INTERNATIONAL COMMISSION ON NON-IONIZING RADIATION PROTECTION

ICNIRP GUIDELINES

FOR LIMITING EXPOSURE TO ELECTROMAGNETIC FIELDS (100 kHz TO 300 GHz)

PUBLISHED IN: **HEALTH PHYS 118(5): 483–524; 2020**

PUBLISHED AHEAD OF PRINT IN MARCH 2020: **HEALTH PHYS 118(00):000–000; 2020**

ICNIRP PUBLICATION – 2020

OPEN

Special Submission

GUIDELINES FOR LIMITING EXPOSURE TO ELECTROMAGNETIC FIELDS (100 kHz to 300 GHz)

International Commission on Non-Ionizing Radiation Protection (ICNIRP)[1]

Abstract—**Radiofrequency electromagnetic fields (EMFs) are used to enable a number of modern devices, including mobile telecommunications infrastructure and phones, Wi-Fi, and Bluetooth. As radiofrequency EMFs at sufficiently high power levels can adversely affect health, ICNIRP published Guidelines in 1998 for human exposure to time-varying EMFs up to 300 GHz, which included the radiofrequency EMF spectrum. Since that time, there has been a considerable body of science further addressing the relation between radiofrequency EMFs and adverse health outcomes, as well as significant developments in the technologies that use radiofrequency EMFs. Accordingly, ICNIRP has updated the radiofrequency EMF part of the 1998 Guidelines. This document presents these revised Guidelines, which provide protection for humans from exposure to EMFs from 100 kHz to 300 GHz.**
Health Phys. 118(5):483–524; 2020

INTRODUCTION

THE GUIDELINES described here are for the protection of humans exposed to radiofrequency electromagnetic fields (EMFs) in the range 100 kHz to 300 GHz (hereafter "radiofrequency"). This publication replaces the 100 kHz to 300 GHz part of the ICNIRP (1998) radiofrequency guidelines, as well as the 100 kHz to 10 MHz part of the ICNIRP (2010) low-frequency guidelines. Although these guidelines are based on the best science currently available, it is recognized that there may be limitations to this knowledge that could have implications for the exposure restrictions. Accordingly, the guidelines will be periodically revised and updated as advances are made in the relevant scientific knowledge. The present document describes the guidelines and their rationale, with Appendix A providing further detail concerning the relevant dosimetry and Appendix B providing further detail regarding the biological and health effects reported in the literature.

PURPOSE AND SCOPE

The main objective of this publication is to establish guidelines for limiting exposure to EMFs that will provide a high level of protection for all people against substantiated adverse health effects from exposures to both short- and long-term, continuous and discontinuous radiofrequency EMFs. However, some exposure scenarios are defined as outside the scope of these guidelines. Medical procedures may utilize EMFs, and metallic implants may alter or perturb EMFs in the body, which in turn can affect the body both directly (via direct interaction between field and tissue) and indirectly (via an intermediate conducting object). For example, radiofrequency ablation and hyperthermia are both used as medical treatments, and radiofrequency EMFs can indirectly cause harm by unintentionally interfering with active implantable medical devices (see ISO 2012) or altering EMFs due to the presence of conductive implants. As medical procedures rely on medical expertise to weigh potential harm against intended benefits, ICNIRP considers such exposure managed by qualified medical practitioners (i.e., to patients, carers and comforters, including, where relevant, fetuses), as well as the utilization of conducting materials for medical procedures, as beyond the scope of these guidelines (for further information, see UNEP/WHO/IRPA 1993). Similarly, volunteer research participants are deemed to be outside the scope of these guidelines, providing that an institutional ethics committee approves such participation following consideration of potential harms and benefits. However,

[1]ICNIRP, c/o BfS, Ingolstaedter Landstr. 1, 85764, Oberschleissheim, Germany;

The International Commission on Non-Ionizing Radiation Protection (ICNIRP) collaborators are listed in the Acknowledgement section.

ICNIRP declares no conflict of interest.

For correspondence contact: Gunde Ziegelberger, c/o BfS, Ingolstaedter Landstr. 1, 85764 Oberschleissheim, Germany, or email at **info@icnirp.org**.

(*Manuscript accepted* 3 *September* 2019)

0017-9078/20/0

Copyright © 2020 The Author(s). Published by Wolters Kluwer Health, Inc. on behalf of the Health Physics Society. This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

DOI: 10.1097/HP.0000000000001210

occupationally exposed individuals in both the clinical and research scenarios are defined as within the scope of these guidelines. Cosmetic procedures may also utilize radiofrequency EMFs. ICNIRP considers people exposed to radiofrequency EMFs as a result of cosmetic treatments without control by a qualified medical practitioner to be subject to these guidelines; any decisions concerning potential exemptions are the role of national regulatory bodies. Radiofrequency EMFs may also interfere with electrical equipment more generally (i.e., not only implantable medical equipment), which can affect health indirectly by causing equipment to malfunction. This is referred to as electromagnetic compatibility, and is outside the scope of these guidelines (for further information, see IEC 2014).

PRINCIPLES FOR LIMITING RADIOFREQUENCY EXPOSURE

These guidelines specify quantitative EMF levels for personal exposure. Adherence to these levels is intended to protect people from all substantiated harmful effects of radiofrequency EMF exposure. To determine these levels, ICNIRP first identified published scientific literature concerning effects of radiofrequency EMF exposure on biological systems, and established which of these were both harmful to human health[3] and scientifically substantiated. This latter point is important because ICNIRP considers that, in general, reported adverse effects of radiofrequency EMFs on health need to be independently verified, be of sufficient scientific quality and consistent with current scientific understanding, in order to be taken as "evidence" and used for setting exposure restrictions. Within the guidelines, "evidence" will be used within this context, and "substantiated effect" used to describe reported effects that satisfy this definition of evidence. The reliance on such evidence in determining adverse health effects is to ensure that the exposure restrictions are based on genuine effects, rather than unsupported claims. However, these requirements may be relaxed if there is sufficient additional knowledge (such as understanding of the relevant biological interaction mechanism) to confirm that adverse health effects are reasonably expected to occur.

For each substantiated effect, ICNIRP then identified the "adverse health effect threshold;" the lowest exposure level known to cause the health effect. These thresholds were derived to be strongly conservative for typical exposure situations and populations. Where no such threshold could be explicitly obtained from the radiofrequency health literature, or where evidence that is independent from the radiofrequency health literature has (indirectly) shown that harm could occur at levels lower than the "EMF-derived threshold," ICNIRP set an "operational threshold." These are based on additional knowledge of the relation between the primary effect of exposure (e.g., heating) and health effect (e.g., pain), to provide an operational level with which to derive restriction values in order to attain an appropriate level of protection. Consistent with previous guidelines from ICNIRP, reduction factors were then applied to the resultant thresholds (or operational thresholds) to provide exposure restriction values. Reduction factors account for biological variability in the population (e.g., age, sex), variation in baseline conditions (e.g., tissue temperature), variation in environmental factors (e.g., air temperature, humidity, clothing), dosimetric uncertainty associated with deriving exposure values, uncertainty associated with the health science, and as a conservative measure more generally.

These exposure restriction values are referred to as "basic restrictions." They relate to physical quantities that are closely related to radiofrequency-induced adverse health effects. Some of these are physical quantities inside an exposed body, which cannot be easily measured, so quantities that are more easily evaluated, termed "reference levels," have been derived from the basic restrictions to provide a more-practical means of demonstrating compliance with the guidelines. Reference levels have been derived to provide an equivalent degree of protection to the basic restrictions, and thus an exposure is taken to be compliant with the guidelines if it is shown to be below either the relevant basic restrictions or relevant reference levels. Note that the relative concordance between exposures resulting from basic restrictions and reference levels may vary depending on a range of factors. As a conservative step, reference levels have been derived such that under worst-case exposure conditions (which are highly unlikely to occur in practice) they will result in similar exposures to those specified by the basic restrictions. It follows that in the vast majority of cases, observing the reference levels will result in substantially lower exposures than the corresponding basic restrictions allow. See "Reference Levels" section for further details.

The guidelines differentiate between occupationally-exposed individuals and members of the general public. Occupationally-exposed individuals are defined as adults who are exposed under controlled conditions associated with their occupational duties, trained to be aware of potential radiofrequency EMF risks and to employ appropriate harm-mitigation measures, and who have the sensory

[3] Note that the World Health Organization (1948) definition of "health" is used here. Specifically, "health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity."

and behavioral capacity for such awareness and harm-mitigation response. An occupationally-exposed worker must also be subject to an appropriate health and safety program that provides the above information and protection. The general public is defined as individuals of all ages and of differing health statuses, which includes more vulnerable groups or individuals, and who may have no knowledge of or control over their exposure to EMFs. These differences suggest the need to include more stringent restrictions for the general public, as members of the general public would not be suitably trained to mitigate harm, or may not have the capacity to do so. Occupationally-exposed individuals are not deemed to be at greater risk than the general public, providing that appropriate screening and training is provided to account for all known risks. Note that a fetus is here defined as a member of the general public, regardless of exposure scenario, and is subject to the general public restrictions.

As can be seen above, there are a number of steps involved in deriving ICNIRP's guidelines. ICNIRP adopts a conservative approach to each of these steps in order to ensure that its limits would remain protective even if exceeded by a substantial margin. For example, the choice of adverse health effects, presumed exposure scenarios, application of reduction factors and derivation of reference levels are all conducted conservatively. The degree of protection in the exposure levels is thus greater than may be suggested by considering only the reduction factors, which represent only one conservative element of the guidelines. There is no evidence that additional precautionary measures will result in a benefit to the health of the population.

SCIENTIFIC BASIS FOR LIMITING RADIOFREQUENCY EXPOSURE

100 kHz to 10 MHz EMF Frequency Range: Relation Between the Present and Other ICNIRP Guidelines

Although the present guidelines replace the 100 kHz to 10 MHz EMF frequency range of the ICNIRP (2010) guidelines, the science pertaining to direct radiofrequency EMF effects on nerve stimulation and associated restrictions within the ICNIRP (2010) guidelines has not been reconsidered here. Instead, the present process evaluated and set restrictions for adverse health effects *other than* direct effects on nerve stimulation from 100 kHz to 10 MHz, and for all adverse health effects from 10 MHz to 300 GHz. The restrictions relating to direct effects of nerve stimulation from the 2010 guidelines were then added to those derived in the present guidelines to form the final set of restrictions. Health and dosimetry considerations related to direct effects on nerve stimulation are therefore not provided here [see ICNIRP (2010) for further information].

Quantities, Units and Interaction Mechanisms

A brief overview of the electromagnetic quantities and units employed in this document, as well as the mechanisms of interaction of these with the body, is provided here. A more detailed description of the dosimetry relevant to the guidelines is provided in Appendix A, "Quantities and Units" section.

Radiofrequency EMFs consist of oscillating electric and magnetic fields; the number of oscillations per second is referred to as "frequency," and is described in units of hertz (Hz). As the field propagates away from a source, it transfers power from its source, described in units of watt (W), which is equivalent to joule (J, a measure of energy) per unit of time (t). When the field impacts upon material, it interacts with the atoms and molecules in that material. When a biological body is exposed to radiofrequency EMFs, some of the power is reflected away from the body, and some is absorbed by it. This results in complex patterns of electromagnetic fields inside the body that are heavily dependent on the EMF characteristics as well as the physical properties and dimensions of the body. The main component of the radiofrequency EMF that affects the body is the electric field. Electric fields inside the body are referred to as induced electric fields (E_{ind}, measured in volt per meter; V m^{-1}), and they can affect the body in different ways that are potentially relevant to health.

Firstly, the induced electric field in the body exerts a force on both polar molecules (mainly water molecules) and free moving charged particles such as electrons and ions. In both cases a portion of the EMF energy is converted to kinetic energy, forcing the polar molecules to rotate and charged particles to move as a current. As the polar molecules rotate and charged particles move, they typically interact with other polar molecules and charged particles, causing the kinetic energy to be converted to heat. This heat can adversely affect health in a range of ways. Secondly, if the induced electric field is below about 10 MHz and strong enough, it can exert electrical forces that are sufficient to stimulate nerves, and if the induced electric field is strong and brief enough (as can be the case for pulsed low frequency EMFs), it can exert electrical forces that are sufficient to cause dielectric breakdown of biological membranes, as occurs during direct current (DC) electroporation (Mir 2008).

From a health risk perspective, we are generally interested in how much EMF power is absorbed by biological tissues, as this is largely responsible for the heating effects described above. This is typically described as a function of a relevant dosimetric quantity. For example, below about 6 GHz, where EMFs penetrate deep into tissue (and thus

require depth to be considered), it is useful to describe this in terms of "specific energy absorption rate" (SAR), which is the power absorbed per unit mass (W kg^{-1}). Conversely, above 6 GHz, where EMFs are absorbed more superficially (making depth less relevant), it is useful to describe exposure in terms of the density of absorbed power over area (W m^{-2}), which we refer to as "absorbed power density" (S_{ab}). In these guidelines, SAR is specified over different masses to better match particular adverse health effects; SAR_{10g} represents the power absorbed (per kg) over a 10-g cubical mass, and whole-body average SAR represents power absorbed (per kg) over the entire body. Similarly, absorbed power density is specified over different areas as a function of EMF frequency. In some situations, the rate of energy deposition (power) is less relevant than the total energy deposition. This may be the case for brief exposures where there is not sufficient time for heat diffusion to occur. In such situations, specific energy absorption (SA, in J kg^{-1}) and absorbed energy density (U_{ab}, in J m^{-2}) are used, for EMFs below and above 6 GHz, respectively. SAR, S_{ab}, SA, U_{ab}, and E_{ind} are the quantities used in these guidelines to specify the basic restrictions.

As the quantities used to specify basic restrictions can be difficult to measure, quantities that are more easily evaluated are also specified, as reference levels. The reference level quantities relevant to these guidelines are incident electric field strength (E_{inc}) and incident magnetic field strength (H_{inc}), incident power density (S_{inc}), plane-wave equivalent incident power density (S_{eq}), incident energy density (U_{inc}), and plane-wave equivalent incident energy density (U_{eq}), all measured outside the body, and electric current inside the body, I, described in units of ampere (A). Basic restriction and reference level units are shown in Table 1, and definitions of all relevant terms provided in Appendix A, in the "Quantities and Units" section.

Radiofrequency EMF Health Research

In order to set safe exposure levels, ICNIRP first decided whether there was evidence that radiofrequency EMFs impair health, and for each adverse effect that was substantiated, both the mechanism of interaction and the minimum exposure required to cause harm were determined (where available). This information was obtained primarily from major international reviews of the literature on radiofrequency EMFs and health. This included an in-depth review from the World Health Organization on radiofrequency EMF exposure and health that was released as a draft Technical Document (WHO 2014), and reports by the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR 2015) and the Swedish Radiation Safety Authority (SSM 2015, 2016, 2018). These reports have reviewed an extensive body of literature, ranging from experimental research to epidemiology, and include consideration of health in children and those individuals thought to be sensitive to radiofrequency EMFs. To complement those reports, ICNIRP also considered research published since those reviews. A brief summary of this literature is provided in Appendix B, with the main conclusions provided below.

As described in Appendix B, in addition to nerve stimulation (described in ICNIRP 2010), radiofrequency EMFs can affect the body via two primary biological effects: changes in the permeability of membranes and temperature rise. Knowledge concerning relations between thermal effects and health, independent of the radiofrequency EMF literature, is also important and is described below. ICNIRP considers this appropriate given that the vast majority of radiofrequency EMF health research has been conducted

Table 1. Quantities and corresponding SI units used in these guidelines.

Quantity	Symbol[a]	Unit
Absorbed energy density	U_{ab}	joule per square meter (J m^{-2})
Incident energy density	U_{inc}	joule per square meter (J m^{-2})
Plane-wave equivalent incident energy density	U_{eq}	joule per square meter (J m^{-2})
Absorbed power density	S_{ab}	watt per square meter (W m^{-2})
Incident power density	S_{inc}	watt per square meter (W m^{-2})
Plane-wave equivalent incident power density	S_{eq}	watt per square meter (W m^{-2})
Induced electric field strength	E_{ind}	volt per meter (V m^{-1})
Incident electric field strength	E_{inc}	volt per meter (V m^{-1})
Incident electric field strength	E_{ind}	volt per meter (V m^{-1})
Incident magnetic field strength	H_{inc}	ampere per meter (A m^{-1})
Specific energy absorption	SA	joule per kilogram (J kg^{-1})
Specific energy absorption rate	SAR	watt per kilogram (W kg^{-1})
Electric current	I	ampere (A)
Frequency	*f*	hertz (Hz)
Time	*t*	second (s)

[a]*Italicized* symbols represent variables; quantities are described in scalar form because direction is not used to derive the basic restrictions or reference levels.

using exposures substantially lower than those shown to produce adverse health effects, with relatively little research addressing adverse health effect thresholds from known interaction mechanisms themselves. Thus, it is possible that the radiofrequency health literature may not be sufficiently comprehensive to ascertain precise thresholds. Conversely, where a more extensive literature is available that clarifies the relation between health and the primary biological effects, this can be useful for setting guidelines. For example, if the thermal physiology literature demonstrated that local temperature elevations of a particular magnitude caused harm, but radiofrequency exposure known to produce a similar temperature elevation had not been evaluated for harm, then it would be reasonable to also consider this thermal physiology literature. ICNIRP refers to thresholds derived from such additional literature as *operational* adverse health effect thresholds.

It is important to note that ICNIRP only uses operational thresholds to set restrictions where they are lower (more conservative) than those demonstrated to adversely affect health in the radiofrequency literature, or where the radiofrequency literature does not provide sufficient evidence to deduce an adverse health effect threshold. For the purpose of determining thresholds, evidence of adverse health effects arising from all radiofrequency EMF exposures is considered, including those referred to as 'low-level' and 'non-thermal', and including those where mechanisms have not been elucidated. Similarly, as there is no evidence that continuous (e.g., sinusoidal) and discontinuous (e.g., pulsed) EMFs result in different biological effects (Kowalczuk et al. 2010; Juutilainen et al. 2011), no theoretical distinction has been made between these types of exposure (all exposures have been considered empirically in terms of whether they adversely affect health).

Thresholds for Radiofrequency EMF-Induced Health Effects

Nerve stimulation. Exposure to EMFs can induce electric fields within the body, which for frequencies up to 10 MHz can stimulate nerves (Saunders and Jeffreys 2007). The effect of this stimulation varies as a function of frequency, and it is typically reported as a "tingling" sensation for frequencies around 100 kHz. As frequency increases, heating effects predominate and the likelihood of nerve stimulation decreases; at 10 MHz the effect of the electric field is typically described as "warmth." Nerve stimulation by induced electric fields is detailed in the ICNIRP low frequency guidelines (2010).

Changes to permeability of cell membranes. When (low frequency) EMFs are pulsed, the power is distributed across a range of frequencies, which can include radiofrequency EMFs (Joshi and Schoenbach 2010). If the pulse is sufficiently intense and brief, exposure to the resultant EMFs may cause cell membranes to become permeable, which in turn can lead to other cellular changes. However, there is no evidence that the radiofrequency spectral component from an EMF pulse (without the low-frequency component) is sufficient to cause changes in the permeability of cell membranes. The restrictions on nerve stimulation in the ICNIRP (2010) guidelines (and used here) are sufficient to ensure that permeability changes do not occur, so additional protection from the resultant radiofrequency EMFs is not necessary. Membrane permeability changes have also been shown to occur with 18 GHz continuous wave exposure (e.g., Nguyen et al. 2015). This has only been demonstrated in vitro, and the effect requires very high exposure levels (circa 5 kW kg^{-1}, over many minutes) that far exceed those required to cause thermally-induced harm (see "Temperature rise" section). Therefore, there is also no need to specifically set restrictions to protect against this effect, as the restrictions designed to protect against smaller temperature rises described in the "Temperature Rise" section will also provide protection against this.

Temperature rise. Radiofrequency EMFs can generate heat in the body and it is important that this heat is kept to a safe level. However, as can be seen from Appendix B, there is a dearth of radiofrequency exposure research using sufficient power to cause heat-induced health effects. Of particular note is that although exposures (and resultant temperature rises) have occasionally been shown to cause severe harm, the literature lacks concomitant evidence of the lowest exposures required to cause harm. For very low exposure levels (such as within the ICNIRP (1998) basic restrictions) there is extensive evidence that the amount of heat generated is not sufficient to cause harm, but for exposure levels above those of the ICNIRP (1998) basic restriction levels, there is limited research. Where there is good reason to expect health impairment at temperatures lower than those shown to impair health via radiofrequency EMF exposure, ICNIRP uses those lower temperatures as a basis for its restrictions (see "Radiofrequency EMF health research" section).

It is important to note that these guidelines restrict radiofrequency EMF exposure to limit temperature rise rather than absolute temperature, whereas health effects are primarily related to absolute temperature. This strategy is used because it is not feasible to limit absolute temperature, which is dependent on many factors that are outside the scope of these guidelines, such as environmental temperature, clothing and work rate. This means that if exposure caused a given temperature rise, this could improve, not affect, or impair health depending on a person's initial temperature. For example, mild heating can be pleasant if a person is cold, but unpleasant if they are already very hot. The restrictions are therefore set to avoid significant increase in temperature, where "significant" is considered in light of both potential harm and normal physiological temperature variation. These guidelines differentiate between steady-state temperature rises (where temperature increases

slowly, allowing time for heat to dissipate over a larger tissue mass and for thermoregulatory processes to counter temperature rise), and brief temperature rises (where there may not be sufficient time for heat to dissipate, which can result in larger temperature rises in small regions given the same absorbed radiofrequency energy). This distinction suggests the need to account for steady-state and brief exposure durations separately.

Steady-state temperature rise

Body core temperature. Body core temperature refers to the temperature deep within the body, such as in the abdomen and brain, and varies substantially as a function of such factors as sex, age, time of day, work rate, environmental conditions and thermoregulation. For example, although the mean body core temperature is approximately 37°C (and within the "normothermic" range[4]), this typically varies over a 24-h period to meet physiological needs, with the magnitude of the variation as large as 1°C (Reilly et al. 2007). As thermal load increases, thermoregulatory functions such as vasodilation and sweating can be engaged to restrict body core temperature rise. This is important because a variety of health effects can occur once body core temperature has increased by more than approximately 1°C (termed "hyperthermia"). For example, risk of accident increases with hyperthermia (Ramsey et al. 1983), and at body core temperatures >40°C it can lead to heat stroke, which can be fatal (Cheshire 2016).

