the state of perfusion. They determined that the arbitrary safety factor of 10 used in the IEEE standard provides a safety factor relative to tissue damage ranging between 10.9 and 31.2 for the most common hot-spot location as well as the tissue exhibiting the lowest SAR required for reaching the tissue damage level of temperature (i.e., muscle tissue). Using heating factors found by the authors, the upper tier DRL of this standard (10 W/kg) could be expected to increase the temperature of nonperfused tissue by approximately 0.5 °C and perfused tissue by approximately 0.3 °C in 5 cm to 20 cm muscle spheres simulating different sizes of fetuses, lending additional support to the argument that exposure at the upper tier of IEEE Std C95.1 should be protective against adverse health effects for both pregnant women and the fetus.

B.7.11 Use of mobile telephones by children

B.7.11.1 General

Concern about the use of mobile phones by children was documented in the 2000 report of the Independent Expert Group on Mobile Phones (IEGMP) titled “Mobile Phones and Health” [B673] and the NRPB report on “Mobile Phones and Health 2004” [B1085]. 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 several studies involving RF exposure during gestation through young adulthood that are considered to be relevant to the use of mobile phones by children (see C.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 much 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 [B1198]). 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 after exposure *in utero* and during the first year of life. To emphasize studies of particular relevance for children's use of mobile phones, literature previously reviewed in C.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: 1) the developing nervous system, 2) long-term exposure including lifetime exposure, and 3) greater absorption of RF energy in the young.

B.7.11.2 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 °C to 2.5 °C, mostly following exposures of tens of minutes up to 1 h or so. Higher temperature elevations, of up to ~5 °C, are effective at shorter exposure durations (Edwards et al. [B385]). 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 MHz) of the pregnant rat on day 9 of gestation caused encephalocele, microphthalmia, and other defects in the head of fetus (Brown-Woodman et al. [B203]). By increasing the duration of RF exposure to elevate the maternal body temperature from 2.5 °C (no abnormalities) to 5 °C, the incidence of these defects increased. The authors noted that the teratogenicity of RF energy deposition is primarily related to hyperthermia because the RF-induced defects were similar to those obtained by heating rats on the same day of gestation in a water bath (Brown-Woodman et al. [B203]). 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. [B201]) and to very low-level CW RF fields (Tofani et al. [B1412]). 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 [B710], [B711]; Jensh et al. [B712], [B713], [B714], [B715]). Pregnant rats were exposed for ~20 % of the total gestation period of 21 days. In the teratology studies at 3.6 W/kg (915 MHz), 3.6 W/kg to 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 W/kg to 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 [B710]), 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."

After prenatal exposure or pre- plus postnatal exposure, 30-day-old 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. [B487]). The maximum fetal exposure was 4 W/kg (3 h/d from days 5 to 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 to males). The fetus could have received up to 4 W/kg for 3 h/d from days 5 to 20 of gestation. After birth, the pups were exposed from 2 days to 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 and Carter [B138]; Berman et al. [B146], [B139]; Jensh [B710]).

Young mice were evaluated for development on 1 day, 5 days, 10 days, 12 days, 15 days, and 17 days of age after *in utero* exposure at 16.5 W/kg for 100 min on days 6 to 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. [B139]). 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 days, 20 days, 30 days, and 40 days of age after prenatal and postnatal exposure (3 h/d, 2450 MHz) from day 4 of gestation to 40 days of age (except for two days; Inouye et al. [B681]). The *in utero* exposure of 1.76 W/kg to the pregnant animals occurred on days 4 to 21 of gestation. In offspring aged 15 to 40 days, the brain SAR ranged from 1.9 W/kg to 9.5 W/kg. The brain development markers were the cortical architecture of the cerebral cortex and hippocampal formation, the germinal layer along the lateral ventricles, myelination of corpus callosum, and the external germinal layer of the cerebellar cortex. In addition, in 40-day-old rats, quantitative measurements of neurons were made (i.e., spine density of the pyramidal cells in cortex). Other endpoints included the density of the Purkinje cells and the extent of the Purkinje cell layer in the cerebellum. This extensive investigation of mammalian brain development after exposure of the rat prenatally and postnatally at 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. [B681]).

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 [B48]; Albert et al. [B47]). The primates were exposed at 2450 MHz and 3.4 W/kg (3 h/d, 5 d/wk), 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. [B47]) 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 d/wk) from days 2 to 18 of gestation at 0.4 W/kg (2.45 GHz). No microcephalous 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. [B1002]). 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 DRL (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 W/kg to 0.075 W/kg. The offspring were tested as adults (11 weeks to 12 weeks of age) for learning deficits. No measurable cognitive deficits were observed (Bornhausen and Scheingraber [B180]).

B.7.11.3 Studies of other physiological changes possible after long-term RF exposure

B.7.11.3.1 CNS effects

In addition to the studies described earlier 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. [B32], [B33]; Anderson et al. [B66]). 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. [B32], [B33]); the measured peak brain SAR ranged from 1.8 W/kg to 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 d/wk) 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 d/wk, 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 W/kg 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. [B66]). The results of these three long-term exposure studies provide no support for the hypothesis that the tested forms of RF energy act as a carcinogen or a cancer promoter in CNS tissues, including the brain, when RF exposure occurred during critical periods of CNS development in the fetus, as well as throughout young and adult life.

B.7.11.3.2 Blood-brain barrier, body weight, and other biological studies

Another lifetime study examined BBB permeability in mice exposed for 1 h/d, 5 d/wk, for two years at four SAR levels (0.25 W/kg, 1 W/kg, 2 W/kg, 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. [B419]). 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. [B314]; see C.6.1). 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 after almost continuous 970 MHz exposure during gestation (22 h/d during days 1 to 19) showed a decrease (12 %) in fetal body weight at 4.8 W/kg but no effect at 2.4 W/kg and 0.07 W/kg (Berman et al. [B146]). In a related study, no change in fetal weight was seen in 22-day rat fetuses after exposure of pregnant rats at 3.6 W/kg (915 MHz) for 6 h/d during days 1 to 21 of gestation (Jensh et al. [B715]). At a higher frequency (6 GHz), an exposure of 7.3 W/kg for ~20 % of the gestational period was sufficiently intense to decrease fetal body weight (Berman and Carter [B138]; Jensh [B710]). Another study reporting reduced weight of fetal rats after exposure at 2450 MHz and 6 W/kg for 100 min/d during days 6 to 15 of gestation (Berman and Carter [B138]) 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 W/kg, 0.34 W/kg, and 3.4 W/kg) for 3 h/d, 5 d/wk beginning the second trimester of pregnancy (Kaplan et al. [B736]). 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, electroencephalogram (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, very few animals in this group were 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. [B736]). 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. [B736]).

B.7.11.4 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 WBA 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 (Anderson [B67]; Bit-Babik et al. [B159]; Foster and Chou [B428], [B427]; Gandhi and Kang [B495]; Gandhi et al. [B494]; Hadjem et al. [B556]; Martinez-Burdalo et al. [B974]; Schonborn et al. [B1263]; Wang and Fujiwara [B1474]; Wang et al. [B1476]). The consensus from the more recent studies is that the size and shape of children's heads do not cause a significant difference in peak SAR compared with the adult for exposed tissues of the head. It is true that due to the thinner skulls of children, the SAR in the brain is higher in children than adults, but the SAR in the brain is always lower than the peak SAR in 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; that is, 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. [B1443]).

B.7.11.5 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 whole-body 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. [B1443]). 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 (Albert et al. [B49]) includes the statement that, "The scientific evidence does not show a danger to users of wireless phones, including children and teenagers." Thus, in 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 studies have found no effect on RF exposure from mobile phones on cognitive function in children (Haarala et al. [B552]; Preece et al. [B1160]).

B.7.12 Macular degeneration

The question of whether a person suffering from macular degeneration would be at increased risk from a temperature increase from exposure where the local SAR is below the DRL for psSAR 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 psSAR of this standard (2 W/kg and 10 W/kg for the lower and upper tier, respectively) should not be considered problematic for the production of or worsening of macular degeneration.

Annex C

(informative)

Identification of levels of exposure associated with adverse effects—summary of the literature

C.1 Introduction

In IEEE Std C95.1™-2005 [B668], Annex B is a summary of the literature based on critical reviews of studies within the IEEE radio frequency (RF) literature database.[44] Detailed review papers were drafted for 12 general subject areas by individual members of IEEE ICES TC95/SC4 and were published together at the end of 2003 as Supplement 6 of the journal *Bioelectromagnetics* [B155].[45]

The literature review conducted for IEEE Std C95.1-2005 [B668] 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 as "nonthermal" 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 false-positive effects even at lower RF field levels.

As stated in A.1.6, the International Committee on Electromagnetic Safety (ICES) literature review working group (LRWG) followed the systematic review guidelines developed by ICES and took advantage of many expert reviews plus the World Health Organization (WHO) environmental health criteria to come up with comprehensive reviews supporting the current revision. Since all expert reviews confirm the protectiveness of the current limits, and the fact that the only changes in limits in this standard are the dosimetric reference limits (DRL) and exposure reference levels (ERL) above 6 GHz, in this annex, only reviews of scientific papers dealing with effects at frequencies higher than 6 GHz are included. When the WHO "Environmental Health Criteria" (EHC) document becomes available, the LRWG will provide a summary of the EHC literature review, provide comments, and review any papers missing in the WHO EHC document that are considered relevant. This will be used to update this annex in the next revision of this standard or via an addendum.

A review of the extensive literature on biological effects of electromagnetic fields reveals adverse health effects can occur as electrostimulation at low frequencies and thermal effects at high frequencies. This conclusion is consistent with those reached by other scientific expert groups and government agencies including the following reviews or reports published up to the end of 2017:

- American Cancer Society [B63]
- Australian Radiation Protection and Nuclear Safety Agency [B91]
- European Code Against Cancer, 4th Edition [B404]
- European Commission's Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) [B407]
- European Economic and Social Committee [B408]

[44] http://ieee-emf.com/
[45] https://onlinelibrary.wiley.com/journal/1521186x

- Health Canada [B582], [B584]
- Health Council of the Netherlands [B588], [B587], [B590]
- IEEE Committee on Man and Radiation [B663]
- Institution of Engineering and Technology [B684]
- International Agency for Research on Cancer [B686]
- Japanese government research program [B1390]
- New Zealand Ministry of Health [B1071]
- Nordic Radiation Safety Authorities [B1078]
- Royal Society of Canada [B1221]
- Swedish Radiation Safety Authority [B1367], [B1369], [B1370], [B1371]
- U.S. Centers for Disease Control and Prevention [B1434]
- U.S. Federal Communications Commission [B1436]
- U.S. Food and Drug Administration [B1431]
- U.S. National Cancer Institute [B1437]
- World Health Organization [B1494], [B1495]

Further examination of the RF literature reveals no confirmed adverse health effects below current exposure limits that would occur even under unusually high heat loads from ambient thermal conditions and workload. The scientific consensus is that no accepted theoretical mechanisms exist that would suggest the existence of such effects. This consensus further supports the analysis presented in this annex, that is, that established harmful effects 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), which 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 [B68], 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 ERLs and DRLs in this standard are broadly protective against established adverse health effects.

C.2 Identification of levels of RF exposure responsible for adverse effects—Summary of the literature (IEEE Std C95.1-2005, Annex B)

C.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 nonhuman primates, and under diverse exposure conditions. The disruption of behavior is often (but not always) accompanied by an increase in core body temperature of ~1.0 °C. These accommodating responses to a thermal challenge, while not considered detrimental, can be compared with 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 deemed significant in establishing the basis for this standard although it falls in the innocuous range. Furthermore, 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 shorter depth of penetration at the higher frequencies deposits energy closer to the skin where most thermoreceptors are located. The threshold of cutaneous thermal pain is 44 °C to 45 °C and, if generated by RF energy, results in a prompt effort to escape from the field, thereby preserving normothermia.

C.2.2 Noncancer-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 °C to 2.5 °C. This mostly occurs after exposures of tens of minutes up to 1 h 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 they 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 ERLs 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 (nonthermal) exposure levels (i.e., below the ERLs), 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 it can fail to support a repeatable low-level effect. Based on computational modeling studies, the localized exposure limit for the lower tier produces an increase in brain temperature of ~0.2 °C (Bernardi et al. [B149], [B150]; Gandhi and Kang [B495]; Van de Kamer and Lagendijk [B1440]; Van Leeuwen et al. [B1442]; see C.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 SAR < 4 W/kg have not been confirmed and no exposure- or dose-response relationship is evident.

Adverse effects of RF exposure of the eye (e.g., cataracts) are associated only with significant temperature increases due to the absorption of RF energy. There is no evidence of other significant ocular effects (including cancer) that would support a change in the adverse effect level of 4 W/kg.

The phenomenon of RF hearing in humans is a well-established biological effect with no known adverse health consequence. The RF-induced sounds are similar to other common sounds. A quiet environment is needed for the sounds to be heard.

A few studies have reported effects of RF exposure on *in vitro* membrane function and protein leakage through artificial and cellular membranes. However, significant variability and a lack of a consistent correlation with SAR were common in these responses. It is possible that the RF exposure resulted in local temperature increases, which might 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 [B1495] 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. A National Radiological Protection Board (NRPB) report [B1087] 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 continuous-wave (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. Several 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 noncancer 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 unreplicated 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, electroencephalogram (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.

C.2.3 Cancer-related studies

Long-term animal studies including lifetime exposures up to 4 W/kg did not provide evidence of physiological, pathological, or disease-specific effects. Some positive studies are followed up with a better exposure system and dosimetry, and the effects are not repeated. These long-term studies indicate a lack of clear evidence that RF exposure causes or promotes tumor induction or any other life-shorting disease. Chronic RF exposures at SARs in the range of 0.5 W/kg to 4 W/kg also did not result in an adverse effect on longevity or body mass.

A review of numerous supportive studies addressing cancer and basic cellular interactions show no consistent evidence for a reproducible biological effect of low-level (nonthermal) RF exposure. These studies include examination of DNA breaks, mutation, specific DNA absorption, chromosome aberration induction, micronucleus formation, sister chromatid exchange induction, DNA repair synthesis, inhibition of DNA repair synthesis, phenotypic mutagenesis, transformation, cell cycle elongation, cell toxicity, proliferation, growth rate, cell cycle analysis, gene and protein expression and activity, and oxidative stress. The majority of studies report no effect. The magnitude of the reported effects are generally very small, often in the range of variability that normally occurs in clinical laboratory tests ordered by physicians, and thus, the direct health implication of such reports would 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 general absence of potentially adverse effect results of the long-term animal studies.

C.2.4 Electrostimulation and effects below 100 kHz

At frequencies below 100 kHz, electrostimulation reaction thresholds are typically 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 root-mean-square (rms) value] is proportional to the square root of the duty factor.

C.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 the 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 might not have confident support from the experimental literature. However, a speculative mechanism can be useful for designing experiments that allow for an experimental determination of biological activity. A well-established effect on biological cells might be speculative because its application to intact human beings is not currently 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.

Several speculative and established mechanisms of RF interactions with biological systems have been proposed and representative samples are listed as follows:

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 100 GHz

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) Nonresonant interactions (dynamical, chemical, statistical, etc.)
   1) Electric dipole interaction (E)
      i) Cooperative interactions (including dipole-dipole interactions suggested by Fröhlich [B468])
      ii) Magnetic dipole interaction, for example, atomic magnetic moments and 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, and so on
   4) Anomalous energy diffusion via normal modes
   5) Nonequilibrium 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

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.

The speculative mechanisms among those listed 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 [B27], [B28], [B29]; Astumian et al. [B86]; Weaver and Astumian [B1483]). Establishment of the mechanisms (e.g., electrostimulation and thermal) that can 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.

C.4 Improvements in dosimetry

Accurate dosimetry is essential for an understanding of biological effects, since even uniform exposures lead to nonuniform 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 estimate *in situ* electric fields and SARs reliably 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 magnetic resonance images (MRI). 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 B.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 might be important for assessment of a biological effect.

It bears emphasis that SAR is a measure of the time 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 can 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 distributions of electrical forces that influence excitable membrane effects are much 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.

C.5 Established effects forming the basis of this standard

C.5.1 General

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.

C.5.2 Thermoregulation

C.5.2.1 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 °C to 40 °C, and it 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. [B954], [B955], [B956]; Webster [B1487]). 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 can occur. At this level, protein denaturation can begin and cells can be damaged. Sustained

exposure to this level in humans often leads to irreversible neurological and cardiac damage (Britt et al. [B198]; Hales et al. [B558]; Mambo et al. [B960]).

Other consequences of severe and prolonged hyperthermia include confusion, unconsciousness, increased heart rate, lowered blood pressure (Gathiram et al. [B507]), elevated enzyme activity, and damage to the heart and kidneys. Thermoregulatory responses can cease above 43 °C (heat stroke), after which body temperature can 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 they can be described quantitatively based on Arrhenius analysis (Dewhirst et al. [B355]). 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 °C to 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 [B9]). 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 [B304]), peripheral and deep blood vessels dilate causing peripheral and/or local blood flow to increase as much as 10-fold (Adair [B5]; Adair et al. [B25]; Bruce-Wolfe and Adair [B205]; Candas et al. [B225]; Gordon [B520]; Gordon et al. [B528], [B529]; Jauchem and Frei [B696]; Lotz and Saxton [B920], [B921]). 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 [B575]). Sweating is activated when the ambient temperature rises above ~30 °C or the internal body temperature rises above ~37 °C (Wenger [B1491]), although the rate of sweating can 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 initially rise and then stabilize at an elevated level. If thermoregulatory mechanisms are impaired, the maximal SAR at which thermal equilibrium can be maintained is 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, and so on. 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 [B1074]). 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 a background RF of less than 10 mW/m^2 (1 $\mu W/cm^2$) (Mantiply et al. [B966]; Tell and Mantiply [B1396]). At the resonant frequency range for humans, this would represent a WBA SAR of 0.0004 W/kg, or ~0.03 % of the normal resting metabolic rate. Even the current WBA SAR limit of 0.4 W/kg for exposure in a restricted environment represents only 35 % of the resting metabolic rate in humans. Heating at this level would be comparable to donning a light sweater and would be of little or no physiological significance during most daily activities.

The potential effects of RF exposure, and the mechanism of elevated body temperatures in febrile individuals, should 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) brain-stem 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 [B1300]). Stitt [B1351] 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 et al. [B14] 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 [B1156] 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 should be performed cautiously as smaller animals, particularly rodents, require a high metabolic heat production to maintain thermal balance. This is due to their larger surface-area-to-volume ratio and lack of efficient mechanisms for heat dissipation (Gordon [B520], [B521], [B524]). 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 (Adair and Adams [B9]; Adair et al. [B14], [B25], [B26]; Candas et al. [B225]; Frei and Jauchem [B439]; Frei et al. [B437], [B442], [B443], [B444]; Gordon [B520], [B521], [B523], [B524], [B525]; Gordon and Ali [B526]; Gordon and Ferguson [B527]; Gordon et al. [B528], [B529]; Guy et al. [B549]; Ho and Edwards [B629]; Jauchem and Frei [B695], [B698]; Jauchem et al. [B695], [B700], [B702], [B703], [B704], [B705]; Lu et al. [B926]; Morrissey et al. [B1042]; Phillips et al. [B1148]; Walters et al. [B1469], [B1471]). Studies on rats (Spiers and Adair [B1337]; Chou et al. [B265]) and monkeys (Adair et al. [B25]) 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 (Hesslink et al. [B602]; Lloyd and Olsen [B912]; Olsen [B1101], [B1102]; Olsen and David [B1104]; Olsen et al. [B1099]; Pound [B1156]). Thermoregulation in nonhuman primates has been studied in detail (Adair [B4], [B8]; Adair and Adams [B9], [B10], [B11]); Adair et al. [B12], [B25], [B26]; Bruce-Wolfe and Adair [B204]; Candas et al. [B225]) and has shown that while thermoregulation is somewhat less efficient in response to RF exposure at resonance (Adair et al. [B10]; de Lorge [B334]; Krupp [B788]; Lotz and Saxton [B920], [B921]), 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 [B7]). Computerized thermoregulatory models, based on physiological data, have predicted human thermoregulatory responses with good accuracy (Adair and Berglund [B14], [B15], [B17]; Stolwijk [B1353]). Exposures of neonates have demonstrated the young rat's ability to maintain a constant body temperature through efficient thermoregulatory mechanisms (Guillet and Michaelson [B544]; Spiers and Adair [B1336], [B1337]). In studies of sheep exposed to MRI, involving head and WBA SARs of up to 4 W/kg for 20 min to 104 min, no apparent adverse consequences or significant core body temperature increases were observed (Barber et al. [B107]). 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 [B520]).