Detailed guidelines are available for minimizing adverse health risk associated with hyperthermia within the occupational setting (ACGIH 2017). These aim to modify work environments in order to keep body core temperature within +1°C of normothermia, and require substantial knowledge of each particular situation due to the range of variables that can affect it. As described in Appendix B, body core temperature rise due to radiofrequency EMFs that results in harm is only seen where temperature increases more than +1°C, with no clear evidence of a specific threshold for adverse health effects. Due to the limited literature available, ICNIRP has adopted a conservative temperature rise value as the operational adverse health effect threshold (the 1°C rise of ACGIH 2017). It is important to note that significant physiological changes can occur when body core temperature increases by 1°C. Such changes are part of the body's normal thermoregulatory response (e.g., Van den Heuvel et al. 2017), and thus do not *in themselves* represent an adverse health effect.

Recent theoretical modeling and generalization from experimental research across a range of species predicts that exposures resulting in a whole-body average SAR of approximately 6 W kg^{-1}, within the 100 kHz to 6 GHz range, over at least a 1-hour interval under thermoneutral conditions[5] (28°C, naked, at rest), is required to induce a 1°C body core temperature rise in human adults. A higher SAR is required to reach this temperature rise in children due to their more-efficient heat dissipation (Hirata et al. 2013). However, given the limited measurement data available, ICNIRP has adopted a conservative position and uses 4 W kg^{-1} averaged over 30 min as the radiofrequency EMF exposure level corresponding to a body core temperature rise of 1°C. An averaging time of 30 min is used to take into account the time it takes to reach a steady-state temperature (for more details, see Appendix A, "Temporal averaging considerations" section). As a comparison, a human adult generates a total of approximately 1 W kg^{-1} at rest (Weyand et al. 2009), nearly 2 W kg^{-1} standing, and 12 W kg^{-1} running (Teunissen et al. 2007).

As EMF frequency increases, exposure of the body and the resultant heating becomes more superficial, and above about 6 GHz this heating occurs predominantly within the skin. For example, 86% of the power at 6 and 300 GHz is absorbed within 8 and 0.2 mm of the surface respectively (Sasaki et al. 2017). Compared to heat in deep tissues, heat in superficial tissues is more easily removed from the body because it is easier for the thermal energy to transfer to the environment. This is why basic restrictions to protect against body core temperature rise have traditionally been limited to frequencies below 10 GHz (e.g., ICNIRP 1998). However, research has shown that EMF frequencies above 300 GHz (e.g., infrared radiation) can increase body core temperature beyond the 1°C operational adverse health effect threshold described above (Brockow et al. 2007). This is because infrared radiation, as well as lower frequencies within the scope of the present guidelines, cause heating within the dermis, and the extensive vascular network within the dermis can transport this heat deep within the body. It is therefore appropriate to also protect against body core temperature rise above 6 GHz.

ICNIRP is not aware of research that has assessed the effect of 6 to 300 GHz EMFs on body core temperature, nor of research that has demonstrated that it is harmful. However, as a conservative measure, ICNIRP uses the 4 W kg^{-1} corresponding to the operational adverse health effect threshold for frequencies up to 6 GHz, for the >6 to 300 GHz range also. In support of this being a conservative value, it has been shown that 1260 W m^{-2} (incident power density) infrared radiation exposure to one side of the body results in a 1°C body core temperature rise (Brockow et al., 2007). If we related this to the exposure of a 70 kg adult with an exposed surface area of 1 m^2 and no skin reflectance, this would result in a whole-body exposure of approximately 18 W kg^{-1}; this is far higher than the 4 W kg^{-1} exposure level for EMFs below 6 GHz that is taken to represent a 1°C body

[4]Normothermia refers to the thermal state within the body whereby active thermoregulatory processes are not engaged to either increase or decrease body core temperature.

[5]Thermoneutral refers to environmental conditions that allow body core temperature to be maintained solely by altering skin blood flow.

core temperature rise. This is viewed as additionally conservative given that the Brockow et al. study reduced heat dissipation using a thermal blanket, which would underestimate the exposure required to increase body core temperature under typical conditions.

Local temperature. In addition to body core temperature, excessive localized heating can cause pain and thermal damage. There is an extensive literature showing that skin contact with temperatures below 42°C for extended periods will not cause pain or damage cells (e.g., Defrin et al. 2006). As described in Appendix B, this is consistent with the limited data available for radiofrequency EMF heating of the skin [e.g., Walters et al. (2000) reported a pain threshold of 43°C using 94 GHz exposure], but fewer data are available for heat sources that penetrate beyond the protective epidermis and to the heat-sensitive epidermis/dermis interface. However, there is also a substantial body of literature assessing thresholds for tissue damage which shows that damage can occur at tissue temperatures >41–43°C, with damage likelihood and severity increasing as a function of time at such temperatures (e.g., Dewhirst et al. 2003; Yarmolenko et al. 2011; Van Rhoon et al. 2013).

The present guidelines treat radiofrequency EMF exposure that results in local temperatures of 41°C or greater as potentially harmful. As body temperature varies as a function of body region, ICNIRP treats exposure to different regions separately. Corresponding to these regions, the present guidelines define two tissue types which, based on their temperature under normothermal conditions, are assigned different operational adverse health effect thresholds; "Type-1" tissue (all tissues in the upper arm, forearm, hand, thigh, leg, foot, pinna and the cornea, anterior chamber and iris of the eye, epidermal, dermal, fat, muscle, and bone tissue), and "Type-2" tissue (all tissues in the head, eye, abdomen, back, thorax, and pelvis, excluding those defined as Type-1 tissue). The normothermal temperature of Type 1 tissue is typically <33–36 °C, and that of Type-2 tissue <38.5 °C (DuBois 1941; Aschoff and Wever 1958; Arens and Zhang 2006; Shafahi and Vafai 2011). These values were used to define operational thresholds for local heat-induced health effects; adopting 41 °C as potentially harmful, the present guidelines take a conservative approach and treat radiofrequency EMF-induced temperature rises of 5°C and 2°C, within Type-1 and Type-2 tissue, respectively, as operational adverse health effect thresholds for local exposure.

It is difficult to set exposure restrictions as a function of the above tissue-type classification. ICNIRP thus defines two regions and sets separate exposure restrictions, where relevant, for these regions: "Head and Torso," comprising the head, eye, pinna, abdomen, back, thorax and pelvis, which includes both Type-1 and Type-2 tissue, and the "Limbs," comprising the upper arm, forearm, hand, thigh, leg and foot, which only includes Type-1 tissue. Exposure levels have been determined for each of these regions such that they do not result in temperature rises of more than 5°C and 2°C, in Type-1 and Type-2 tissue, respectively. As the Limbs, by definition, do not contain any Type-2 tissue, the operational adverse health effect threshold for the Limbs is always 5°C.

The testes can be viewed as representing a special case, whereby reversible, graded, functional change can occur within normal physiological temperature variation if maintained over extended periods, with no apparent threshold. For example, spermatogenesis is reversibly reduced as a result of the up to 2°C increase caused by normal activities such as sitting (relative to standing; Mieusset and Bujan 1995). Thus, it is possible that the operational adverse health effect threshold for Type-2 tissue may result in reversible changes to sperm function. However, there is currently no evidence that such effects are sufficient to impair health. Accordingly, ICNIRP views the operational adverse health effect threshold of 2°C for Type-2 tissue, which is within the normal physiological range for the testes, as appropriate for them also. Note that the operational adverse health effect threshold for Type-2 tissue, which includes the abdomen and thus potentially the fetus, is also consistent with protecting against the fetal temperature rise threshold of 2°C for teratogenic effects in animals (Edwards et al. 2003; Ziskin and Morrissey 2011).

Within the 100 kHz to 6 GHz EMF range, average SAR over 10 g provides an appropriate measure of the radiofrequency EMF-induced steady-state temperature rise within tissue. A 10-g mass is used because, although there can initially be EMF-induced temperature heterogeneity within that mass, heat diffusion rapidly distributes the thermal energy to a much larger volume that is well-represented by a 10-g cubic mass (Hirata and Fujiwara 2009). In specifying exposures that correspond to the operational adverse health effect thresholds, ICNIRP thus specifies an average exposure over a 10-g cubic mass, such that the exposure will keep the Type-1 and Type-2 tissue temperature rises to below 5 and 2°C respectively. Further, ICNIRP assumes realistic exposures (exposure scenarios that people may encounter in daily life, including occupationally), such as from EMFs from radio-communications sources. This method provides for higher exposures in the Limbs than in the Head and Torso. A SAR_{10g} of at least 20 W kg^{-1} is required to exceed the operational adverse health effect thresholds in the Head and Torso, and 40 W kg^{-1} in the Limbs, over an interval sufficient to produce a steady-state temperature (from a few minutes to 30 min). This time interval is operationalized as a 6-min average as it closely matches the thermal time constant for local exposure.

Within the >6 to 300 GHz range, EMF energy is deposited predominantly in superficial tissues; this makes SAR_{10g},

which includes deeper tissues, less relevant to this frequency range. Conversely, absorbed power density (S_{ab}) provides a measure of the power absorbed in tissue that closely approximates the superficial temperature rise (Funahashi et al. 2018). From 6 to 10 GHz there may still be significant absorption in the subcutaneous tissue. However, the maximum and thus worst-case temperature rise from 6 to 300 GHz is close to the skin surface, and exposure that will restrict temperature rise to below the operational adverse health effect threshold for Type-1 tissue (5°C) will also restrict temperature rise to below the operational adverse health effect threshold for Type-2 tissue (2°C). Note that there is uncertainty with regard to the precise frequency for the change from SAR to absorbed power density. Six GHz was chosen because at that frequency, most of the absorbed power is within the cutaneous tissue, which is within the upper half of a 10-g SAR cubic volume (that is, it can be represented by the 2.15 cm × 2.15 cm surface of the cube). Recent thermal modeling and analytical solutions suggest that for EMF frequencies between 6 and 30 GHz, the exposure over a square averaging area of 4 cm^2 provides a good estimate of local maximum temperature rise (Hashimoto et al. 2017; Foster et al. 2017). As frequency increases further, the averaging area needs to be reduced to account for the possibility of smaller beam diameters, such that it is 1 cm^2 from approximately 30 GHz to 300 GHz. Although the averaging area that best corresponds to temperature rise would therefore gradually change from 4 cm^2 to 1 cm^2 as frequency increases from 6 to 300 GHz, ICNIRP uses a square averaging area of 4 cm^2 for >6 to 300 GHz as a practical protection specification. Moreover, from >30 to 300 GHz (where focal beam exposure can occur), an additional spatial average of 1 cm^2 is used to ensure that the operational adverse health effect thresholds are not exceeded over smaller regions.

As 6 minutes is an appropriate averaging interval (Morimoto et al. 2017), and as an absorbed power density of approximately 200 W m^{-2} is required to produce the Type-1 tissue operational adverse health effect threshold of a 5°C local temperature rise for frequencies of >6 to 300 GHz (Sasaki et al. 2017), ICNIRP has set the absorbed power density value for local heating, averaged over 6 min and a square 4-cm^2 region, at 200 W m^{-2}; this will also restrict temperature rise in Type-2 tissue to below the operational adverse health effect threshold of 2°C. An additional specification of 400 W m^{-2} has been set for spatial averages of square 1-cm^2 regions, for frequencies >30 GHz.

Rapid temperature rise

For some types of exposure, rapid temperature rise can result in "hot spots," heterogeneous temperature distribution over tissue mass (Foster et al. 2016; Morimoto et al. 2017; Laakso et al. 2017; Kodera et al. 2018). This suggests the need to consider averaging over smaller time-intervals for certain types of exposure. Hot spots can occur for short duration exposures because there is not sufficient time for heat to dissipate (or average out) over tissue. This effect is more pronounced as frequency increases due to the smaller penetration depth.

To account for such heterogeneous temperature distributions, an adjustment to the steady-state exposure level is required. This can be achieved by specifying the maximum exposure level allowed, as a function of time, in order to restrict temperature rise to below the operational adverse health effect thresholds.

From 400 MHz to 6 GHz, ICNIRP specifies the restriction in terms of specific energy absorption (SA) of any 10-g cubic mass, where SA is restricted to $7.2[0.05+0.95(t/360)^{0.5}]$ kJ kg^{-1} for Head and Torso, and $14.4[0.025+0.975(t/360)^{0.5}]$ kJ kg^{-1} for Limb exposure, where t is exposure interval in seconds (Kodera et al. 2018). Note that for this specification, exposure from any pulse, group of pulses, or subgroup of pulses in a train, as well as from the total (sum) of exposures (including non-pulsed EMF), delivered in t seconds, must not exceed the below formulae (in order to ensure that the temperature thresholds are not exceeded).

There is no brief-interval exposure level specified below 400 MHz because, due to the large penetration depth, the total SA resulting from the 6-minute local SAR average cannot increase temperature by more than the operational adverse health effect threshold (regardless of the particular pattern of pulses or brief exposures).

Above 6 GHz, ICNIRP specifies the exposure level for both Head and Torso, and Limbs, in terms of absorbed energy density (U_{ab}) over any square averaging area of 4 cm^2, such that U_{ab} is specified as $72[0.05+0.95(t/360)^{0.5}]$ kJ m^{-2}, where t is the exposure interval in seconds (extension of Kodera et al. 2018).

An additional exposure level for square 1-cm^2 averaging areas is applicable for EMFs with frequencies of >30 to 300 GHz to account for focused beam exposure and is given by $144[0.025+0.975(t/360)^{0.5}]$ kJ m^{-2}.

The SA and U_{ab} values are conservative in that they are not sufficient to raise Type 1 or Type 2 tissue temperatures by 5 or 2°C, respectively.

GUIDELINES FOR LIMITING RADIOFREQUENCY EMF EXPOSURE

As described in the "Scientific Basis for Limiting Radiofrequency Exposure" section, radiofrequency EMF levels corresponding to operational adverse health effects were identified. Basic restrictions have been derived from these and are described in the "Basic Restrictions" section below. The basic restrictions related to nerve stimulation

Table 2. Basic restrictions for electromagnetic field exposure from 100 kHz to 300 GHz, for averaging intervals ≥6 min.[a]

Exposure scenario	Frequency range	Whole-body average SAR (W kg^{-1})	Local Head/Torso SAR (W kg^{-1})	Local Limb SAR (W kg^{-1})	Local S_{ab} (W m^{-2})
Occupational	100 kHz to 6 GHz	0.4	10	20	NA
	>6 to 300 GHz	0.4	NA	NA	100
General public	100 kHz to 6 GHz	0.08	2	4	NA
	>6 to 300 GHz	0.08	NA	NA	20

[a]Note:

1. "NA" signifies "not applicable" and does not need to be taken into account when determining compliance.
2. Whole-body average SAR is to be averaged over 30 min.
3. Local SAR and S_{ab} exposures are to be averaged over 6 min.
4. Local SAR is to be averaged over a 10-g cubic mass.
5. Local S_{ab} is to be averaged over a square 4-cm^2 surface area of the body. Above 30 GHz, an additional constraint is imposed, such that exposure averaged over a square 1-cm^2 surface area of the body is restricted to two times that of the 4-cm^2 restriction.

for EMF frequencies 100 kHz to 10 MHz, from ICNIRP (2010), were then added to the present set of basic restrictions, with the final set of basic restrictions given in Tables 2–4. Reference levels were derived from those final basic restrictions and are described in the "Reference Levels" section, with details of how to treat multiple frequency fields in terms of the restrictions in the "Simultaneous Exposure to Multiple Frequency Fields" section. Contact current guidance is provided in the "Guidance for Contact Currents", and health considerations for occupational exposure are described in the "Risk Mitigations Considerations for Occupational Exposure" section. To be compliant with the present guidelines, for each exposure quantity (e.g., E-field, H-field, SAR), and temporal and spatial averaging condition, either the basic restriction or corresponding reference level must be adhered to; compliance with both is not required. Note that where restrictions specify particular averaging intervals, 'all' such averaging intervals must comply with the restrictions.

Basic Restrictions

Basic restriction values are provided in Tables 2–4 with an overview of their derivation described below. As described above, the basic restrictions from ICNIRP (2010) for the frequency range 100 kHz to 10 MHz have not been re-evaluated here; these are described in Table 4. A more detailed description of issues pertinent to the basic restrictions is provided in Appendix A, in the "Relevant Biophysical Mechanisms" section. Note that for the basic restrictions described below, a pregnant woman is treated as a member of the general public. This is because recent modeling suggests that for both whole-body and local exposure scenarios, exposure of the mother at the occupational basic restrictions can lead to fetal exposures that exceed the general public basic restrictions.

Whole-body average SAR (100 kHz to 300 GHz). As described in the "Body core temperature" section, the guidelines take a whole-body average SAR of 4 W kg^{-1},

Table 3. Basic restrictions for electromagnetic field exposure from 100 kHz to 300 GHz, for integrating intervals >0 to <6 min.[a]

Exposure scenario	Frequency range	Local Head/Torso SA (kJ kg^{-1})	Local Limb SA (kJ kg^{-1})	Local U_{ab} (kJ m^{-2})
Occupational	100 kHz to 400 MHz	NA	NA	NA
	>400 MHz to 6 GHz	$3.6[0.05+0.95(t/360)^{0.5}]$	$7.2[0.025+0.975(t/360)^{0.5}]$	NA
	>6 to 300 GHz	NA	NA	$36[0.05+0.95(t/360)^{0.5}]$
General public	100 kHz to 400 MHz	NA	NA	NA
	>400 MHz to 6 GHz	$0.72[0.05+0.95(t/360)^{0.5}]$	$1.44[0.025+0.975(t/360)^{0.5}]$	NA
	>6 to 300 GHz	NA	NA	$7.2[0.05+0.95(t/360)^{0.5}]$

[a]Note:

1. "NA" signifies "not applicable" and does not need to be taken into account when determining compliance.
2. t is time in seconds, and restrictions must be satisfied for all values of t between >0 and <360 s, regardless of the temporal characteristics of the exposure itself.
3. Local SA is to be averaged over a 10-g cubic mass.
4. Local U_{ab} is to be averaged over a square 4-cm^2 surface area of the body. Above 30 GHz, an additional constraint is imposed, such that exposure averaged over a square 1-cm^2 surface area of the body is restricted to $72[0.025+0.975(t/360)^{0.5}]$ for occupational and $14.4[0.025+0.975(t/360)^{0.5}]$ for general public exposure.
5. Exposure from any pulse, group of pulses, or subgroup of pulses in a train, as well as from the summation of exposures (including non-pulsed EMFs), delivered in t s, must not exceed these levels.

Table 4. Basic restrictions for electromagnetic field exposure from 100 kHz to 10 MHz, for peak spatial values.[a]

Exposure scenario	Frequency range	Induced electric field; E_{ind} (V m^{-1})
Occupational	100 kHz to 10 MHz	$2.70 \times 10^{-4} f$
General public	100 kHz to 10 MHz	$1.35 \times 10^{-4} f$

[a]Note:

1. f is frequency in Hz.

2. Restriction values relate to any region of the body, and are to be averaged as root mean square (rms) values over 2 mm × 2 mm × 2 mm contiguous tissue (as specified in ICNIRP 2010).

averaged over the entire body mass and a 30-minute interval, as the exposure level corresponding to the operational adverse health effect threshold for an increase in body core temperature of 1°C. A reduction factor of 10 was applied to this threshold for occupational exposure to account for scientific uncertainty, as well as differences in thermal physiology across the population and variability in environmental conditions and physical activity levels. Variability in an individual's ability to regulate their body core temperature is particularly important as it is dependent on a range of factors that the guidelines cannot control. These include central and peripherally-mediated changes to blood perfusion and sweat rate (which are in turn affected by a range of other factors, including age and certain medical conditions), as well as behavior and environmental conditions.

Thus the basic restriction for occupational exposure becomes a whole-body average SAR of 0.4 W kg^{-1}, averaged over 30 min. Although this means that SAR can be larger for smaller time intervals, this will not affect body core temperature rise appreciably because the temperature will be "averaged-out" within the body over the 30-min interval, and it is this time-averaged temperature rise that is relevant here. Further, as both whole-body and local restrictions must be met simultaneously, exposures sufficiently high to be hazardous locally will be protected against by the local restrictions described below.

As the general public cannot be expected to be aware of exposures and thus to mitigate risk, a reduction factor of 50 was applied for the general public, making the whole-body average SAR restriction for the general public 0.08 W kg^{-1}, averaged over 30 min.

It is noteworthy that the scientific uncertainty pertaining to both dosimetry and potential health consequences of whole-body radiofrequency exposure have reduced substantially since the ICNIRP (1998) guidelines. This would justify less conservative reduction factors, but as ICNIRP considers that the benefits of maintaining stable basic restrictions outweighs any benefits that subtle changes to them would provide, ICNIRP has retained the same reduction factors as before for the whole-body average basic restrictions. Similarly, although temperature rise is more superficial as frequency increases (and thus it is easier for the resultant heat to be lost to the environment), the whole-body average SAR restrictions above 6 GHz have been conservatively set the same as those ≤6 GHz.

Local SAR (100 kHz to 6 GHz)

Head and Torso

As described in the "Local temperature" section within the 100 kHz to 6 GHz range, the guidelines take a SAR of 20 W kg^{-1}, averaged over a 10-g cubic mass and 6-min interval, as the local exposure level corresponding to the operational adverse health effect threshold for the Head and Torso (5°C in Type-1 tissue and 2°C in Type-2 tissue). A reduction factor of 2 was applied to this for occupational exposure to account for scientific uncertainty, as well as differences in thermal physiology across the population and variability in environmental conditions and physical activity levels. Reduction factors for local exposure are smaller than for whole-body exposure because the associated health effect threshold is less dependent on environmental conditions and the highly variable centrally-mediated thermoregulatory processes, and because the associated health effect is less serious medically. Thus, the basic restriction for occupational exposure becomes a SAR_{10g} of 10 W kg^{-1}, averaged over a 6-min interval. As the general public cannot be expected to be aware of exposures and thus to mitigate risk, and also recognizing greater differences in thermal physiology in the general population, a reduction factor of 10 was applied for the general public, reducing the general public basic restriction to a SAR_{10g} of 2 W kg^{-1} averaged over a 6-min interval.

Limbs

As described in the "Local temperature" section, within the 100 kHz to 6 GHz range, the guidelines take a SAR of 40 W kg^{-1}, averaged over a 10-g cubic mass and 6-min interval, as the local exposure level corresponding to the operational adverse health effect threshold for the Limbs of a 5°C rise in local temperature. As with the Head and Torso restrictions, a reduction factor of 2 was applied to this threshold for occupational exposure to account for scientific uncertainty, as well as differences in thermal physiology across the population and variability in environmental conditions and physical activity levels. This results in a basic restriction for occupational exposure of a SAR_{10g} of 20 W kg^{-1}. As the general public cannot be expected to be aware of exposures and thus to mitigate risk, and also to recognize greater differences in thermal physiology in the general population, a reduction factor of 10 was applied for the general public, reducing the general public restriction to 4 W kg^{-1} averaged over a 6-min interval.

Local SA (400 MHz to 6 GHz). As described in the "Rapid temperature rise" section, within the >400 MHz to 6 GHz range, an additional constraint is required to ensure that the cumulative energy permitted by the 6-minute

average SAR_{10g} basic restriction is not absorbed by tissues too rapidly. Accordingly, ICNIRP sets an SA level for exposure intervals of less than 6 min, as a function of time, to limit temperature rise to below the operational adverse health effect thresholds. This SA level, averaged over a 10-g cubic mass, is given by $7.2[0.05+0.95(t/360)^{0.5}]$ kJ kg^{-1} for the Head and Torso, and $14.4[0.025+0.975(t/360)^{0.5}]$ kJ kg^{-1} for the Limbs, where t is exposure duration in seconds.

As with the SAR_{10g} basic restrictions, a reduction factor of 2 was applied to these exposure levels for occupational exposure to account for scientific uncertainty, as well as differences in thermal physiology across the population and variability in environmental conditions and physical activity levels. This results in a basic restriction for the Head and Torso of $3.6[0.05+0.95(t/360)^{0.5}]$ kJ kg^{-1}, and for the Limbs of $7.2[0.025+0.975(t/360)^{0.5}]$ kJ kg^{-1}. As the general public cannot be expected to be aware of exposures and thus to mitigate risk, and to recognize greater differences in thermal physiology in the general population, a reduction factor of 10 was applied for the general public. This makes the general public restriction $0.72[0.05+0.95(t/360)^{0.5}]$ kJ kg^{-1} for the Head and Torso, and $1.44[0.025+0.975(t/360)^{0.5}]$ kJ kg^{-1} for the Limbs.

Note that for these brief exposure basic restrictions, the exposure from any pulse, group of pulses, or subgroup of pulses in a train, as well as from the summation of exposures (including non-pulsed EMFs), delivered in t seconds, must not exceed these local SA values.

Local absorbed power density (>6 GHz to 300 GHz). As described in the "Local temperature" section, within the >6 to 300 GHz range, the guidelines take an absorbed power density of 200 W m^{-2}, averaged over 6 min and a square 4-cm^2 surface area of the body, as the local exposure corresponding to the operational adverse health effect threshold for both the Head and Torso, and Limb regions (5 and 2°C local temperature rise in Type-1 and Type-2 tissue, respectively). As with the local SAR restrictions, a reduction factor of 2 was applied to this exposure level for occupational exposure to account for scientific uncertainty, as well as differences in thermal physiology across the population and variability in environmental conditions and physical activity levels. This results in a basic restriction for occupational exposure of 100 W m^{-2}, averaged over 6 min and a square 4-cm^2 surface area of the body.

As the general public cannot be expected to be aware of these exposures and thus to mitigate risk, and to recognize greater differences in thermal physiology in the general population, a reduction factor of 10 was applied, which reduces the general public basic restriction to 20 W m^{-2}, averaged over 6 min and a square 4-cm^2 surface area of the body.

Further, to account for focal beam exposure from >30 to 300 GHz, absorbed power density averaged over a square 1-cm^2 surface area of the body must not exceed 2 times that of the 4-cm^2 basic restrictions for workers or the general public.

Local absorbed energy density (>6 GHz to 300 GHz). As described in the "Rapid temperature rise" section, within the >6 to 300 GHz range, an additional constraint is required to ensure that the cumulative energy permitted by the 6-min average absorbed power density basic restriction is not absorbed by tissue too rapidly. Accordingly, for both the Head and Torso, and Limbs, ICNIRP set a maximum absorbed energy density level for exposure intervals of less than 6 minutes, as a function of time, to limit temperature rise to below the operational adverse health effect thresholds for both Type-1 and Type-2 tissues. This absorbed energy density level, averaged over any square 4-cm^2 surface area of the body, is given by $72[0.05+0.95(t/360)^{0.5}]$ kJ m^{-2}, where t is exposure duration in seconds. To account for focal beam exposure from >30 to 300 GHz, the absorbed energy density level corresponding to the operational adverse health effect threshold, averaged over a square 1-cm^2 surface area of the body, is given by $144[0.025+0.975(t/360)^{0.5}]$ kJ m^{-2}. Note that for these basic restrictions for brief exposures, the exposure from any pulse, group of pulses, or subgroup of pulses in a train, as well as from the summation of exposures (including non-pulsed EMFs), delivered in t seconds, must be used to satisfy this formula.

As with the absorbed power density basic restrictions, a reduction factor of 2 was applied to this exposure level for occupational exposure to account for scientific uncertainty, as well as differences in thermal physiology across the population and variability in environmental conditions and physical activity levels. This results in a basic restriction for occupational exposure of $36[0.05+0.95(t/360)^{0.5}]$ kJ m^{-2}, over any square 4-cm^2 surface area of the body. From >30 to 300 GHz, an additional basic restriction for occupational exposure is $72[0.025+0.975(t/360)^{0.5}]$ kJ m^{-2}, averaged over any square 1-cm^2 surface area of the body. As the general public cannot be expected to be aware of exposures and thus to mitigate risk, and to recognize greater differences in thermal physiology in the general population, a reduction factor of 10 was applied for the general public, reducing the general public restriction to $7.2[0.05+0.95(t/360)^{0.5}]$ kJ m^{-2}, averaged over any square 4-cm^2 surface area of the body. From >30 to 300 GHz, an additional basic restriction for the general public is $14.4[0.025+0.975(t/360)^{0.5}]$ kJ m^{-2}, averaged over any square 1-cm^2 surface area of the body.

Basic restriction tables. To be compliant with the basic restrictions, radiofrequency EMF exposure must not exceed the restrictions specified for that EMF frequency in Table 2, 3 or 4. That is, for any given radiofrequency EMF frequency, relevant whole-body SAR, local

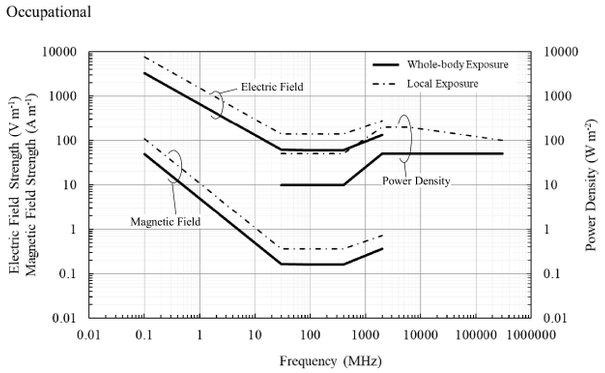

FIGURE 1. Reference levels for time averaged occupational exposures of ≥6 min, to electromagnetic fields from 100 kHz to 300 GHz (unperturbed rms values; see Tables 5 and 6 for full specifications).

SAR, S_{ab}, SA, U_{ab} and induced E-field[6] restrictions must be met simultaneously.

Reference Levels

Reference levels have been derived from a combination of computational and measurement studies to provide a means of demonstrating compliance using quantities that are more-easily assessed than basic restrictions, but that provide an equivalent level of protection to the basic restrictions for worst-case exposure scenarios. However, as the derivations rely on conservative assumptions, in most exposure scenarios the reference levels will be more conservative than the corresponding basic restrictions. Further details regarding the reference levels are provided in Appendix A, the "Derivation of Reference Levels" section.

Reference levels are provided in Tables 5–9. Figures 1 and 2 provide graphical representations of the occupational and general public reference level values for extended durations of exposure (≥6 min). Table 5 reference levels are averaged over a 30-min interval, and correspond to the whole-body average basic restrictions. Table 6 (averaged over a 6-min interval), Table 7 (integrated over intervals between >0 and <6 min), and Table 8 (peak instantaneous field strength measures) each relate to basic restrictions that are averaged over smaller body regions. Additional limb current reference levels have been set to account for effects of grounding near human body resonance frequencies (Dimbylow 2001) that might otherwise lead to reference levels underestimating exposures within tissue at certain EMF frequencies (averaged over 6 min; Table 9). Limb current reference levels are only relevant in exposure scenarios where a person is not electrically isolated.

[6]Note that although the term internal is used in place of induced in ICNIRP (2010), induced is used here for consistency within the present document.

Tables 5–9 specify averaging and integrating times of the relevant exposure quantities to determine whether personal exposure level is compliant with the guidelines. These averaging times are not necessarily the same as the measurement times needed to estimate field strengths or other exposure quantities. Depending on input from technical standards bodies, actual measurement times used to provide an appropriate estimate of exposure quantities may be shorter than the intervals specified in these tables.

An important consideration for the application of reference levels is to what degree the quantities used to assess compliance with the reference levels (i.e., E_{inc}, H_{inc}, S_{inc}, U_{inc}, S_{eq}, U_{eq}, I) adequately predict the quantities used to assess compliance with the basic restrictions. In situations where reference level quantities are associated with greater uncertainty, reference levels must be applied more conservatively. For the purposes of the guidelines, the degree of adequacy strongly depends on whether external EMFs can be considered to be within the far-field, radiative near-field or reactive near-field zone. Accordingly, in most cases, different reference level assessment rules have been set for EMFs as a function of whether they are within the far-field, radiative or reactive near-field zone.

A difficulty with this approach is that other factors may also affect the adequacy of estimating basic restriction quantities from reference level quantities. These include the EMF frequency, physical dimensions of the EMF source and its distance from the resultant external EMFs assessed, as well as the degree to which the EMFs vary over the space to be occupied by a person. Taking into account such sources of uncertainty, the guidelines have more conservative rules for exposure in the reactive and radiative near-field than far-field zone. It is noted that there is no simple delineation of the far-field, radiative and reactive near-field zones that is sufficient for ensuring that reference levels will adequately correspond to the basic restrictions. Accordingly, although a definition of these zones is provided in

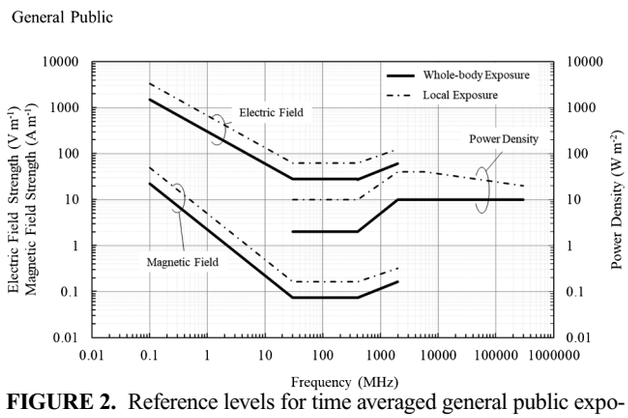

FIGURE 2. Reference levels for time averaged general public exposures of ≥6 min, to electromagnetic fields from 100 kHz to 300 GHz (unperturbed rms values; see Tables 5 and 6 for full specifications).

Table 5. Reference levels for exposure, averaged over 30 min and the whole body, to electromagnetic fields from 100 kHz to 300 GHz (unperturbed rms values).[a]

Exposure scenario	Frequency range	Incident E-field strength; E_{inc} (V m^{-1})	Incident H-field strength; H_{inc} (A m^{-1})	Incident power density; S_{inc} (W m^{-2})
Occupational	0.1 – 30 MHz	$660/f_M^{0.7}$	$4.9/f_M$	NA
	>30 – 400 MHz	61	0.16	10
	>400 – 2000 MHz	$3f_M^{0.5}$	$0.008f_M^{0.5}$	$f_M/40$
	>2 – 300 GHz	NA	NA	50
General public	0.1 – 30 MHz	$300/f_M^{0.7}$	$2.2/f_M$	NA
	>30 – 400 MHz	27.7	0.073	2
	>400 – 2000 MHz	$1.375f_M^{0.5}$	$0.0037f_M^{0.5}$	$f_M/200$
	>2 – 300 GHz	NA	NA	10

[a]Note:

1. "NA" signifies "not applicable" and does not need to be taken into account when determining compliance.

2. f_M is frequency in MHz.

3. S_{inc}, E_{inc}, and H_{inc} are to be averaged over 30 min, over the whole-body space. Temporal and spatial averaging of each of E_{inc} and H_{inc} must be conducted by averaging over the relevant square values (see eqn 8 in Appendix A for details).

4. For frequencies of 100 kHz to 30 MHz, regardless of the far-field/near-field zone distinctions, compliance is demonstrated if neither E_{inc} or H_{inc} exceeds the above reference level values.

5. For frequencies of >30 MHz to 2 GHz: (a) within the far-field zone: compliance is demonstrated if either S_{inc}, E_{inc} or H_{inc}, does not exceed the above reference level values (only one is required); S_{eq} may be substituted for S_{inc}; (b) within the radiative near-field zone, compliance is demonstrated if either S_{inc}, or both E_{inc} and H_{inc}, does not exceed the above reference level values; and (c) within the reactive near-field zone: compliance is demonstrated if both E_{inc} and H_{inc} do not exceed the above reference level values; S_{inc} cannot be used to demonstrate compliance, and so basic restrictions must be assessed.

6. For frequencies of >2 GHz to 300 GHz: (a) within the far-field zone: compliance is demonstrated if S_{inc} does not exceed the above reference level values; S_{eq} may be substituted for S_{inc}; (b) within the radiative near-field zone, compliance is demonstrated if S_{inc} does not exceed the above reference level values; and (c) within the reactive near-field zone, reference levels cannot be used to determine compliance, and so basic restrictions must be assessed.

Appendix A in the "General Considerations for Reference Levels" section this is only intended as a guide, and information from a technical standards body, designed to specify external exposures for each EMF source type to more adequately match the basic restrictions, should be utilized to improve reference level assessment procedures.

Related to the near- and far-field zone distinctions, for some exposure conditions the less onerous plane wave equivalent incident power density (S_{eq}) and plane wave equivalent incident energy density (U_{eq}) quantities can be used in place of S_{inc} and U_{inc}, respectively; where this is permitted, it is specified below. In such cases, the *plane wave equivalent incident energy densities* are to be averaged in the same way as described in Tables 5–7 for the corresponding *incident power densities*.

In terms of electromagnetic fields in the far-field zone, the following rules apply. For EMF frequencies from >30 MHz to 2 GHz, ICNIRP requires compliance to be demonstrated for only one of the E-field, H-field or S_{inc} quantities in order to be compliant with that particular reference level. Further, S_{eq} can be substituted for S_{inc}. Similarly, for EMF frequencies >400 MHz where the restrictions are specified in terms of U_{inc}, these can be substituted for by U_{eq}. EMF frequencies from 100 kHz to 30 MHz are treated as always being within the near-field zone; see next paragraph.

In terms of electromagnetic fields in the near-field zones, the following rules apply. From 100 kHz to 30 MHz, relevant personal exposures from present radiofrequency EMF sources are typically within the near-field zone. The present guidelines treat *all* exposures within this frequency range as near-field, and requires compliance with both the E-field and H-field reference level values in order to be compliant with the reference levels. For EMF frequencies from >30 MHz to 2 GHz, personal exposure within either the radiative or reactive near-field zones is treated as compliant if both the E-field and H-field strengths are below the reference level values described in the tables. For frequencies >30 MHz to 300 GHz, personal exposure within the radiative near-field zone is treated as compliant if S_{inc} (or, where relevant U_{inc}) is below the reference level value. However, for exposure within the >2 to 300 GHz range, within the reactive near-field the quantities applied for the reference level values are treated as inadequate to ensure compliance with the basic restrictions. In such cases, compliance with the basic restrictions must be assessed.

ICNIRP is aware that for some exposure scenarios, radiofrequency EMFs at the reference levels specified below could potentially result in exposure that exceeds basic restrictions. Where such scenarios were identified, ICNIRP determined whether the reference levels needed to be reduced by considering the magnitude of the difference between the resultant tissue exposure and corresponding basic restriction (including comparison with the associated dosimetric uncertainty), and whether the violation was likely to adversely affect health (including consideration of the degree of conservativeness in the associated basic

Table 6. Reference levels for local exposure, averaged over 6 min, to electromagnetic fields from 100 kHz to 300 GHz (unperturbed rms values).[a]

Exposure scenario	Frequency range	Incident E-field strength; E_{inc} (V m^{-1})	Incident H-field strength; H_{inc} (A m^{-1})	Incident power density; S_{inc} (W m^{-2})
Occupational	0.1 – 30 MHz	$1504/f_M^{0.7}$	$10.8/f_M$	NA
	>30 – 400 MHz	139	0.36	50
	>400 – 2000 MHz	$10.58f_M^{0.43}$	$0.0274f_M^{0.43}$	$0.29f_M^{0.86}$
	>2 – 6 GHz	NA	NA	200
	>6 – <300 GHz	NA	NA	$275/f_G^{0.177}$
	300 GHz	NA	NA	100
General public	0.1 – 30 MHz	$671/f_M^{0.7}$	$4.9/f_M$	NA
	>30 – 400 MHz	62	0.163	10
	>400 – 2000 MHz	$4.72f_M^{0.43}$	$0.0123f_M^{0.43}$	$0.058f_M^{0.86}$
	>2 – 6 GHz	NA	NA	40
	>6 – 300 GHz	NA	NA	$55/f_G^{0.177}$
	300 GHz	NA	NA	20

[a] Note:

1. "NA" signifies "not applicable" and does not need to be taken into account when determining compliance.

2. f_M is frequency in MHz; f_G is frequency in GHz.

3. S_{inc}, E_{inc}, and H_{inc} are to be averaged over 6 min, and where spatial averaging is specified in Notes 6–7, over the relevant projected body space. Temporal and spatial averaging of each of E_{inc} and H_{inc} must be conducted by averaging over the relevant square values (see eqn 8 in Appendix A for details).

4. For frequencies of 100 kHz to 30 MHz, regardless of the far-field/near-field zone distinctions, compliance is demonstrated if neither peak spatial E_{inc} or peak spatial H_{inc}, over the projected whole-body space, exceeds the above reference level values.

5. For frequencies of >30 MHz to 6 GHz: (a) within the far-field zone, compliance is demonstrated if one of peak spatial S_{inc}, E_{inc} or H_{inc}, over the projected whole-body space, does not exceed the above reference level values (only one is required); S_{eq} may be substituted for S_{inc}; (b) within the radiative near-field zone, compliance is demonstrated if either peak spatial S_{inc}, or both peak spatial E_{inc} and H_{inc}, over the projected whole-body space, does not exceed the above reference level values; and (c) within the reactive near-field zone: compliance is demonstrated if both E_{inc} and H_{inc} do not exceed the above reference level values; S_{inc} cannot be used to demonstrate compliance; for frequencies >2 GHz, reference levels cannot be used to determine compliance, and so basic restrictions must be assessed.

6. For frequencies of >6 GHz to 300 GHz: (a) within the far-field zone, compliance is demonstrated if S_{inc}, averaged over a square 4-cm^2 projected body surface space, does not exceed the above reference level values; S_{eq} may be substituted for S_{inc}; (b) within the radiative near-field zone, compliance is demonstrated if S_{inc}, averaged over a square 4-cm^2 projected body surface space, does not exceed the above reference level values; and (c) within the reactive near-field zone reference levels cannot be used to determine compliance, and so basic restrictions must be assessed.

7. For frequencies of >30 GHz to 300 GHz, exposure averaged over a square 1-cm^2 projected body surface space must not exceed twice that of the square 4-cm^2 restrictions.

restriction). Where the difference was small, and where it would not adversely affect health, reference levels were retained that can potentially result in exposures that exceed the basic restrictions.

This situation has been shown to occur in terms of the reference levels corresponding to whole-body average SAR basic restrictions, which, in the frequency range of body resonance (up to 100 MHz) and from 1 to 4 GHz, can potentially lead to whole-body average SARs that exceed the basic restrictions (ICNIRP 2009). The exposure scenario where this can potentially occur is very specific, requiring a small stature person (such as a 3-years-old child) to be extended (e.g., standing still and straight with arms above the head) for at least 30 min, while being subject to a plane wave exposure within the above frequency ranges, incident to the child from front to back. The resultant SAR elevation is small relative to the basic restriction (15–40%), which is similar to or smaller than the whole-body average SAR measurement uncertainty (Flintoft et al. 2014; Nagaoka and Watanabe 2019), there are many levels of conservativeness built into the basic restriction derivation itself, and importantly, this will not impact on health. This latter point is important because the basic restriction that this relates to was set to protect against body core temperature rises of greater than 1°C, and being of small stature, the individual in this hypothetical exposure scenario would more easily dissipate heat to the environment than a larger person due to their increased body "surface area-to-mass ratio" (Hirata et al. 2013). Within a small stature person the net effect of this "increased whole-body average SAR" and "increased heat loss" would be a smaller temperature rise than would occur in a person of larger stature who did not exceed the basic restriction, and in both cases would be substantially smaller than 1°C. ICNIRP has thus not altered the reference levels to account for this situation.