RF exposure can influence the action of various psychoactive drugs, ethanol, corticosteroids, anesthetics, and other agents that normally influence the thermoregulatory balance (Blackwell [B170]; Cleary and Wangemann [B284]; Hjeresen et al. [B626], [B627]; Jauchem et al. [B695], [B700]; Lai et al. [B808], [B810], [B811], [B812], [B813], [B814], [B815], [B816], [B817], [B818], [B819], [B821], [B822], [B823], [B826]; Lotz and Michaelson [B918], [B919]; Michaelson [B1007]; Putthoff et al. [B1165]; Smialowicz et al. [B1315], [B1319]; Spiers et al. [B1336]). 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. [B1336]) 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 [B6]; Adair et al. [B19], [B20], [B21], [B24]; Walters et al. [B1469]). Whole-body exposures at 100 MHz and 220 MHz and partial-body exposures at 450 MHz 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. [B19], [B20], [B21], [B23], [B24]); 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 [B667]. No consistent difference in response to pulsed-wave

(PW) and CW exposures at comparable average field strengths has been observed (Lu and de Lorge [B926]). Humans exposed to MRI (64 MHz, peak SARs of 2 W/kg to 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 [B14], [B15]; Gordon [B520]; Schaefer [B1255]; Shellock [B1287]; Shellock and Crues [B1288], [B1289]; Shellock et al. [B1293], [B1294], [B1296]). Local high-power RF exposures were used in China to heat testicular tissue to 40 ºC to 42 ºC for short periods of time for human contraceptive applications (Chiang et al. [B252]; Liu et al. [B908], [B908]) with no apparent adverse or long-term tissue effects. Science-based simulation models of human physiological responses have predicted that the scenario after 100 W of power were deposited in the head for 30 min, 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 [B6]; Adair and Berglund [B15]; Stolwijk [B1353], [B1354]; Stolwijk and Hardy [B1356]). Even with skin blood flow restrictions of up to 67 %, an MRI scan of the trunk at an SAR = 4 W/kg for 40 min 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 and Westerman [B635]; Hocking et al. [B632]), 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 [B1346]) and millimeter waves (Frei et al. [B445]; Ryan et al. [B1222]) 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 are 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 (Adair et al. [B20], [B22]; Blick et al. [B171]; Cook [B295], [B296]; Eijkman and Vendrik [B387]; Hendler [B598]; Hendler and Hardy [B598]; Hendler et al. [B600]; Justesen et al. [B727]; Michaelson [B1006]; Riu et al. [B1202]; Schwan et al. [B1268]; Sienkowicz et al. [B1308]; Vendrik and Vos [B1447]; Walters et al. [B1469]), using brief exposures (≤ 10 s) and exposures of restricted areas of the forehead, back, or forearm skin. In general, the shorter the wavelength, the less energy that is required to produce a cutaneous thermal sensation. Using Pennes' bioheat equation as the basis for a theoretical analysis, Riu et al. [B1202] 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 ºC ± 1.0 °C (Cook [B295], [B296]), although this threshold depended on the area exposed, exposure time, initial skin temperature, anatomical site, and thermal conductivity.

C.5.2.2 Summary of thermoregulation

Significant core temperature increases (on the order of ~1 °C or more) can be induced in laboratory rodents and nonhuman 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 ºC to 47 °C, and if generated by RF energy, the pain results in a prompt effort to escape from the field to preserve normothermia.

C.5.3 Animal behavior, neurochemistry, and neuropathology

C.5.3.1 Animal behavior

C.5.3.1.1 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 C95.1-1982 [B68]; ICNIRP [B646]; NCRP [B1060]) and studies of human thermal sensation of RF exposures (Adair et al. [B20], [B22]; Blick et al. [B171]; Brown et al. [B199]; Cook [B295], [B296]; Eijkman and Vendrik [B387]; Hendler [B598]; Hendler and Hardy [B598], [B600]; Justesen [B726]; Justesen et al. [B724]; Michaelson [B1006]; Riu et al. [B1202]; Schwan et al. [B1268]; Vendrik and Vos [B1447]; Walters et al. [B1469]) 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 (Frei et al. [B445]; Guy and Chou [B545]; Justesen [B724]; Modak et al. [B1029]; Phillips et al. [B1148]). RF effects on behavior, however, can reflect an animal's attempts to engage in other thermoregulatory activities (Stern [B1344]). Furthermore, hot spots generated in certain parts of the body at nonresonant frequencies and in locations where blood flow is minimal (D'Andrea et al. [B319], [B320], [B321]; Gandhi [B490]; Grandolfo et al. [B535]; Lin et al. [B892]), as well as RF hearing effects that occur with high peak pulses (see C.6.5), might 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 MHz to 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 (D'Andrea et al. [B322], [B323]; de Lorge and Ezell [B338]; Gordon [B520], [B521], [B523], [B524], [B525]; Gordon and Ali [B526]; Gordon and Ferguson [B527]; Gordon et al. [B528], [B529]; Mitchell et al. [B1021], [B1022]; Smialowicz [B1315], [B1319]). In a series of studies, de Lorge and colleagues disrupted learned behavior in mice, rats, and monkeys with acute RF exposures at various frequencies (D'Andrea and de Lorge [B316]; de Lorge [B334], [B337]; de Lorge and Ezell [B338]; Knepton and de Lorge [B761]; Knepton et al. [B762]; Nelson [B1068]; Sanza and de Lorge [B1250]). WBA SAR greater than ~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 greater than ~4 W/kg with either CW or high peak power pulses (Akyel et al. [B39]; Bornhausen and Scheingraber [B180]; Brown et al. [B199]; D'Andrea et al. [B315], [B324], [B325]; Quock et al. [B1166], [B1167]; Schrot et al. [B1266]). Most studies at low levels of RF exposure, and even some at thermal levels, report no effects on behavior (Akyel et al. [B39]; Gage [B475]; Gage and Guyer [B477]; Gage et al. [B476]; Lebovitz [B846], [B847]; Liddle et al. [B878]; Sagan and Medici [B1229]; Thomas et al. [B1401], [B1405], [B1406]), although positive reports of behavioral changes at near-thermal (Schrot et al. [B1266]) and apparent nonthermal acute (Frey and Spector [B463]) and chronic (Bruderer and Bolt [B207]) exposure levels do exist. Studies of acute RF exposure effects on cognitive performance generally report no effects (Dubreuil and Edeline [B372]; Sienkowicz et al. [B1305]) unless exposures reach the thermal range (Mickley and Cobb [B1012]; Mickley et al. [B1013]; Thuroczy et al. [B1409]) 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. [B826]; Wang and Lai [B1472]). The high peak pulses used in these later studies might have generated RF hearing effects. Recent and well-documented efforts by two laboratories to confirm the maze result were unsuccessful (Cassel et al. [B239]; Cobb et al. [B285]; Cosquer et al. [B299]).

Some enhancement of active and passive avoidance behavior in mice acutely exposed to RF energy at thermal levels has been reported (Beel [B128]; Luttges [B939]), while continued daily repeated exposures lead to performance deterioration (Beel [B128]). The ability of acute high peak pulsed RF energy to influence

aversive and escape behavior has produced equivocal results (Carroll et al. [B236]; Justesen [B724], [B725], [B726]; Justesen et al. [B728]; King et al. [B756]; Levinson et al. [B861]; Monahan and Henton [B1031], [B1032]; Monahan and Ho [B1033]). 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 [B724] has suggested that the inability of animals to learn an escape response in the presence of intense RF fields indicates 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 [B460]; Lebovitz [B847]; Thomas et al. [B1405]), although the possibility of an RF auditory effect specific to high peak power PW exposures in these later studies cannot be ruled out (Stern [B1344]).

Acute RF exposure can affect changes in thermoregulatory response and behavior as well (Adair [B4], [B8]; Adair and Adams [B9], [B10], [B11]; Adair et al. [B12], [B14], [B25]; Berglund [B133]; Candas et al. [B225]; Gordon [B520]; Lotz [B917]; Lotz and Saxton [B917]; Lu et al. [B926]; Nielsen and Nielsen [B1074]; Shimada and Stitt [B1300]; Stern et al. [B1345]; Vitulli et al. [B1458], [B1459]). In studies with resonant versus nonresonant 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 (Bruce-Wolfe and Adair [B204]; Marr et al. [B973]; Vitulli et al. [B1458], [B1459]). Studies have also reported on the ability of acute RF exposures to interact with the thermoregulatory action of various drugs (Lai et al. [B808], [B810], [B811], [B812], [B813], [B814], [B815], [B816], [B817], [B818], [B819], [B820], [B821], [B822], [B823], [B826]; Lotz [B917]; Lotz and Saxton [B917]; Monahan and Henton [B1031], [B1032]; Monahan and Ho [B1033]; Thomas [B1403]; Thomas et al. [B1401], [B1404]).

Reports on the effects of chronic low-level RF exposure have been generally negative (Chou et al. [B265]; D'Andrea et al. [B317], [B318], [B322], [B323]; DeWitt [B356]; Lebovitz [B846], [B847]), although positive effects at near-thermal levels have been reported (Mitchell et al. [B1023]). Reports from Eastern Europe and the Soviet Union (summarized in D'Andrea and de Lorge [B316]) 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. [B487]; Kaplan et al. [B736]), 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 [B709], [B710], [B711]; Jensh et al. [B712], [B713], [B714], [B715]; O'Connor [B1092]).

C.5.3.1.2 Summary of animal behavior studies

A threshold WBA SAR of ~4 W/kg for disruption of complex behavioral performance in several animal species, including nonhuman 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 W/kg and 4 W/kg, depending on the frequency and the size of the animal. Essentially all behavioral changes due to RF exposure at these levels are reversible, and no consistent evidence exists for long-term or permanent effects. Thermoregulatory behavior in the presence of RF fields appears to be efficient in most species and under most conditions, even at SARs equal to twice the resting metabolic rate, although exceptions might 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 on perception of intense RF fields, the animal data might tend to underestimate the threshold levels for safety for humans.

C.5.3.2 Neurochemistry

C.5.3.2.1 General

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 [B1327]); lower concentrations of norepinephrine, serotonin, and dopamine (Merritt et al. [B1002], [B1004]); changes in norepinephrine and acetylcholine (Gandhi and Ross [B498]); and reduced norepinephrine, increased 5-hydroxyindoleacetic acid, and no change in serotonin (Inaba et al. [B678]). Reduced brain acetylcholine levels were measured in rats after RF exposure producing brain temperature increases of 2 ºC to 4 ºC (Modak et al. [B1029]) and at 6.5 W/kg but not at 3.5 W/kg (2450 MHz CW) and 0.3 W/kg (800 MHz) (Testylier et al. [B1399]). Mausset et al. [B978] showed that SARs of 4 W/kg and 32 W/kg reduced gamma-ami-nobutyric acid (GABA) levels in the rat cerebellum. Under exposure conditions (2.86 GHz PW, 10 mW/cm^2 for 4 h/d, 5 d/wk, for up to 4 weeks 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. [B1540]). Browning and Haycock [B204] showed that neither acute nor chronic RF exposure at nonhyperthermia levels had any effect on rat brain synapsin I, an indicator of neurotoxicity.

Lai [B807] 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 WBA exposure of 0.6 W/kg, as follows: 1) exposure enhanced morphine-induced catalepsy in the rat (Lai et al. [B825]); 2) exposure attenuated the naloxone-induced, wet-dog shake, a morphine withdrawal symptom, in morphine-dependent rats (Lai et al. [B823]; 3) narcotic antagonist blocked a transient increase in body temperature after exposure (Lai et al. [B824]); 4) the effect of acute exposure on amphetamine-induced hyperthermia (Lai et al. [B808]) and ethanol-induced hypothermia (Lai et al. [B821]) 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. [B819], [B810]); 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. [B814]); and 7) three major subtypes of opioid receptors are involved in the effect of RF exposure on HACU (Lai et al. [B813]). 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. [B817]), and waveforms. An example of the latter was the finding that HACU was affected by PW fields and not by CW fields. As explained by Lai [B807], the differential effect due to waveform was possibly a result of the auditory response to pulsed RF fields (see C.6.5).

In addition to the studies on cholinergic systems mentioned, Lai published other studies on these systems because of their role in many physiological and behavioral functions (Lai et al. [B808], [B809], [B810]. 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, which was a response similar to acute restraint-induced stress (Lai et al. [B819], [B822]). A learning deficit was found to be correlated to the decrease in cholinergic activity (Lai et al. [B811]). 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. [B814]). All three subtypes of opioid receptors were affected (Lai et al. [B810], [B812]). Based on his results, Lai [B812] 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 the corticotropin-releasing factor, which in turn caused a decrease in activity of cholinergic innervations in the frontal cortex and hippocampus (Lai et al. [B808]). The endogenous opioids, via three receptors, are the intermediate step before the hippocampal change occurs. The activation process might be a stress response. Lai et al. [B813] 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 [B807] speculated that low-level RF exposure is a "stressor" (Lai et al. [B808]) because of the similarity

of RF effects and those of established sources of stress and 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. [B933] 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 mW/cm^2 to 70 mW/cm^2 for 1 h to 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 finding is consistent with the observation that none of the endocrine changes occurred without a thermogenic RF exposure.

In other neurochemical studies, Hjeresen et al. [B627] investigated the effects of RF exposure on ethanol-induced interactions with neurotransmitter systems and Monahan [B1030] reported that 1 W/kg and 10 W/kg affected the cholinergic drug scopolamine and physostigmine on shock latency and motor activity of mice. The results from the latter study suggest RF enhancement of cholinergic activity (D'Andrea et al. [B314]). Ashani et al. [B83] 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 and Joines [B1239] and Sanders et al. [B1240], [B1241], [B1242] 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. [B253]) and that pulsed RF fields induced subtle changes in succinate dehydrogenase levels in the developing mouse brain (Chiang and Yao [B251]).

In an *in vitro* study, Gandhi and Ross [B488] described changes in the metabolism of inositol phospholipids in rat brain synaptosomes exposed at 10 and 30 W/kg. Millar et al. [B1020] 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 W/kg to 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. [B377] reported different responses including increased and decreased AChE activity and no effect over a range of SARs from 0.001 W/kg to 0.1 W/kg. In young rats exposed at 0.1 W/kg to 0.4 W/kg, decreased brain AChE was found (Kunjilwar and Benhari [B795]).

Mausset et al. [B979] 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 after 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/d, 5 d/wk 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. [B352]) and no effect was measured on melatonin in subjects exposed at the maximum power of commercially available mobile phones (de Seze et al. [B350]). Mann et al. [B964] 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. [B1169] 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-h periods (across night and day) in a double blind study. In rats and hamsters exposed to 900 MHz (CW and PW) at 0.04 W/kg to 0.36 W/kg, Vollrath et al. [B1461] also failed to find nocturnal melatonin changes. Reviews that address neurochemical effects of RF exposure include Michaelson et al. [B1009], Lai [B807], Vander Vorst and Duhamel [B1441], Hermann and Hossmann [B601], Hossmann and Hermann [B639] and D'Andrea et al. [B314].

C.5.3.2.2 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 nonhyperthermic RF levels, for example, the effects on brain energy metabolism (Sanders et al. [B1240], [B1241], [B1242]), have not been confirmed/replicated by independent investigators. Some effects were reported to occur after pulsed, but not CW, RF exposure (Lai [B807]). It is known that the auditory system is sensitive to pulsed RF energy (see C.6.5), and Lai [B807] 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. [B808]) because of the similarity of RF effects and those of established sources of stress, Lai [B807] 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 earlier show no changes in a variety of neurochemicals after exposure of the head to pulsed 900 MHz and 1800 MHz signals used in telecommunications.

C.5.3.3 Neuropathology

C.5.3.3.1 General

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. [B520]). A study in the Western literature, however, found no histologic changes after acute RF exposure causing brain temperature increases of 4.4 °C to 6.5 ºC (Lin et al. [B892]).

The rationale for a series of histologic studies by Albert and his colleagues (Albert and DeSantis, [B42]; Albert et al. [B47], [B48]) was based in part on the results of the research mentioned earlier. In Chinese hamsters, Albert and DeSantis [B42] 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 and Sherif [B40]; Albert et al. [B47], [B48]). In rats, exposure to two frequencies (100 MHz and 2450 MHz) resulted in a decrease in the number of Purkinje cells. At 2450 MHz, rats exposed postnatally (5 days, 7 h/d) 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 [B40]) 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. [B48]). 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; that is, pregnant rats were exposed from gestation day 6 through the end of pregnancy, and their offspring were exposed for 97 days for 4 h/d at 100 MHz (Albert et al. [B48]). In a nonhuman primate study, Albert et al. [B47] examined Purkinje cells in the offspring of pregnant squirrel monkeys exposed at 3.4 W/kg (2450 MHz) for 3 h/d, 5 d/wk, 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. [B47] and D'Andrea et al. [B314]), 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, which is a level that is 8.5 times greater than the limit for controlled environments.

As described in more detail in B.7.11.2, an extensive investigation of mammalian brain development found no histological changes in the developing rat brain (Inouye et al. [B681]). In contrast to the effect reported by Albert et al. [B48], there were no changes in Purkinje cells. In this study, rats were exposed prenatally and postnatally to brain SARs up to about five times greater than the threshold SAR for established adverse effects.

Most importantly, histopathological analysis of the brain and other central nervous system (CNS) tissues was a special focus of lifetime RF exposure studies in rats (Zook and Simmens [B1550]), some of which included exposure of the animals during gestation (Adey et al. [B32], [B33]; Anderson et al. [B66]). These studies are described in detail in B.7.11.3.1. 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 [B545] reported histological changes in the brains of rats exposed to a single, high-intensity microwave pulse at 915 MHz (10 kW at 60 ms 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. [B1486]), while another study found minor changes in cellular structure in snail ganglia exposed at 12.9 W/kg, which is a level more than three times greater than the adverse effect level found in live animals (Arber et al. [B80]).

C.5.3.3.2 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. [B48] reported a reduction in Purkinje cell density in the cerebellum of exposed *in utero* (0.5 W/kg to 6 W/kg range; average ~2 W/kg) and examined 40 days later; however, as discussed, this effect is not supported by results from Inouye et al. [B681] in rats exposed *in utero* to 1.76 W/kg from day 15 to day 40 postnatally or in the Albert et al. [B47] study of the cerebellum of squirrel monkeys exposed *in utero* and for 9.5 months afterward to 3.4 W/kg.

C.5.4 Review of 3 kHz to 100 kHz studies

C.5.4.1 General

There are now many major reviews of the RF literature, including those of the Advisory Group on Non-ionising Radiation of the U.K. National Radiological Protection Board [B36], the Health Council of the Netherlands [B585], the Institution of Electrical Engineers [B662], the International Commission on Non-Ionizing Radiation Protection [B646], the U.S. National Research Council [B1082], and the European Commission's Scientific Committee on Emerging and Newly Identified Health Risks [B407].

None of these reviews established a hazard from long-term RF exposure, however. 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. ICES 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. ICES will also continue to evaluate new research on short-term effects and modeling.

C.5.4.2 Short-term exposures (3 kHz to 100 kHz)

In the frequency range from 3 kHz 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 field and magnetic field exposures. These have been described in IEEE Std C95.6™-2002 [B671]. These established mechanisms fall within the category of short-term effects known as electrostimulation, which refers to the induction of a propagating action potential (a "nerve impulse") in excitable tissue (nerve and muscle) by an applied electrical stimulus. Such effects are understood in terms of recognized interaction mechanisms. The

standard regarding such effects does not apply to exposure encountered during medical procedures, nor does it necessarily protect against interference of medical devices or problems involving metallic implants.

Maximum exposure limits in this frequency range are based on avoidance of short-term reactions of electrostimulation. A review of the literature pertaining to electrostimulation effects, and the rationale for the ERLs, is provided in this standard and the following reactions are discussed: 1) aversive or painful stimulation of sensory or motor neurons, 2) muscle excitation that could lead to injury while performing potentially hazardous activities, and 3) cardiac excitation.

C.6 Noncancer-related studies

C.6.1 Teratogenicity, reproduction, and development

C.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 °C to 2.5 °C (mostly after exposures of tens of minutes up to 1 h or so). Higher temperature increases, of up to ~5 °C, for shorter durations are teratogenic (Edwards et al. [B385]). Fetal malformations were observed in offspring of pregnant rodents (mice, rats, and Syrian hamsters) exposed to WBA SARs $\geq$ 9 W/kg (Berman et al. [B141]; Brown-Woodman and Hadley [B203]; Chazan et al. [B245]; Lary et al. [B837], [B838], [B840]; Rugh et al. [B1225]). The teratogenic effects of RF exposure were attributed to thermal stress because many of the studies recorded elevations of 2 °C or more in the maternal core body temperature.

Exposures at lower SARs (3.6 W/kg to 7.3 W/kg) did not cause deformities in rats (Berman et al. [B138]; Jensh [B710], [B711]; Jensh et al. [B714], [B715]). Reduced fetal body weight in rats was observed at 7.3 W/kg (Jensh [B710]) and 4.8 W/kg, but not at 2.4 W/kg (Berman et al. [B146]), after long-term exposure of pregnant rats. The studies involved almost continuous exposure during gestation. These studies and another report (Berman and Carter [B138]) support the observation that exposure levels of 4.8 W/kg to 7.3 W/kg (i.e., levels somewhat less than those causing malformations) result in reduced fetal mass in rats. In comparison with the rat, higher SARs are required for teratogenicity and reduced fetal mass in the mouse because the smaller animal can dissipate heat more efficiently (Berman et al. [B142]; Inouye et al. [B682]; Nawrot et al. [B1057], [B1058]).

Very high SARs for short periods of time (Chernovetz et al. [B247], [B248]) or low SARs for long periods of time (causing no significant thermal stress) have generally not been associated with teratogenic effects (Chiang and Yao [B251]; Cobb et al. [B286]; Lary et al. [B838]; Schmidt et al. [B1261]).

Several studies have investigated the interaction between RF exposure and known teratogens such as ionizing radiation (e.g., Mortazavi et al. [B1043]), 2-methoxyethanol (Nelson and Conover [B1063]; Nelson et al. [B1063], [B1065], [B1066]), salicylic acid (Nelson and Snyder [B1067]), and arabinoside (Marcickiewicz et al. [B967]). 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. [B967])].

A few reports (Berman et al. [B145]; Brown-Woodman and Hadley [B201]; Tofani et al. [B1412]) 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. [B596]) 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 after continuous exposure to 20 kHz magnetic fields of pregnant mice during gestation day 0 to day 18, such as those associated with video display terminals (VDTs; Huuskonen et al. [B643]). In humans, no association between VDT exposure and teratogenesis was found (Kurppa et al. [B796]).

In addition to mammalian models, avian and insect species have been examined for teratogenic effects after 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 °C (Hamrick and McRee [B564]; McRee and Hamrick [B989]), although effects could be precipitated with exposures causing higher temperature elevations (Byman et al. [B218]; Clarke and Justesen [B272]; Hills et al. [B611]). Hatchability of chicken eggs was not affected at 2.9 W/kg (Braithwaite et al. [B193]). Those studies reporting terata in avian models in the absence of RF heating (Saito and Suzuki [B1232]; Saito et al. [B1233]; Fisher et al. [B421]; Youbicier-Simo et al. [B1538]) 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 (Carpenter and Livstone [B233]; Green et al. [B537]; Lindauer et al. [B899]; Liu et al. [B907]; Olsen [B1099], [B1100]; Pickard and Olsen [B1150]; Schwartz et al. [B1272]).