Simultaneous Exposure to Multiple Frequency Fields

It is important to determine whether, in situations of simultaneous exposure to fields of different frequencies, these

Table 7. Reference levels for local exposure, integrated over intervals of between >0 and <6 minutes, to electromagnetic fields from 100 kHz to 300 GHz (unperturbed rms values).[a]

Exposure scenario	Frequency range	Incident energy density; U_{inc} (kJ m^{-2})
Occupational	100 kHz – 400 MHz	NA
	>400 – 2000 MHz	$0.29f_M^{0.86} \times 0.36[0.05+0.95(t/360)^{0.5}]$
	>2 – 6 GHz	$200 \times 0.36[0.05+0.95(t/360)^{0.5}]$
	>6 – <300 GHz	$275/f_G^{0.177} \times 0.36[0.05+0.95(t/360)^{0.5}]$
	300 GHz	$100 \times 0.36[0.05+0.95(t/360)^{0.5}]$
General public	100 kHz – 400 MHz	NA
	>400 – 2000 MHz	$0.058f_M^{0.86} \times 0.36[0.05+0.95(t/360)^{0.5}]$
	>2 – 6 GHz	$40 \times 0.36[0.05+0.95(t/360)^{0.5}]$
	>6 – <300 GHz	$55/f_G^{0.177} \times 0.36[0.05+0.95(t/360)^{0.5}]$
	300 GHz	$20 \times 0.36[0.05+0.95(t/360)^{0.5}]$

[a]Note:

1. "NA" signifies "not applicable" and does not need to be taken into account when determining compliance.

2. f_M is frequency in MHz; f_G is frequency in GHz; t is time interval in seconds, such that exposure from any pulse, group of pulses, or subgroup of pulses in a train, as well as from the summation of exposures (including non-pulsed EMFs), delivered in t seconds, must not exceed these reference level values.

3. U_{inc} is to be calculated over time t, and where spatial averaging is specified in Notes 5–7, over the relevant projected body space.

4. For frequencies of 100 kHz to 400 MHz, >0 to <6-min restrictions are not required and so reference levels have not been set.

5. For frequencies of >400 MHz to 6 GHz: (a) within the far-field zone: compliance is demonstrated if peak spatial U_{inc}, over the projected whole-body space, does not exceed the above reference level values; U_{eq} may be substituted for U_{inc}; (b) within the radiative near-field zone, compliance is demonstrated if peak spatial U_{inc}, over the projected whole-body space, does not exceed the above reference level values; and (c) within the reactive near-field zone, reference levels cannot be used to determine compliance, and so basic restrictions must be assessed.

6. For frequencies of >6 GHz to 300 GHz: (a) within the far-field or radiative near-field zone, compliance is demonstrated if U_{inc}, averaged over a square 4-cm^2 projected body surface space, does not exceed the above reference level values; (b) within the reactive near-field zone, reference levels cannot be used to determine compliance, and so basic restrictions must be assessed.

7. For frequencies of >30 GHz to 300 GHz: exposure averaged over a square 1-cm^2 projected body surface space must not exceed $275/f_G^{0.177} \times 0.72[0.025+0.975(t/360)^{0.5}]$ kJ m^{-2} for occupational and $55/f_G^{0.177} \times 0.72[0.025+0.975(t/360)^{0.5}]$ kJ m^{-2} for general public exposure.

exposures are additive in their effects. Additivity should be examined separately for the effects of thermal and electrical stimulation, and restrictions met after accounting for such additivity. The formulae below apply to relevant frequencies under practical exposure situations. As the below reference level summation formulae assume worst-case conditions among the fields from multiple sources, typical exposure situations may in practice result in lower exposure levels than indicated by the formulae for the reference levels.

The following issues are noted. In terms of the reference levels, the largest ratio of the E-field strength, H-field strength or power density, relative to the corresponding reference level values, should be evaluated to demonstrate compliance. Reference levels are defined in terms of external physical quantities and have transitions, in terms of quantities, at specific frequencies. For example, field strengths are used below 30 MHz, whereas both field strength and incident power density are applicable from 30 MHz to 2 GHz. Where the exposure includes frequency components below and above the transition, additivity should be used to account for this. The same principle applies for basic restrictions. Field values entering the below equations must be derived using the same spatial and temporal constraints referred to in the basic restriction and reference level tables. The summation equations for basic restrictions and reference levels are presented separately below. However, for practical compliance purposes,

Table 8. Reference levels for local exposure to electromagnetic fields from 100 kHz to 10 MHz (unperturbed rms values), for peak values.[a]

Exposure scenario	Frequency range	Incident E-field strength; E_{inc} (V m^{-1})	Incident H-field strength; H_{inc} (A m^{-1})
Occupational	100 kHz – 10 MHz	170	80
General public	100 kHz – 10 MHz	83	21

[a]Note:

1. Regardless of the far-field/near-field zone distinction, compliance is demonstrated if neither peak spatial E_{inc} or peak spatial H_{inc}, over the projected whole-body space, exceeds the above reference level values.

Table 9. Reference levels for current induced in any limb, averaged over 6 min, at frequencies from 100 kHz to 110 MHz. [a]

Exposure scenario	Frequency range	Electric current; I (mA)
Occupational	100 kHz – 110 MHz	100
General public	100 kHz – 110 MHz	45

[a]Note

1. Current intensity values must be determined by averaging over the relevant square values (see eqn 8 in Appendix A for details).

2. Limb current intensity must be evaluated separately for each limb.

3. Limb current reference levels are not provided for any other frequency range.

4. Limb current reference levels are only required for cases where the human body is not electrically isolated from a ground plane.

the evaluation by basic restriction and reference level can be combined. For example, the second term in eqn (2) can be replaced by the fourth term in eqn (4) for frequency components above 6 GHz. To be compliant with the guidelines, the summed values in each of Eqn (1) to (7) must be less than 1.

Basic restrictions for intervals ≥6 min. For practical application of the whole-body average basic restrictions, SAR should be added according to

$$\sum_{i=100\ \mathrm{kHz}}^{300\ \mathrm{GHz}} \frac{\mathrm{SAR}_i}{\mathrm{SAR}_{\mathrm{BR}}} \leq 1, \quad (1)$$

where SAR_i and $\mathrm{SAR}_{\mathrm{BR}}$ are the whole-body average SAR levels at frequency i and the whole-body average SAR basic restrictions given in Table 2, respectively.

For practical application of the local SAR and local absorbed power density basic restrictions, values should be added according to

$$\sum_{i=100\ \mathrm{kHz}}^{6\ \mathrm{GHz}} \frac{\mathrm{SAR}_i}{\mathrm{SAR}_{\mathrm{BR}}} + \sum_{i>6\ \mathrm{GHz}}^{30\ \mathrm{GHz}} \frac{\mathrm{S}_{\mathrm{ab,4cm},i}}{\mathrm{S}_{\mathrm{ab,4cm,BR}}} + \sum_{i>30\ \mathrm{GHz}}^{300\ \mathrm{GHz}} \mathrm{MAX}\left\{ \left(\frac{\mathrm{S}_{\mathrm{ab,4cm},i}}{\mathrm{S}_{\mathrm{ab,4cm,BR}}} \right), \left(\frac{\mathrm{S}_{\mathrm{ab,1cm},i}}{\mathrm{S}_{\mathrm{ab,1cm,BR}}} \right) \right\} \leq 1, \quad (2)$$

where, SAR_i and $\mathrm{SAR}_{\mathrm{BR}}$ are the local SAR level at frequency i and the local SAR basic restriction given in Table 2, respectively; $\mathrm{S}_{\mathrm{ab,4cm},i}$ and $\mathrm{S}_{\mathrm{ab,4cm,BR}}$ are the 4-cm^2 absorbed power density level at frequency i and the 4-cm^2 absorbed power density basic restriction given in Table 2, respectively; $\mathrm{S}_{\mathrm{ab,1cm},i}$ and $\mathrm{S}_{\mathrm{ab,1cm,BR}}$ are the 1-cm^2 absorbed power density level at frequency i and the 1-cm^2 absorbed power density basic restriction given in Table 2, respectively; inside the body, S_{ab} terms are to be treated as zero; when evaluating the summation of SAR and S_{ab} over the body surface, the center of the SAR averaging space is taken to be x,y,z, such that the x,y plane is parallel to the body surface (z = 0) and z = −1.08 cm (approximately half the length of a 10-g cube), and the center of the S_{ab} averaging area is defined as x,y,0; eqn (2) must be satisfied for every position in the human body.

Reference levels for intervals ≥6 min. For practical application of the whole-body average reference levels, incident electric field strength, incident magnetic field strength and incident power density values should be added according to;

$$\sum_{i=100\ \mathrm{kHz}}^{30\ \mathrm{MHz}} \left\{ \left(\frac{\mathrm{E}_{\mathrm{inc},i}}{\mathrm{E}_{\mathrm{inc,RL},i}} \right)^2 + \left(\frac{\mathrm{H}_{\mathrm{inc},i}}{\mathrm{H}_{\mathrm{inc,RL},i}} \right)^2 \right\} + \sum_{i>30\ \mathrm{MHz}}^{2\ \mathrm{GHz}} \mathrm{MAX}\left\{ \left(\frac{\mathrm{E}_{\mathrm{inc},i}}{\mathrm{E}_{\mathrm{inc,RL},i}} \right)^2, \left(\frac{\mathrm{H}_{\mathrm{inc},i}}{\mathrm{H}_{\mathrm{inc,RL},i}} \right)^2, \left(\frac{\mathrm{S}_{\mathrm{inc},i}}{\mathrm{S}_{\mathrm{inc,RL},i}} \right) \right\} + \sum_{i>2\ \mathrm{GHz}}^{300\ \mathrm{GHz}} \left(\frac{\mathrm{S}_{\mathrm{inc},i}}{\mathrm{S}_{\mathrm{inc,RL}}} \right) \leq 1, \quad (3)$$

where, $\mathrm{E}_{\mathrm{inc},i}$ and $\mathrm{E}_{\mathrm{inc,RL},i}$ are the whole-body average incident electric field strength and whole-body average incident electric field strength reference level given in Table 5, at frequency i, respectively; $\mathrm{H}_{\mathrm{inc},i}$ and $\mathrm{H}_{\mathrm{inc,RL},i}$ are the whole-body average incident magnetic field strength and whole-body average incident magnetic field strength reference level given in Table 5, at frequency i, respectively; $\mathrm{S}_{\mathrm{inc},i}$ and $\mathrm{S}_{\mathrm{inc,RL},i}$ are the whole-body average incident power density and whole-body average incident power density reference level given in Table 5, at frequency i, respectively. Note that the second term is not appropriate for the reactive near-field zone, and so cannot be used in eqn (3).

For practical application of the local reference levels, incident electric field strength, incident magnetic field strength and incident power density values should be added according to

$$\sum_{i=100\ \mathrm{kHz}}^{30\ \mathrm{MHz}} \mathrm{MAX}\left\{ \left(\frac{\mathrm{E}_{\mathrm{inc},i}}{\mathrm{E}_{\mathrm{inc,RL},i}} \right)^2, \left(\frac{\mathrm{H}_{\mathrm{inc},i}}{\mathrm{H}_{\mathrm{inc,RL},i}} \right)^2 \right\} + \sum_{i>30\ \mathrm{MHz}}^{2\ \mathrm{GHz}} \mathrm{MAX}\left\{ \left(\frac{\mathrm{E}_{\mathrm{inc},i}}{\mathrm{E}_{\mathrm{inc,RL},i}} \right)^2, \left(\frac{\mathrm{H}_{\mathrm{inc},i}}{\mathrm{H}_{\mathrm{inc,RL},i}} \right)^2, \left(\frac{\mathrm{S}_{\mathrm{inc},i}}{\mathrm{S}_{\mathrm{inc,RL},i}} \right) \right\} + \sum_{i>2\ \mathrm{GHz}}^{6\ \mathrm{GHz}} \left(\frac{\mathrm{S}_{\mathrm{inc},i}}{\mathrm{S}_{\mathrm{inc,RL},i}} \right) + \sum_{i>6\ \mathrm{GHz}}^{30\ \mathrm{GHz}} \left(\frac{\mathrm{S}_{\mathrm{inc,4cm},i}}{\mathrm{S}_{\mathrm{inc,4cm,RL},i}} \right) + \sum_{i>30\ \mathrm{GHz}}^{300\ \mathrm{GHz}} \mathrm{MAX}\left\{ \left(\frac{\mathrm{S}_{\mathrm{inc,4cm},i}}{\mathrm{S}_{\mathrm{inc,4cm,RL},i}} \right), \left(\frac{\mathrm{S}_{\mathrm{inc,1cm},i}}{\mathrm{S}_{\mathrm{inc,1cm,RL},i}} \right) \right\} \leq 1, \quad (4)$$

where, $\mathrm{E}_{\mathrm{inc},i}$ and $\mathrm{E}_{\mathrm{inc,RL},i}$ are the local incident electric field strength and local incident electric field strength reference level given in Table 6, at frequency i, respectively; $\mathrm{H}_{\mathrm{inc},i}$ and $\mathrm{H}_{\mathrm{inc,RL},i}$ are the local incident magnetic field strength and local incident magnetic field strength reference level given in Table 6, at frequency i, respectively; $\mathrm{S}_{\mathrm{inc},i}$ and $\mathrm{S}_{\mathrm{inc,RL},i}$ are the local incident power density and local incident power density reference level given in Table 6, at

frequency i, respectively; inside the body above 6 GHz, S_{inc} terms are to be treated as zero; eqn (4) must be satisfied for every position in the human body.

For practical application of the limb current reference levels, limb current values should be added according to

$$\sum_{i=100\ \mathrm{kHz}}^{110\ \mathrm{MHz}} \left(\frac{I_i}{I_{RL}}\right)^2 \leq 1, \quad (5)$$

where I_i is the limb current component at frequency i; and I_{RL} is the limb current reference level value from Table 9. If there are non-negligible contributions to the local SAR around limbs over 110 MHz, these need to be considered by combining corresponding terms in eqns (2) or (4).

Basic restrictions for intervals <6 min. For practical application of the local basic restrictions for time intervals (t)<6 min, SAR, SA and absorbed energy density values should be added according to:

$$\sum_{i=100\ \mathrm{kHz}}^{400\ \mathrm{MHz}} \int_t \frac{SAR_i(t)}{360 \times SAR_{BR}}\,dt + \sum_{i>400\ \mathrm{MHz}}^{6\ \mathrm{GHz}} \frac{SA_i(t)}{SA_{BR}(t)} + \sum_{i>6\ \mathrm{GHz}}^{30\ \mathrm{GHz}} \frac{U_{ab,4cm,i}(t)}{U_{ab,4cm,BR}(t)} + \sum_{i>30\ \mathrm{GHz}}^{300\ \mathrm{GHz}} \mathrm{MAX}\left\{\left(\frac{U_{ab,4cm,i}(t)}{U_{ab,4cm,BR}(t)}\right), \left(\frac{U_{ab,1cm,i}(t)}{U_{ab,1cm,BR}(t)}\right)\right\} \leq 1, \quad (6)$$

where, $SAR_i(t)$ and $SAR_{BR}(t)$ are the local SAR level at frequency i and the local SAR basic restriction given in Table 2, over time t, respectively; $SA_i(t)$ and $SA_{BR}(t)$ are the local SA level at frequency i and the local SA basic restriction given in Table 3, over time t, respectively; $U_{ab,4cm,i}(t)$ and $U_{ab,4cm,BR}(t)$ are the 4-cm^2 absorbed power density level at frequency i and the 4-cm^2 absorbed power density basic restriction given in Table 3, over time t, respectively; $U_{ab,1cm,i}(t)$ and $U_{ab,1cm,BR}(t)$ are the 1-cm^2 absorbed power density level at frequency i and the 1-cm^2 absorbed power density basic restriction given in Table 3, over time t, respectively; inside the body, U_{ab} terms are to be treated as zero; when evaluating the summation of SAR and/or SA, and U_{ab}, over the body surface, the center of the SAR and/or SA averaging space is taken to be x,y,z, such that the x,y plane is parallel to the body surface (z = 0) and z = −1.08 cm (approximately half the length of a 10-g cube), and the center of the U_{ab} averaging area is defined as x,y,0; eqn (6) must be satisfied for every position in the human body; for simultaneous exposure of brief and extended exposures, SAR, SA and U_{ab} must all be accounted for in this equation.

Reference levels for intervals <6 min. For practical application of the local reference levels for time intervals (t) <6 min, incident electric field strength, incident magnetic field strength, incident power density and incident energy density values should be added according to:

$$\sum_{i>100\ \mathrm{kHz}}^{30\ \mathrm{MHz}} \mathrm{MAX}\left\{\left(\int_t \frac{E^2_{inc,i}(t)}{360{*}E^2_{inc,RL,i}}\,dt\right), \left(\int_t \frac{H^2_{inc,i}(t)}{360{*}H^2_{inc,RL,i}}\,dt\right)\right\} + \sum_{i>30\ \mathrm{MHz}}^{400\ \mathrm{MHz}} \mathrm{MAX}\left\{\left(\int_t \frac{E^2_{inc,i}(t)}{360{*}E^2_{inc,RL,i}}\,dt\right), \left(\int_t \frac{H^2_{inc,i}(t)}{360{*}H^2_{inc,RL,i}}\,dt\right), \left(\int_t \frac{S_{inc,i}(t)}{360{*}S_{inc,RL,i}}\,dt\right)\right\} + \sum_{i>400\ \mathrm{MHz}}^{6\ \mathrm{GHz}} \frac{U_{inc,i}(t)}{U_{inc,RL,i}(t)} + \sum_{i=6\ \mathrm{GHz}}^{30\ \mathrm{GHz}} \frac{U_{inc,4cm,i}(t)}{U_{inc,4cm,RL,i}(t)} + \sum_{i>30\ \mathrm{GHz}}^{300\ \mathrm{GHz}} \mathrm{MAX}\left\{\left(\frac{U_{inc,4cm,i}(t)}{U_{inc,4cm,RL,i}(t)}\right), \left(\frac{U_{inc,1cm,i}(t)}{U_{inc,1cm,RL,i}(t)}\right)\right\} \leq 1, \quad (7)$$

where $E_{inc,i}(t)$ and $E_{inc,RL,i}$ are the local E_{inc} level over time t and the local E_{inc} reference level given in Table 6, at frequency i, respectively; $H_{inc,i}(t)$ and $H_{inc,RL,i}$ are the local H_{inc} level over time t and the local H_{inc} reference level given in Table 6, at frequency i, respectively; $S_{inc,i}(t)$ and $S_{inc,RL,i}$ are the local S_{inc} level over time t and the local S_{inc} reference level given in Table 6, at frequency i, respectively; $U_{inc,i}(t)$ and $U_{inc,RL}(t)$ are the incident energy density level and the incident energy density reference level, over time t, at frequency i, given in Table 7, respectively; $U_{inc,4cm,i}(t)$ and $U_{inc,4cm,RL}(t)$ are the 4-cm^2 incident energy density level and the 4-cm^2 incident energy density reference level, over time t, at frequency i, given in Table 7, respectively; $U_{inc,1cm,i}(t)$ and $U_{inc,1cm,RL}(t)$ are the 1-cm^2 incident energy density level and the 1-cm^2 incident energy density reference level, over time t, at frequency i, given in Table 7, respectively; inside the body, U_{inc} terms are to be treated as zero; eqn (7) must be satisfied for every position in the human body.

Guidance for Contact Currents

Within approximately the 100 kHz to 110 MHz range, contact currents can occur when a person touches a conducting object that is within an electric or magnetic field, causing current flow between object and person. At high levels these can result in nerve stimulation or pain (and potentially tissue damage), depending on EMF frequency (Kavet et al. 2014; Tell and Tell 2018). This can be a particular concern around large radiofrequency transmitters, such as those that are found near high power antennas used for broadcasting below 30 MHz and at 87.5–108 MHz, where there have been sporadic reports of pain and burn-related accidents. Contact currents occur at the region of contact, with smaller contact

regions producing larger biological effects (given the same current). This is due to the larger current density (A m^{-2}), and consequently the higher localized SAR in the body.

Exposure due to contact currents is indirect, in that it requires an intermediate conducting object to transduce the field. This makes contact current exposure unpredictable, due to both behavioral factors (e.g., grasping versus touch contact) and environmental conditions (e.g., configuration of conductive objects), and it reduces ICNIRP's ability to protect against them. Of particular importance is the heterogeneity of the current density passing to and being absorbed by the person, which is due not only to the contact area, but also to the conductivity, density and heat capacity of the tissue through which the current passes, and most importantly the resistance between conducting object and contacting tissue (Tell and Tell 2018).

Accordingly, these guidelines do not provide restrictions for contact currents, and instead provide "guidance" to assist those responsible for transmitting high-power radiofrequency fields to understand contact currents, the potential hazards, and how to mitigate such hazards. For the purpose of specification, ICNIRP here defines high-power radiofrequency EMFs as those emitting greater than 100 V m^{-1} within the frequency range 100 kHz to 100 MHz at their source.

There is limited research available on the relation between contact currents and health. In terms of pain, the health effect arising from the lowest contact current level, the main data comes from Chatterjee et al. (1986). In that study sensation and pain were assessed in a large adult cohort as a function of contact current frequency and contact type (grasping versus touch contact). Reversible, painful heat sensations were reported to occur with average (touch contact) induced current thresholds of 46 mA within the 100 kHz to 10 MHz range tested, which required at least 10 s of exposure to be reported as pain. Thresholds were frequency-independent within that range, and thresholds for grasping contact were substantially higher than those for touch contact.

However, given that the threshold value reported was an average across the participants, and given the standard deviation of the thresholds reported, ICNIRP considers that the lowest threshold across the cohort would have been approximately 20 mA. Further, modeling from that data suggests that children would have lower thresholds; extrapolating from Chatterjee et al. (1986) and Chan et al. (2013), the lowest threshold in children would be expected to be within the range of 10 mA. The upper frequency of contact current capable of causing harm is also not known. Although the ICNIRP (1998) guidelines specified reference levels to account for contact currents from 100 kHz to 110 MHz, Chatterjee et al. (1986) only tested up to 10 MHz, and Tell and Tell (2018) reported strong reductions in contact current sensitivities from about 1 MHz to 28 MHz (and did not assess higher frequencies). Thus, it is not clear that contact currents will remain a health hazard across the entire 100 kHz to 110 MHz range.

In determining the likelihood and nature of hazard due to potential contact current scenarios, ICNIRP views the above information as important for the responsible person in managing risk associated with contact currents within the frequency range 100 kHz to 110 MHz. This may also assist in conducting a risk-benefit analysis associated with allowing a person into a radiofrequency EMF environment that may result in contact currents. The above information suggests that risk of contact current hazards can be minimized by training workers to avoid contact with conducting objects, but that where contact is required, the following factors are important. Large metallic objects should be connected to ground (grounding); workers should make contact via insulating materials (e.g., radiofrequency protective gloves); and workers should be made aware of the risks, including the possibility of "surprise," which may impact on safety in ways other than the direct impact of the current on tissue (for example, by causing accidents).

Risk Mitigation Considerations for Occupational Exposure

To justify radiofrequency EMF exposure at the occupational level, an appropriate health and safety program is required. Part of such a program requires an understanding of the potential effects of radiofrequency EMF exposure, including consideration of whether biological effects resulting from the exposure may add to other biological effects that are unrelated to radiofrequency EMF. For example, where body core temperature is already elevated due to factors unrelated to EMF, such as through strenuous activity, radiofrequency EMF-induced temperature rise needs to be considered in conjunction with the other sources of heating. Similarly, it is also important to consider whether a person has an illness or condition that might affect their capacity to thermoregulate, or whether environmental impediments to heat dissipation might be present.

The relevant health effects that the whole-body SAR restrictions protect against are increased cardiovascular load (due to the work that the cardiovascular system must perform in order to restrict body core temperature rise), and where temperature rise is not restricted to a safe level, a cascade of functional changes that may lead to both reversible and irreversible effects on tissues (including brain, heart, and kidney). These effects typically require body core temperatures greater than 40°C (or an increase of approximately 3°C relative to normothermia). Large reduction factors have thus been used to make it extremely unlikely that radiofrequency-induced temperature rise would exceed 1°C (occupational restrictions have been set that would, under normothermic conditions, lead to body core temperature rises of <0.1°C), but care must be exercised when other factors are present that may affect body core temperature. These include high environmental temperatures, high physical activity, and impediments to normal thermoregulation (such as the use of thermally insulating clothing or certain medical conditions). Where significant heat is expected from other sources, it is advised that workers have a suitable means

of verifying their body core temperature (see ACGIH 2017 for further guidance).

The relevant health effects that the localized basic restrictions protect against are pain and thermally-mediated tissue damage. Within Type-1 tissue, such as in the skin and limbs, pain (due to stimulation of nociceptors) and tissue damage (due to denaturation of proteins) typically require temperatures above approximately 41°C. Occupational exposure of the Limbs is unlikely to increase local temperature by more than 2.5°C, and given that Limb temperatures are normally below 31–36°C, it is unlikely that radiofrequency EMF exposure of Limb tissue, in itself, would result in either pain or tissue damage. Within Type-2 tissue, such as within regions of the Head and Torso (excluding superficial tissue), harm is also unlikely to occur at temperatures below 41°C. As occupational exposure of the Head and Torso tissue is unlikely to increase temperature by more than 1°C, and given that body core temperature is normally around 37–38°C, it is unlikely that radiofrequency EMF exposure would lead to temperature rises sufficient to harm Type-2 tissue or tissue function.

However, care must be exercised when a worker is subject to other heat sources that may add to that of the radiofrequency EMF exposure, such as those described above in relation to body core temperature. For superficial exposure scenarios, local thermal discomfort and pain can be important indicators of potential thermal tissue damage. It is thus important, particularly in situations where other thermal stressors are present, that the worker understands that radiofrequency EMF exposure can contribute to their thermal load and is in a position to take appropriate action to mitigate potential harm.

Acknowledgments—Collaborators: Rodney Croft, ICNIRP and Australian Centre for Electromagnetic Bioeffects Research, Illawarra Health & Medical Research Institute, University of Wollongong, Australia; Maria Feychting, ICNIRP and Karolinska Institutet, Sweden; Adèle C Green, ICNIRP and QIMR Berghofer Medical Research Institute, Brisbane, Australia and CRUK Manchester Institute, University of Manchester, Manchester, UK; Akimasa Hirata, ICNIRP and Nagoya Institute of Technology, Japan; Guglielmo d'Inzeo, ICNIRP and La Sapienza University, Rome, Italy; Kari Jokela†, ICNIRP SEG and STUK – Radiation and Nuclear Safety Authority, Finland; Sarah Loughran, ICNIRP SEG and Australian Centre for Electromagnetic Bioeffects Research, Illawarra Health & Medical Research Institute, University of Wollongong, Australia; Carmela Marino, ICNIRP and Agency for New Technologies, Energy and Sustainable Economic Development (ENEA), Italy; Sharon Miller, ICNIRP; Gunnhild Oftedal, ICNIRP and Norwegian University of Science and Technology (NTNU); Tsutomu Okuno, ICNIRP; Eric van Rongen, ICNIRP and Health Council, The Netherlands; Martin Röösli, ICNIRP and Swiss Tropical and Public Health Institute, Switzerland; Zenon Sienkiewicz, ICNIRP; John Tattersall, ICNIRP SEG; Soichi Watanabe, ICNIRP and National Institute of Information and Communications Technology (NICT), Japan.

The views expressed by the collaborators in this publication do not necessarily reflect the views or policies of the organizations they are professionally affiliated with. The mention of commercial products, their sources, or their use in connection with material reported herein is not to be construed as either an actual or implied endorsement of such products by ICNIRP or any of the organizations with which the ICNIRP members are affiliated.

The support received by the German Federal Ministry for the Environment (BMU), the European Union Programme for Employment and Social Innovation "EaSI" (2014–2020), the International Radiation Protection Association (IRPA), the Australian Radiation Protection and Nuclear Safety Agency (ARPANSA), and the New Zealand Ministry of Health is gratefully acknowledged.

In regard to the EU funds, for further information please consult: http://ec.europa.eu/social/easi. The information contained in this publication does not necessarily reflect the official position of the European Commission, or any other donors. All information concerning the support received by ICNIRP is available at www.icnirp.org.

The guidelines were prepared by the ICNIRP Commission members and the scientific experts of the ICNIRP Project Group on RF: Rodney Croft (Chair), Maria Feychting, Akimasa Hirata, Guglielmo d'Inzeo, Kari Jokela†, Sarah Loughran, Carmela Marino, Gunnhild Oftedal, Tsutomu Okuno, Eric van Rongen, Martin Röösli, Zenon Sienkiewicz, John Tattersall, and Soichi Watanabe.

The guidelines were submitted to public consultation in 2018 and approved by the commission in August 2019. At the time of approval, the commission included the following members: Eric van Rongen (Chair), Rodney Croft, Maria Feychting, Adèle C Green, Akimasa Hirata, Guglielmo d'Inzeo, Carmela Marino, Sharon Miller, Gunnhild Oftedal, Tsutomu Okuno, Martin Röösli, Zenon Sienkiewicz, and Soichi Watanabe.

REFERENCES

American Conference of Governmental Industrial Hygienists. TLVs and BEIs: based on the documentation of the threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, OH: ACGIH; 2017.

Arens E, Zhang H. Skin's role in human thermoregulation and comfort. In: Pann N, Gibson P, eds. Thermal and moisture transport in fibrous materials. Cambridge, England: Woodhead Publishing Ltd; 2006: 560–602.

Aschoff J, Wever R. Kern und Schale im Wärmehaushalt des Menschen. Naturwissenschaften 20:477–487; 1958 (in German).

Brockow T, Wagner A, Franke A, Offenbacher M, Resch KL. A randomized controlled trial on the effectiveness of mild water-filtered near infrared whole-body hyperthermia as an adjunct to a standard multimodal rehabilitation in the treatment of fibromyalgia. Clinical J Pain 23:67–75; 2007.

Chan KH, Hattori J, Laakso I, Hirata A, Taki M. Computational dosimetry for grounded and ungrounded human models due to contact current. Phys Med Biol 58:5153–5172; 2013.

Chatterjee I, Wu D, Gandhi OP. Human body impedance and threshold currents for perception and pain for contact hazards analysis in the VLF-MF band. IEEE Trans Biomed Engineer 33:486–494; 1986.

Cheshire WP Jr. Thermoregulatory disorders and illness related to heat and cold stress. Autonomic Neurosci: Basic and Clinical 196:91–104; 2016.

Defrin R, Shachal-Shiffer M, Hadgadg M, Peretz C. Quantitative somatosensory testing of warm and heat-pain thresholds: the effect of body region and testing method. Clinical J Pain 22: 130–136; 2006.

Dewhirst MW, Viglianti BL, Lora-Michiels M, Hanson M, Hopes PJ. Basic principles of thermal dosimetry and thermal thresholds for tissue damage from hyperthermia. Internat J Hyperthermia 19:267–294; 2003.

Dimbylow P. The relationship between localised SAR in the arm and wrist current. Radiat Protect Dosim 95:177–179; 2001.

DuBois EF. The temperature of the human body in health and disease. In: Temperature: its measurement and control in science and industry. New York: American Institute of Physics, Reinhold Publishing Corporation; 1941: 24–40.

Edwards MJ, Saunders RD, Shiota K. Effects of heat on embryos and fetuses. Internat J Hyperthermia 19:295–324; 2003.

Flintoft M, Robinson MP, Melia GCR, Marvin AC, Dawson JF. Average absorption cross-section of the human body measured at 1–12 GHz in a reverberant chamber: results of a human volunteer study. Phys Med Biol 59:3297–3317; 2014.

Foster KR, Ziskin MC, Balzano Q. Thermal modeling for the next generation of radiofrequency exposure limits: commentary. Health Phys 113:41–53; 2017.

Foster KR, Ziskin MC, Balzano Q. Thermal response of human skin to microwave energy: a critical review. Health Phys 111:528–541; 2016.

Funahashi D, Hirata A, Kodera S, Foster KR. Area-averaged transmitted power density at skin surface as metric to estimate surface temperature elevation. IEEE Access 6:77665–77674; 2018.

Hashimoto Y, Hirata A, Morimoto R, Aonuma S, Laakso I, Jokela K, Foster KR. On the averaging area for incident power density for human exposure limits at frequencies over 6 GHz. Phys Med Biol 62:3124–3138; 2017.

Hirata A, Fujiwara O. The correlation between mass-averaged SAR and temperature elevation in the human head model exposed to RF near-fields from 1 to 6 GHz. Phys Med Biol 54:7171–7182; 2009.

Hirata A, Laakso I, Oizumi T, Hanatani R, Chan KH, Wiart J. The relationship between specific absorption rate and temperature elevation in anatomically based human body models for plane wave exposure from 30 MHz to 6 GHz. Phys Med Biol 58:903–921; 2013.

International Commission on Non-Ionizing Radiation Protection. Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Phys 74:494–522; 1998.

International Commission on Non-Ionizing Radiation Protection. ICNIRP Statement on the "Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz)". Health Phys 97:257–58; 2009.

International Commission on Non-Ionizing Radiation Protection. Guidelines for limiting exposure to time-varying electric and magnetic fields (1 Hz to 100 kHz). Health Phys 99:818–836; 2010.

IEC. Medical electrical equipment—part 1–2: general requirements for basic safety and essential performance—collateral standard: electromagnetic disturbances—requirements and tests. Geneva: IEC; 60601-1-2:2014; 2014.

ISO. Active implantable medical devices—electromagnetic compatibility—EMC test protocols for implantable cardiac pacemakers, implantable cardioverter defibrillators and cardiac resynchronization devices. Geneva: ISO; 14117:2012; 2012.

Joshi RP, Schoenbach KH. Bioelectric effects of intense ultrashort pulses. Critical Rev Biomed Engineer 38:255–304; 2010.

Juutilainen J, Höytö, Kumlin T, Naarala J. Review of possible modulation-dependent biological effects of radiofrequency fields, Bioelectromagnetics 32(7):511–34; 2011.

Kavet R, Tell RA, Olsen RG. Radiofrequency contact currents: sensory responses and dosimetry. Radiat Protect Dosim 162:268–279; 2014.

Kodera S, Hirata A, Funahashi D, Watanabe S, Jokela K, Croft RJ. Temperature rise for brief radio-frequency exposure below 6 GHz. IEEE Access 6:65737–65746; 2018.

Kowalczuk C, Yarwood G, Blackwell R, Priestner M, Sienkiewicz Z, Bouffler S, Ahmed I, Abd-Alhameed R, Excell P, Hodzic V, Davis C, Gammon R, Balzano Q. Absence of nonlinear responses in cells and tissues exposed to RF energy at mobile phone frequencies using a doubly resonant cavity. Bioelectromagnetics 31(7):556–565; 2010.

Laakso I, Morimoto R, Heinonen J, Jokela K, Hirata A. Human exposure to pulsed fields in the frequency range from 6 to 100 GHz. Phys Med Biol 62:6980–6992; 2017.

Mir LM. Application of electroporation gene therapy: past, current, and future. Meth Molecular Biol 423:3–17; 2008.

Mieusset R, Bujan L. Review: testicular heating and its possible contributions to male infertility. Internat J Androl 18:169–184; 1995.

Morimoto R, Hirata A, Laakso I, Ziskin M, Foster R. Time constants for elevation in human models exposed to dipole antenna and beams in the frequency range from 1 to 30 GHz. Phys Med Biol 62:1676–1699; 2017.

Nagaoka T, Watanabe S. Development of voxel models adjusted to ICRP reference children and their whole-body averaged SARs for whole-body exposure to electromagnetic fields from 10 MHz to 6 GHz. IEEE Access, in press.

Nguyen THP, Shamis Y, Croft RJ, Wood A, McIntosh RL, Crawford RJ, Ivanova EP. 18 GHz electromagnetic field induces permeability of Gram-positive cocci. Nature: Scientific Reports 16:10980; 2015.

Ramsey JD, Buford C, Beshir M, Jensen RC. Effects of workplace thermal conditions on safe work behavior. J Safety Res 14:105–114; 1983.

Reilly T, Atkinson G, Edwards B, Waterhouse J, Farrelly K, Fairhurst E. Diurnal variation in temperature, mental and physical performance, and tasks specifically related to football (soccer). Chronobiol Internat 24:507–519; 2007.

Sasaki K, Mizuno M, Wake K, Watanabe S. Monte Carlo simulations of skin exposure to electromagnetic field from 10 GHz to 1 THz. Phys Med Biol 62:6993–7010; 2017.

Saunders RD, Jefferys JG. A neurobiological basis for ELF guidelines. Health Phys 92:596–603; 2007.

Scientific Committee on Emerging and Newly Identified Health Risks. Opinion on potential health effects of exposure to electromagnetic fields (EMF). Luxembourg: European Commission; 2015.

Shafahi M, Vafai K. Human eye response to thermal disturbances. J Heat Transfer 133:011009–011009-7; 2011.

SSM. Recent research on EMF and health risk. Tenth report from SSM's Scientific Council on Electromagnetic Fields. Stockholm: Strålsäkerhetsmyndigheten; SSM Report 19; 2015.

SSM. Recent research on EMF and health risk. Eleventh report from SSM's Scientific Council on Electromagnetic Fields. Stockholm: Strålsäkerhetsmyndigheten; SSM Report 15; 2016.

SSM. Recent research on EMF and health risk. Twelfth report from SSM's Scientific Council on Electromagnetic Fields. Stockholm: Strålsäkerhetsmyndigheten; SSM Report 09; 2018.

Tell RA, Tell CA. Perspectives on setting limits for RF contact currents: a commentary. Biomed Engineer Online 17:2; 2018.

Teunissen LP, Grabowski A, Kram R. Effects of independently altering body weight and body mass on the metabolic cost of running. J Experimental Biol 210:4418–4427; 2007.

United Nations Environment Programme/World Health Organization/International Radiation Protection Association. Electromagnetic fields (300 Hz to 300 GHz). Geneva: World Health Organization; Environmental Health Criteria 137; 1993.

Van den Heuvel AMJ, Haberley BJ, Hoyle DJR, Taylor NAS, Croft RJ. The independent influences of heat strain and dehydration upon cognition. Euro J Appl Physiol 117:1025–1037; 2017.

Van Rhoon GC, Samaras T, Yarmolenko PS, Dewhirst MW, Neufeld E, Kuster N. CEM43°C thermal dose thresholds: a potential guide for magnetic resonance radiofrequency exposure levels? Euro Radiol 23:2215–2227; 2013.

Walters TJ, Blick DW, Johnson LR, Adair ER, Foster KR. Heating and pain sensation produced in human skin by millimetre waves: comparison to a simple thermal model. Health Phys 78:259–267; 2000.

Weyand PG, Smith BR, Sandell RF. Assessing the metabolic cost of walking: the influence of baseline subtractions. In: Annual International Conference of the IEEE Engineering in Medicine and Biology Society. Minneapolis, MN: IEEE; 2009: 6878–6881.

World Health Organization. Constitution of the World Health Organization [online]. 1948. Available at ***https://www.who.int/governance/eb/who_constitution_en.pdf***. Accessed 1 April 2019.

World Health Organization. Radiofrequency fields. Geneva: WHO; Public Consultation Document; 2014.

Yarmolenko PS, Moon EJ, Landon C, Manzoor A, Hochman DW, Viglianti BL, Dewhirst MW. Thresholds for thermal damage to normal tissues: an update. International J Hyperthermia 27:320–343; 2011.

Ziskin MC, Morrissey J. Thermal thresholds for teratogenicity, reproduction, and development. International J Hyperthermia 27:373–387; 2011.

■■

APPENDIX A: BACKGROUND DOSIMETRY

Introduction

This appendix provides additional dosimetry information that is directly relevant to the derivation of the radiofrequency exposure restrictions that form the basis of the present guidelines. As described in the main document, the operational adverse health effects resulting from the lowest radiofrequency exposure levels are due to heating (nerve stimulation is discussed within the low frequency guidelines; ICNIRP 2010). Accordingly, this appendix details the choice of quantities used to restrict temperature rise to the operational adverse health effect thresholds described in the main document, the methods used to derive these restrictions (including, where relevant, the associated uncertainty), the spatial and temporal averaging methods used to represent temperature rise, and the derivation of the basic restrictions and reference levels themselves (including, where relevant, the associated uncertainty). The operational adverse health effect thresholds considered are 1°C body core temperature rise for exposures averaged over the whole body, and 5°C and 2°C local temperature rise over more-localized regions for "Type-1" and "Type-2" body tissue, respectively.[7]

QUANTITIES AND UNITS

Detailed explanations for the basic quantities, e.g., **E**, **H**, I, T, and *t* are found elsewhere (see ICNIRP 1985, 2009a, 2009, 2010). In this section, the other quantities used in the guidelines are detailed (i.e., SAR, SA, S_{inc}, S_{ab}, S_{eq}, U_{inc}, U_{ab}, and U_{eq}). Vector quantities are presented in **bold** font.

It is noted that radiofrequency basic restrictions and reference levels are based on the lowest radiofrequency exposure levels that may cause an adverse health effect. Since the health effects are related to the temperature rises caused by the exposure, it is determined by energy or power of the radiofrequency exposure. Therefore, squared values of **E**, **H**, and I are considered for time or spatial integration, or where summation of multiple frequencies is applied. The following equation is an example of the spatial average of **E** over a volume *V*:

$$E_{spatial_average} = \sqrt{\frac{1}{V}\int_v |\mathbf{E}|^2 dv}, \tag{8}$$

where *V* is the volume of the integration ($V = \int_v dv$).

Specific Energy Absorption Rate (SAR) and Specific Energy Absorption (SA)

SAR is defined as the time derivative of the incremental energy consumption by heat, δW, absorbed by or dissipated in an incremental mass, δm, contained in a volume element, δV, of a given mass density of the tissue (kg m^{-3}), ρ, and is expressed in watt per kilogram (W kg^{-1}):

$$\mathrm{SAR} = \frac{\delta}{\delta t}\left(\frac{\delta W}{\delta m}\right) = \frac{\delta}{\delta t}\left(\frac{\delta W}{\rho \delta V}\right). \tag{9}$$

Dielectric properties of biological tissues or organs are generally considered as dielectric lossy material and magnetically transparent because the relative magnetic permeability (μ_r) is 1. Therefore, the SAR is usually derived from the following equation:

$$\mathrm{SAR} = \frac{\sigma |\mathbf{E}|^2}{\rho}, \tag{10}$$

where σ is the conductivity (S m^{-1}) and **E** is the internal electric-field (root mean square (rms) value).

Temperature rise is strongly correlated with SAR. Under conditions where heat loss due to processes such as conduction is not significant, SAR and temperature rise are directly related as follows;

$$\mathrm{SAR} = C\frac{d\mathrm{T}}{dt}, \tag{11}$$

where *C* is specific heat capacity (J kg^{-1} $°C^{-1}$) of the tissue, T is temperature (°C) and *t* is the duration of exposure (s). For most realistic cases, a large amount of heat energy rapidly diffuses during the exposure. Therefore, eqn (11) cannot be routinely applied to human exposure scenarios. However, eqn (11) is useful for brief exposure scenarios where heat loss is not significant.

SAR is used as a basic restriction in the present guidelines. The SAR basic restrictions are defined as spatially averaged values; that is, whole-body average SAR and SAR_{10g}. The whole-body average SAR is the total power absorbed in the whole body divided by the body mass:

$$\text{Whole-body average SAR} = \frac{(Total\ power)_{WB}}{(Total\ mass)_{WB}} = \frac{\left[\int_{WB}\sigma|\mathbf{E}|^2 dv\right]_{WB}}{\int_{WB}\rho dv}. \tag{12}$$

SAR_{10g} is defined as the total power absorbed in a 10-g cubic volume divided by 10 g (see the "Spatial averaging considerations" section):

$$\mathrm{SAR}_{10g} = \frac{(Total\ power)_{V_{10g}}}{(Total\ mass)_{V_{10g}}} = \frac{\left[\int_{V_{10g}}\sigma|\mathbf{E}|^2 dv\right]_{V_{10g}}}{\int_{V_{10g}}\rho dv}. \tag{13}$$

A 10-g volume (V_{10g}) is approximately computed as a 2.15 cm × 2.15 cm × 2.15 cm cube, based on the assumption that the tissue has the same mass density as water, or 1,000 kg m^{-3}.