C.6.1.2 Reproduction

Sterility can occur when mammalian testes, which are normally at a temperature of 33 °C to 35 °C, are heated by a variety of methods (e.g., hot water, infrared radiation, and ultrasound) to temperatures approaching normal abdominal temperature (37 °C to 38 °C). 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. [B532]; Kowalczuk et al. [B774]). Permanent changes in reproductive efficiency in rats have been associated with RF exposures causing temperatures in the testes greater than 45 °C (Fahim et al. [B410]). At less extreme RF exposure conditions, temporary sterility has been demonstrated in male rodents with core temperatures of ~41 °C and intra-testicular temperatures ≥ 37.5 °C (Berman et al. [B143]; Lebovitz and Johnson [B848], [B849]; Lebovitz et al. [B850]). A lower sperm count and necrosis of testicular tissue was observed in testes heated to 39 °C or more by either microwave heating or through the use of a water bath (Reed et al. [B1177]). An RF exposure at an SAR of 6.3 W/kg, which caused a body temperature increase of ~1.5 °C, did not affect spermatogenesis in rats (Johnson et al. [B719]).

After reporting that exposure of rats to mobile phone emissions caused a reduction in the diameter of seminiferous tubules (Akdag et al. [B38]; Dasdag et al. [B328]), the same laboratory performed “a more thorough study” that failed to confirm the effect and failed to find effects on additional measures of testicular function and structure (Dasdag et al. [B329]). A study reporting effects of low-level RF exposure on reproductive ability in rodents (Magras and Xenos [B947]) 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. [B202]) 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, that is, exposures that are much higher than the established adverse effects threshold of 4 W/kg (Cleary et al. [B278]).

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. [B713]).

In avian studies, the number and fertility of sperm maintained at their normal temperature during RF exposures at 10 W/kg and 50 W/kg were not affected (Hall et al. [B559], [B560]). Reports of effects on fecundity in chickens are not useful because the exposures took place in metal cages (Giarola and Krueger [B511]; Krueger et al. [B787]). Reproductive parameters in quail exposed during development are discussed in C.7.3.

In Drosophila melanogaster, RF fields produced reproductive effects but only at very high exposure levels (Pay et al. [B1131], [B1132]).

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 (Larsen [B835]; Michaelson [B1006]; Nurminen and Kurppa [B1088]; Schnorr et al. [B1262]; Taskinen et al. [B1392]), including miscarriage (Bryant and Love [B209]; Ericson and Kallen [B401], [B402]), while other studies found an increased risk of infertility (Smith et al. [B1325]) and a slightly elevated risk of miscarriage (Goldhaber et al. [B517]; McDonald et al. [B983]). Studies of female MRI workers concluded that there was no major elevation in risk of adverse reproductive outcomes (Evans et al. [B409]; Kanal et al. [B734]). Work with RF heat sealers reportedly did not affect male semen quality or hormone levels (Grajewski et al. [B534]). In China, intentional RF exposures of human testes, sufficient to cause scrotum surface temperatures of 40 °C to 42 °C, have been reported to be an effective contraception method (Liu et al. [B908]).

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 MHz and 2450 MHz; Ouellet-Hellstrom and Stewart [B1116]; Stewart and Ouellet-Hellstrom [B1348]). However, a commentary on the exposure–response relationship showed that there was no association between absorbed RF energy and the reported effect (see Hocking and Joyner [B632] and Ouellet-Hellstrom and Stewart [B1117]).

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. [B859]) and dead or malformed infants (Kallen et al. [B732]). In Danish physiotherapists (Larsen et al. [B836]), use of high-frequency electromagnetic devices was associated with a higher ratio of female births and a 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. [B543]). In Finland, no firm evidence of increased spontaneous abortions or congenital malformations was found in the offspring of female physiotherapists (Taskinen et al. [B1392]). Daels [B307] 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 °C.

A possible association between the incidence of Down's syndrome and paternal radar exposure (Sigler et al. [B1310]) was not confirmed in an extended study by the investigators (Cohen et al. [B288]). Lower sperm concentration, motility, and number of normal sperm have been reported in RF workers (Lancranjan et al. [B831]). Schrader et al. [B1264] and Weyandt et al. [B1492], however, could not confirm their own finding of a decrease in sperm numbers in U.S. soldiers exposed to radar.

C.6.1.3 Development

In an important long-term study, squirrel monkeys were exposed at 2450 MHz at three SARs (0.034 W/kg, 0.34 W/kg, 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. [B736]). 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, which was an effect that was not replicated

by the same laboratory (Kaplan et al. [B736]). Exposure of rats during gestation to 2450 MHz fields at thermal levels (16.6 W/kg to 22 W/ kg) resulted in lower brain weight (Berman et al. [B140]; Shore et al. [B1302]). Long-term, continuous exposure of rats during gestation at 0.4 W/kg (2450 MHz PW) caused no effect on development, fetal body weight, brain weight, or the DNA, RNA, or protein content of the brain (Merritt et al. [B1003]). A decrease in Purkinje cells in the cerebellum of rats after 100 MHz and 2450 MHz exposures at ~3 W/kg (Albert et al. [B48] could not be confirmed in squirrel monkeys exposed at 2450 MHz by the same laboratory (Albert et al. [B47]). Histologic examination of the brains of rats at 15 days, 20 days, 30 days, and 40 days of age after prenatal and postnatal 2450 MHz exposure from day 4 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-day-old to 40-day-old rats, and the WBA SAR was 1.76 W/kg (Inouye et al. [B681]). 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 [B358]). Effects on the adrenal gland were observed in neonatal rats exposed to 2450 MHz at 9 W/kg to 10 W/kg (Guillet and Michaelson [B544]).

Rats exposed at 2450 MHz prenatally (days 5 to 20 of gestation) and perinatally (days 5 to 20 of gestation plus days 2 to 20 postnatally) had a larger body mass and less swimming endurance at 30 but not at 100 days of age. The estimated SAR in the fetal rats was 4 W/kg, and the SAR of rats aged 2 days to 20 days was 5.5 W/kg to 16.5 W/kg (Galvin et al. [B487]). Exposure of rats throughout pregnancy at 3.6 W/kg to 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. [B715]). 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 on open field tests (Jensh [B711]). The SARs associated with behavioral effects are consistent with the conclusion that the threshold for such effects is ~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 [B180]). RF fields at SARs of 0.2 W/kg, 1.0 W/kg, and 5 W/kg had no effect on development of rat embryos grown in culture (Klug et al. [B760]).

A series of studies at 2450 MHz investigated the development of the quail embryo exposed *in ovo* (Clark et al. [B271]; Galvin et al. [B482], [B481]; Gildersleeve et al. [B512], [B513], [B514], [B515]; Hamrick and McRee [B564], [B565]; Hamrick et al. [B566]; Inouye et al. [B680]; McRee and Hamrick [B989]; McRee et al. [B990], [B991]). Continuous exposure of quail embryos during the first eight days of incubation at 4 W/kg and 16 W/kg had no effect on the development of the heart (Galvin et al. [B482]). Brief exposures from 0.3 W/kg to 3 0 W/kg (CW and PW) had no effect on the heart rate of quail embryos that could not be attributed to temperature changes (Hamrick and McRee [B565]). After 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 (Gildersleeve et al. [B512]) and changes limited to one sex were found in corticosterone levels (Gildersleeve et al. [B514]) and leucopoiesis (Clark et al. [B271]). 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 8 weeks of age, no morphological changes in Purkinje cells were noted (Inouye et al. [B680]); 2) produced hematological changes (McRee and Hamrick [B989]); and 3) reduced male reproductive capacity (McRee et al. [B991]), but 4) did not affect the immune response in both sexes (Galvin et al. [B481]; Gildersleeve et al. [B515]; Hamrick et al. [B566]). 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 (Gildersleeve et al. [B512]). 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 °C to 3 °C. At an ambient temperature of 37 °C, the RF exposure caused the temperature of the eggs to rise to 39.5 °C to 40 °C, and only 7 % of the eggs hatched. Therefore, to maintain the egg at the normal incubation temperature of 37 °C, the ambient temperature was reduced to 35.5 °C during exposure at 4 W/kg. This procedure was used in several of the studies summarized and resulted in a higher hatchability in exposed eggs compared with control eggs (McRee and Hamrick [B989]). In related studies with chicken eggs, 2450 MHz exposure during incubation at 2.9 W/kg did not affect hatchability (Braithwaite et al. [B193]), and temperature increases of 0.25 °C to 2.3 °C were measured in embryonic and amniotic fluid in eggs exposed at 1250 MHz to 1.45 W/kg to 10.44 W/kg (Talau et al. [B1391]).

C.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 °C to 2.5 °C, mostly after exposures of tens of minutes up to 1 h or so (Edwards et al. [B385]). 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 earlier. 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 DRLs and ERLs recommended in IEEE Std C95.1-1999 [B667] and those recommended in this standard.

C.6.2 Hematology and endocrinology

C.6.2.1 Hormone changes

A handful of reports cite changes in melatonin and various other hormones (Abhold et al. [B2]; Deschaux and Pelissier [B349]; Gildersleeve et al. [B512], [B513], [B514], [B515]; Saddiki-Traki and Lescoat [B1227]) and neurotransmitters (Mausset et al. [B978]) 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. [B931], [B932], [B933], [B934], [B935]; Merritt et al. [B1002], [B1004]; Michaelson et al. [B1009], [B1010]). In some cases, (Deschaux and Pelissier [B349]; Saddiki-Traki and Lescoat [B1227]), it is difficult to determine whether exposure levels were 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. Numerous other studies reported no change in hormones after low-level, nonthermal RF exposures (Bonasera et al. [B178]; Heikkinen and Juutilianen [B593]; Toler et al. [B1414]; Vollrath et al. [B1461]). In humans, a marginal melatonin increase was associated with a study of occupational mobile phone use (Burch et al. [B215]), 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. [B352]; Mann et al. [B964]; Radon et al. [B1169]).

C.6.2.2 Immune function and hematology

Several 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 (Braithwaite et al. [B192]; Chagnaud and Veyret [B241]; Chou et al. [B260]; Djordjevich et al. [B364]; Gandhi and Ross [B498]; Guy et al. [B548]; Liddle et al. [B875], [B877], [B878]; Smialowicz et al. [B1315]). This is also the case in isolated cell lines of hematopoietic origin or primary lymphocytes (Brown and Marshall [B200]; Cleary et al. [B274]; Roberts and Michaelson [B1203]). 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 (Dasdag [B327]; Elekes et al. [B392]; Nakamura et al. [B1052]), and increased (Shao and Chiang [B1283]), decreased (Lyle et al. [B940]), or mixed effects (Veyret et al. [B1449]) in immunoglobulin titers and cellular immunity function. One study (Liburdy and Wyant [B874]) reported a possible RF-induced shape change in immunoglobulin proteins exposed to low levels of RF energy in an liquid gel chromatography fractionation column.

A series of studies from a single laboratory in Poland reported that exposure of rabbits and guinea pigs to low RF levels depressed erythrocyte numbers and erythroblast proliferation, while conversely the same exposure was reported to stimulate lymphocyte proliferation. The exposure also was reported to cause mitotic disturbances and changes in nuclear structure, and it generated various other effects in combination with drugs on CNS function (Baranski [B100], [B101], [B102]; Baranski and Edelwejn [B105]). Little information was provided on the actual conditions of exposure, making interpretation and confirmation of nonthermal conditions impossible.

When thermal levels of RF exposure are used, some studies continue to find no effect on autoimmune response (Anane and Veyret [B65]) or other hematologic or immunologic endpoints (Dunscombe et al. [B373]; Galvin et al. [B481],[B484]Ortner and Gavin [B1108]; Ragan et al. [B1172]) in animals and tissue culture. Many more studies at thermal levels of exposure report either increased or decreased immune cell function (Bogolyubov et al. [B174], [B175]; Deschaux et al. [B348]; Dwivedi et al. [B382]; Galvin and Ortner [B483]; Huang and Mold [B640]; Liburdy [B866], [B867], [B868]; Logani et al. [B914], [B915]; McRee et al. [B988]; Nakamura et al. [B1052]; Ortner and Galvin [B1108]; Pazderova-Vejlupkova and Frank [B1133]; Pazderova-Vejlupkova and Josifko [B1134]; Rama Rao et al. [B1173], [B1174], [B1175]; Rotkovska et al. [B1217], [B1218]; Smialowicz [B1314]; Smialowicz et al. [B1315], [B1316], [B1320], [B1321], [B1322], [B1323], [B1324]; Takashima and Asakura [B1388]; Wiktor-Jedrzejczak et al. [B1505], [B1506], [B1507], [B1508]; Yang et al. [B1531]), as well as the induction of stress markers (Cleary et al. [B281]; Nakamura et al. [B1052]; Pazderova-Vejlupkova and Frank [B1133]; Wangemann and Cleary [B1477]), which is similar to the effects of non-RF heating to elevated temperatures (Rama Rao et al. [B1173], [B1174], [B1175]). Other studies have shown no effect of RF exposure using Global System for Mobile Communications (GSM) signals on the immune system *in vitro* (Sultan et al. [B1361], [B1362]).

In a single Italian study of women living near radio-television broadcasting towers (500 kHz to 3 GHz) with electric field strengths of 4.3 V/m ± 1.4 V/m on their balconies (Boscolo et al. [B182]), 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. [B1418]) 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 in 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. [B1419]).

C.6.2.3 Summary of hematology and endocrinology

While studies have reported effects on hematological and immunologic endpoints in animals and *in vitro* models, most 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 (nonthermal) 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.

C.6.3 Blood-brain barrier (BBB) permeability

C.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. [B759]; Polyashuck [B1152]). The first article on this subject in the Western literature appeared in 1975 when Frey et al. [B462] reported that an SAR of approximately 1 W/kg caused increased BBB permeability in rats. In 1977, Oscar and Hawkins [B1112] 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. [B1164] that the changes in Oscar and Hawkins [B1112] might have been due to blood flow changes, Oscar et al. [B1111] measured and found increased local brain blood flow after RF exposure. Because of this finding, Oscar et al.

concluded that their earlier BBB permeability effects might have been smaller than reported. Oscar then co-authored the paper by Gruenau et al. [B541], 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 [B1112], other investigators (Preston and Prefontaine [B1163]; Preston et al. [B1164]) could find no effect of RF exposure on BBB permeability at WBA SARs estimated to be 0.02 W/kg to 6 W/kg, or at SARs in the head ranging from 0.08 W/kg to 1.8 W/kg. Additional attempts to replicate or confirm the effects in Oscar and Hawkins [B1112] and Frey et al. [B462] have been unsuccessful (Lin and Lin [B894]; Merritt et al. [B1001]; Ward and Ali [B1478]; Ward et al. [B1479]). Frey [B456] 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 (Frey [B459]).

A series of studies from Albert's laboratory (see Albert [B40], [B41]; Albert and Kerns [B45]; and Albert et al. [B44], [B48]) reported increased BBB permeability using a different technique (electron microscopy) than those used by Frey et al. [B462] and Oscar and Hawkins [B1112]. Effects were reported in rats and hamsters exposed at SARs ranging from 0.9 W/kg to 2.5 W/kg, but later work (Tsurita et al. [B1417]) failed to confirm the effects. Ward et al. [B1479] used RF exposure conditions similar to those of Albert [B40] and found no increase in permeation, after correcting the data for thermal effects due to absorbed RF energy.

Sutton et al. [B1363] 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 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 1-h exposure at 4 W/kg (whole-body) or after lifetime exposures at SARs ranging from 0.25 W/kg, 0.5 W/kg, 1.0 W/kg, to 4.0 W/kg (whole body; Finnie et al. [B419], [B420]).

Sutton and Carroll [B1364] found that RF exposure of the rat head, which produced a brain temperature of 40 °C or more, caused increased BBB permeation. When the body core temperature of the rat was kept at 30 °C 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. [B1001] showed that BBB permeation was affected in rats heated to 40 °C by hot air or RF exposure, and they concluded that hyperthermia was the causative factor, not RF energy *per se*. In a series of four papers, Williams et al. [B1509], [B1513], [B1514], [B1515] concluded that RF effects on the BBB are mediated by temperature-dependent changes and are not a direct nonthermal effect of the RF energy. Similarly, Fritze et al. [B466] found BBB 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 (Goldman et al. [B518]; Lin and Lin [B894], [B895]; Moriyama et al. [B1040]; Neilly and Lin [B1062]; Ohmoto et al. [B1096]).

Two papers describe the 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 °C or more reduced survival after inoculation with Japanese encephalitis virus; the expression of lethality of this virus requires entry into the central nervous system (Lange and Sedmak [B832]). High-level RF exposure (45.5 W/kg) facilitated drug action by increasing BBB permeability in mice (Quock et al. [B1166]). 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 can alter BBB permeability (Prato et al. [B1158]). However, no effect on the BBB was found when exposure was to the RF signal only (Garber et al. [B505]). Other reports are not consistent with the evidence presented earlier. Schirmacher et al. [B1260] reported an increase in permeability in a cell culture model of the BBB, when it was exposed at a low SAR. Neubauer et al. [B1070] found that 2 W/kg, but not 1 W/kg, caused a BBB change in rats. Chang et al. [B243] reported that one of six RF exposure levels affected BBB permeability in dogs, although no exposure-response relationship was found. Persson et al. [B1140] 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 approximately threefold, the change did not follow an SAR–response relationship over four ranges spanning 0.02 W.kg to 8.3 W/kg. The results with modulated RF fields also were not SAR dependent. The lowest SAR range (0.0004 W/kg to 0.008 W/kg) showed the highest increases at all modulations (4 Hz, 8.3 Hz, 16 Hz, 50 Hz, and 217 Hz), and at the highest SAR range (1.7 W/kg to 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 that 217 Hz was effective at the other two ranges, including the lowest SAR range. The 1997 paper by Persson et al. [B1140] stated that their earlier reports (Persson et al. [B1141]; Salford et al. [B1235], [B1237]) 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 [B1234] from this group describes effects on neurons and the BBB in rats exposed at SARs $\leq$ 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 BBB 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 BBB 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 $\leq$ 4 W/kg.

C.6.3.2 Summary of 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 produces an increase in localized brain temperature of ~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 earlier. 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.

C.6.4 Eye pathology

C.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 (Hirsch et al. [B625]; Williams et al. [B1512]). Cataracts can also be produced by localized (near-field), high-intensity exposures of the eyes of the dog (Baillie [B94]; Baillie et al. [B95]; Daily et al. [B308]) and rabbit (Carpenter and Van Ummersen [B234]; Cogan et al. [B287]; Hagan and Carpenter [B557]). Continuous and pulsed RF exposures at the same average power were shown to be equally effective in producing cataracts in rabbits (Birenbaum et al. [B156]), which is 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 ºC to 2.7 °C (Carpenter et al. [B231], [B232]; Foster et al. [B435]); whole-body exposure at these levels produced extreme body temperatures resulting in death (Appleton [B72]; Appleton et al. [B73]).

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 °C in or near the lens of the rabbit eye (Carpenter [B229]; Carpenter et al. [B230]; Guy et al. [B550]; Kramar et al. [B777], [B779]). At the same frequency, cataracts were not observed in the monkey eye exposed to similar high-intensity fields (Kramar et al. [B778]). 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 nonhuman 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 °C 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 [B1384]) and 2450 MHz (Neelakantaswamy and Ramakrishnan [B1061]) 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 ~6 °C (Taflove and Brodwin [B1384]). 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. [B1231]). Two relevant modeling studies of the human eye showed that for 50 W/m^2, the maximum permissible exposure at 1.5 GHz for controlled environments (e.g., FCC [B1435]) and a temperature change in the lens of less than 0.3 °C at frequencies from 0.6 GHz to 6 GHz (Hirata et al. [B617], [B623]) would be expected.

RF exposures that produced lens opacities in rabbits almost always caused inflammation of the iris (Birenbaum et al. [B157]). Other ocular effects, including corneal lesions, retinal effects, and changes in vascular permeability, were reported in nonhuman primates by Kues' laboratory after both CW and PW exposures (Kues and Monahan [B792]; Kues et al. [B790], [B793]). However, the inconsistencies in Kues' results, the failure by Kamimura et al. [B733] to independently confirm corneal lesions after CW exposure, the failure by Lu et al. [B930] to independently confirm retinal effects after PW exposure, and the absence of functional changes in vision (Lu et al. [B930]; McAfee et al. [B981]) 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. [B790] 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 [B1511]). A high-intensity pulsed RF exposure causing a temperature rise to 40 °C 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. [B1130]) was observed. In contrast to these findings, Frey [B456] reported increased permeability of the blood-vitreous humor barrier in rats exposed to pulsed fields at low average power. An appropriate control group was not used for one exposure group, however, and no information on SAR or temperature in the eye was provided.

A comparative study (Hagan and Carpenter [B557]) of relative effects at 2.45 GHz and 10 GHz found that the cataractogenic potential was greater at the lower frequency, which is 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 GHz and 70 GHz, that did not cause opacities in the lens (Rosenthal et al. [B1215]), 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 [B1444]) and ascorbic acid levels (Kinoshita et al. [B757]) 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 ºC to 3 ºC rise in the interior of the eye, are attributed to thermal effects (Bernat [B151]). 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. [B1489]). Stewart-DeHaan and colleagues (Creighton et al. [B301]; Stewart-DeHaan et al. [B1349], [B1350]) reported effects in the rat lens exposed *in vitro* to RF energy, but the usefulness of these studies, and a related modeling study (Wyeth [B1524]), 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 (Juan et al. [B723]; Ye et al. [B1534]) 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. [B1439]), rabbits (Guy et al. [B549]), and monkeys (McAfee et al. [B980], [B981]) did not cause cataracts or other ocular effects. In these studies, rats were exposed at 0.25 W/kg to 4.0 W/kg, rabbits received 17 W/kg in the head, and monkeys received 20 W/kg and 40 W/kg in the face. These and other study results (Chou et al. [B260], [B266]) 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 [B691]) 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 (Aurell and Tengroth [B88]; Cleary and Pasternack [B282]; Hollows and Douglas [B636]; Majewska [B949]; Odland [B1094]). Several other studies reported no ocular effects in human populations (Appleton and McCrossan [B75]; Appleton et al. [B74]; Cleary et al. [B283]; Hathaway et al. [B579]; Shacklett et al. [B1281]; Siekierzynski et al. [B1303]). One of these studies (Hathaway et al. [B579]) did not confirm the retinal effects reported in an earlier study (Aurell and Tengroth [B88]). The data in Appleton and McCrossan [B75] was analyzed by Frey [B454], 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 and Martin [B1503]). This independent statistical analysis by Wike and Martin [B1503] confirmed the results of studies of U.S. military personnel, which showed no association between RF exposure and ocular effects (Appleton and McCrossan [B75]; Appleton et al. [B74]).

An ocular effect (abnormal cone function) was reported in a man exposed twice for 15 min to 6000 MHz energy while inspecting a satellite antenna (Lim et al. [B881]). 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 (Holly et al. [B637]; Stang et al. [B1342]). 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. [B1342]). In an attempt to confirm these observations, Johansen et al. [B717] 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. [B717] 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 (Johansen et al. [B716], [B717]; Stang et al. [B1342]) and mobile phone use therefore failed to provide clear supporting evidence for the results described in the earliest study (Holly et al. [B637]).

C.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.

C.6.5 Auditory pathology and RF hearing

C.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 (Airborne Instrument Labs [B37]; Frey [B452], [B457]; Frey and Messenger [B463]), which of itself has no known adverse health consequence. RF-induced sound has been characterized as a click, buzz, hiss, knock, or chirp, and it is best detected in extremely quiet environments, often with subjects inserting earplugs to reduce background noise (Cain and Rissmann [B224]; Constant [B294]; Frey [B452], [B455], [B458]; Guy et al. [B547]; Ingalls [B679]; Khizhnyak et al. [B747]; Tyazhelov et al. [B1421]). RF hearing requires the ability of the exposed person to detect high-frequency acoustic waves in the range of ~5 kHz to 8 kHz as well as bone-conduction hearing responding to lower acoustic frequencies (Airborne Instrument Labs [B37]; Cain and Rissmann [B224]; Frey [B452], [B457]; Rissmann and Cain [B1200]). The fundamental frequencies able to produce RF sound in the human head, based on animal data and modeling are similar, for example, 7 kHz to 10 kHz (Chou et al. [B263]), 8 kHz to 15 kHz (Lin [B883], [B887], [B890]), and 7 kHz to 9 kHz (Watanabe et al. [B1480]). Effective radio frequencies are reported in the literature range from 2.4 MHz to 10 000 MHz (Cain and Rissmann [B224]; Frey [B455], [B458]; Ingalls [B679]; Roschmann [B1214]). 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 GHz to 300 GHz is unknown (Gandhi and Riazi [B497]).

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 can be recorded by electrodes in neurons at various locations along the auditory pathway. Chou et al. [B257] reported recording of cochlear microphonics from RF-exposed animals after two other attempts were unsuccessful (Frey [B453]; Guy et al. [B547]). 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 [B455]). 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. [B262]; Frey [B453]; Lebovitz and Seaman [B851], [B852]; Lin et al. [B896]; Taylor and Ashleman [B1394]). 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 [B256]; Guy et al. [B547]; Taylor and Ashleman [B1394]; Wilson et al. [B1517]). Several studies have reported thresholds for the RF-induced auditory sensation in laboratory animals (Cain and Rissmann [B224]; Guy et al. [B547]; Lebovitz and Seaman [B852]; Seaman and Lebovitz [B1275]).

The RF hearing phenomenon depends on the energy in a single pulse and not on the average power density. Guy et al. [B547] 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 [B430]; Gournay [B533]; Sommer and von Gierke [B1329]; White [B1493]). 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 ~10 000 W/kg peak (ARPANSA [B90]). The experimental weight-of-evidence, and the results of modeling studies, support the thermoelastic expansion theory (Chou and Guy [B259]; Chou et al. [B264], [B267]; Foster and Finch [B430]; Frey and Messenger [B463]; Guy et al. [B547]; Joines and Wilson [B721]; Lebovitz and Seaman [B851], [B852]; Lin [B883]; Lin et al. [B897], [B898]; Olsen and Lin [B1105], [B1106]; Roschmann [B1214]). This evidence does not support an alternative proposal by Frey [B453], [B455] that pulses of RF energy directly stimulate the central nervous system. The failure (Frey and Coren [B460]) 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. [B261]). No published report supports the suggestion by Tyazhelov et al. [B1421] 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-wave fields (Wilson et al. [B1517]). 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 W/kg and 1 W/kg (Marino et al. [B971]).

Additional information on RF hearing is available in reviews and fact sheets listed in the following references: ARPANSA [B90]; Chou et al. [B262]; Elder and Cahill [B390]; Elder and Chou [B391]; Lin [B882], [B884], [B885], [B886], [B888], [B889]; Postow and Swicord [B1155]; and Stewart [B1347].

C.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 approximately 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 [B1214]). 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. [B1480]). Based on this comparison, the exposure limit in the IEEE Std C95.1-1999 [B667] 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 [B391]).

C.6.6 Membrane biochemistry

A few studies have reported the effects of RF exposure on *in vitro* membrane function (Alekseev and Ziskin [B54]; Philippova et al. [B1145], [B1146]) and protein leakage through artificial and cellular membranes (Savopol et al. [B1253]). 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 in changes in cell surface charge distribution, in a manner dependent on the amplitude-modulated (AM) modulation (Somosy et al. [B1330], [B1331]). 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 might 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 (Allis and Sinha [B59],

[B60]; Arber and Lin [B76]; Baranski et al. [B106]; Barsoum and Pickard [B112], [B113]; Benz and Zimmerman [B133]; Bergqvist et al. [B136]; Bliss et al. [B172]; Brunkard and Pickard [B208]; Eibert et al. [B386]; Fesenko and Gluvstein [B413], [B414]; Friend et al. [B465]; Galvin et al. [B485]; Kim et al. [B752]; Liburdy and Magin [B869]; Liburdy and Penn [B870]; Liburdy and Vanek [B872]; Liu and Cleary [B906]; Neshev and Kirilova [B1069]; Olcerst et al. [B1098]; Orlando et al. [B1107]; Phelan et al. [B1144]; Philippova et al. [B1145], [B1146]; Pickard and Barsoum [B1149]; Portella et al. [B1154]; Saalman et al. [B1226]; Sandblom and Thenander [B1238]; Sandweiss [B1245]; Shnyrov et al. [B1301]; Tyazhelov et al. [B1420]; Weaver [B1482]; Weaver et al. [B1484]; Webber et al. [B1486]).

C.6.7 Calcium studies and neuron conduction

C.6.7.1 Calcium studies

C.6.7.1.1 General

A paper published in 1975 described changes in calcium ions associated with chick brain samples exposed *in vitro* to AM RF fields (Bawin et al. [B123]). This was called the “calcium efflux effect,” which was 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 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 Hz, 9 Hz, 11 Hz, 16 Hz, and 20 Hz, with the maximal response at 16 Hz, and no effects for an unmodulated field or for modulation were found at 0.5 Hz, 3 Hz, 25 Hz, 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 Hz, 6 Hz, 9 Hz, and 16 Hz (Bawin et al. [B123]). The effect also appeared to be power dependent (Blackman et al. [B161]; Sheppard et al. [B1298]), 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. [B123]) on calcium efflux describes a result that is often overlooked in interpreting the physiological significance of this effect; that is, 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 [B390]), 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 [B400]). These EPA reports addressed chick brain studies that were published in the period from 1975 to 1991 (Albert et al. [B49]; Bawin and Adey [B117]; Bawin et al. [B120], [B123]; Blackman et al. [B161], [B162], [B163], [B164], [B165], [B166], [B168], [B169]; Joines and Blackman [B720]; Shelton and Merritt [B1297]; Sheppard et al. [B1298]).

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. [B31]); 2) increased calcium efflux and increased ornithine decarboxylase activity were found in the brains of rats exposed *in vivo* (Paulraj et al. [B1129]); 3) with electron microscopy, examination of the brains of mice exposed *in vivo* showed a modified Ca++-ATPase activity and a redistribution of calcium at the synapse; that is, 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. [B758]); 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. [B378], [B379]); and 5) increased calcium efflux was reported in rat brain synaptosomes exposed *in vitro* (Lin-Liu and Adey [B900]).

In contrast to the AM studies, RF fields pulsed at repetition rates numerically equal to the frequency of sinusoidal modulations (e.g., 8 Hz, 16 Hz, 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. [B1005]; Shelton and Merritt [B1297]) or from the brains of rats exposed *in vivo* (Merritt et al. [B1003]).

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. [B42]). 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. [B123]). 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 [B1271]). 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. [B1270]). Exposure of frog hearts *in vitro* to 16 Hz modulated CW and pulsed fields had no effect on the beating rate (Yee et al. [B1535]) and that 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. [B1536]). An increase was observed in the inter-beat interval of chick cardiac cells exposed *in vitro* to unmodulated (CW) RF fields at SARs $\geq$ 1.2 W/kg, while fields with a modulating square-wave frequency of 16 Hz had no effect (Seaman and DeHaan [B1274]). 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 MHz, 900 MHz, 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. [B901]).

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. [B1518]). 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. [B300]). 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-A10 irradiation[46] used with the fluorescent assay for $[Ca^{++}]_i$ (Ihrig et al. [B674], [B675]).

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 [B470]). 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 [B1176]).

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 earlier. The IEEE database includes additional related papers (Athey [B87]; Bawin and Adey [B117], [B118]; Bawin et al. [B122]; Fisher et al. [B423]; Geletyuk et al. [B508]; Greengard et al. [B538]; Kaczmarek and Adey [B729]; McLeod et al. [B985]; Prasad et al. [B1157]). Detailed reviews of this literature are also available (NRPB [B1083]; UNEP/WHO/IRPA [B1427]).

[46] UV-A is defined as wavelengths in the ultraviolet portion of the electromagnetic spectrum between 315 nm and 400 nm.

C.6.7.1.2 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 AM RF fields cannot be used in setting RF exposure standards. In its review, a UNEP/WHO/IRPA [B1427] 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 [B1083] 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 [B1085] did not mention effects of AM RF fields on calcium efflux.

C.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 on the exposure level (Tattersall et al. [B1393]). Amplitude modulation at 16 Hz in this system had no effect on the results. In contrast, Pakhomov et al. [B1119] 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 and Lin [B76], [B77], [B78], [B79]; Arber et al. [B80]; Lin and Arber [B891]) seem to contradict reports that RF exposure either increased the firing rate (Beasond and Semm [B126]; Shchurov et al. [B1286]) or had no effect (Wang et al. [B1473]) on isolated neurons. Furthermore, several 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 [B992], [B993], [B994]; Seaman [B1273]; Wachtel et al. [B1464]). No effects of even very high levels of RF exposure were observed if cooling techniques were used to prevent temperature elevation (Chou and Guy [B258]).

C.6.8 Other animal studies

Various other noncancer endpoints affected by acute thermal exposures to animals have included altered digestive function (Santini [B1246]), increased serum triglyceride and beta-lipoprotein levels (Deficis et al. [B333]), increased rate of liver regeneration (Ottani et al. [B1114], [B1115]), and increased tissue water content and conductivity (Mikolajczyk et al. [B1014]). These unreplicated 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 C.7.1).

C.6.9 Human provocation studies

C.6.9.1 Cognitive function and memory

Studies have reported that mobile phone RF exposures result in either improved (Jech et al. [B707]; Kellnyi et al. [B743]; Koivisto et al. [B767]); Lee et al. [B854]; Preece et al. [B1161]) or hindered (Lass et al. [B841]) 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 its earlier results (Haarala et al. [B553]; Koivisto et al. [B768]). 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 [B802]), although serious flaws in the study design might have introduced artifacts. Other studies report that mobile phone RF exposure has no effect on memory performance or cognitive function (Freude et al. [B450], [B451]; Kelly et al. [B744]; Krause et al. [B782]; Preece et al. [B1161]). Two recent studies have found no effect on RF exposure from mobile phones on cognitive function in children (Haarala et al. [B552]; Preece et al. [B1161]).

C.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 (Lebedeva et al. [B843], [B844]; Reiser et al. [B1195]; von Klitzing [B1462]), mainly around the 10 Hz alpha frequency EEG band (Borbély et al. [B179]; Huber et al. [B641]; Lebedeva et al. [B843], [B844]). Other reports after similar RF exposures have either observed decreases in these same alpha frequency EEG bands (Croft et al. [B302], [B303]) or no effects (de Seze [B350]; Hietanen et al. [B606]; Kim [B753]). In studies looking at event-related potentials (ERP), some effects have been observed (Freude et al. [B450], [B451]).

Initial studies by Mann and Röschke [B961] and Mann et al. [B963] using mobile phone signals for RF exposure did indicate effects on shortening sleep onset time, as well as a 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 to 250 times higher than those used in previous studies (Röschke and Mann [B1213]; Wagner et al. [B1465], [B1466]), although the authors suggest that different characteristics associated with RF exposure could have caused the seemingly discrepant findings. In awake EEG studies, no statistically significant effects on EEG were reported (Röschke and Mann [B1213]). 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. [B964]). In a summary review, the authors conclude from their findings that while there might be some slight biological effects, their data do not suggest any adverse consequences associated specifically with mobile phone exposure (Mann and Röschke [B962]).

Disruption of sleep has been reported in subjects exposed to RF energy either occupationally (Bielski [B153]) or living in the vicinity of RF broadcasting towers (Altpeter et al. [B62]; Santini et al. [B1248], [B1249]). Some studies of event-related brain potentials in humans have reported mixed effects (Eulitz et al. [B403]; Freude et al. [B450], [B451]; Kellenyi et al. [B743]), while other studies reported no effects (Hladký et al. [B628]; Kim [B753]; Urban et al. [B1428]). 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. [B707]). Initial findings (Krause et al. [B781], [B782]) of ERP changes during working memory tasks were not repeatable in a double blind replication study by the same investigators (Krause et al. [B780]). 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.

C.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 (Chia et al. [B249], [B250]; Hocking [B630]; Oftedal et al. [B1095]; Sandstrom et al. [B1243]; Santini et al. [B1248], [B1249]). 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 $\leq 50\ W/m^2$ exhibited no headaches, fatigue, or irritability attributable to the microwave exposures (Djordjevich et al. [B362], [B363]). In addition, two controlled laboratory provocation studies examining the effects of RF exposure on headaches have reported no effect (Koivisto et al. [B766]; Paredi et al. [B1122]).

C.6.9.4 Hypersensitivity

Hypersensitivity has been reported in individuals in association with exposure to computer VDU display units (Sandstrom et al. [B1244]; Stenberg et al. [B1343]), as well as with RF from occupational (Bini et al. [B154]) and other external sources (Choy et al. [B268]; Flodin et al. [B425]). An early Polish study of RF-exposed industrial workers reported an increase in certain subjective endpoints including irritability, perspiration, dizziness, and other bioeffects (Bielski [B153]), 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. [B766]) and self-claimed hypersensitive subjects (Hietanen et al. [B605]) have reported no association between the self-reported hypersensitivity and RF exposure from mobile phones, Hocking [B630] and Hocking and Westerman [B634] reported links between various subjective symptoms and mobile phone exposure. A study from Japan (Kimata [B754]) reported that subjects with a history of eczema and dermatitis (AEDS) showed increased allergic reaction in a skin wheal assay after exposure for 60 min to mobile phone emissions, although the suggestion was made in a follow-up study that the larger effect might be associated with the annoyance of the ringing phone and its disruption on normal activities (Kimata [B755]). A preliminary study in Spain (Navarro et al. [B1055]) 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."

C.6.9.5 Effects on blood pressure/heart rate

While a provocation study by Braune et al. [B195] initially reported an increase in blood pressure (BP) and heart rate (HR) after exposure to a GSM mobile phone operating at 900 MHz, these effects were subsequently found not to be repeatable by the same group (Braune et al. [B194]). The BP and HR increases were not confirmed by independent studies in other laboratories (Tahvanainen et al. [B1385]). A single study (Lu et al. [B929]) 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. [B195] and Lu et al. [B926] studies did not cause any change in heart rate (Adair et al. [B24]). 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 and Jauchem [B439]; Frei et al. [B437], [B442], [B443]; Jauchem [B695]; Jauchem et al. [B699], [B700], [B702], [B703]; Phillips et al. [B1148]).

C.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.

C.7 Cancer-related studies

C.7.1 Animal cancer bioassays

C.7.1.1 General

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 United States 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 are 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 noncarcinogens.

C.7.1.2 Long-term animal bioassays

Several long-term animal bioassays have been performed exposing rats and mice to different RF signals for various daily periods. Most studies performed at both low-level and thermal levels have indicated no pathological or cancer effects (Adey et al. [B32], [B33]; Frei et al. [B437], [B440]; Jauchem et al. [B706]; La Regina and Roti Roti [B833]; Spalding et al. [B1333]; Toler et al. [B1415]; Zook and Simmens [B1550]). A 1962 study (Prausnitz and Susskind [B1159]) reported that exposed animals survived longer but reported cancer of white cells; a review of this work (Roberts and Michaelson [B1203]) 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. [B875]). As one arm of a large study by Utteridge et al. [B1439], mice were exposed (120 per group) to RF energy at WBA SARs of 0.25 W/kg, 0.5 W/kg, 1.0 W/kg, or 4.0 W/kg for 1 h/d, 5 d/wk, for 104 weeks. This study, with improved exposure methods, was to verify whether the positive effect reported by Repacholi et al. [B1197] is repeatable. No pathological or cancer effects were observed by Utteridge et al. [B1439]. The only report of a tumor increase due to long-term RF exposure at low levels was by Chou et al. [B265]. A slight increase in overall tumor incidence was reported in rats exposed for 2 years to 2450 MHz at low SAR levels (0.15 W/kg to 0.4 W/kg). A possible increase in pheochromocytoma (based on only 7 tumors in exposed versus 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 earlier. There was also no effect in the Chou et al. study [B265] 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. [B718]). It is impossible, however, to make any conclusion from a finding in a single animal without even a control.

C.7.1.3 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 ethylnitrosourea (ENU)-initiated rat brain tumors (Adey et al. [B32], [B33]; Zook and Simmens [B1550]), benz(a)pyrene-initiated rat sarcomas (Chagnaud et al. [B240]), 7,12-dimethylbenz[a]anthracene (DMBA)-initiated rat mammary tumors (Bartsch et al. [B114]), DMBA-initiated ± TPA co-promoted skin pappillomas in SENCAR mice (Mason et al. [B977]), DEN-induced GSTp(+) rat hepatomas (Imaida et al. [B677]), dimethylhydrazine-induced mouse colon tumors (Wu et al. [B1522]), and ionizing radiation-induced mouse lymphomas (Heikkinen et al. [B593]). These have consistently demonstrated an absence of tumor promotion by RF fields. In a recent study (Anane et al. [B64]) using DMBA-initiated rat mammary tumors, 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. [B1382] and Szudzinski et al. [B1383]. 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. [B1197] using transgenic Pim-1 mice did report an association between long-term RF exposure and mortality from a certain subtype of lymphoma (follicular), but it did not report a statistically significant increase in lymphoblastoid lymphomas. The Pim-1 transgenic model was specifically reported to use the appearance of the latter type of lymphoma to reveal carcinogens in a shorter time frame than that used for the detection of the follicular

lymphomas. A subsequent study, performed at multiple exposure levels with a more uniform and better characterized exposure field, was not able to confirm the initial Pim-1 findings (Utteridge et al. [B1439]). Hybrid transgenic mice designed to overexpress ornithine decarboxylase (ODC) and wild-type mice were initiated for skin tumors with UV radiation. They were then exposed to 900 MHz [GSM and Digital Advanced Mobile Phone System (DAMPS)] RF for 5 h/d, 5 d/wk for 1 year at an SAR of 0.5 W/kg (WBA; Heikkinen et al. [B594]). RF exposure did not result in a statistically significant effect on the development of skin tumors in either the transgenic or the nontransgenic mice. No effects on body weight, survival, urinary 6-hydroxymelatonin sulfate levels, polyamine levels, or ODC activity were found. Another transgenic mouse model (pKZ-1) was used in the investigation of intrachromosomal recombination inversions after 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. [B1374]).

C.7.1.4 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 (Preskorn et al. [B1162]; Rozkowski et al. [B1216]; Santini et al. [B1247]). In studies using 2 W/kg to 3 W/kg and 6 W/kg to 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. [B1382]). Similarly designed studies in other laboratories using different tumor cell lines reported no such effects (Higashikubo et al. [B607]; Salford et al. [B1234]).

C.7.1.5 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. [B676], [B677]) looked at liver tumor formation in rats exposed to 929 MHz (PDC) signals for 90 min/d, 5 d/wk, for 6 weeks at SAR values of 1.7 W/kg to 2.0 W/kg maximal in the liver (0.6 W/kg to 0.8 W/kg WBA) 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/d, 5 d/wk for 13 weeks 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. [B260]). No effects were observed on body mass, cataract formation, blood chemistry, blood protein, lymphocyte activation, or tissue pathology (indicating no evidence of cancer cells).

C.7.1.6 Summary of animal cancer-related studies

The scientific weight-of-evidence from the 35 animal bioassay studies discussed earlier 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. [B1439]). 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 follow-up 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 W/kg to 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.

C.7.2 Other animal and *in vitro* studies addressing cancer

C.7.2.1 General

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 are 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 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.

C.7.2.2 DNA single strand breaks (SSB) and/or DNA double strand breaks (DSB)

Studies by Lai and Singh [B827], [B828] have reported DNA breaks in the brain cells of rats exposed at 2450 MHz. These studies have 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. [B957]) 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. [B806]). A major *in vitro* investigation performed at mobile phone frequencies and modulations with even higher SARs (Tice et al. [B1411]) resulted in the absence of induction of DNA SSB. Careful examination of the actual data in another *in vitro* paper (Phillips et al. [B1147]), 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 (Alekseev and Ziskin [B52]; Li et al. [B864]; Maes et al. [B944]; McNamee et al. [B986], [B987]; Malyapa et al. [B957], [B959]; Tice et al. [B1411]; Vijayalaxmi et al. [B1453], [B1455]).