SA (J m^{-3}) is derived as the time integral of SAR during the time from t_1 to t_2:

[7] Type-1 tissue refers to all tissues in the upper arm, forearm, hand, thigh, leg, foot, pinna and the cornea, anterior chamber and iris of the eye, epidermal, dermal, fat, muscle, and bone tissue. Type-2 tissue refers to all tissues in the head, eye, abdomen, back, thorax, and pelvis, excluding those defined as Type-1 tissue.

$$SA = \int_{t_1}^{t_2} SAR(t)dt. \quad (14)$$

Absorbed Power Density (S_{ab}) and Absorbed Energy Density (U_{ab})

SAR_{10g} is no longer an appropriate surrogate for local temperature rise at frequencies above 6 GHz. Therefore, the absorbed power and energy densities are introduced in the guidelines for basic restrictions at such frequencies, where the radiofrequency power or energy absorption is largely confined within very superficial regions of the body. For example, the penetration depths are approximately 8.1 mm and 0.23 mm at 6 GHz and 300 GHz, respectively (see also Table 10). The absorbed power density (W m^{-2}) is defined at the body surface:

$$S_{ab} = \iint_A dxdy \int_0^{Z_{max}} \rho(x,y,z) \cdot SAR(x,y,z)\, dz/A, \quad (15)$$

where the body surface is at $z = 0$, A is the averaging area (in m^2), and Z_{max} is depth of the body at the corresponding region; where Z_{max} is much larger than the penetration depth, infinity can be substituted for Z_{max}. Considering heat diffusion, a square 2 cm × 2 cm region (from 6 to 300 GHz) is used for the averaging area of the absorbed power and energy density basic restrictions.

A more rigorous formula for absorbed power density is based on the Poynting vector (**S**):

$$S_{ab} = \iint_A \mathrm{Re}[\mathbf{S}] \cdot d\boldsymbol{s}/A = \iint_A \mathrm{Re}[\mathbf{E} \times \mathbf{H}^*] \cdot d\boldsymbol{s}/A, \quad (16)$$

where Re[X] and X* are the real part and the complex conjugate of a complex value "X," respectively, and ***ds*** is the integral variable vector with its direction normal to the integral area A on the body surface.

Similar to the relationship between SAR and SA, the absorbed energy density is derived as the temporal integration of the absorbed power density (J m^{-2}):

$$U_{ab} = \int_{t_1}^{t_2} S_{ab}(t)dt. \quad (17)$$

Incident Power Density (S_{inc}) and Incident Energy Density (U_{inc})

The incident power and energy densities are used as reference levels in the guidelines. The incident power density is defined as the modulus of the complex Poynting vector:

$$S_{inc} = |\mathbf{E} \times \mathbf{H}^*|. \quad (18)$$

In the case of the far-field or transverse electromagnetic (TEM) plane wave, the incident power density is derived as:

$$S_{inc} = \frac{|\mathbf{E}|^2}{Z_0} = Z_0 |\mathbf{H}|^2, \quad (19)$$

where Z_0 is the characteristic impedance of free space, i.e., 377 Ω. The above equation is also used for the evaluation of the plane wave equivalent incident power density (S_{eq}).

S_{inc} is also related to S_{ab} using the reflection coefficient Γ:

$$S_{ab} = \left(1 - |\Gamma|^2\right) S_{inc}. \quad (20)$$

The reflection coefficient (Γ) is derived from the dielectric properties of the tissues, shape of the body surface, incident angle, and polarization.

Similar to the relationship between SAR and SA, the incident energy density is derived as the temporal integration of the incident power density during the time from t_1 to t_2:

$$U_{inc} = \int_{t_1}^{t_2} S_{inc}(t)\, dt. \quad (21)$$

In near-field exposure scenarios, the components of the Poynting vector are not real values but complex ones. In such cases a detailed investigation of the Poynting vector components may be necessary to calculate the incident power density relevant to radiofrequency safety.

RELEVANT BIOPHYSICAL MECHANISMS

Whole-Body Average Exposure Specifications

Relevant quantity. Health effects due to whole-body exposure are related to body core temperature rise. It is, however, difficult to predict body core temperature rise based on exposure of the human body to radiofrequency EMFs.

Body core temperature depends on the whole-body thermal energy balance. Radiofrequency energy absorbed by the body is transferred to the body core via blood flow, which can activate thermoregulatory responses to maintain the body core temperature (Adair and Black 2003). This means that the time rate of the energy balance is essential for the body core temperature dynamics. Accordingly, whole-body average SAR is used as the physical quantity relating to body core temperature rise.

The relationship between the total energy absorption and the body core temperature is in general independent of frequency. However, at frequencies higher than a few GHz, core temperature does not generally elevate as much as with the same level of whole-body average SAR at lower frequencies because of larger heat transfer from the body surface to air via convection or radiative emission, which

Table 10. Penetration depth of human skin tissue (dermis), for frequencies 6 to 300 GHz.

Frequency (GHz)	Relative permittivity	Conductivity (S/m)	Penetration depth (mm)
6	36	4.0	8.1
10	33	7.9	3.9
30	18	27	0.92
60	10	40	0.49
100	7.3	46	0.35
300	5.0	55	0.23

includes the effect of vasodilation in the skin (Hirata et al. 2013). The power absorption is confined primarily within skin surface tissues where localized temperature rise is more significant than the body core temperature rise (Laakso and Hirata 2011). However, it has also been reported that infrared radiation (IR) exposure can cause significant body core temperature rise (Brockow et al. 2007). Infrared radiation refers to electromagnetic waves with frequencies between those of radiofrequency EMF and visible light. This means that despite the penetration depth of infrared radiation being very small or comparable to the high GHz radiofrequency EMFs (or millimeter waves) it is still possible for infrared radiation exposure to raise body core temperature significantly. For conservative reasons, therefore, ICNIRP set equal whole-body average limits for frequencies both above and below 6 GHz. This is especially important for cases of multiple-frequency exposure of both higher and lower frequencies. Thus, the applicable frequency is defined as the entire frequency range considered in the guidelines.

Temporal averaging considerations. The definition of the time constant for body core temperature is not clear. However, under simplified conditions that produce a reasonable estimate of the time constant (e.g., assuming a first order lag), temperature dynamics can be described as follows:

$$T(t) = T_0 + (T_\infty - T_0)\left(1 - e^{\frac{-t}{\tau}}\right), \qquad (22)$$

where T is the temperature as a function of time t, T_0 and T_∞ are the initial and steady-state temperatures, respectively, and τ is the time constant. In this case, the time constant corresponds to the time taken for 63% of the temperature rise, from initial temperature to steady state temperature, to be reached. In the present guidelines, the time to reach a steady-state of 80–90% of the equilibrium temperature, from the initial temperature, is considered for guideline setting; this is almost two times the time constant in eqn (22).

Further, the time needed to reach the steady-state body core temperature depends on the level of heat load, which in this case relates to the whole-body average SAR. Hirata et al. (2007) numerically simulated the body core temperature rise of a naked body exposed to a plane wave at 65 MHz and 2 GHz, and reported that in both cases it takes at least 60 min to reach a 1°C body core temperature rise for whole-body average SARs of 6 to 8 W kg^{-1}. This time is also dependent on the sweating rate, with strong sweating increasing this time by 40–100 min (Hirata et al. 2008; Nelson et al. 2013). Consequently, the time to reach the steady state temperature rise due to whole-body exposure to radiofrequency EMFs below 6 GHz is 30 min or longer.

As described above, power absorption is mainly confined within the surface tissues at frequencies above 6 GHz (see Table 10). Thermoregulatory responses are thus initiated by the skin temperature rise rather than body core temperature rise. However, the time needed for the steady state temperature rise is not significantly affected by this, and so is not taken into account. It is thus reasonable to keep the averaging time above 6 GHz the same as that below 6 GHz, because there is no quantitative investigation on the time constant of body core temperature rise above 6 GHz.

Whole-body average SAR needed to raise body core temperature by 1°C. Thermoregulatory functions are activated if a human body is exposed to significant heating load, which often results in non-linear relations between whole-body average SAR and body core temperature rise.

Adair and colleagues have experimentally investigated body core temperature (via esophageal temperature measurement) during whole-body exposure. They have reported no or minor increases of the esophageal temperature (<0.1°C) during the whole-body exposure at 100 MHz, 220 MHz, and 2450 MHz, with whole-body average SAR ranging from 0.54 to 1 W kg^{-1} in normal ambient temperature conditions, from 24°C to 28°C (Adair et al. 2001, 2003, 2005).

They also reported a relatively high body core temperature rise (0.35°C) for whole-body average SAR at 220 MHz of 0.675 W kg^{-1} in a hot ambient temperature (31°C) condition, although this was found in only one person and the mean of the body core temperature rises (6 persons) was not appreciable. There is no data on body core temperature rise for whole-body exposure to radiofrequency EMFs above 6 GHz. The only available data are on infrared radiation (Brockow et al. 2007). The conservativeness for whole-body exposure at higher frequencies is discussed in the main text.

There are two main factors affecting body core temperature rise due to radiofrequency exposure: sweating and mass-to-body surface ratio.

Evaporative heat loss due to sweating reduces body core temperature efficiently and needs to be accounted for when estimating body core temperature rise due to EMF. For example, Hirata et al. (2007) reported that 4.5 W kg^{-1} is required to increase the body core temperature by 1°C for a person with a lower sweat rate, such as an elderly person, while 6 W kg^{-1} is required for a person with a normal sweat rate. The decline of sweat rate in elderly people is primarily due to degradation of thermal sensation (Dufour and Candas, 2007).

Similarly, heat exchange between the body surface and external air is also very important. Hirata et al. (2009) found that the steady-state body core temperature rise due to whole-body radiofrequency EMF exposure is proportional to the ratio of the (whole-body) power absorption to the surface area of the body. The ratio of the mass to the surface area is smaller for smaller-dimension bodies such as children, and so greater whole-body average SAR is required to elevate their body core temperature.

This coincides with the finding that smaller persons have a lower body core temperature rise for the same whole-body average SAR. For example, Hirata et al. (2008) numerically evaluated the body core temperature rise in 8-months-old and 3-years-old child models and found that their body core temperature rises were 35% smaller than that of an adult female model for the same whole-body average SAR. They concluded that the higher ratio of a child's surface area to body mass is the reason for more effective cooling resulting from heat loss to the environment. Consequently, the body core temperature rise in the child is smaller than that of the adult at the same whole-body average SAR.

Addressing the issue more broadly, theoretical modeling and generalization from experimental research across a range of species has shown that within the 100 kHz to 6 GHz range, whole-body average SARs of at least 6 W kg^{-1}, for exposures of at least 1 h at moderately high ambient temperature (28°C), are necessary to increase body core temperature by 1°C for healthy adults and children (Hirata et al. 2013), and at least 4.5 W for those with lower sweat rates, such as the elderly (Hirata et al. 2007).

Considerations for fetal exposure. The primary thermoregulatory mechanism for a fetus is body core heat exchange with the mother via blood flow through the umbilical cord. The fetal temperature is therefore tightly controlled by maternal temperature, and it takes longer to reach thermal equilibrium than in adults (Gowland and De Wilde 2008). The body core temperature of the fetus is typically 0.5°C higher than that of the mother (Asakura 2004). This relationship is not changed significantly by radiofrequency EMF exposure of the mother at 26 weeks gestation, as reported by Hirata et al. (2014). In the frequency range from 40 MHz to 500 MHz, they computed steady-state fetal temperature, taking the thermal exchange between mother and fetus into account, and reported that the fetal temperature rise was only 30% higher than that of the mother, even when the power absorption was focused around the fetus. At lower frequencies, the SAR distribution becomes more homogeneous because of the longer wavelength and penetration depth, which results in more homogeneous temperature rise over the whole-body of the mother and fetus. At higher frequencies, the SAR distribution becomes more superficial because of the shorter penetration depth. This results in a smaller SAR of the young fetus or embryo, as it is generally located in the deep region of the abdomen of the mother, as well as resulting in a smaller whole-body SAR of the older fetus because the size of the fetus is larger than the penetration depth. This suggests that EMF whole-body exposure to the mother will result in a similar body core temperature rise in the fetus relative to that of the mother, even at frequencies outside those investigated in that study.

It follows that an EMF-induced body core temperature rise within the mother will result in a similar rise within the fetus, and thus an exposure at the occupational whole-body average SAR basic restriction would result in a similar body core temperature rise in mother and fetus. Therefore, to maintain fetal temperature to the level required by the general public, a pregnant woman is considered a member of the general public in terms of the whole-body average SAR basic restriction.

ICNIRP's decision on the occupational whole-body average SAR for pregnant women is significantly conservative compared with the established teratogenic fetal temperature threshold (2°C: Edwards et al. 2003; Ziskin and Morrissey 2011). ICNIRP also recognizes that the body core temperature of the fetus, especially during early stage one or embryonic development, is not clearly defined, and that there is no direct evidence that occupational whole-body exposure of the pregnant worker will harm the fetus. It is thus acknowledged that the decision to treat a pregnant worker as a member of the general public is conservative. ICNIRP also notes that there are some mitigating techniques that can be considered in order to allow pregnant workers to enter areas where radiofrequency EMFs are at occupational exposure levels, without exceeding the general public restrictions. For example, within a 30-min averaging interval, a pregnant worker could be within an area at the occupational exposure restriction level for 6 min, providing that the SAR averaged over 30 min (which includes this 6-min interval) does not exceed the general public restrictions. In considering such mitigating techniques, local region exposure restrictions for the pregnant worker are also important, and are described in the "Considerations for fetal exposure" in "Exposure Specifications for Local Regions (100 kHz to 6 GHz)" and in "Exposure Specifications for Local Regions (>6 GHz to 300 GHz)" sections.

Exposure Specifications for Local Regions (100 kHz to 6 GHz)

Relevant quantity. For cases of exposure to radiofrequency EMF over localized body regions, temperature can rise in part of the body without altering body core temperature. Local temperature rise must therefore also be restricted. The maximum local temperature rise generally appears on the surface of the body, and local SAR is a useful surrogate for local temperature rise due to localized radiofrequency EMF exposure. However, other factors, such as clothing, environmental conditions, and physiological states can have more impact on local temperature than SAR itself.

The transition frequency between local SAR and area-averaged absorbed power density is chosen as 6 GHz (Funahashi et al. 2018). This was done as a practical compromise suitable for the conditions relevant to the spatial and temporal averaging described in the following subsections,

because no optimal single frequency exists for this transition. For frequencies lower than the transition frequency, the SAR is a metric for simultaneously protecting both the internal tissues (e.g., brain) and the skin, as explained in the "Spatial averaging considerations" section. At higher frequencies (especially above 10 GHz), the absorbed power density is a surrogate for maximum skin temperature rise.

Spatial averaging considerations. Different averaging schemes (e.g., cubic, spherical, contiguous single tissue) and masses have been assessed in terms of their ability to predict local temperature rise (Hirata and Fujiwara 2009; McIntosh and Anderson 2011). These suggest that the effect of the size of the averaging mass is more crucial than the shape of the averaging volume, and that SAR varies with different averaging schemes by a factor of approximately 2 (Hirata et al. 2006). It has also been shown that SAR averaged over a single tissue provides somewhat worse correlation with local temperature than that for multiple tissues, because the heat generated in biological tissue can diffuse up to a few centimeters (i.e., across multiple tissue types). Consequently, a cubic averaging mass of 10 g, including all tissues, is used as an appropriate spatial averaging regime for frequencies up to 6 GHz. This metric has been shown to be applicable even for plane wave exposures, in that local temperature rise in the Head and Torso, and Limbs, is correlated with SAR when this averaging mass is used (Razmadze et al. 2009; Bakker et al. 2011; Hirata et al. 2013).

Temporal averaging considerations. Time to reach steady-state temperature, given the balance between rate of radiofrequency power deposition on one hand, and heat diffusion and conduction on the other, is characterized by the time constant of temperature rise. The time constant primarily depends on heat convection due to blood flow and thermal conduction. Van Leeuwen et al. (1999), Wang and Fujiwara (1999), and Bernardi et al. (2000) report that the time needed for 80–90% of the steady-state temperature rise, at 800 MHz to 1.9 GHz, is 12–16 min. These guidelines take 6 min as a suitable, conservative averaging time for steady-state temperature rise up to 6 GHz for local exposures.

Local SAR required to increase local Type-1 and Type-2 tissue temperature by 5 and 2˚C, respectively. Although early research provided useful rabbit eye data concerning the relation between 2.45 GHz exposure and local temperature rise (e.g., Guy et al. 1975; Emery et al. 1975), research with more accurate techniques has demonstrated that the rabbit is an inappropriate model for the human eye (Oizumi et al. 2013). However, given the concern about potential radiofrequency harm to the eye, there are now several studies that provide more-accurate information about radiofrequency-induced heating of the human eye. Expressed as heating factors for the SAR averaged over 10 g of tissue (the °C rise per unit mass, per W of absorbed power), the computed heating factors of a human eye have been relatively consistent [0.11–0.16°C kg W^{-1}: Hirata (2005); Buccella et al. (2007); Flyckt et al. (2007); Hirata et al. (2007); Wainwright (2007); Laakso (2009); Diao et al. (2016)]. In most studies, the heating factor was derived for the SAR averaged over the eyeball (contiguous tissue). The SAR averaged over the cubic volume (which includes other tissues) is higher than that value (Diao et al. 2016), resulting in lower heating factors.

There is also a considerable number of studies on the temperature rise in the head exposed to mobile phone handset antennas (Van Leeuwen et al. 1999; Wang and Fujiwara 1999; Bernardi et al. 2000; Gandhi et al. 2001; Hirata and Shiozawa 2003; Ibrahim et al. 2005; Samaras et al. 2007). Hirata and Shiozawa (2003) reported that heating factors are 0.24 or 0.14°C kg W^{-1} for the local SAR averaged over a 10-g contiguous volume, with and without the pinna, respectively. Other studies considering the local SAR averaged over a 10-g cubic volume including the pinna reported heating factors of the head in the range of 0.11–0.27°C kg W^{-1} (Van Leeuwen et al. 1999; Bernardi et al. 2000; Gandhi et al. 2001). Fujimoto et al. (2006) studied the temperature rise in a child head exposed to a dipole antenna and found that it is comparable to that in the adult when the same thermal parameters were used. The heating factor in the brain (the ratio of the temperature rise in the brain to peak SAR in the head) is 0.1°C kg W^{-1} or smaller (Morimoto et al. 2016). Only one study reported the temperature rise in the trunk for body-worn antennas (Hirata et al. 2006). This study showed that the heating factor in the skin is in the range of 0.18–0.26 °C kg W^{-1}. Uncertainty factors associated with the heating factors are attributable to the energy absorbed in the pinna (for mobile phones) and other surrounding structures (for example, see Foster et al. 2018) as well as the method for spatial averaging of SAR.

Those studies are consistent with research showing that, within the 100 kHz–6 GHz range, numerical estimations converge to show that the maximum heating factor is lower than 0.25°C kg W^{-1} in the skin and 0.1°C kg W^{-1} in the brain for exposures of at least approximately 30 min. Based on these heating factors, the operational adverse health effect thresholds for the eye and brain (Type 1) and for the skin (Type 2) will not be exceeded for local SARs of up to 20 W kg^{-1}.

Considerations for fetal exposure. Local SAR heating factors for the fetus, as a function of gestation stage and fetal posture and position, have been determined that take heat exchange between mother and fetus into account (Akimoto et al. 2010; Tateno et al. 2014; Takei et al. 2018). This research used numerical models of 13-week, 18-week,

and 26-week pregnant women. The heating factors of the fetus were several times lower than those of the mother in most cases. However, the largest heating factor was observed when the fetal body position is very close to the surface of the abdomen (i.e., middle and later stages of gestation). These provide 0.1°C kg W^{-1} as a conservative heating factor for the fetus.

Based on these findings, exposure of the mother at the occupational basic restriction of 10 W kg^{-1} will result in a temperature rise in the fetus of approximately 1°C, which is lower than the operational adverse health effect threshold for the Head and Torso, but results in a smaller reduction factor (i.e., 2) than that considered appropriate for the general public (i.e., 10). It follows that a localized occupational radiofrequency EMF exposure of the mother would cause the temperature to rise in the fetus to a level higher than that deemed acceptable for the general public. Therefore, to maintain fetal temperature to the level required by the general public local SAR restrictions, a pregnant woman is considered a member of the general public in terms of the local SAR restriction.

It is noted that the above-mentioned case appears only in the middle and late pregnancy stages (18 to 26-week gestation), while the heating factor of the fetus in the early pregnancy stage (12-week gestation) is at most 0.02°C kg W^{-1} (Tateno et al. 2014; Takei et al. 2018). This 12-week gestation fetal temperature rise is 100 times lower than the threshold (2°C) for teratogenic effects in animals (Edwards et al. 2003; Ziskin and Morrissey 2011).

Exposure Specifications for Local Regions (>6 GHz to 300 GHz)

Relevant quantity. In a human body exposed to radiofrequency EMF, an electromagnetic wave exponentially decays from the surface to deeper regions. This phenomenon is characterized according to penetration depth, as described below:

$$S_{ab} = PD_0 \int_0^{Z_{max}} e^{-\frac{2z}{\delta}}\, dz, \qquad (23)$$

where S_{ab} is the absorbed power density, the body surface is at $z = 0$, δ is the penetration depth from the body surface in the z direction (defined as the distance from the surface where 86% of the radiofrequency power is absorbed), and Z_{max} is depth of the body at the corresponding region; where Z_{max} is much larger than the penetration depth, infinity can be substituted for Z_{max}. PD_0 is the specific absorbed power averaged over the area A at $z = 0$, as described below:

$$PD_0 = \iint_A \rho(x,y,0) \cdot SAR(x,y,0)\, dxdy/A. \qquad (24)$$

The penetration depth depends on the dielectric properties of the medium, as well as frequency. As frequency increases, the penetration depth decreases, and is predominantly within the surface tissues at frequencies higher than about 6 GHz. Table 10 lists the penetration depths based on the dielectric properties of skin tissue (dermis) measured by Sasaki et al. (2017) and Sasaki et al. (2014).