C.7.2.3 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 due to some type of induction of a reactive chemical species resulting from (and during or after) the RF exposure. Some published papers have theorized that DNA can absorb RF energy (Van Zandt et al. [B1445]), and some have also reported experimental evidence for specific absorption of RF energy at specific frequencies (Davis et al. [B330]; Edwards et al. [B383], [B384]; Sagripanti et al. [B1230]; Swicord and Davis [B1373]). Careful subsequent studies by two laboratories independently failed to confirm these observations (Foster et al. [B429]; Gabriel et al. [B471], [B472]). The initial results appear to have been the result of a measurement artifact.

C.7.2.4 Chromosome aberration induction

In vitro investigations of the possible induction of chromosomal damage have a long history in the field of RF bioeffects. Early studies presented the case for chromosome aberrations and “erosion” (Heller [B597]), and subsequent studies advocated for RF effects on chromosome aberrations in several mammalian systems (Chen et al. [B246]; Yao [B1532]). These studies had technical and analysis problems; Chen et al. [B246], 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. [B910], [B911] reported no increase due to RF exposures. A very careful and extensive examination was undertaken by Kerbacher et al. [B746]. Chinese hamster ovary cells were exposed to pulsed-wave, 2450 MHz fields for 2 h 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 °C. 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 °C incubated cells or temperature control shams. The authors went one step further and explored the hypothesis that the high SAR RF exposure could cause an increase in the extent of chromosome aberrations induced by two known chemical clastogens, mitomycin C and Adriamycin. The result again was the absence of a statistically significant difference of any of the indices compared with 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, several 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 (Maes et al. [B944], [B945], [B946]; Vijayalaxmi et al. [B1450], [B1454], [B1455]). There are reports of RF exposures causing chromosome aberrations *in vitro* (Garaj-Vrhovac et al. [B502], [B503], [B504]); these studies typically have inherent technical flaws or experimental ambiguities based on the exposure system employed.

C.7.2.5 Micronucleus formation

The examination of exposed cells for micronuclei is a newer 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 numerous 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. [B312]; Tice et al. [B1411]). At the same time, there is a much more abundant literature describing the absence of the induction of MN (Bisht et al. [B158]; McNamee et al. [B986], [B987]; Vijayalaxmi et al. [B1450], [B1455], [B1456]) 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. [B1411] 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. [B1452]) not to be statistically significant in a chronic animal RF tumor induction experiment, the initial publication was corrected (Vijayalaxmi et al. [B1451]) 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 exposed chronically to RF who did have tumors (although the RF was shown not to be responsible for the tumors present).

C.7.2.6 Sister chromatid exchange (SCE) induction

The assay for sister chromatid induction might or might 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. [B269], [B270] at 2450 MHz, pulsed wave, with a 0.1 duty cycle and a reported SAR of 33.8 W/kg. There was no evidence (even with a temperature

increase in the medium due to the RF exposure) of any increase in the frequency of SCEs. Expanding the hypothesis to look for an interaction between the RF exposure and simultaneous treatment with chemical mutagens known to induce SCEs (mitomycin C or Adriamycin), no statistically significant increase was found for the 2-h chemical and RF exposure compared to the chemical exposure alone. In a series of studies by Maes et al., the authors' results were inconsistent. After initially reporting that an RF exposure caused an increase in the SCEs induced by a subsequent mitomycin C treatment (Maes et al. [B943]), the subsequent study was inconsistent (Maes et al. [B944]), with the last two studies reporting that the effect was not present (Maes et al. [B945], [B946]).

C.7.2.7 DNA repair synthesis

There is only one published study (Meltz et al. [B1000]) examining the possible induction of DNA repair synthesis resulting from RF exposures. Cells were exposed at three frequencies at 10 W/m^2, 50 W/m^2, and/or 100 W/m^2 (SAR 0.39 W/kg to 4.5 W/kg depending on frequency) for 1 h to 3 h. The results of the series of experiments, using a normal human fibroblast cell line, at frequencies of 350 MHz, 850 MHz, 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.

C.7.2.8 Inhibition of DNA repair synthesis

In the same study examining the possible induction of DNA repair synthesis in preexisting DNA as a result of RF exposures of normal human fibroblast cells, experiments were also performed to determine whether RF exposures could interfere with the DNA repair synthesis process after the DNA of the cells was damaged by an acute UV-C[47] exposure (Meltz et al. [B1000]). The result of this investigation was that the RF exposures had no effect on the repair rate of the UV-C-damaged DNA; the evidence is that RF exposure does not interfere with this important type of DNA repair, which occurs after DNA base damage.

C.7.2.9 Phenotypic mutagenesis

Most of the evidences provided earlier are related to assessment of direct and immediate damage of the DNA and the genetic apparatus of the cell or to postexposure 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 might or might 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. [B998], [B999] 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 as "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. [B1386]). The exposures of the "Big Blue Mice" to RF energy were for 4 weeks. No statistical evidence was found for RF-induced mutagenesis.

[47] UV-C is defined as wavelengths in the UV portion of the electromagnetic spectrum between 190 nm and 280 nm.

C.7.2.10 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 should 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 [B97], [B98], [B99]), 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 consistent with the weight-of-evidence indicating that the RF is not mutagenic. An independent attempt to transform mammalian cells (Roti Roti et al. [B1219]) by RF exposures at mobile phone frequencies failed to demonstrate transformation.

C.7.2.11 DNA damage, cell cycle elongation, cell toxicity, and decreased cell proliferation

When the DNA of a normal cell is damaged, several significant events will occur. Since the cell is normal, it has a functioning p53 gene, and therefore, the stress response to the agent causing the mutation includes 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, temporarily ceases. 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 to 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, micronuclei formation, 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 (Adey et al. [B32], [B33]; Chou et al. [B265]; Frei et al. [B437], [B440]; Repacholi et al. [B1197]; Takahashi et al. [B1386]; Toler et al. [B1415]; Utteridge et al. [B1439]).

C.7.2.12 Proliferation, growth rate, and cell cycle analysis

Most 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 (Higashikubo et al. [B608]; Hoque and Gandhi [B638]; Stagg et al. [B1341]) or other RF signals (Czerski et al. [B305]; Fuhr et al. [B469]; Hamrick and Fox [B563]; Krause et al. [B783]; Vijayalaxmi et al. [B1454]; Wiktor-Jedrzejczak et al. [B1505], [B1508]). A few *in vitro* studies have reported effects at low levels, although these are inconsistent in their findings and include both increases (Daniells et al. [B326]; de Pomerai et al. [B344], [B345]; Donnellan et al. [B367]; Stodolnik-Baranska [B1352]) and decreases (French et al. [B448]; Garaj-Vrhovac et al. [B502]; Kwee and Raskmark [B799]; Szabo et al. [B1377]; Velizarov et al. [B1446]), in growth and proliferation, as well as nonlinear and frequency-dependent changes in the cell cycle time of yeast (Grundler [B542]).

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. [B968], [B969], [B970]). 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. [B274], [B276], [B279], [B280], and by Cleary and Wangemann [B284] 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. [B1120]).

In studies using bacterial cells, increased growth was reported from RF exposures that resulted in small localized temperature increases (Grospietsch et al. [B539]), while larger temperature effects resulted in decreased growth (Morozov et al. [B1041]). Experiments designed to investigate the possibility of using microwaves for spore inactivation found no nonthermal microwave response (Welt et al. [B1490]); the authors reported that the effects observed were indistinguishable from conventional heating. A study in nematodes (de Pomerai et al. [B344]) reported that extremely low levels of RF exposure resulted in a ~10 % size increase (hypothesized as a faster rate of progression through the life cycle) and in a 30 % to 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.

C.7.2.13 Gene and protein expression and activity

Several 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 (Bush et al. [B216]; Goswami et al. [B531]; Ivaschuk et al. [B692]; Li et al. [B863]; Morrissey et al. [B1042]; Parker et al. [B1123]; Stagg et al. [B1340]), but some do report changes in expression after low-level (Romano-Spica et al. [B1211]; Weisbrot et al. [B1488]) or high-level (~thermal) RF exposures (Fritze et al. [B467]; Natarajan et al. [B1053]). In some cases (Makrides et al. [B954]), 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 (Baranski [B103]; Byus et al. [B221], [B222]; Chiang and Yao [B251]; Dutta et al. [B377], [B380], [B381]; Fisher et al. [B422], [B423]; Kim et al. [B751]; Krause et al. [B783]; Kubinyi et al. [B789]; La Cara et al. [B803]; Litovitz et al. [B902], [B903]; Markkanen et al. [B972]; Pacini et al. [B1118]; Paulraj et al. [B1129]; Penafiel et al. [B1138]; Porcelli et al. [B1153]; Seaman et al. [B1276]; Somosy et al. [B1330], [B1331]; Szabo et al. [B1377]; Verma and Dutta [B1448]); these are also not consistent in their effects. In addition, many studies looking at endpoints similar to those earlier report either no effect (Allis and Fromme [B58]; Allis and Sinha-Robinson [B61]; Galvin et al. [B485]; Makheja et al. [B953]; Millar et al. [B1020]; Takashima [B1387]; Yeargers et al. [B1533]) or an effect only at thermal levels of exposure (Gandhi and Ross [B498]; Saffer and Profenno [B1228]). While three studies reported that RF exposure might accelerate denaturization and/or polymerization of proteins (Bohr and Bohr [B177]; Lubec [B936]), such effects were not repeatable in other laboratories (Oscar et al. [B1111]; Petrucelli and Fisher [B1143]). 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. [B326]; de Pomerai et al. [B342], [B343], [B345]), 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 after RF exposure, but the increased expression required much higher SAR levels than in the nematode study, and could have been due to local thermal conditions (Leszczynski et al. [B860]). Another study performed at extremely low levels of exposure reported decreases in hsp 70 (di Carlo et al. [B357]). 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 (Cleary et al. [B275]; Parker et al. [B1123]). When increased hsp gene expression has been observed, it is often at much higher levels of exposure that produce thermal conditions (Fritze et al. [B467]; Tian et al. [B1410]).

Exposure of rat brain synaptosomes at high SAR levels was reported to result in an increase in phosphorylation (Gandhi and Ross [B488]). 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 [B204]). A series of studies reported effects on immune parameters and protein synthesis but only at thermal levels (Wiktor-Jedrzejczak et al. [B1505], [B1506],

[B1507], [B1508]). One study reported sporadically distributed increases and decreases in adenosine diphosphate (ADP) ribosylation among various tissues of rats in a manner that was not linked to any obvious dose–response curve after exposure to very low RF exposure levels (Singh et al. [B1313]); 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 downregulation of a secretory protein (Kremer et al. [B785]). In other *in vitro* studies of respiratory enzymes and phage growth, no effects were observed until thermal levels were reached (Lukashevsky et al. [B938]; Melnick et al. [B996]). One group exposed 23-day-old rats to 147 MHz (CW and modulated) and its two subharmonics and reported changes in Na^+-K^+-ATPase activity in brain tissue (Behari et al. [B129]). The SAR was reported to be 6 W/kg to 9 W/kg. There was no report of measurement of core temperature or of adequate ventilation in the transverse electromagnetic cell exposure system; these effects, therefore, were most likely thermally induced.

C.7.2.14 Oxidative stress

Although oxidative stress has been proposed as a potential mechanism for RF effects (Lai and Singh [B827], [B828], [B829]), 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 dithiothreitol), but the effect did not show any obvious linear correlation with SAR (Philippova et al. [B1145], [B1146]). 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 [B916]). A study using lower levels of RF energy exposure reported protection against temperature-induced oxidative hemolysis in human red blood cells (RBC) (Kiel and Erwin [B748]). A series of studies using extremely high exposure levels of RF energy demonstrated that significant temperature elevations could affect membrane fluidity, permeability, and protein shedding in a manner that might be related to oxidative stress (Liburdy and Magin [B869]; Liburdy and Penn [B870]; Liburdy and Vanek [B872], [B873]; Liburdy et al. [B871]). 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, furthermore, that hyperthermia-induced auto-oxidation could be partially reversed by co-exposure with RF energy (Kiel and Erwin [B748]; Kiel et al. [B750]). 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 °C), and hydrogen peroxide (5.6 mM) were all effective in promoting the oxidation conversion. RF exposure inhibited thermally induced autooxidation by 28.6 %, but it did not affect oxidation by hydrogen peroxide (Kiel et al. [B750]).

C.7.2.15 Elevated temperature and carcinogenesis

Boukamp et al. [B183] showed that long-term exposure of already immortalized and p53 mutated human HaCaT keratinocyte cells to a significantly elevated temperature (40 °C or 104 °F) for up to 11 passages resulted in no significant tumor formation when injected into nude mice, although after 13 passages or more 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 °C 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 °C to 34 °C, it is unrealistic to imagine an area of skin on a living human being maintained at 40 °C 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 as follows. Skin cells exposed to RF energy, or any other heat source sufficient to maintain temperature levels at 40 °C for up to 11 passages, conditions loosely equivalent to a human being maintaining a localized skin temperature of 104 °F 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. [B354] who concluded that, "The bulk of the data presented indicate that hyperthermia alone is not carcinogenic."

C.7.2.16 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 (nonthermal) RF exposure. Most studies report no effect on a wide variety of biological endpoints. The magnitude of the reported effects is generally very small, often in the range of biological/physiological variability with no known health implications. In contrast, many studies described in this annex support the basis for this standard.

C.7.3 Cancer-related epidemiology studies

C.7.3.1 General

Epidemiology is "the study of the distribution and determinants of disease in human populations" (MacMahon and Pugh [B942]). Such studies provide the most relevant information for determining possible associations between exposure to a chemical or a 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 [B609]) 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 [B394] and other relevant detailed reviews by Moulder et al. [B1046] and Bergqvist et al. [B137].

C.7.3.2 Review of epidemiology studies

Epidemiological studies of RF exposure and cancer fall into the following five groups:

a) Studies of disease clusters

b) Studies of general populations exposed to RF sources [TV, radio, communication transmissions]

c) Studies of occupational groups

d) Case control studies

e) Studies of mobile phone users

Cluster studies, such as the one performed in Sutton Coldfield in the United Kingdom in response to a cluster of leukemia and lymphoma in adults living close to an RF broadcasting transmitter (Dolk et al. [B366]), 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 (Cooper et al. [B297]; Dolk et al. [B365]). Inconsistent effects have been reported between residential proximity to other RF broadcast towers and adverse health endpoints (Altpeter et al. [B62]; Bielski [B153]; Boscolo et al. [B182]; Hallberg and Johansson [B561], [B562]; Maskarinec et al. [B975]; Michelozzi et al. [B1011]; Selvin and Merrill [B1278]), 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. [B631]), although subsequent reanalysis of the data showed the results

could have been influenced by other confounding variables within the study location (McKenzie et al. [B984]).

While scattered reports of adverse health effects associated with occupational exposure to RF do exist (Cantor et al. [B226]; Davis and Mostofi [B331]; Demers et al. [B339]; Grayson [B536]; Hayes et al. [B581]; Holly et al. [B637]; Kurt and Milham [B797]; Pearce [B1135]; Pearce et al. [B1136], [B1137]; Richter et al. [B1199]; Speers et al. [B1335]; Thomas et al. [B1407]; Tynes et al. [B1422], [B1423]), these studies are mostly inconsistent with each other in terms of the adverse health endpoints affected, and they 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 (Armstrong et al. [B81]; Coleman [B290]; Coleman et al. [B291]; Czerski et al. [B306]; Gallagher et al. [B478]; Groves et al. [B540]; Hill [B610]; Kaplan et al. [B737]; Lagorio et al. [B805]; Lilienfeld et al. [B880]; Morgan et al. [B1036]; Muhm [B1047]; Robinette and Silverman [B1205], Robinette et al. [B1206]; Siekierzynski et al. [B1303], [B1304]; Tornqvist et al. [B1416]; Wiklund [B1504]; Wright et al. [B1520]). While micronuclei formation in workers occupationally exposed from broadcast antennas has been reported (Garaj-Vrhovac [B501]; Lalic et al. [B830]), these findings are not verified in a larger study of more than 40 Australian linemen exposed under similar conditions (Garson et al. [B506]). No clear association could be established between occupational exposures of parents to several agents, including RF, and effects (neuroblastoma) in their offspring (De Roos et al. [B346]; Spitz and Johnson [B1339]). 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. [B1407]). A study of a Polish military cohort reported a substantial excess of total cancer and several cancer subtypes with jobs associated with RF exposure (Szmigielski [B1379]; Szmigielski and Kubacki [B1380]), although questions have been raised about severe bias in the exposure assessment of this study (Bergqvist [B137]; Elwood [B393]; Stewart [B1347]). Studies by Milham of U.S. amateur radio operators reported an excess in one of nine types of leukemia assessed (see [B1015], [B1016], [B1017], [B1018], and [B1019]), but not for total tumors, total leukemia, or brain tumors, and potential confounding factors might have included exposure to soldering fumes, degreasing agents, and overrepresentation 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: 1) the difficulty of obtaining and/or reconstructing accurate and detailed individual exposures associated with mobile phone use over many years, and 2) the short period of time the technology has been in widespread use versus the long latency periods associated with many disease endpoints (e.g., various forms of cancer). Large cohort studies of tumor incidence (Johansen et al. [B717]) and mortality (Dreyer et al. [B371]) have shown no association with mobile phone use. A report by Stang et al. [B1342] that drew on data gathered on multiple disease endpoints from a larger cohort reported an association between mobile phone use and melanoma of the eye. A similar analysis drawn from a large set of cohort data by Johansen et al. [B716] reported no such association. Both analyses of ocular melanoma were based on 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 (Inskip et al. [B683]; Muscat et al. [B1048], [B1049]). A series of studies by Hardell et al. [B572], [B573], [B574] first reported no association between mobile phone use and brain tumors. 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. [B567], [B568], [B570]). After reanalysis of the same data set, malignant astrocytoma was then found to correlate with analog as well as with GSM mobile phone and cordless phone use (Hardell et al. [B569]). In the latest analysis of the same study group and data set, an association was reported between analog mobile phone use and vestibular schwannoma (VS; Hardell et al. [B567]). Hardell et al. correlated these findings on VS with the subjective studies by Oftedal et al. [B1095] and Sandstrom et al. [B1243] that reported increased complaints of tinnitus (a precondition of VS) in Norway. Hardell also provided three 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. [B574] reported a case study of angiosarcoma of the scalp associated with the use of cordless telephones. Recent studies by Auvinen et al. [B92] and Kahn et al. [B730] did not confirm the findings of Hardell et al. [B574] with respect to nonmalignant acoustic neuroma or tumor laterality. Auvinen et al. [B92] did report a slight

association between malignant gliomas (but not other brain tumors or salivary gland tumors) and analog 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) of 1.02, 95 % confidence interval (CI) of 0.85 to 1.23] or maximum mobile phone use [combined OR of 1.08, 95 % CI of 0.75 to 1.57] (see Table 5.6 of Elwood [B394]). Another preliminary study reported chromosomal aberrations in a small number of mobile phone users (Gadhia et al. [B473]).

C.7.3.3 Summary of epidemiology studies

The epidemiological evidence to date does not show clear or consistent evidence to indicate a causal role of RF exposures in connection with human cancer or other disease endpoints. Many of the relevant studies, however, are weak in terms of their design, their lack of detailed exposure assessment, and have potential biases in the data. While the available results do not indicate a strong causal association, they cannot establish the absence of a hazard. They do indicate that for commonly encountered RF exposures, any health effects, if they exist, must be small. Even though epidemiological evidence cannot rule out a causal relationship, the overall weight-of-evidence is consistent with the results of the long-term animal studies showing no evidence of physiological, pathological, or disease-specific effects.

C.8 Reviews of the literature (frequencies between 6 GHz and 300 GHz)

C.8.1 General

For most topics, systematic searches were conducted on various databases, such as PubMed, IEEE *Xplore*, and Web of Science, using applicable keywords (e.g., thermoregulation, electromagnetic field, microwaves, and radio frequency). Selection and inclusion of relevant studies was also contingent on the quality of dosimetry documented in the respective publications.

C.8.2 Thermoregulation

A systematic search produced 148 journal articles in refereed journals. Subsequent filtering produced 17 applicable articles relevant to thermoregulation for exposures above 6 GHz. No result would suggest a need for making any of the applicable exposure limits more restrictive.

Most notably, Kanezaki et al. [B735] found that the heat transfer coefficient between the body surface and air was the dominant factor influencing the surface temperature elevation. Millenbauch et al. [B1024] showed that millimeter waves (MMW) induce the same thermoregulatory responses as a warm ambient environment. Alekseev et al. [B50] showed that MMWs at sufficient intensities could thermally affect thermosensitive structures located in the skin and underlying tissue. Alekseev and Ziskin [B53] showed that the presence of a fat layer resulted in the appearance of a significant temperature gradient between the dermis and the muscle layer that increased with the fat layer thickness, and that dermal and muscle blood flows were the most influential parameters affecting the skin temperature elevations produced by MMW exposure. Hirata et al. [B615] showed that the core temperature elevation even for WBA SAR of 4 W/kg with a duration of 1 h was at most 0.8 °C. Hirata et al. [B615] showed that body-core temperature elevation in a 3-year-old child computational model was 35 % smaller than in an adult female model, due to the child's higher body surface-area-to-mass ratio.

C.8.3 Neurophysiology and neuropathology

Millimeter waves are widely used in Russia, China, and many Eastern European countries for the treatment of more than 30 diseases, including peptic ulcers, cardiovascular diseases, skin disorders, neurological diseases, and pain relief (Rojavin and Ziskin [B1210]). Numerous early studies, mostly in the former Soviet Union, were carried out exploring the effects of MMWs on the nervous system (Alekseev and Ziskin [B51]; Rojavin and Ziskin [B1210]). Alekseev et al. [B55] presented the results of a study of MMW effects on the firing rate of intact pacemaker neurons in the isolated nerve ring of the snail *Lymnaea stagnalis*. The neurons were exposed to MMWs at a frequency of 75 GHz, and local SAR ranging from 600 W/kg to 4200 W/kg, causing temperature rises of 0.3 °C and 2.2 °C, respectively. At an SAR = 4200 W/kg, exposure to MMWs produced biphasic changes in the firing rate.

Shapiro et al. [B1284] showed that a 64 mW, 60 GHz MMW stimulation significantly increased the action potential firing rate in oocytes coexpressing voltage-gated sodium and potassium channels. The time course of change in the spiking interval upon application of millimeter waves paralleled that of a temperature rise of 6 °C.

Pikov et al. [B1151] exposed slices of cortical tissue located an exposure chamber to 60.1 GHz at the highest incident power density of 900 W/m^2. A 1-min exposure caused the reduction of the firing rate, narrowing of the width of the action potential, and decrease of the membrane resistance. These effects were accompanied by MMW heating of the bath solution by 3 °C.

Most studies showed the changes in the electrical activity and structure of the excitable cells induced by a thermal effect of MMWs. It was shown that the rate of heating plays a significant role in eliciting the electrical response of nerve cells (Alekseev et al. [B55]).

Radzievsky et al. [B1171] evaluated the pain relief effect of MMWs in humans in a double-blinded, randomized, cross-over prospective experiment conducted using a cold pressor test as a model of tonic aching pain. The subjects were exposed to 42.25 GHz at 250 W/m^2 for 30 min. The MMW exposure produced a significant suppression of pain sensation with an average 37.7 % gain in pain tolerance. Similar results were obtained in volunteers exposed to 42.25 GHz at 172 W/m^2 (Partyla et al. [B1126]).

Numerous experiments were performed in mice to study the mechanism by which the MMW exposure could produce the hypoalgesic effect. The murine paw was exposed to 61.22 GHz with the average incident power density of 150 W/m^2. Dissection of the local nerve resulted in a full loss of the hypoalgesic effect. This result directly indicated the involvement of the peripheral nervous system in MMW-induced hypoalgesia. The pretreatment of mice with naloxone, a nonselective antagonist of opioid receptors, also fully blocked the hypoalgesic effect of MMWs (Rojavin et al. [B1209]). This result indicated that the antinociceptive effect of MMWs was mediated through endogenous opioids. Moreover, the involvement of δ and κ endogenous opioids in the MMW-induced hypoalgesia was demonstrated using selective blockers of δ- and κ-opioid receptors and the direct ELISA measurement of endogenous opioids in CNS tissue (Radzievsky et al. [B1170]). The authors concluded that endogenous opioids seems to play an important role in the systemic effects of MMWs involving the CNS.

Millimeter waves produce systemic effects on humans and animals affecting the immune and nervous systems and involving the endogenous opioids. In most studies, a hypoalgesic effect and an effect on the immune system were obtained at thermal exposure intensities (> 100 W/m^2). It was suggested that the systemic effect was initiated by stimulation of free nerve endings in the skin. Then the peripheral signal is conveyed to the CNS where it modulates the neural activity resulting in the development of various biological effects. No adverse health effects to the nervous system were found at exposures of millimeter waves below 100 W/m^2 (Alekseev et al. [B50]).

C.8.4 Hematology and endocrinology

C.8.4.1 Hormone changes

A handful of reports cite changes in melatonin and various other hormones (Abhold et al. [B2]; Deschaux and Pelissier [B349]; Gildersleeve et al. [B512], [B513], [B514], [B515]; Saddiki-Traki and Lescoat [B1227]) and neurotransmitters (Mausset et al. [B978]) 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. [B928], [B931], [B932], [B933], [B934], [B935]; Merritt et al. [B1002], [B1004]; Michaelson et al. [B1009], [B1010]). In some cases (Deschaux and Pelissier [B349]; Saddiki-Traki and Lescoat [B1227]), it is difficult to determine whether exposure levels were thermal or not because of the absence of temperature measurement, inadequate temperature measurement, or inadequate reporting/description of dosimetric measurements. A small sample size is frequently a problem. Several other studies reported no change in hormones after low-level, nonthermal RF exposures (Bonasera et al. [B178]; Heikianen and Juutilianen [B593]; Toler et al. [B1414]; Vollrath et al. [B1461]). In humans, a marginal melatonin increase was associated with a study of occupational mobile phone use (Burch et al. [B215]), 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 [B350]; de Seze et al. [B352]; Mann et al. [B964]; Radon et al. [B1169]).

C.8.4.2 Immune function and hematology

A limited number of studies on the effects on immune system and hematological parameters of radio frequencies in the range of 6 GHz to 300 GHz has been carried out in the considered period (2003 to November 2017).

Ziskin's group published some articles on *in vivo* effects of MMWs in mice treated with the immuno-suppressive chemotherapeutic drug cyclophosphamide (CPA). Overall these articles show that MMW local exposure of the nose (42 GHz to 60 GHz; incident power density 31 mW/cm^2 to 38 mW/cm^2; 30 min/d, 3 days; temperature increase in the nose area of 1 °C to 1.5 °C) restores some immune parameters. In particular, the authors showed a recovery in T-cell proliferation, in the production of the Th1-type cytokine IFN-gamma, and NK cell activity without effects on IL-10 production and B-cell proliferation (Logani et al. [B913]; Makar et al. [B950], [B951], [B952]).

Effects induced by thermal increase were found in an *in vitro* study (Sypniewska et al. [B1375]) performed treating a rat macrophage cell line (NR8383) with plasma from rats exposed to environmental heat (42 °C) or to MMW (35 GHz; 75 mW/cm^2) until the core temperature reached 41 °C. Results showed an increase in the expression of 11 proteins associated with inflammation, oxidative stress, and energy metabolism. An increase in NF-κB activation and expression of pro-inflammatory cytokines (interleukin(IL)-1, IL-6 and IFN-gamma), was described in a study with the mouse macrophage cell line RAW264.7 exposed for 1 h to MMW (8.15 GHz to 18 GHz; incident power density of 1.6 mW/cm^2). At variance, two *in vivo* studies from Gapeyev (Gapeyev et al. [B499], [B500]) done using a model of zymosan-induced footpad inflammation showed that treatment with frequencies in the range of 42.2 GHz to 65 GHz (power intensity 0.1 mW/cm^2 to 0.7 mW/cm^2) induced a reduction in the paw edema and local (inflammation-induced) hyperthermia. The effects were reported to be dependent on carrier and modulation frequencies.

In an *in vitro* study (Szabo et al. [B1376]) exposing human blood to 42.2 GHz, in the incident power density 0.55 mW/cm^2 to 1.23 mW/cm^2 range was found that MMW induce phosphatidylserine externalization to the cell membrane external layer, which is an early event in the apoptotic death. This finding was also observed in isolated RBCs and in the human lymphoblastoid cell line Jurkat. The same result could not be obtained with MMW-independent thermal heating up to 40 °C.

In a study on human volunteers mainly aimed at evaluating the effects on genotoxic endpoints, no effects of 120 GHz to 130 GHz also on lymphocyte proliferation were observed (Scarfi et al. [B1254]).

C.8.4.3 Summary of hematology and endocrinology

A limited number of studies on the effects on immune system and hematological parameters of radiofrequencies in the range of 6 GHz to 300 GHz has been carried out in the considered period (2003 to November 2017). A few of these studies were performed with an adequate dosimetry and experimental design. While there are some *in vivo* studies indicating stimulating effects of MMW in chemotherapeutically immunodepressed animals, other studies showed a reduction in the induction of inflammation *in vivo*. However, no extensive studies addressed the issue deeply. *In vitro* studies seem to potentiate some inflammatory responses, but it is difficult to translate their relevance to *in vivo* models and even less to human health. In a study with human volunteers, no effects on cell proliferation were observed. Noteworthy, no specific studies on skin-associate immune cells and skin-associated immune responses were performed despite the relevance for this frequency range.

C.8.5 Eye pathology

In recent years, with the spread of smartphones, the speedup of wireless communication, and the increase in capacity, demand for RF bandwidths has been rapidly rising. Accompanying this demand, development of new RF utilization systems based on advanced RF technology is progressing. Quasi-millimeter-wave and millimeter-wave technologies have come into widespread use in daily life, including in high-speed wireless communications, sensing, high-resolution radar imaging, spectroscopy, and automobile collision prevention systems. For example, wireless gigabit (WiGig) products, which operate in the 60 GHz frequency band, are now commercially available, with additional MMW frequency bands expected to be used in fifth-generation (5G) wireless communication technologies (Colombi et al. [B292]; Obara et al. [B1091]; Thors et al. [B1408]).

Most of the eyeball is buried in the orbit. There are eyelids in front, which are thin folds of skin that cover and protect the human eye. Since the 1960s, it has been known that cataracts develop due to RF exposure above a threshold (Guy et al. [B550]). However, recent reviews have reported no conclusive evidence of RF induced cataractogenesis (Demers et al. [B339]). The onset threshold of cataract differs between rabbit and monkey, and it was reported that facial burns occur in the monkey before cataract develops (Hirata [B614]; Kramar et al. [B777]). Recent results of the dosimetry have reported that the temperature around the eyes becomes 43 ºC before reaching the lens temperature of cataract threshold at 41 ºC (Oizumi et al. [B1097]).

At frequencies above 6 GHz, energy absorption occurs mainly in the uppermost layer of surface tissues (e.g., skin and cornea). Rise in ocular temperature (cornea, lens, and vitreous) during exposure to frequencies of 18 GHz, 22 GHz, 26.5 GHz, 35 GHz, and 40 GHz at 200 mW/cm^2 for 3 min was observed in rabbit (Kojima et al. [B771]). A temperature elevation was dependent on various factors, including penetration depth (Sasaki et al. [B1251]), and convection of aqueous humor. It is well known that corneal injury occurs from exposure to millimeter-waves exceeding a threshold level dose in experimental animals (Chalfin et al. [B242]; Kojima et al. [B769]; Rosenthal et al. [B1215]). These animal experiments studied animals in which protective responses such as blinking were suppressed. Blinking intervals of rabbits have been reported to range widely, including 5 min to 20 min (Korb et al. [B772]; Ludwig and Reimann [B937], Schwartz [B1269]). In contrast, blinking intervals of humans are much shorter, ranging from 3 s to 10 s (Bernard [B147]; Ludwig and Reimann [B937]; Schwartz [B1269]). Therefore, even if humans were exposed to the dose levels used in rabbit experiments, it is considered that humans would be at less risk than rabbits. There is no evidence of ocular disorder due to RF exposure below the present international guidelines.

C.8.6 Auditory pathology and RF hearing

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 with evidence reported by multiple laboratories around the world in studies involving human subjects (Airborne Instrument Labs [B37]; Constant [B294]; Frey [B452], [B455], [B457]; Frey and Messenger [B463]; Guy et al. [B547]; Ingalls [B679]; Khizhnyak et al. [B747]; Tyazhelov et al. [B1421]) and various animals (rats, guinea pigs, and cats), which of itself has no known adverse health consequence. Most studies reported hearing effects at 200 MHz to 3 GHz carrier frequency. Only two reports in the database show a hearing effect at above 6 GHz.

Ingalls [B679] reported hearing radar at 1 GHz, 3 GHz, and 10 GHz. Measurements of hearing threshold at 1 GHz and 3 GHz were made but not at 10 GHz. No reason was given. Guy et al. [B547] studied the threshold energy of microwave-evoked auditory responses in cats exposed to 915 MHz, 2450 MHz, and 8.67 GHz to 9.16 GHz pulses of various durations. The required energy per pulse to elicit the responses using the 8.67 GHz to 9.16 GHz pulses was significantly higher than that required at 915 MHz and 2450 MHz. The exposure also had to be through the open hole in the skull.

Due to the superficial absorption of energy above 6 GHz, the hearing of microwave pulses becomes more difficult as shown in the cat study of Guy et al. [B547]. The phenomenon of RF hearing in humans is a well-established biological effect with no known adverse health consequence. The RF-induced sounds by thermoelastic pressure 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. Although the RF hearing occurs at low intensity levels that do not produce measurable tissue heating, the nature of the effect is thermal.

C.8.7 Other animal and *in vitro* studies addressing cancer

C.8.7.1 Cell proliferation and apoptosis in human and animal cells exposed to above 6 GHz and into the THz frequency range

The investigated range of exposure frequency is very wide from MMWs to terahertz (< 6 GHz to 60 THz); however, only 19 publications have been identified dealing with the biological endpoints of cell proliferation (including all relevant investigations such as cell cycle regulation and cell survival) and apoptosis. A general observation is that within these studies, very different exposure conditions were employed, including numerous frequencies and power density levels, as well as both short-term (minutes) and medium-term (several hours) exposure durations. Furthermore, using these frequencies to expose cells, a temperature rise has to be very well controlled since temperature is increasing quickly within water-containing solutions depending on the applied power density. The presented data consider the so-called nonthermal (low-level) effects, "biological responses," according to the authors. Thermal effects were considered to be "no response" or mentioned as "thermal effect/response."

Consequently, we prioritized as a relevant quality characteristic of the studies the presence or absence of controlled temperature conditions. Out of 19 studies, 14 detected responses after electromagnetic field (EMF) exposure. Remarkably, among these studies, 60 % did not control the temperature change. Thus it is difficult to identify whether these responses are a result of the temperature rise induced by the EMF exposure, or as the authors themselves declared, these responses are nonthermal.

The LWRG also analyzed other relevant quality characteristics. It shows that only 68 % applied sham control, 58 % performed dosimetry, 42 % used positive control, and only 5 % of the studies were performed in a blinded manner, in this case, a single study.

A more detailed analysis of the available studies is not possible since there is no appropriate number of publications available investigating similar biological endpoints and applying comparable physical parameters (exposure conditions).

Scrutinizing the overall performance of the *in vitro* studies of cell proliferation and apoptosis, the quality of the experimental controls need strong improvement. Thus, to draw any conclusions regarding possible health related bioeffects is not appropriate.

Beneduci et al. [B131] investigated frequency and the exposure time-dependent anti-proliferative effect of low-power MMW on the RPMI 7932 human melanoma cell line and detected evidence of the anti-proliferative effects on tumor cells induced by a 53.57 GHz to 78.33 GHz wide-band frequency range for 3 h. The monochromatic frequencies (51.05 GHz and the 65.00 GHz), however, did not affect the growth of RPMI 7932 cells. In another study by Beneduci et al. [B132], the erythromyeloid leukemia cell line K562 was used to the wide-band exposure showing a similar significant decrease of the proliferation rate without significant cell death increment.

Gallerano et al. [B479] applied a frequency range of 100 GHz to 150 GHz to investigate the effects on human fibroblasts. No effects were detected on the cell cycle kinetics. However, Li et al. [B865] identified that MMW exposure (physical parameter not provided) promotes Sprague Dawley (SD) rat chondrocyte proliferation via G1/S cell cycle transition and significantly enhances chondrocyte viability. In addition, after MMW exposure, cyclin D1, CDK4, and CDK6 expression was significantly upregulated, whereas p21 expression was significantly downregulated.

It was shown by Zhadobov et al. [B1542] that MMW exposure at 60.42 GHz and with a maximum incident power density of 1 mW/cm^2 does not alter the cell viability, gene expression, and protein conformation.

Phosphatidylserine (PS) is a common marker for apoptosis induction. Szabo et al. [B1376] investigated whether MMW induces the externalization of PS molecules of adherent human keratinocytes (HaCaT) and murine melanoma cells (B16F10). The MMW exposure (42.25 GHz) at 1.23 W/cm^2 induced a reversible externalization of PS molecules in cells within the beam area without detectable membrane damage. Nonadherent Jurkat cells exposed to MMW at 34.5 mW/cm^2 also showed reversible PS externalization, both when the cell temperature was held constant or permitted to rise. The same group investigated the effects on human keratinocytes HaTaC, after exposure to 61.2 GHz and at an SAR of 770 W/kg. No significant changes on the viability of adherent keratinocytes were observed (Szabo et al. [B1377], [B1378]).

Volkova et al. [B1460] studied the effects on the morphology and cellular function of human cryopreserved spermatozoa. Exposure to MMW (frequency not specified) energy at 0.03 mW/cm^2 density for 5 min in normozoospermia and for 15 min in asthenozoospermia leads to an increase of the fraction of mobile spermatozoa without impairing the membrane integrity and nuclear chromatin status and without apoptosis generation. Wu et al. [B1521] investigated the inhibition of sodium nitroprussiate (SNP)-induced apoptosis in SD rat chondrocytes after exposure to a therapeutic instrument (KFA-100A MW, incidence power density: 4 mW/cm^2, wavelength coverage: 7.5 mm to 10.0 mm). The MMW treatment inhibited the loss of plasma membrane asymmetry (externalization of PS), collapse of mitochondrial membrane potential, and activation of caspase-9 and caspase-3.

A contradictory result was shown by Zhao et al. [B1544]. These authors found that MMW exposure (frequency not specified) to 10 mW/cm^2, 30 mW/cm^2, and 50 mW/cm^2 for 5 min of NK-92 cells induced apoptosis and cell cycle arrest, in a dose-dependent manner.

In vitro THz exposure has been investigated in seven publications. Rat glial cells exposed to continuous THz radiation (0.12 THz to 0.18 THz, average power density of 3.2 mW/cm^2) showed a dose-dependent increase (1.5 times to 2 times) of apoptotic cells (Borovkova et al. [B181]). Exposure to 0.106 THz (continuous wave, 0.043 mW/cm^2 to 4.3 mW/cm^2, 30 min) of human–hamster hybrid cells resulted in the appearance of spindle disturbances at the anaphase and telophase (especially lagging and nondisjunction of single chromosomes) of cell divisions (Hintzsche et al. [B613]). De Amicis et al. [B332] exposed human fetal fibroblasts to low-frequency THz radiation (0.1 THz to 0.15 THz, 1 min) using a Compact Free Electron Laser for 20 min. No

induction of apoptosis or changes in pro-survival signaling proteins was detected. No cell survival effect was seen by Korenstein-Ilan et al. [B773] in human lymphocytes (CW 0.1 THz (0.031 mW/cm^2 for 1 h, 2 h, and 24 h) and by Hintzsche et al. [B612] on HaCaT and HDF cells after exposure for 2 h, 8 h, and 24 h with different power intensities ranging from 0.04 mW/cm^2 to 2 mW/cm^2. Using 1 mW average power free-electron laser radiation in the frequency range of 120 GHz to 140 GHz for 20 min, no alteration in cell cycle kinetics of human peripheral blood-lymphocyte was identified by Scarfi et al. [B1254]. Using three different cell types, namely, human corneal epithelial cells (HCE-T), human retinal pigment epithelial cells (ARPE-19), and human embryonic stem cells (hES07), no biological response was detected after exposure to a high-peak-power, low-average-power THz radiation produced by the ALICE (Daresbury Laboratory, U.K.) synchrotron source (Williams et al. [B1510]).

C.8.7.2 Gene and protein expression in human and animal cells exposed to above 6 GHz and into the THz frequency range

Only 14 publications have been identified dealing with the biological endpoints gene and protein expression. The employed frequency range is between 42.25 GHz and 10 THz, the power density range between 0.1 mW/cm^2 and 84 mW/cm^2 (in one publication 1670 mW/cm^2), and the exposure duration varied between minutes and several hours. Using this frequency range to expose cell cultures, the temperature can increase fast depending on the applied power density. The presented data consider the so-called nonthermal (low-level) effects, “biological responses,” according to the authors. Thermal effects were considered to be “no response.”

Within these 14 publications, all used sham control, dosimetry, positive controls, and temperature control. However, none of them was performed in a blinded manner.

None of the investigations detected induced responses after MMW exposure; however, due to the small number of publications available, the evaluation or any kind of analysis is not possible. Similarly, since only five publications deal with THz exposure, a general evaluation is not appropriate.

Haas et al. [B554] exposed the Neuroscreen 1 cell line (a PC12 subclone) to 60.4 GHz MMW with an incident power density of 10 mW/cm^2. The absence of changes in protein expression of HSP70, the Transient Receptor Potential cation channel subfamily Vanilloid member 1 and 2 (TRPV1, TRPV2), and the purinergic receptor P2X, ligand-gated ion channel, was detected. Moreover, no specific cell subpopulations were found to express any of the studied markers at a different level, compared with the rest of the cell populations. However, a slight insignificant increase in HSP70 expression and an increase in protein expression variability within cell populations were observed in exposed cells ascribed to thermal effect.

Habauzit et al. [B555] exposed primary cultures of human keratinocytes to 60 GHz MMW energy at 20 mW/cm^2 and detected 665 genes that were differentially expressed in a whole gene expression approach. The results were validated by reverse transcription polymerase chain reaction (RT-PCR), and the detected effects disappeared under controlled temperature conditions. The authors suggested that a heat control did not mimic the MMW effect exactly. Rather a slight specific electromagnetic effect of MMW was detected, which is associated with the cellular response to hyperthermia as shown on 34 differentially expressed genes.

Koyama et al. [B775] did not detect any effects on the expression of Hsp 27, Hsp 70, and Hsp 90 α in human corneal epithelial (HCE-T) and human lens epithelial (SRA01/04) cells exposed to 60 GHz MMW energy for 24 h (1 mW/cm^2).

Similarly, no effects were detected by Le Quement et al. [B858] investigating human skin cell lines (A375 melanoma cell line, HaCaT keratinocyte cell line) exposed to 60.4 GHz, with incident power densities between 1 mW/cm^2 and 20 mW/cm^2. These authors examined the expression of specific endoplasmic reticulum (ER)-stress sensors, namely, BIP and ORP150, by real-time RT-PCR. MMW exposures did not change the BIP or ORP150 mRNA basal levels, irrespective of the cell line, the exposure duration, or the IPD level. However, when cells were co-exposed with the ER-stress inducer thapsigargin (TG) and

20 mW/cm^2 exposure, the inhibition of TG-induced BIP and ORP150 overexpression was detected. Experimental controls showed that this inhibition is linked to the thermal effect resulting from the MMW exposure.