As a result, the local SAR averaged over a 10-g cubical mass with side lengths of 2.15 cm is no longer a good proxy for local temperature rise; that is, the power deposition is limited to within a few millimeters of the surface tissues. Conversely, the power density absorbed in the skin provides a better approximation of the superficial temperature rise from 6 GHz to 300 GHz (Foster et al. 2016; Funahashi et al. 2018).

Spatial averaging considerations. Thermal modeling (Hashimoto et al. 2017) and analytical solutions (Foster et al. 2016) suggest that a square averaging area of 4 cm^2 or smaller provides a close approximation to local maximum temperature rise due to radiofrequency heating at frequencies greater than 6 GHz. This is supported by computations for realistic exposure scenarios (He et al. 2018). An important advantage of the 4-cm^2 averaging area is the consistency at 6 GHz between local SAR and absorbed power density; the face of an averaging 10-g cube of SAR is approximately 4 cm^2.

Because the beam area can usually only be focused to the size of the wavelength, the averaging area of the absorbed power density relevant to the temperature rise depends on frequency; smaller averaging areas are necessary as frequency increases. Therefore, a smaller averaging area is sometimes necessary for extremely focused beams at higher frequencies. An additional criterion is therefore imposed for frequencies above 30 GHz for the spatial peak (maximum) absorbed power density averaged over 1 cm^2, such that it must not exceed 2 times the value for the averaging area of 4 cm^2 (Foster et al. 2016).

Temporal averaging considerations. As well as the cases of localized exposure at frequencies lower than 6 GHz, the temperature rise due to localized exposure to radiofrequency EMF over 6 GHz also achieves an equilibrium state with a particular time constant. Morimoto et al. (2017) demonstrated that the same averaging time as the local SAR (6 min) is appropriate for localized exposure from 6 GHz to 300 GHz. The time needed for steady-state local temperature rise decreases gradually as frequency increases, but no notable change is observed at frequencies higher than 15 GHz (Morimoto et al. 2017). The time needed to reach 80–90% of the maximum temperature rise is approximately 5–10 min at 6 GHz and 3–6 min at 30 GHz. However, it is noted that the time constant becomes shorter if brief or irregular exposure is considered, which is discussed in the "Brief Exposure Specifications for Local Regions (>6 GHz to 300 GHz)" section. In the present guidelines, 6 min is chosen as the averaging time, with additional

restrictions for briefer or irregular exposures subjected to additional constraints as a conservative measure.

Absorbed power density required to increase local Type-1 tissue temperature by 5˚C. Above 6 GHz, power absorption is primarily restricted to superficial tissue and cannot result in tissue temperatures that exceed operational adverse health effect thresholds for Type-2 tissues without also exceeding those for the more superficial Type-1 tissues (e.g., Morimoto et al. 2016). Therefore, exposure level must be chosen to ensure that temperature rise in the more superficial Type-1 tissue does not exceed the operational threshold of 5°C.

Tissue heating, as a function of absorbed power density over 6 GHz, is dependent on a variety of factors, as it is for lower frequencies. A comprehensive investigation of the heating factors for absorbed power density [in terms of the temperature rise (°C) over a unit area (m^2), per W of absorbed power] has been conducted in the case of a plane wave incident to a multi-layered slab model as an extreme uniform exposure condition (Sasaki et al. 2017). In that study, Monte Carlo statistical estimation of the heating factor was conducted where it was shown that the maximum heating factor for absorbed power density is 0.025°C m^2 W^{-1}. This value is more conservative (larger) than results from other studies on the temperature rise in the skin (Alekseev et al. 2005; Foster et al. 2016; Hashimoto et al. 2017) and the eye (Bernardi et al. 1998; Karampatzakis and Samaras 2013). Thus, to increase temperature by 5°C requires an absorbed power density of 200 W m^{-2}.

Considerations for fetal exposure. As discussed in the "Considerations for fetal exposure" of the "Exposure Specifications for Local Regions (100 kHz to 6 GHz)" section in relation to the frequency characteristics of the SAR distribution, the contribution of surface heating due to radiofrequency EMF exposure above 6 GHz to fetal temperature rise is likely very small (and smaller than that from below 6 GHz). This suggests that the fetus will not receive appreciable heating from localized exposure above 6 GHz. However, there is currently no study that has assessed this. ICNIRP thus takes a conservative approach for exposures above 6 GHz and requires that the pregnant worker is treated as a member of the general public in order to ensure that the fetus will not be exposed above the general public basic restrictions.

Brief exposure specifications for local regions (100 kHz to 6 GHz)

The 6-min averaging scheme for localized exposure allows greater strength of the local SAR if the exposure duration is shorter than the averaging time. However, if the exposure duration is significantly shorter, heat diffusion mechanisms are inadequate to restrict temperature rise. This means that the 6-min averaged basic restriction can temporarily cause higher temperature rise than the operational adverse health effect thresholds if the exposure period is shorter than 6 min.

A numerical modeling investigation for brief exposure to radiofrequency EMF from 100 MHz to 6 GHz, using a multi-layer model and an anatomical head model, found that the SA corresponding to the allowable temperature rise is greatly variable depending on a range of factors (Kodera et al. 2018). Based on that study and empirical equations of the SA corresponding to the operational adverse health effect threshold for the skin (5°C), the exposure corresponding to this temperature rise is derived from the following equations for Head and Torso:

$$\mathrm{SA}(t) = 7.2\left(0.05 + 0.95\sqrt{t/360}\right)\ \left(\mathrm{kJ\ kg^{-1}}\right), \qquad (25)$$

where t is time in seconds and applicable for t<360, and SA (t) is spatially averaged over any 10-g cubic tissue, considering the continuity of the SAR at 6 min. The averaging procedure of SA is in the same manner as SAR in eqn (13). For Limbs, the following equation should be satisfied:

$$\mathrm{SA}(t) = 14.4\left(0.025 + 0.975\sqrt{t/360}\right)\ \left(\mathrm{kJ\ kg^{-1}}\right). \quad (26)$$

It is noted that the above logic results in slightly different time functions for brief exposure below and above 6 GHz; the resultant time functions below 6 GHz are more conservative than for above 6 GHz (i.e., eqns 27 and 28).

The numerical modeling study by Kodera et al. (2018) also shows that the temperature rise in Type-2 tissue (e.g., brain) is also kept below 1°C by the SA restriction defined in eqn (25). They furthermore reported that the SA corresponding to the allowable temperature rise increases as frequency decreases. At 400 MHz or lower, the SA derived from the local 6-min SAR basic restriction [10 (W kg^{-1}) × 360 (s) = 3.6 (kJ kg^{-1})] does not cause the temperature rise corresponding to the operational adverse health effect threshold for the Head and Torso to be exceeded. Accordingly, this SA limit is only required for exposures above 400 MHz.

It should be noted that eqns (25) and (26) must be met for all intervals up to 6 min, regardless of the particular pulse or non-pulsed continuous wave patterns. That is, exposure from any pulse, group of pulses, or subgroup of pulses in a train, as well as from the summation of exposures (including non-pulsed EMFs), delivered in t seconds, must not exceed that specified in eqns (25) to (26), as exposure to a part of the exposure pattern can be more critical than exposure to a single pulse or the exposure averaged over t. For example, if two 1-s pulses are separated by 1 s, the levels

provided by eqns (25) and (26) must be satisfied for each of the 1-s pulses as well as for the total 3-s interval.

The above discussion on brain temperature rise suggests that the temperature rise in the fetus will also be lower than that assumed for the steady-state (6-min) exposure. That is, as the Type-2 tissue temperature rise will be kept below the operational adverse health effect threshold by applying eqn (25), this will presumably also be the case for temperature rises for the fetus due to brief exposures. However, there is no study available that has considered the effect of brief exposure of pregnant women up to the occupational limit on the fetus. ICNIRP thus maintains the same conservative policy for <6-min exposure as for >6-min exposure (see "Considerations for fetal exposure of Exposure Specifications for Local Regions (100 kHz to 6 GHz)" section), and requires the pregnant worker to be subject to the general public restrictions.

Brief Exposure Specifications for Local Regions (>6 GHz to 300 GHz)

Similar to the situation for frequencies up to 6 GHz, temperature rise can be enhanced for intense short pulses or discontinuous exposures above 6 GHz, relative to a continuous exposure with the same absorbed power density averaged over a 6-min interval. This becomes significant at frequencies higher than 30 GHz (Foster et al. 2016). Considering the robustness and consistency of simple multilayer models, the basic restrictions for the brief exposures are derived based on investigations using simple models (Foster et al. 2016; Morimoto et al. 2017). Unlike continuous wave exposure, the effect of diffraction, or interference of waves reflected from protruding parts of the body back to the skin, may be apparent for brief pulses. Although the effect of diffraction to the absorbed power density is yet to be fully determined, the resultant temperature rise is estimated to be up to 3 times higher if pulsed than that due to the same absorbed power density spread evenly over a 6-min interval (Laakso et al. 2017).

Considering these factors, absorbed energy density basic restrictions (U_{ab}) have been set as a function of the square root of the time interval, to account for heterogeneity of temperature rise (Foster et al. 2016). These have been set to match the operational adverse health effect threshold for Type 1 tissue, as well as to match the absorbed energy density derived from the absorbed power density basic restriction for 360 s. As per the brief interval exposure limits for frequencies up to 6 GHz, the superficial nature of the resultant temperature rise will not result in temperatures that exceed Type-2 tissue operational adverse health effect thresholds, and so only the Type-1 tissue threshold of 5°C needs to be considered here.

Consequently, an extension of the formula from Kodera et al. (2018) for frequencies up to 6 GHz, specifies the maximum absorbed energy density level for brief exposures corresponding to the 5°C temperature rise as follows:

$$U_{ab}(t) = 72\left(0.05 + 0.95\sqrt{t/360}\right)\ \left(\text{kJ m}^{-2}\right) \quad \text{averaged over 2 cm} \times \text{2 cm}, \tag{27}$$

where t is the time interval in seconds and is applicable for t<360s. Above 30 GHz, an additional criterion is given for 1 cm × 1 cm averaging areas, such that absorbed energy density must not exceed the value specified in eqn (28):

$$U_{ab}(t) = 144\left(0.025 + 0.975\sqrt{t/360}\right)\ \left(\text{kJ m}^{-2}\right) \quad \text{averaged over 1 cm} \times \text{1 cm}. \tag{28}$$

It should be noted that eqns (27) and (28) must both be met for all intervals up to 6 min, regardless of the particular pulse or non-pulsed continuous wave patterns. That is, exposure from any pulse, group of pulses, or subgroup of pulses in a train, as well as from the summation of exposures (including non-pulsed EMFs), delivered in t seconds, must not exceed that specified in eqns (27) and (28), as exposure to a part of the exposure pattern can be more critical than exposure to a single pulse or the exposure averaged over t. For example, if two 1-s pulses are separated by 1 s, the levels provided by eqns (27) and (28) must be satisfied for each of the 1-s pulses, as well as for the total 3-s interval.

As discussed above, in relation to the frequency characteristics of the SAR distribution, the contribution of the surface heating due to radiofrequency EMF above 6 GHz to fetal temperature rise is likely smaller than that below 6 GHz. This is the same for cases of brief exposure. However, as there is no study on the fetus relating to exposure of a pregnant woman to radiofrequency EMF above 6 GHz, ICNIRP adopts a conservative approach and treats a pregnant worker as a member of the general public to ensure that the fetal exposure will not exceed that of the general public.

DERIVATION OF REFERENCE LEVELS

General Considerations for Reference Levels

As described in the main guidelines document, the reference levels have been derived as a practical means of assessing compliance with the present guidelines. The reference levels for **E**-field strength, **H**-field strength and incident power density have been derived from dosimetric studies assuming whole-body exposure to a uniform field distribution, which is generally the worst-case scenario. Due to the strongly conservative nature of the reference levels in most exposure scenarios, reference levels may often be exceeded without exceeding the corresponding basic restrictions, but this should always be verified to determine compliance.

Different reference level application rules have been set for exposure in the far-field, radiative near-field and reactive near-field zones. The intention of ICNIRP's distinction between these zones is to provide assurance that the reference levels are generally more conservative than the basic restrictions. In so far as the distinction between the zones is concerned, the principle (but not only) determinant of this is the degree to which a field approximates plane wave conditions. A difficulty with this approach is that other factors may also affect the adequacy of estimating reference level quantities from basic restriction quantities. These include the EMF frequency, physical dimensions of the EMF source and its distance from the resultant external EMFs assessed, as well as the degree to which the EMFs vary over the space to be occupied by a person. Taking into account such sources of uncertainty, the guidelines have more conservative rules for exposure in the reactive and radiative near-field than far-field zone. This makes it difficult to specify whether, for the purpose of compliance, an exposure should be considered reactive near-field, radiative near-field or far-field without consideration of a range of factors that cannot be easily specified in advance. As a rough guide, distances $> 2D^2/\lambda$ (m), between $\lambda/(2\pi)$ and $2D^2/\lambda$ (m), and $< \lambda/(2\pi)$ (m) from an antenna correspond approximately to the far-field, radiative near-field and reactive near-field, respectively, where D and λ refer to the longest dimension of the antenna and wavelength, respectively, in meters. However, it is anticipated that input from technical standards bodies should be utilized to better determine which of the far-field/near-field zone reference level rules should be applied so as to provide appropriate concordance between reference levels and basic restrictions.

E-Field and H-Field Reference Levels up to 30 MHz

In the ICNIRP (1998) guidelines, the reference levels in this frequency region were derived from the whole-body average SAR for whole-body exposure to plane waves. However, Taguchi et al. (2018) demonstrated that whole-body exposure to the decoupled **H**-field results in a whole-body average SAR significantly lower than that calculated for the whole-body exposure to plane-waves with the same **H**-field strength. The whole-body exposure to the decoupled **E**-field was also calculated and it was found that the whole-body average SARs are almost the same as those for the plane wave with the same direction and strength as the **E**-field. The reference levels relevant to the whole-body average SAR basic restrictions below 30 MHz in these guidelines are therefore based on the numerical calculations of the whole-body average SAR for the whole-body exposure to the decoupled uniform **E**-field and **H**-field, separately. Taguchi et al. (2018) also concluded that local SAR basic restrictions, including in the ankle, will also be satisfied when the whole-body SAR basic restrictions are satisfied. This means that compliance with the whole-body average reference levels in this frequency region will result in exposures that do not exceed the whole-body average and local SAR basic restrictions.

In the low frequency guidelines (ICNIRP 2010) where reference levels for frequencies up to 10 MHz are set to protect against nerve cell stimulations, a reduction factor of 3 was applied to account for uncertainty associated with the numerical modeling of the relation between the external fields and the induced (internal) electric fields. The reason for this is that 2-mm cube-averaged values (within a specific tissue) were evaluated in the low frequency guidelines, which are significantly affected by computational artifact.

In the present guidelines, however, the uncertainty of the numerical simulation is not significant because the spatial averaging procedure applied in evaluating the whole-body average and local SAR significantly decreases the uncertainty of the computational artifact. Therefore, additional reduction factors due to computational uncertainty do not need to be considered in deriving the reference levels relevant to the local and whole-body average SAR basic restrictions below 30 MHz in these guidelines.

E-Field, H-Field and Power Density Reference Levels From >30 MHz to 6 GHz

The ICNIRP (1998) whole-body average SAR for exposure to a field strength equal to the reference level becomes close to the basic restrictions around the whole-body resonant frequency (30–200 MHz) and post resonant frequency region (1,500–4,000 MHz).

The resonance frequency appears at a frequency where half of the wavelength in free space is close to the height (vertical dimension of a person standing) of the human body in free space, or where a quarter of the wavelength in free space is close to the height of a human body standing on the ground plane (Durney et al. 1986), resulting in higher whole-body average SARs. Whole-body resonance appears only for the case of vertically polarized plane wave incidence. If different polarizations are assumed, the resultant whole-body average SAR is significantly (a few orders of magnitude) lower than that of the case of the vertical polarization around the whole-body resonant frequency (Durney et al. 1986). Whole-body resonance has been confirmed by numerical computations (Dimbylow 1997; Nagaoka et al. 2004; Dimbylow 2005; Conil et al. 2008; Kühn et al. 2009; Hirata et al. 2010).

Above the whole-body resonant frequency, especially above a few GHz, the differences in the whole-body average SARs due to polarization are not significant compared with those at the whole-body resonant frequency. Hirata et al. (2009) reported that the whole-body average SAR in child models from 9 months to 7 years old, exposed to horizontally polarized plane wave incidence, is only slightly higher

(up to 20%) than the vertically polarized plane wave at frequencies from 2 GHz to 6 GHz. A similar tendency has been reported in other studies (Vermeeren et al. 2008; Kühn et al. 2009).

ICNIRP had concluded that, given the same external field, the child whole-body average SAR can be 40% higher than those of adults (ICNIRP 2009). After that ICNIRP statement, Bakker et al. (2010) reported similar (but slightly higher) enhancements (45%) of the child whole-body average SAR. The effects of age dependence of dielectric properties of the tissues and organs have also been investigated, but no significant effect relevant to whole-body average SAR has been found (Lee and Choi 2012). It is noted that the increased whole-body average SARs have been reported from calculations using very thin child models, which were scaled from adult, and very young (infant) models. Those studies assumed that the child or infant maintains their posture for a substantial time interval so as to match an extreme case condition, in order for their whole-body SAR to exceed the basic restriction. Further, a more recent study using child models that have used the standard dimensions specified by the International Commission on Radiological Protection (ICRP), rather than scaled versions of adults, showed that the increases of the whole-body average SARs in the standard child models are not significant (at most 16%; Nagaoka et al. 2019). Similarly, the relation between whole-body average SAR and whole-body mass has been investigated and it has been found that the whole-body average SAR in low body mass index (BMI) adults can increase in a similar manner to the case of the child (Hirata et al. 2010, 2012; Lee and Choi 2012).

As discussed in the "Considerations for fetal exposure" of the "Whole-body Average Exposure Specifications" section, the temperature of the fetus is similar to the body core temperature of the mother. The whole-body average SAR, which is used to restrict body core temperature rise, is defined as the power absorption in the whole body divided by the whole-body mass. Therefore, the whole-body average SAR of a pregnant woman, whose mass is larger, is generally the same as, or lower than, that of a non-pregnant woman in this frequency region. Nagaoka et al. (2007) reported that the whole-body average SAR of a 26-week pregnant woman model exposed to the vertically polarized plane wave from 10 MHz to 2 GHz was almost the same as, or lower than, the non-pregnant woman model for the same exposure condition.

Dimbylow (2007) reported that, using a simplified pregnant woman model, the whole-body average SAR in both the fetus and mother is highest for ungrounded conditions, at approximately 70 MHz. A similar tendency was found for anatomical fetus models of second and third trimester conditions, with the whole-body average SARs in a fetus of 20, 26, and 29 week gestation periods approximately 80%, 70%, and 60% of those in the mother, respectively (Nagaoka et al. 2014). The whole-body average SARs of the fetus, while still embryonic, are comparable to or lower than the whole-body average SARs in the mother, because the embryo is located deep within the abdomen of the mother (Kawai et al. 2009). The pregnant woman is therefore not considered independently from the fetus in terms of reference levels and is subject to the general public restrictions.

As described above, there are numerous databases relevant to whole-body average SAR for whole-body exposure in this frequency region. These include a considerable number reported since the ICNIRP (1998) guidelines, which are generally consistent with the database used as the basis for the ICNIRP (1998) guidelines. ICNIRP uses a combination of the older and newer databases to derive the reference levels, taking into account some incongruences discussed below.

Since publishing the ICNIRP (1998) guidelines it has been shown that the whole-body average SAR basic restrictions can be exceeded for exposure levels at the reference level for children or small stature people. As reviewed above, the whole-body average SAR is exceeded by no more than 45%, and only for very specific child models, and more recent modeling using realistic, international standardized child models shows only a modest increase of 16% at most (Nagaoka et al. 2019). This deviation is comparable with the uncertainty expected in the numerical calculations. For example, Dimbylow et al. (2008) reported that differences in the procedure or algorithm used for the whole-body averaging results in 15% variation of the whole-body average SARs at 3 GHz, and that the assignment of the dielectric properties of the skin conditions (dry or wet) reported also results in 10% variation in the whole-body average SARs at 1.8 GHz (Gabriel et al. 1996).

As reviewed in the "Considerations for fetal exposure" of the "Whole-body Average Exposure Specifications" section, the heating factor of children is generally lower than that of adults. It follows that the increased SAR will not result in a larger temperature rise than is allowed for adults, and so will not affect health. Given the magnitude of uncertainty and the lack of health benefit in reducing the reference levels to account for small stature people, this has not resulted in ICNIRP altering the reference levels in the frequency range >30 MHz to 6 GHz.

It is also noted that there are other conditions where the whole-body average reference levels can result in whole-body average SARs that exceed the basic restrictions by up to 35%. This occurs in human models with unusual postures that would be difficult to maintain for a sufficient duration in order to cause the elevated SAR (Findlay and

Dimbylow 2005; Findlay et al. 2009). However, the elevated SAR is small compared with the associated uncertainties and the conservative nature of the basic restrictions themselves, the postures are not likely to be routinely encountered, and there is no evidence that this will result in any adverse health effects.

Reference Levels From >6 GHz to 300 GHz for Whole-Body Exposure

Above 6 GHz, radiofrequency EMFs generally follow the characteristics of plane wave or far-field exposure conditions; incident power density or equivalent incident power density is used as the reference level in this frequency region. The reactive near-field exists very close to a radiofrequency source in this frequency region. The typical boundary of the reactive near-field and the radiative near-field is defined as $\lambda/(2\pi)$ (e.g., 8 mm at 6 GHz). Because the incident power density used for the reference levels above 6 GHz does not appropriately correlate with the absorbed power density used for the basic restrictions in the reactive near-field region, reference levels cannot be used to determine compliance in the reactive near field; basic restrictions need to be assessed for such cases.