Nicolaz et al. [B1073] exposed the human glial cell line U-251 MG to different frequencies ranging from 59.16 GHz to 61.15 GHz, for 24 h with a peak incident power density of 0.14 mW/cm^2. The authors did not identify any effect in exposed cells compared to sham exposed ones on the mRNA levels (RT-PCR) of two endogenous ER-stress biomarkers, namely the chaperones BiP/GRP78 and ORP150/GRP170. In another study of the same group [B1072] investigated the modifications in ER protein folding and secretion and the XBP1 or ATF6 transcription factors maturation in 60.4 GHz exposed (0.14 mW/cm^2) U-251 MG cells. Again, the absence of significant changes in mRNA levels for BiP/GRP78, an endoplasmic reticulum stress sensor, was detected.

Koyama et al. [B776] exposed human corneal epithelial (HCE-T) cells derived from the human eye to 0.12 THz radiation at 5 mW/cm^2 for 24 h, and no effect was detected on the expression of Hsp 27, Hsp 70, and Hsp 90 α. Szabo et al. [B1377] employed human epidermal HaKaT keratinocytes under two different exposure conditions designated as low power, LP (61.2 GHz ± 2.1 GHz at 29 mW/cm^2 ± 2 mW/cm^2) and high power, HP (42.25 GHz at 1.67 W/cm^2) MMW exposure to evaluate the effect on Hsp 70 production. LP-MMW exposure did not alter Hsp70 production, unlike exposure to HP MMWs or hyperthermia (43 °C; 1 h). Zhadobov et al. [B1543] exposed U-251 MG human glial cell line to 60 GHz MMW at two different power densities (5.4 mW/cm^2 or 0.54 mW/cm^2) for different durations (1 h to 33 h) to evaluate stress-sensitive gene expression of molecular chaperones, clusterin (CLU), and HSP70. Results indicated the absence of significant modifications in gene transcription, mRNA, and protein amount for the stress-sensitive genes in all the conditions tested.

Applying 2.52 THz exposure (84.8 mW/cm^2) for 5 min, 10 min, 20 min, 40 min, or 80 min on human dermal fibroblasts, no effect on heat shock protein expression was found under nonthermal conditions by Wilmink et al. [B1516].

Bock et al. [B173] exposed mouse mesenchymal stem cells (MSC) to a broadband THz field (~10 THz), average power density of 1 mW/cm^2 for 2 h, 4 h, 6 h, and 9 h. By investigating the global gene expression (Affymetrix mouse genome microarray), many MSC genes did not respond (89 %); however, certain genes were activated (6 %), while other genes were repressed (5 %) significantly after 9-h irradiation. In the group of activated genes that were confirmed by using RT-PCR, the overexpression of transcription factor for adipocyte differentiation was detected. This suggests that a specific THz exposure condition enhances the differentiation process toward an adipocyte-like phenotype in MSC.

Alexandrov et al. [B56] investigated the effects of both pulsed and CW THz fields on hyperthermia genes (i.e., genes that usually respond to temperature increases in the cell) in mouse stem cells. Low-power exposure from both a pulsed broad-band (centered at 10 THz) source (1 mW/cm^2) and from a CW laser (2.52 THz) source (~3 mW/cm^2) was applied for 2 h and 9 h. Modeling, empirical characterization, and monitoring techniques were applied to minimize the impact of exposure-induced increases in temperature. QRT-PCR was used to evaluate changes in the transcriptional activity of selected hyperthermia genes. Temperature increases were minimal, and the differential expression of the investigated heat shock proteins (HSP105, HSP90, and CPR) resulted unaffected, while the expression of certain other genes (Adiponectin, GLUT4, and PPARG) showed clear effects of the THz irradiation after prolonged, broad-band exposure. In a follow-up study by Alexandrov et al. [B57], the authors reported that THz irradiation of mouse MSCs with a single-frequency, 2.52 THz laser or pulsed broad-band source (centered at 10 THz) results in irradiation-specific heterogenic changes in gene expression, depending on type of THz source, duration, and degree of stem cells differentiation. Microarray survey and RT-PCR experiments demonstrated that the prolonged broad-band THz exposure drives the mMSCs toward differentiation.

In the study of Bogomazova et al. [B176], human embryonic stem cells were exposed to narrow-band THz exposure (2.3 THz) under strict temperature control conditions. The transcription of approximately 1 % of genes was subtly increased after THz irradiation. Functional annotation enrichment analysis of differentially expressed genes revealed 15 functional classes, which were mostly related to mitochondria function.

Annex D

(informative)

Practical examples for compliance determinations—Applications

D.1 Introduction

D.1.1 General

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 and IEEE Std C95.3.1.

Generally, determining compliance can be accomplished in the following two ways:

— Theoretical analyses

— Measurements

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 can be nonuniform or high induced currents can be present), it is extremely difficult to determine analytically the levels that could be present. Also, measurement might 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 to emissions. As such, analysis and/or measurement results that indicate levels in excess of the exposure reference level (ERL) do not necessarily imply that persons will be exposed to such levels. This can depend on the exact environment and on the safety program, if one is associated with that particular radio frequency (RF) environment. See IEEE Std C95.7-2014 [B672] for recommended guidelines for establishing RF safety programs.

D.1.2 Characterizing exposure to nonuniform fields

D.1.2.1 Practical constraints

Exposure to nonuniform fields may be characterized as exposure to fields over a specified volume of space, in which there exists a highly localized area of intense RF energy. Nonuniform fields can 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 can be characterized by rapid changes in field strength with distance. Localized exposures result from exposure to nonuniform fields leading to nonuniform SAR distributions with high spatial peak specific absorption rate (SAR) values (nonuniform energy absorption). Localized exposures can also result from the exposure to a nonuniform field, with the exposure dependent on the size and orientation of the person in the field. Nonuniform fields can result in localized exposures in excess of the ERL.

In the reactive near-field region, there is no simple relationship between the electric field strength (E) and the magnetic field strength (H); the impedance (E/H) differs from 377 Ω. The linear decrease in field strength with distance and the decrease in power density with distance squared that is characteristic of the far field do not apply in the near-field region.

The reactive near field contains stored RF energy rather than radiated RF energy, and the fields often vary rapidly with distance. Issues that should be considered are as follows:

a) **ERLs:** The ERLs are based on the assumption of uniform exposure and are expressed in terms of field strength or plane-wave-equivalent power density of the incident field (i.e., the electric field strengths 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 can strongly affect the nature of the fields. For example, placing a probe near a source or standing near a source while carrying out measurements can change the characteristics of the fields considerably.

Measurements to determine adherence to the recommended ERLs should take into account the fact that several factors influence the field values obtained from measurements and the associated uncertainty in exposure assessments. More details on measurement uncertainty are provided in IEEE Std C95.3 [B670]. Exposure assessments should apply best practices in terms of the technical approach used and should minimize and document to the extent practical uncertainty in the process. These factors include the following:

— Variation of probe impedance with proximity to nearby reflective surfaces

— Capacitive coupling between the probe and the field source

— Nonuniform illumination of the sensing elements that make up the probe

— Perturbation of the field caused by the presence of the observer performing the measurement

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 and often in erroneously high readings. Accordingly, measurements should be made at a distance no closer than three probe-diameters between the center of the probe and any object.

Whole-body exposure is defined as a situation in which the entire body is exposed to electric, magnetic, or electromagnetic fields. This is the typical case for most environmental exposures. For “local exposure,” the area for assessment should be the approximate projected area of exposure on the body due to the source(s). However, this type of exposure assessment might not be appropriate when the transmitting source is in close proximity to the body, for example, exposure due to hand-held portable and mobile devices (see IEEE Std 1528™-2013 [B664], IEC 62209-1 [B654], and IEC 62209-2 [B655] for body-mounted devices).

For frequencies below 100 kHz, the assessed value for whole-body exposure should be the spatial average of the root-mean-square (rms) electric field strength as measured over the projected height of the human body.

For frequencies from 100 kHz to 6 GHz, the rms values of the squares of the electric field strength (E) or the magnetic field strength (H) or, for frequencies exceeding 300 MHz, the plane-wave-equivalent power density should be averaged over an area equivalent to the projected cross section of the human body for whole-body exposure or the projected area of exposure on the body for local exposure. For these frequencies, a vertical scan of the fields over an appropriate distance (e.g., 1.8 m for a standing adult for whole-body exposure or the vertical dimension of the projected area of exposure, as noted, is normally appropriate and should be sufficient for determining compliance with the ERLs). Alternatively, other methods, for convenience in assessing exposure, may be acceptable including a measure of the spatial maximum value that is always equal to or greater than the spatial average and, thus, conservative for determining compliance with the ERLs.

For frequencies from 6 GHz to 300 GHz, the rms value of the plane-wave-equivalent power density should be averaged over any square area of 4 cm^2. For small exposed areas above 30 GHz, if the exposed area on the body surface is small (< 1 cm^2 as defined by −3 dB contours relative to the peak exposure), the epithelial power density is allowed to exceed the values of Table 6 and Table 11 by a factor of 2, with an averaging area of 1 cm^2 (defined as area in the shape of a square at the body surface).

When a broadband survey instrument is used for the frequency range of 100 kHz to 6 GHz, spatially averaged exposure levels may be determined by slowly moving the probe while scanning over an area approximately equivalent to the vertical cross section (projected area) of the human body (1.8 m) or projected area of exposure. An average can be estimated by observing the meter reading during this scanning process or by reading directly on instruments that provide for spatial averaging. However, when using an instrument with an automatic spatial averaging feature, it is crucial that the probe be physically scanned at a uniform speed to ensure an accurate measure of the spatial average value.

Another alternative method is to average a series of 10 measurements performed in a vertical line with uniform spacing starting at ground level up to a height of 1.8 m or over the projected area of exposure. Other methods for spatial averaging in this frequency range have been described in IEC 62232 [B656] and may also be acceptable.

When performing exposure assessments, several factors should be considered such as minimum separation distance between the measurement probe and a source, measurement uncertainty, and operator interaction, as discussed in this annex and in IEEE Std C95.3.

In all cases, noncompliance with the spatially averaged or spatial maximum ERLs may be overturned by demonstrating compliance with the dosimetric reference limits (DRL) using appropriate measurement or computational dosimetry methods. However, such methods are typically more complex, and it is usually simpler and more practical to test first for compliance with the ERLs.

D.1.2.2 Applying the peak power density limits

D.1.2.2.1 General

As indicated in 4.3.4, 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: In general, for exposures to pulsed RF fields in the range of 100 kHz to 300 GHz, exposures should meet three conditions: 1) the time-averaged (rms) values of WBA exposure should comply with the WBA ERL; and 2) total incident energy density during any 100 ms period within the averaging time should not exceed one-fifth of the total energy density permitted during the entire averaging time for a continuous field; that is,

$$\sum_{i=0\,\mathrm{s}}^{0.1\,\mathrm{s}} \left(S_{i\mathrm{peak}} \times \tau_i \right) \leq \frac{ERL_{\mathrm{local}} \times t_{\mathrm{avg}}}{5}$$

where $S_{i\mathrm{peak}}$ is the peak incident power density of the ith pulse, τ_i is the pulse width of the ith pulse, ERL_{local} is the ERL for local exposure at the relevant frequency, and t_{avg} is 6 min (360 s) and the summation over the number of pulses applies to any 100 ms of exposure; and 3) for exposures exceeding 30 GHz, the maximum local incident energy density (fluence) is limited for a single pulse (e.g., to a value less than $\tau^{1/2}$ kJ/m^2 for restricted environments).

D.1.2.2.2 Extremely-low pulse-repetition-rate source

Scenario: Determine whether the peak power limits for persons permitted in restricted environments are exceeded for a high-power pulsed radiating system with the following characteristics:

- Frequency (f): 10 GHz
- Pulse width (τ): 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: To evaluate compliance, the three exposure criteria described in D.1.2.2.1 should be examined.

a) RMS WBA criterion:

The time-averaged power density for WBA exposure is determined by the conventional application of the radiating system duty cycle δ (10 ms/250 000 ms).

$$\text{rms WBA } S = S_{\text{peak}} \times \delta = 1\ 200\ 000 \times 0.000\ 04 = 48\ \text{W/m}^2$$

Because 48 W/m^2 is less than the WBA ERL at 10 GHz (50 W/m^2), the exposure complies relative to the rms WBA criterion.

b) Energy density criterion:

The summation of the product of peak power density S_{peak} and pulse width τ over any 100 ms period is given by

$$\sum_{i=1} \left(S_{i\text{peak}} \times \tau_i \right) = 1\ 200\ 000 \times 0.01\ \text{s} = 12\ 000\ \text{J/m}^2$$

Because of the very low prf, there is only one pulse that occurs during any 100 ms (0.1 s) period. For compliance with this criterion, this summation should be less than

$$\frac{ERL_{\text{local}} \times t_{\text{avg}}}{5}\ \text{J/m}^2 = \frac{183\ \text{W/m}^2 \times 360\ \text{s}}{5} = 13\ 176\ \text{J/m}^2$$

The ERL for local exposure in restricted environments at 10 GHz (Table 11; see 4.3.3.2) is used in the evaluation since this yields a conservative approach to assessing compliance. Because 12 000 J/m^2 < 13 176 J/m^2, the exposure complies with the energy density criterion.

c) Maximum local incident pulse fluence:

Because the radiating source frequency of 10 GHz is below 30 GHz, the limitation on maximum pulse fluence is not applicable.

Therefore the exposure is compliant with both the rms WBA power density and the local incident energy density criteria of the standard for persons permitted in restricted environments.

NOTE—If the radiating system in this example instead operated above 30 GHz, where the maximum incident single pulse fluence is limited, the fluence would have to be less than $\tau^{1/2}$ kJ/m^2 for compliance. In such a case, with a pulse width of 10 ms, the maximum fluence for any single pulse would be limited to 100 J/m^2 and the system parameters would not support compliance with the specified peak power density.

D.1.2.2.3 Conventional radar

Scenario: Determine whether the peak power density limits for persons permitted in restricted environments are exceeded for a radar with the following characteristics:

- Pulse width (τ): 10 μs
- Pulse repetition frequency (prf): 1200 pulses/s (Hz)
- Beam width (θ): 2°
- Antenna rotation (360°): 6 revolutions/min (r/min)
- Peak power density (S_{peak}): 300 000 W/m^2
- Frequency: 9.4 GHz

Solution:

a) The long-term time-averaged power density (S_{avg}) is

$$S_{avg} = S_{peak} \times \delta_{tx} \times \delta_{ant}$$

where

δ_{tx} = duty factor of the transmitter = prf × τ = 1 200 pulses/s × 10 μs = 0.012
δ_{ant} = duty factor of the rotating antenna = 2°/360° = 0.005 56

thus

S_{avg} = 300 000 × 0.012 × 0.005 56 = 20 W/m^2 ≤ 50 W/m^2

This means that the long-term time-averaged power density complies with the ERL.

b) The integrated energy density in any 0.1 s period (J_{int}) is

$$J_{int} = \int_{0\,s}^{0.1\,s} S_{peak} \times \tau \le \frac{ERL \times t_{avg}}{5} \text{ J/m}^2$$

1) The limit for any 0.1 s period ($ERL_{0.1\,s}$) is

$$ERL_{0.1\,s} = \frac{50 \text{ W/m}^2 \times 1800\ s}{5} = 18\,000 \text{ J/m}^2$$

2) The peak energy density of each 10 μs pulse is equal to

$$300\ 000\ \text{W/m}^2 \times 10\ \mu\text{s} = 3\ \text{J/m}^2$$

3) Next, determine the number of pulses that occur in each sweep of the beam from the rotating antenna.

4) The duration of a single antenna rotation (T_{rot}) is given by

$$T_{\text{rot}} = 60\ \text{s}/6\ \text{rotations} = 10\ \text{s/rotation}$$

5) The duration of the beam illumination is $(2° / 360°) \times 10$ s = 0.0556 s. (Note that during any 0.1 s period of the 30 min averaging time, only one beam illumination occurs.)

6) Thus, based on a prf of 1200 Hz, a total of 1200×0.0556 s = 66.7 pulses occur during each 10 s rotation of the antenna during exposure to the beam.

7) Hence, the total integrated energy density = 3 J/m^2 × 66.7 pulses = 200 J/m^2, which is less than the limit 18 000 J/m^2.

Therefore, the system complies with this criterion.

D.1.2.2.4 Nonsinusoidal waveform

In the preceding subclauses of D.1.2.2, 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 subclause, an example of how to assess compliance when the pulse is a nonsinusoidal 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 ms; see Figure D.1. To assess compliance, it is necessary to test first for compliance with the rms ERL; this is essentially no different than the previous example. Next, test for compliance with either the peak field restriction or the Fourier component restriction.

Peak field: The external magnetic field and electric field are limited by the pulse rise time (since real pulses are never square) expressed as the time rate of change of the magnetic flux density or electric field strength (*dB*/*dt* or *dE*/*dt*). For this simple case, the frequency (*f*) is 10 kHz so the applicable ERL is 0.205 mT rms (from Table 2; see 4.2.2.1) for the magnetic field, and 614 V/m rms (from Table 4; see 4.2.3.1) for the electric field.

$$\dot{B}_{\text{p}} = \sqrt{2} ERL_{B} (2\pi f)$$

$$dB/dt\ (\text{peak}) = \sqrt{2} \times 0.000\ 205\ T \times 2\pi \times 10\ 000\ \text{Hz} = 18.2\ \text{T/s}$$

$$\dot{E}_{\text{p}} = \sqrt{2} ERL_{E} (2\pi f)$$

$$dE/dt\ (\text{peak}) = \sqrt{2} \times 614\ \text{V/m} \times 2\pi \times 10\ 000\ \text{Hz} = 54\ 558\ \text{kV/(m s)}$$

Fourier component: The values of B and E for each Fourier component of the square wave are divided by the respective ERL at each component frequency, then all B terms summed and all E terms summed. Each summation shall be less than unity to comply with this restriction (see 4.2.2.4). 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; see Figure D.2. Since the Fourier components at 110 kHz and 130 kHz fall within the transition region where effects are associated with electrostimulation and heating, the ERLs from Table 2 and Table 8 shall be compared for these frequencies and the most restrictive value of each pair shall be used in the summation (see 4.2.2.4.3). (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 component out to 5 MHz).

To illustrate compliance evaluation based on Fourier components, assume the following values:

$A_1 = 100$ 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, the ERLs are:

ERL = 490 A/m, 10 kHz $\leq f \leq$ 100 kHz, per Table 2

ERL = 148 A/m, f = 110 kHz, per Table 8

ERL = 125 A/m, f = 130 kHz, per Table 8

Therefore, because

$$100/490 + 50/490 + 10/490 + 5/490 + 1/490 + 0.5/148 + 0.1/125 = 0.34 < 1$$

the ERL is not exceeded.

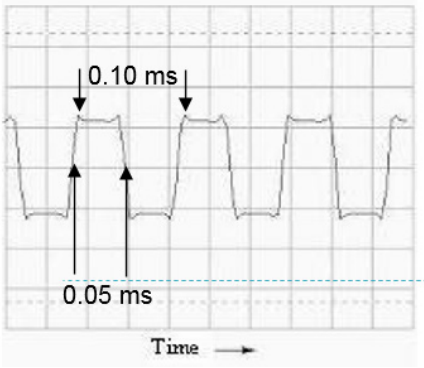


Figure D.1—Square wave in the time domain

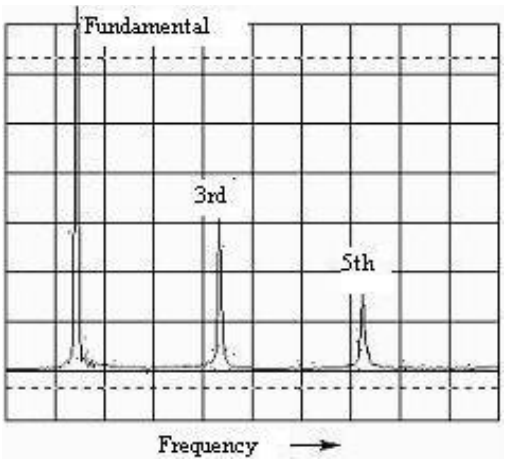


Figure D.2—Square wave in the frequency domain (Fourier spectrum)

D.1.2.3 Heat sealing application at 27 MHz

RF heat-sealing equipment typically operates in the 27 MHz industrial, scientific, and medical (ISM) band. ERLs are obtained from Table 7 and Table 8 (see 4.3.2). 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 11 m). From Table B.12 (see B.7.3), the reactive near field is estimated to extend out approximately 1.6 m from the source. From Table 8, the ERLs at 27 MHz are 68 V/m and 0.6 A/m for the electric field and the magnetic field, respectively. A whole-body measurement at the operator location provided the values in Table D.1 (which are typical).

The WBA value is determined based on the squares of the field strengths for both the electric field and the magnetic field components. The average electric field strength squared (3751 V^2/m^2) does not exceed the corresponding ERL of 4624 V^2/m^2, and the average magnetic field strength squared (0.27 A^2/m^2) does not exceed the corresponding ERL of 0.36 A^2/m^2. In this application, for persons permitted in restricted environments the squares of the peak values observed (81 V/m and 0.97 A/m) do not exceed five times the squares of the ERLs; that is,

$81^2 < 5 \times 68^2$ (6561 < 23 120)

$0.97^2 < 5 \times 0.6^2$ (0.94 < 1.8)

Notwithstanding the compliance with the ERL for the electric field, Figure 6 (see 4.4.2.1) indicates that at 27 MHz a field-to-ERL ratio greater than 16 % could result in an induced body current as well as in a touch contact current that exceeds the induced and contact current limits in Table 12 and Table 13 (see 4.4.1.1). In this example, the ratio (58.5 / 68) is equivalent to 86 %, indicating that both induced current and touch current measurements are also required.

Table D.1—Measured electric field strengths and magnetic field strengths 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)	$\|H\|^2$ $(A/m)^2$
Foot	22	484	0.08	0.0064
Ankle	34	1156	0.23	0.0529
Calf	47	2209	0.7	0.49
Knee	58	3364	0.97	0.9409
Thigh	69	4761	0.82	0.6724
Groin	75	5625	0.59	0.3481
Abdomen	81	6561	0.36	0.1296
Chest	75	5625	0.14	0.0196
Shoulder	66	4356	0.1	0.01
Head	58	3364	0.1	0.01
Average	58.5	3751	0.41	0.27

D.1.2.4 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 an optimum coupling between the body and the incident RF field with a maximum RF energy absorption, but it might 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 et al. [B375]). It is permitted to assess compliance with the DRLs 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 can be caused by an RF field that is not polarized with the long axis of the body, and hence, the resulting SAR can be substantially less than the value resulting from optimum polarization. For example, near the body resonance frequency of an adult in free space, the difference in WBA SAR that can result from optimum alignment of the body with the electric field and a nonoptimum alignment can be more than a factor of 10. If a particular exposure situation is such that it can be assured that nonoptimum polarization exists during the exposure, then RF fields that can exceed the ERLs specified in this standard may be applied after a careful analysis of the dosimetry using as a reference the data contained in Durney et al. [B375]. Users are cautioned, however, to always apply a conservative approach to estimating WBA SARs, especially in occupational exposure scenarios where personnel can be working in close proximity to high-power antennas.

D.2 Multifrequency 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 contributes some percentage of the ERL 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 ERL for the frequency of each source is evaluated. The exposure complies with the ERL if the sum of the ratios is less than unity; that is,

$$\sum_{i=1}^{n} \frac{exposure_i}{ERL_i} < 1$$

NOTE—Although the ERLs in Table 7 and Table 8 (see 4.3.2) are expressed in terms of field strengths (*E* and *H*) and power density, the exposures and the corresponding ERLs shall be expressed in terms of power density in the preceding summation, or in terms of the field-strengths squared (see 4.3.2).

Scenario: Measurements were made in a restricted environment at a point near six sources [three induction heaters (IH) and three dielectric heaters (DH)]. The values shown in columns 2, 3, and 6 in Table D.2 represent the measured frequency and the electric field strengths and magnetic field strengths as averaged over an area equivalent to the vertical cross section of an adult. S_E, ERL_E, S_H, and ERL_H are the electric field and magnetic field plane-wave-equivalent power densities and ERLs, 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_E (W/m^2)	ERL_E (W/m^2)	H (A/m)	S_H (W/m^2)	ERL_H (W/m^2)	Duty factor (%)
DH_1	27.5	90	21.5	11.9	0.1	3.8	132	20
DH_2	7.5	283	212	160	0.2	15.1	1780	60
DH_3	3.5	592	930	735	0.4	60.3	8160	45
IH_4	0.4	15	0.6	9000	8.0	24 100	625 000	100
IH_5	0.9	21	1.2	9000	4.0	6030	123 456	100
IH_6	8.04	30	2.4	140	0.2	15.1	1547	100

NOTE—Power densities are the calculated plane-wave-equivalent.

Solution: To ensure compliance with the ERL for persons permitted in a restricted environment, the sum of the ratios of the time-averaged squares of the measured electric field strength (or plane-wave-equivalent power density, S_E) to the corresponding squares of the ERL (or plane-wave-equivalent power density), and the sum of the ratios of the time-averaged squares of the measured magnetic field strength (or plane-wave-equivalent power density, S_H) to the corresponding squares of the ERL (or plane-wave-equivalent power density), shall not exceed unity (see 4.3.2). That is,

$$\sum_{i=1}^{6}\left(\frac{S_E \times duty\ factor}{ERL_E}\right)_i < 1$$

and

$$\sum_{i=1}^{6}\left(\frac{S_H \times duty\ factor}{ERL_H}\right)_i < 1$$

where

duty factor is the duty factor from Table D.2, expressed as a fraction
S_E is electric field plane-wave-equivalent power density
S_H is the magnetic field plane-wave-equivalent power density
ERL_E is the electric field exposure reference level
ERL_H is the magnetic field exposure reference level
i is the index of Source$_i$ and the Table D.2 row in which the values of the other variables are defined

For this example,

$$\sum_{i=1}^{6}\left(\frac{S_E \times duty\ factor}{ERL_E}\right)_i < 1$$

$$= \frac{21.5\times 0.2}{11.9} + \frac{212\times 0.6}{160} + \frac{930\times 0.45}{735} + \frac{0.6\times 1}{9000} + \frac{1.2\times 1}{9000} + \frac{2.4\times 1}{140} = 1.74 > 1$$

and

$$\sum_{i=1}^{6}\left(\frac{S_H \times duty\ factor}{ERL_H}\right)_i < 1$$

$$= \frac{3.8\times 0.2}{132} + \frac{15.1\times 0.6}{1780} + \frac{60.3\times 0.45}{8160} + \frac{24\ 100\times 1}{625\ 000} + \frac{6030\times 1}{123\ 456} + \frac{15.1\times 1}{1547} = 0.11 < 1$$

To comply with the ERL for persons permitted in restricted environments, both summations are required to 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 ERL for persons permitted in restricted environments.

D.2.2 Induced and contact current

A somewhat similar procedure is applied to the case where induced or contact current is associated with more than one source. In this case, the following provisions apply:

For frequencies $f < 100$ kHz:

$$\sum_{i=1}^{n}\frac{\left(induced\ current\right)_{i,\,f<100\,\mathrm{kHz}}}{ERL_{i,\,f<100\,\mathrm{kHz}}} < 1$$

For frequencies $f \geq 100$ kHz:

$$\sum_{i=1}^{n}\frac{\left(induced\ current\right)^2_{i,\,f\geq 100\,\mathrm{kHz}}}{ERL^2_{i,\,f\geq 100\,\mathrm{kHz}}} < 1$$

where ERL_i represents the induced current ERL for the ith source. For cases where exposure is to currents both below and above 100 kHz, each summation shall be complied with separately (see 4.4).

Scenario: The measured induced currents shown in Table D.3 correspond to those expected in an individual working in the vicinity of several sinusoidal sources. Determine whether the exposure exceeds the induced current ERL for persons permitted in a restricted environment.

Table D.3—Induced rms body current (maximum through either foot) measured in an exposed worker

Source	Frequency (MHz)	Induced Current rms (mA)	ERL_i (mA)
S_1	0.006	3.2	6
S_2	0.070	56.3	70
S_3	2.0	49.6	100
S_4	8.4	62.0	100

Solution:

Step 1: Determine whether electrostimulation limits are exceeded

$$\sum_{i=1}^{2} \frac{\left(induced\ current\right)_i}{ERL_i} = \frac{3.2}{6} + \frac{56.3}{70} = 1.34 > 1$$

The summation exceeds 1, and therefore, the exposure exceeds the induced current ERLs for electrostimulation effects ($f < 100$ kHz) for persons permitted in a restricted environment.

Step 2: Determine whether the RF heating limits are exceeded

$$\sum_{i=1}^{2} \frac{\left(induced\ current\right)_i^2}{ERL_i^2} = \frac{49.6^2}{100^2} + \frac{62.0^2}{100^2} = 0.63 < 1$$

The summation is less than 1, and therefore, the exposure does not exceed the induced current ERLs for heating effects ($f \geq 100$ kHz) for persons permitted in a restricted environment.[48]

D.3 Fluence considerations

RF field exposures consisting of intense pulsed power densities shall comply not only with the WBA ERL and local ERL but also with a limit on the fluence of the pulses (J/m^2 or kJ/m^2; see 4.3.4). For a given pulse, fluence is the product of the power density and the duration of the pulse.

Scenario 1:

A high peak power pulsed system operating at a frequency above 30 GHz produces an incident peak power density exposure for 2 s in a research-facility restricted environment. What is the maximum power density (S) for a single pulse of this type during the averaging time?

Solution:

The limit on pulse fluence = $\sqrt{2}$ kJ/m^2 = 1414 J/m^2.

The fluence associated with the 2 s long pulse is $S \times 2 < 1414$ J/m^2. Hence, $S < 1414 / 2$ W/m^2 < 707 W/m^2. A single pulse having a peak power density of 707 W/m^2 is compliant with the fluence limit. Also, the 6-min time-averaged power density is 707 W/m^2 $\times\ 2 / 360 = 3.93$ W/m^2, which complies with both the WBA (50 W/m^2) and local-exposure (150.5 W/m^2) ERLs.

[48] Note that all currents are rms values and that multiple frequency currents do not simply add algebraically.

Scenario 2:

A high peak power pulsed system produces an incident peak power density (S) of 500 W/m^2 exposure in a restricted environment with only one pulse in any 6 min averaging time. What is the greatest pulse width (τ) that does not exceed the limit on fluence?

Solution:

The fluence limit is $\tau^{1/2}$ kJ/m^2 or 1000 $\tau^{1/2}$ J/m^2, with pulse duration = τ.

Because $S \times \tau$ = fluence, find a value of τ that satisfies the limit for fluence in the following relation:

$$S \times \tau \leq 1000 \times \tau^{1/2}$$

$$S \times \tau^{1/2} \times \tau^{1/2} \leq 1000 \times \tau^{1/2}$$

$$\tau^{1/2} = 1000/500 = 2$$

Hence, τ = 4.0 s.

A pulse lasting 4 s and with a peak power density of 500 W/m^2 just complies with the limit.

D.4 Measurement requirements[49]

D.4.1 Electric field and magnetic field measurements

In general, measurements of both electric field strengths and magnetic field strengths 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 reradiating (reflecting) source.

The far-field region of a simple, electrically large antenna is generally defined as starting at a distance of $2D^2/\lambda$, where D is the largest linear dimension of the antenna and λ is the wavelength (IEEE Std C95.3-2002 [B670]). In the case of an antenna with a parabolic reflector, the far-field distance is estimated as approximately $0.6D^2/\lambda$, where D is the reflector diameter. For an antenna with multiple elements, the radiation pattern of the antenna can be considered to be fully formed at a distance of 10 times the maximum element spacing. Most commonly, measurements of both field components are not required at frequencies above 100 MHz (3 m wavelength) unless multiple emitters are involved or standing waves are produced by the presence of reradiators.

Measurement of electric field strengths and/or magnetic field strengths (or plane-wave-equivalent power densities) very close to RF sources might not adequately infer WBA SAR or even local SAR. Field measurements made closer than 20 cm to an RF source could result in an erroneous implication as to actual RF energy absorption. Care should be taken when performing such measurements with consideration of potential uncertainties about compliance. In some cases, theoretical calculations using appropriate methods can be superior to measurements for compliance assessments when an exposed person is in close proximity to the source.

[49] Subclause D.4 provides a brief overview of some of the issues associated with measurements of electric fields and magnetic fields, induced and contact currents, and contact voltages. More comprehensive and detailed information can be found in IEEE Std C95.3-2002 [B670] and various IEC standards (e.g., IEC 62232:2017 [B656]).

When metallic (conducting) surfaces are immersed in an RF field, currents are induced in those surfaces that, 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. In the absence of a focusing effect (which might be produced by a pair of orthogonally related conducting surfaces), the total absorbed energy is no greater than would be experienced in the absence of the reflecting object.

D.4.2 Induced current measurements

D.4.2.1 General

In some cases, induced current can best indicate exposure. For example, when RF exposure needs to be determined in the near field of an emitter or a reradiating 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 reradiator can 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 energy absorption. In addition, locations where the distribution of electromagnetic energy exhibits a complex pattern, compliance with the ERL can be better determined by measuring the induced current in the subject than by measuring field strength. This condition can occur particularly in locations where multiple emitters, using a variety of frequencies and at different locations, are producing the total exposure environment.

D.4.2.2 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 can conclude that, at some level of electric field strength, induced currents in the human body cannot exceed the standard, thus, making current measurements unnecessary to show compliance with the standard. Applying the work of Gandhi and Chatterjee [B491]; Gandhi et al. [B494]; and Tofani, et al. [B1413], calculations have been made of the threshold field strengths, below which induced current measurements need not be made. Figure 5 and Figure 6 provide percentages of the electric field ERLs, below which measurements of induced current are not required. For induced current measurements, the detailed provisions of IEEE Std C95.3 and IEEE Std C95.3.1 are recommended.

D.4.3 Contact voltage measurements (frequencies above 100 kHz)

RF burns are related to the local current density at the point of contact with a source. The source could be a passive object that is illuminated by incident RF fields or an active source such as a transmitting antenna. After contact is made with the RF current source, the relevant parameter is the contact current. However, a particularly hazardous scenario is presented when the source exhibits a high RF voltage sufficient to produce an electrical arc between the source and the body. If an arc occurs, the localized current density can be so high as to cause immediate burns on the skin surface at points where the arc terminates. Often, there are multiple points on the skin where the arcs terminate. This arcing phenomenon is related to the RF voltage existing on the source at the point of potential contact. A value of 140 V rms has historically been used by the U.S. Navy as a criterion for whether the source voltage has the potential to arc to a person approaching the surface of the source. Measurement of contact voltage (the voltage just prior to contact) can be accomplished with a high-impedance RF voltmeter. This can consist of a conventional digital multimeter equipped with a suitable RF probe having sufficient measurement range (i.e., voltage rating). Alternatively, a portable oscilloscope with sufficient bandwidth to detect the subject frequency may be used to measure the contact voltage. In either case, the measurement is to be made with a very high impedance probe (typically 10 MΩ) so that the RF voltage source is not loaded by the probe and to determine the RF potential difference

between the source and the ground. Referencing to ground presents the potentially greatest voltage value and should be a conservative measurement.

It should be noted that the extent of skin heating, which could lead to an RF burn, is predominately a function of the resistance at the interface of the skin and the object contacted. For a given contact current, higher contact resistance results in greater heating. Hence, a given contact current should not be perceived as resulting in the same potential for an RF burn when different exposure conditions exist. It remains a task in a future revision of this standard to specify limits on RF contact current better based on the sparse data on contact currents but with present insights as to the important factors that influence the thermal effect of contact currents (Tell and Tell [B1397]).

Annex E

(informative)

Bibliography

Bibliographical references are resources that provide additional or helpful material but do not need to be understood or used to implement this standard. Reference to these resources is made for informational use only.[50]

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IEEE Standard for Safety Levels with Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz
Corrigenda 2

STANDARDS

IEEE International Committee on Electromagnetic Safety (SCC39)

IEEE Std C95.1™-2019/Cor2-2020
(Corrigenda to IEEE Std C95.1-2019)

IEEE

IEEE Std C95.1™-2019/Cor2-2020
(Corrigenda to IEEE Std C95.1-2019)

IEEE Standard for Safety Levels with Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz
Corrigenda 2

Developed by the

IEEE International Committee on Electromagnetic Safety (SCC39)

Approved 24 September 2020

IEEE SA Standards Board

Abstract: Technical corrections are addressed in this corrigendum. Figure 1 and Figure 2 in IEEE Std C95.1-2019 were found to be incorrect. They did not accurately portray the limits given in Table 2, Table 3, and Table 4. The figures were corrected with respect to frequency extent and breakpoints to be consistent with the tables.

Keywords: electric field strength, exposure reference level (ERL), IEEE C95.1™, magnetic flux density, restricted environment, unrestricted environment

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Introduction

This introduction is not part of IEEE Std C95.1-2019/Cor2-2020, IEEE Standard for Safety Levels with Respect to Human Exposure to Electric, Magnetic, and Electromagnetic Fields, 0 Hz to 300 GHz—Corrigenda 2.

Figure 1 and Figure 2 in IEEE Std C95.1™-2019 were found to be incorrect. They did not accurately portray the limits given in Table 2, Table 3, and Table 4. The figures were corrected with respect to frequency extent and breakpoints to be consistent with the tables.

Contents

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NOTE—The editing instructions contained in this **corrigenda** define how to merge the material contained therein into the existing base standard and its amendments to form the comprehensive standard.

The editing instructions are shown in ***bold italic***. Four editing instructions are used: change, delete, insert, and replace. ***Change*** is used to make corrections in existing text or tables. The editing instruction specifies the location of the change and describes what is being changed by using ~~strikethrough~~ (to remove old material) and underscore (to add new material). ***Delete*** removes existing material. ***Insert*** adds new material without disturbing the existing material. Insertions may require renumbering. If so, renumbering instructions are given in the editing instruction. ***Replace*** is used to make changes in figures or equations by removing the existing figure or equation and replacing it with a new one. Editing instructions, change markings, and this NOTE will not be carried over into future editions because the changes will be incorporated into the base standard.

4. Exposure limits

4.2.4 Graphs of the ERLs of 4.2.2 and 4.2.3 for exposure to electric and magnetic fields

Replace Figure 1 with new Figure 1 as shown:

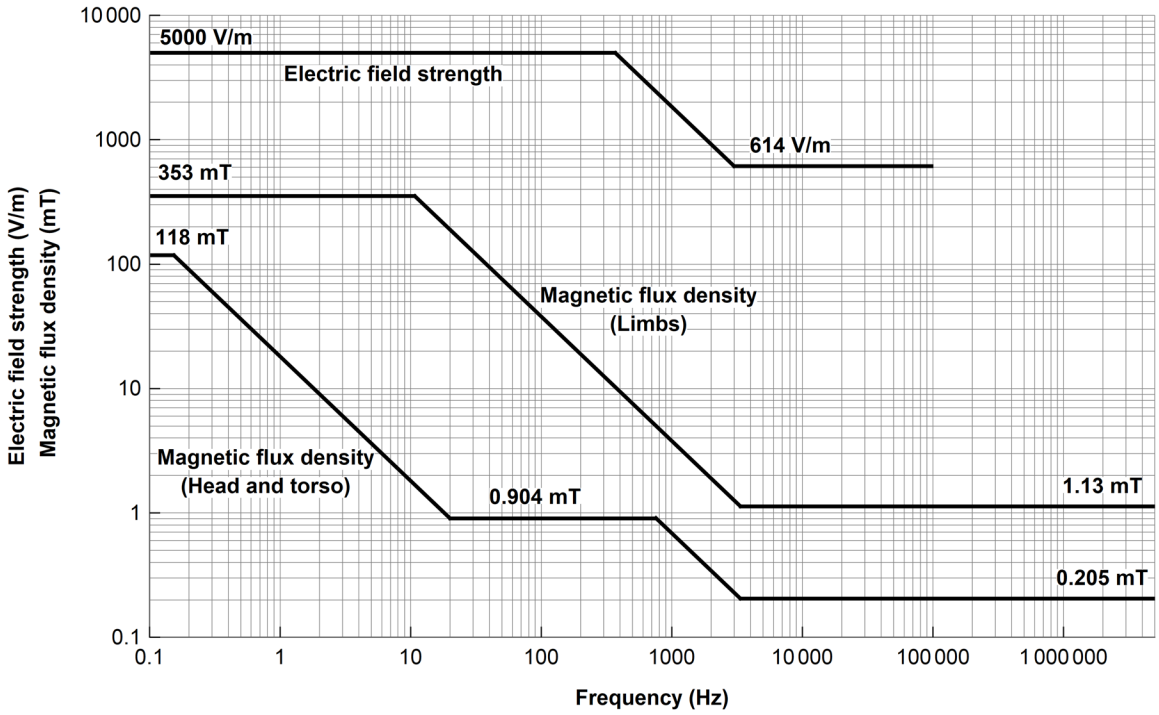

Figure 1—Graphical representations of the ERLs of Table 2, Table 3, and Table 4 for electric and magnetic fields—persons in unrestricted environments

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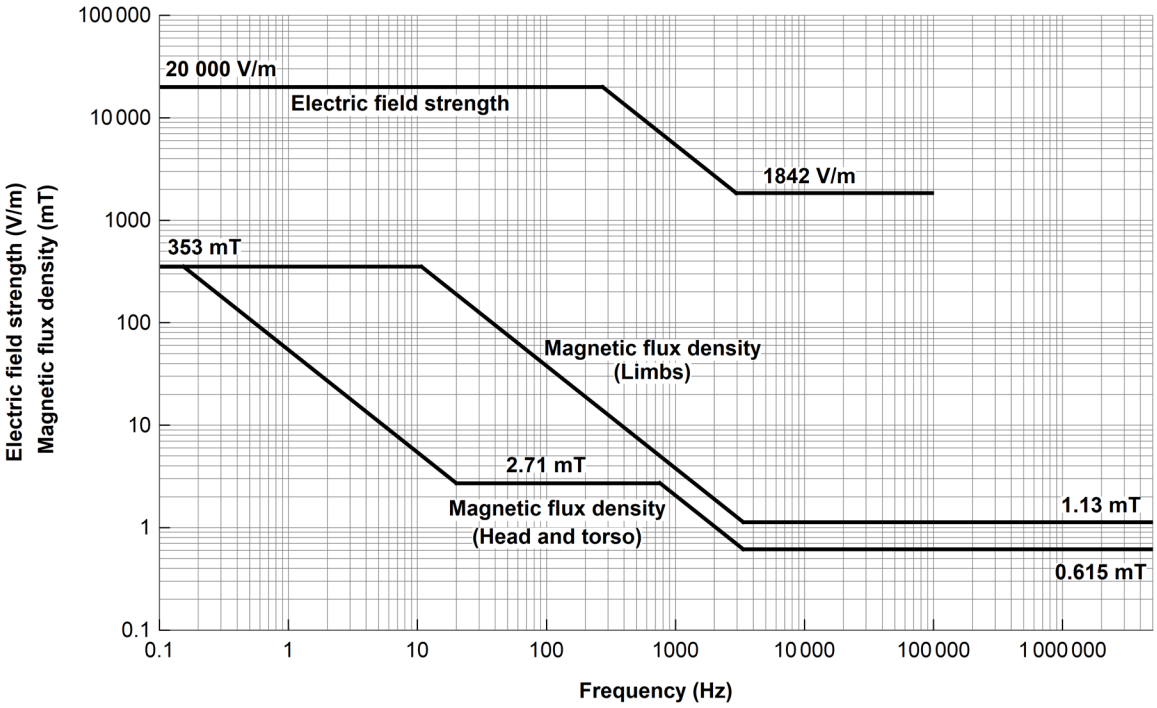

Figure 2—Graphical representations of the ERLs of Table 2, Table 3, and Table 4 for electric and magnetic fields—persons in restricted environments

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