The radiofrequency power absorbed in the body exponentially decays in the direction from the surface to deeper regions (see eqn 23). Therefore, the power absorption is primarily confined within the body surface above 6 GHz, where the total power absorption or the whole-body average SAR is approximately proportional to the exposed area of the body surface (Hirata et al. 2007; Gosselin et al. 2009; Kühn et al. 2009; Uusitupa et al. 2010). For example, an experimental study using a reverberation chamber found a strong correlation between the whole-body average SAR and the surface area of a human body from 1 GHz to 12 GHz (Flintoft et al. 2014).

Because the whole-body average SAR is approximately proportional to the incident power density and body surface area (and is not dependent on EMF frequency), ICNIRP has extended the whole-body reference levels from below 6 GHz, up to 300 GHz. ICNIRP (1998) set whole-body reference levels within this range (up to 10 GHz) at 50 W m^{-2} and 10 W m^{-2} (for occupational and general public exposure, respectively). As there is no evidence that these levels will result in exposures that exceed the whole-body basic restrictions above 6 GHz, or that they will cause harm, these guidelines retain the ICNIRP (1998) reference levels for whole-body exposure conditions.

The same time and spatial average for the whole-body average SAR basic restrictions are applied to these corresponding reference levels. Therefore, the incident power density is to be temporally averaged over 30 min and spatially averaged over the space to be occupied by a human body (whole-body space).

Reference Levels From >6 GHz to 300 GHz for Local Exposure

The incident power density (S_{inc}) reference levels above 6 GHz for local exposure can be derived from the basic restrictions (i.e., from absorbed power density, S_{ab}):

$$S_{inc} = S_{ab} T^{-1} \left(\mathrm{W\ m^{-2}} \right), \tag{29}$$

where T is Transmittance, defined as follows:

$$\text{Transmittance} = 1 - |\Gamma|^2. \tag{30}$$

The reflection coefficient Γ is derived from the dielectric properties of the tissues, shape of the body surface, incident angle and polarization. For transverse electric (TE)-wave incidence, the angle corresponding to the maximum transmittance is the angle normal to the body surface, whereas for transverse magnetic (TM)-wave incidence this occurs at the Brewster angle (the angle of incidence at which there is no reflection of the TM wave). Furthermore, for cases of oblique incidence of the radiofrequency EMF wave, Li et al. (2019) have shown that the incident power and energy densities of TE waves, averaged over the body or boundary surface, overestimate the absorbed power and energy densities, while the absorbed power and energy densities of TM-waves around the Brewster angle approach the incident power and energy densities. They also found that normal incidence is always the worst case scenario regarding temperature rise (Li et al. 2019).

In the present guidelines, the basic restrictions and reference levels are derived from investigations assuming normal incidence to the multi-layered human model. As this represents worst-case modeling for most cases, the results obtained and used in these guidelines will generally be conservative.

The variation and uncertainty of the transmittance for the normal-angle incident condition have been investigated (Sasaki et al. 2017). The transmittance asymptotically increases from 0.4 to 0.8 as the frequency increases from 10 GHz to 300 GHz. Similar tendencies have also been reported elsewhere (Kanezaki et al. 2009; Foster et al. 2016; Hashimoto et al. 2017).

Considering the frequency characteristics of the transmittance, the reference levels for local exposure have been derived as exponential functions of the frequency linking 200 W m^{-2} at 6 GHz to 100 W m^{-2} at 300 GHz (for occupational exposure). The same method is applied for the derivation of reference levels for the general public. For the same reasons given in the "Reference Levels from >6 GHz to 300 GHz for Whole-body Exposure" section, reference levels cannot be used to determine compliance in the reactive near field; basic restrictions need to be assessed for such cases.

The temporal and spatial characteristics are almost the same for incident power density and absorbed power density at the body surface for the scale considered in the basic restrictions, i.e., 6 min, and either 4 cm^2 or 1 cm^2 (an additional criteria above 30 GHz). Therefore, the same averaging conditions are applied to the incident power density reference levels, as for the absorbed power density basic restrictions.

Limb Current Reference Levels

Limb current is defined as the current flowing through the limbs, such as through an ankle or wrist. High local SAR can appear in these parts of the body because of their anatomical composition. The volume ratio of the high conductivity tissues to the low conductivity tissues is small in the ankle and wrist, resulting in the current concentrating into high conductivity tissues such as muscle, and thus greater SAR. This phenomenon is particularly pronounced for cases of a human body standing on the ground plane in a whole-body resonant condition.

The local SAR in limbs (ankle and wrist) is strongly correlated with the current flowing through the limbs. Although the local SAR is generally difficult to measure directly, the limb SAR can be derived from the limb current (I), which can be relatively easily measured, as follows:

$$\mathrm{SAR} = \frac{\sigma \mathrm{E}^2}{\rho} = \frac{\mathrm{J}^2}{\sigma\rho} = \frac{\mathrm{I}^2}{\sigma\rho A^2}, \qquad (31)$$

where J and A are the current density and effective section area, respectively.

The limb current reference levels are therefore set in order to evaluate the local SAR in the ankle and wrist, especially around the ankle in a grounded human body for the whole-body resonant condition. As the frequency increases above the whole-body resonant frequency for the grounded condition, the efficiency of the localization within the limbs gradually decreases. Thus, at higher frequencies, the maximum local SAR does not generally appear around limbs, and is thus not relevant.

Dimbylow (2002) showed that a limb current of 1 A at 10 MHz to 80 MHz causes 530 W kg^{-1} to 970 W kg^{-1} of local SAR averaged over 10 g in the ankles of an adult male model standing on a grounded plane. It is noted that the shape of the averaging region of the 10-g tissue was not cubic, but contiguous, which results in higher SAR values than those of a cube. Based on that study, ICNIRP sets the limb current reference levels at 100 mA and 45 mA for occupational and general public exposures, respectively, to conservatively ensure compliance with the local SAR basic restrictions in the limbs (e.g., the maximum local SAR in the limbs for a 100 mA current would only be 10 W kg^{-1}). Taguchi et al. (2018) confirmed this relation between SAR and ankle current from 10 MHz to 100 MHz in different anatomical models.

Similarly, Dimbylow (2001) computed the 10-g local SAR (with contiguous tissue) for a 100-mA wrist current, which resulted in 27 W kg^{-1} at 100 kHz, decreasing to 13 W kg^{-1} at 10 MHz. Considering the reduction of SAR for the cubic compared to contiguous shape, the 100-mA limb current at the wrist will also conservatively ensure compliance with the local SAR basic restrictions in the wrist. Based on this, ICNIRP has revised the lower frequency range to 100 kHz, from 10 MHz in ICNIRP (1998).

As shown in eqn (31), the local SAR is proportional to the squared value of the limb current. In eqn (31), however, the effective area is a constant to relate the limb current to the 10-g averaged local SAR and depends on not only the actual section area but also tissue distribution/ratio and conductivity. Because the conductivity asymptotically increases as the frequency increases from 100 kHz to 110 MHz, the relationship between local SAR and limb current is not constant across this frequency range. For example, Dimbylow (2002) demonstrated that the local SAR due to a constant limb current halved as frequency increased from 10 MHz to 80 MHz. This suggests that the upper frequency limit for limb current reference levels could potentially be lowered, relative to the upper limit of the 10 MHz to 110 MHz range of ICNIRP (1998). However, due to the lack of research addressing this issue, ICNIRP has kept the same upper frequency range as in ICNIRP (1998).

Because the limb current reference levels are relevant to the local SAR basic restrictions, the same temporal averaging is applied (i.e., 6 min). Further, as the squared value of the limb current is proportional to the local SAR, the squared value of the limb current must be used for time averaging (as described in the "Quantities and Units" section). Note that temperature rise for exposures of less than 6 min is only of concern for frequencies above 400 MHz, which is higher than the upper frequency limit for limb currents. Limb current reference levels are therefore not required for exposures of less than 6 min.

Reference Levels for Brief Exposure (<6 min)

The reference levels for brief exposure are derived to match the brief exposure basic restrictions, which have been set in terms of SA and absorbed energy density, up to and above 6 GHz, respectively.

The reference levels have been derived from numerical computations with the multi-layered human model exposed to a plane wave, or to typical sources used close to the body, such as a dipole antenna.

The reference levels vary as a function of time interval to match the absorbed energy density basic restrictions (above 6 GHz), with a similar function used below 6 GHz to match the SA basic restrictions. It is noted that the time

function of the absorbed energy density basic restrictions and corresponding incident energy density reference levels are more conservative than those for the SA basic restrictions and corresponding incident energy density reference levels. This means that the reference levels are more conservative above than below 6 GHz.

Because the reference levels are based on the multilayered model, the uncertainty included in the dosimetry is not significant. Conversely, this simple modeling is likely overly conservative for a realistic human body shape and structure. This overestimation decreases as the frequency increases because the penetration depth is short relative to the body-part dimensions. Morphological variations are also not significant.

REFERENCES

Adair ER, Blick DW, Allen SJ, Mylacraine KS, Ziriax JM, Scholl DM. Thermophysiological responses of human volunteers to whole body RF exposure at 220 MHz. Bioelectromagnetics 26:448–461; 2005.

Adair ER, Mylacraine KS, Allen SJ. Thermophysiological consequences of whole body resonant RF exposure (100 MHz) in human volunteers. Bioelectromagnetics 24:489–501; 2003.

Adair ER, Mylacraine KS, Cobb BL. Partial-body exposure of human volunteers to 2450 MHz pulsed or CW fields provokes similar thermoregulatory responses. Bioelectromagnetics 22: 246–259; 2001.

Akimoto S, Kikuchi S, Nagaoka T, Saito K, Watanabe S, Takahashi M, Ito K. Evaluation of specific absorption rate for a fetus by portable radio terminal close to the abdomen of a pregnant woman. IEEE Trans Microwave Theory Tech 58:3859–3865; 2010.

Alekseev S, Radzievsky A, Szabo I, Ziskin M. Local heating of human skin by millimeter waves: effect of blood flow. Bioelectromagnetics 26:489–501; 2005.

Asakura H. Fetal and neonatal thermoregulation. J Nippon Med Sch 71:360–370; 2004.

Bakker J, Paulides M, Christ A, Kuster N, Van Rhoon G. Assessment of induced SAR in children exposed to electromagnetic plane waves between 10 MHz and 5.6 GHz. Phys Med Biol 55:3115; 2010.

Bakker JF, Paulides MM, Neufeld E, Christ A, Kuster N, Rhoon GCv. Children and adults exposed to electromagnetic fields at the ICNIRP reference levels: theoretical assessment of the induced peak temperature increase. Phys Med Biol 56: 4967; 2011.

Bernardi P, Cavagnaro M, Pisa S, Piuzzi E. SAR distribution and temperature increase in an anatomical model of the human eye exposed to the field radiated by the user antenna in a wireless LAN. IEEE Trans Microwave Theory Tech 46: 2074–2082; 1998.

Bernardi P, Cavagnaro M, Pisa S, Piuzzi E. Specific absorption rate and temperature increases in the head of a cellular-phone user. IEEE Trans Microwave Theory Tech 48:1118–1126; 2000.

Brockow T, Wagner A, Franke A, Offenbächer M, Resch KL. A randomized controlled trial on the effectiveness of mild water-filtered near infrared whole-body hyperthermia as an adjunct to a standard multimodal rehabilitation in the treatment of fibromyalgia. Clin J Pain 23:67–75; 2007.

Buccella C, De Santis V, Feliziani M. Prediction of temperature increase in human eyes due to RF sources. IEEE Trans Electromagnet Compat 49(4):825–833; 2007.

Conil E, Hadjem A, Lacroux F, Wong MF, Wiart J. Variability analysis of SAR from 20 MHz to 2.4 GHz for different adult and child models using finite-difference time-domain. Phys Med Biol 53:1511–1525; 2008.

Diao Y, Leung SW, He Y, Sun W, Chan KH, Siu YM, Kong R. Detailed modeling of palpebral fissure and its influence on SAR and temperature rise in human eye under GHz exposures. Bioelectromagnetics 37:256–263; 2016.

Dimbylow P. The relationship between localised SAR in the arm and wrist current. Radiat Protect Dosim 95:177–179; 2001.

Dimbylow P. Resonance behaviour of whole-body averaged specific energy absorption rate (SAR) in the female voxel model, Naomi. Phys Med Biol 50:4053–4063; 2005.

Dimbylow P. SAR in the mother and foetus for RF plane wave irradiation. Phys Med Biol 52:3791–3802; 2007.

Dimbylow PJ. FDTD calculations of the whole-body averaged SAR in an anatomically realistic voxel model of the human body from 1 MHz to 1 GHz. Phys Med Biol 42:479–490; 1997.

Dimbylow PJ. Fine resolution calculations of SAR in the human body for frequencies up to 3 GHz. Phys Med Biol 47:2835–2846; 2002.

Dimbylow PJ, Hirata A, Nagaoka T. Intercomparison of whole-body averaged SAR in European and Japanese voxel phantoms. Phys Med Biol 53:5883–5897; 2008.

Dufour A and Candas V. Ageing and thermal responses during passive heat exposure: sweating and sensory aspects. Eur J Appl Physiol 100:19–26; 2007.

Durney CH, Massoudi H, Iskander MF. Radiofrequency radiation dosimetry handbook. Fourth ed. Brooks AFB, TX: USAF School of Aerospace Medicine (USAFSAM-TR-85-73).

Edwards MJ, Saunders RD, Shiota K. Effects of heat on embryos and foetuses. Int J Hypertherm 19:295–324; 2003.

Emery A, Kramar P, Guy A, Lin J. Microwave induced temperature rises in rabbit eyes in cataract research. J Heat Transfer 97:123–128; 1975.

Findlay R, Dimbylow P. Effects of posture on FDTD calculations of specific absorption rate in a voxel model of the human body. Phys Med Biol 50:3825–3835; 2005.

Findlay R, Lee A-K, Dimbylow P. FDTD calculations of SAR for child voxel models in different postures between 10 MHz and 3 GHz. Radiat Protect Dosim 135:226–231; 2009.

Flintoft I, Robinson M, Melia G, Marvin A, Dawson J. Average absorption cross-section of the human body measured at 1–12 GHz in a reverberant chamber: results of a human volunteer study. Phys Med Biol 59:3297–3317; 2014.

Flyckt V, Raaymakers B, Kroeze H, Lagendijk J. Calculation of SAR and temperature rise in a high-resolution vascularized model of the human eye and orbit when exposed to a dipole antenna at 900, 1500 and 1800 MHz. Phys Med Biol 52:2691–2701; 2007.

Foster KR, Ziskin MC, Balzano Q. Thermal response of human skin to microwave energy: a critical review. Health Phys 111: 528–541; 2016.

Foster KR, Ziskin MC, Balzano Q, Bit-Babik G. Modeling tissue heating from exposure to radiofrequency energy and relevance of tissue heating to exposure limits: heating factor. Health Phys 115:295–307; 2018.

Fujimoto M, Hirata A, Wang J, Fujiwara O, Shiozawa T. FDTD-derived correlation of maximum temperature increase and peak SAR in child and adult head models due to dipole antenna. IEEE Trans Electromagnet Compat 48:240–247; 2006.

Funahashi D, Hirata A, Kodera S, Foster KR. Area-averaged transmitted power density at skin surface as metric to estimate surface temperature elevation. IEEE Access 6:77665–77674; 2018.

Gabriel S, Lau RW, Gabriel C. The dielectric properties of biological tissues: III. parametric models for the dielectric spectrum of tissues. Phys Med Biol 41:2271–2293; 1996.

Gandhi OP, Li Q-X, Kang G. Temperature rise for the human head for cellular telephones and for peak SARs prescribed in safety guidelines. IEEE Trans Microwave Theory Tech 49:1607–1613; 2001.

Gosselin M-C, Christ A, Kühn S, Kuster N. Dependence of the occupational exposure to mobile phone base stations on the properties of the antenna and the human body. IEEE Trans Electromagnet Compat 51: 227–235; 2009.

Gowland P, De Wilde J. Temperature increase in the fetus due to radio frequency exposure during magnetic resonance scanning. Phys Med Biol 53:L15–L18; 2008.

Guy AW, Lin JC, Kramar PO, Emery AF. Effect of 2450-MHz radiation on the rabbit eye. IEEE Trans Microwave Theory Tech 23:492–498; 1975.

Hashimoto Y, Hirata A, Morimoto R, Aonuma S, Laakso I, Jokela K, Foster KR. On the averaging area for incident power density for human exposure limits at frequencies over 6 GHz. Phys Med Biol 62:3124–3138; 2017.

He W, Xu B, Gustafsson M, Ying Z, He S. RF compliance study of temperature elevation in human head model around 28 GHz for 5G user equipment application: simulation analysis. IEEE Access 6:830–838; 2018.

Hirata A. Temperature increase in human eyes due to near-field and far-field exposures at 900 MHz, 1.5 GHz, and 1.9 GHz. IEEE Trans Electromagnet Compat 47:68–76; 2005.

Hirata A, Asano T, Fujiwara O. FDTD analysis of human body-core temperature elevation due to RF far-field energy prescribed in the ICNIRP guidelines. Phys Med Biol 52:5013–5023; 2007.

Hirata A, Asano T, Fujiwara O. FDTD analysis of body-core temperature elevation in children and adults for whole-body exposure. Phys Med Biol 53:5223–5238; 2008.

Hirata A, Fujimoto M, Asano T, Jianqing W, Fujiwara O, Shiozawa T. Correlation between maximum temperature increase and peak SAR with different average schemes and masses. IEEE Trans Electromagn Compat 48:569–578; 2006.

Hirata A, Fujiwara O. The correlation between mass-averaged SAR and temperature elevation in the human head model exposed to RF near-fields from 1 to 6 GHz. Phys Med Biol 54:7227–7238; 2009.

Hirata A, Fujiwara O, Nagaoka T, Watanabe S. Estimation of whole-body average SAR in human models due to plane-wave exposure at resonance frequency. IEEE Trans Electromagnet Compat 52:41–48; 2010.

Hirata A, Kodera S, Wang J, Fujiwara O. Dominant factors influencing whole-body average SAR due to far-field exposure in whole-body resonance frequency and GHz regions. Bioelectromagnetics 28:484–487; 2007.

Hirata A, Laakso I, Ishii Y, Nomura T, Chan KH. Computation of temperature elevation in a fetus exposed to ambient heat and radio frequency fields. Numerical Heat Transfer, Part A: Appl 65:1176–1186; 2014.

Hirata A, Laakso I, Oizumi T, Hanatani R, Chan KH, Wiart J. The relationship between specific absorption rate and temperature elevation in anatomically based human body models for plane wave exposure from 30 MHz to 6 GHz. Phys Med Biol 58: 903–921; 2013.

Hirata A, Nagaya Y, Ito N, Fujiwara O, Nagaoka T, Watanabe S. Conservative estimation of whole-body average SAR in infant model for 0.3-6 GHz far-field exposure. Phys Med Biol 129: 2102–2107; 2009.

Hirata A, Shiozawa T. Correlation of maximum temperature increase and peak SAR in the human head due to handset antennas. IEEE Trans Microw Theory Tech 51:1834–1841; 2003.

Hirata A, Sugiyama H, Fujiwara O. Estimation of core temperature elevation in humans and animals for whole-body averaged SAR. Prog Electromagnet Res 99:53–70; 2009.

Hirata A, Watanabe S, Fujiwara O, Kojima M, Sasaki K, Shiozawa T. Temperature elevation in the eye of anatomically based human head models for plane-wave exposures. Phys Med Biol 52:6389–6399; 2007.

Hirata A, Yanase K, Laakso I, Chan KH, Fujiwara O, Nagaoka T, Watanabe S, Conil E, Wiart J. Estimation of the whole-body averaged SAR of grounded human models for plane wave exposure at respective resonance frequencies. Phys Med Biol 57:8427–8442; 2012.

Ibrahim A, Dale C, Tabbara W, Wiart J. Analysis of the temperature increase linked to the power induced by RF source. Prog Electromagn Res 52:23–46; 2005.

International Commission on Non-Ionizing Radiation Protection. Review of concepts, quantities, units, and terminology for non-ionizing radiation protection. Health Phys 49:1329–1362; 1985.

International Commission on Non-Ionizing Radiation Protection. Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). Health Phys 74:494–521; 1998.

International Commission on Non-Ionizing Radiation Protection. Review of scientific evidence on dosimetry, biological effects, epidemiological observations, and health consequences concerning exposure to high frequency electromagnetic fields (100 kHz to 300 GHz). Munich: International Commission on Non-ionizing Radiation Protection; 2009.

International Commission on Non-Ionizing Radiation Protection. Statement on the "guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz)." Health Phys 97:257–258; 2009.

International Commission on Non-Ionizing Radiation Protection. Guidelines for limiting exposure to time-varying electric and magnetic fields (1 Hz to 100 kHz). Health Phys 99:818–836; 2010.

Kühn S, Jennings W, Christ A, Kuster N. Assessment of induced radio-frequency electromagnetic fields in various anatomical human body models. Phys Med Biol 54:875–90; 2009.

Kühn S, Jennings W, Christ A, Kuster N. Assessment of induced radio-frequency electromagnetic fields in various anatomical human body models. Phys Med Biol 54:875–890; 2009.

Kanezaki A, Hirata A, Watanabe S, Shirai H. Effects of dielectric permittivities on skin heating due to millimeter wave exposure. Biomed Eng Online 8:20; 2009.

Karampatzakis A, Samaras T. Numerical modeling of heat and mass transfer in the human eye under millimeter wave exposure. Bioelectromagnetics 34:291–299; 2013.

Kawai H, Nagaoka T, Watanabe S, Saito K, Takahashi M, Ito K. Computational dosimetry in embryos exposed to electromagnetic plane waves over the frequency range of 10 MHz–1.5 GHz. Phys Med Biol 55:N1; 2009.

Kodera S, Hirata A, Funahashi D, Watanabe S, Jokela K, Croft RJ. Temperature rise for brief radio-frequency exposure below 6 GHz. IEEE Access 6:65737–65746; 2018.

Laakso I. Assessment of the computational uncertainty of temperature rise and SAR in the eyes and brain under far-field exposure from 1 to 10 GHz. Phys Med Biol 54:3393–3404; 2009.

Laakso I, Hirata A. Dominant factors affecting temperature rise in simulations of human thermoregulation during RF exposure. Physics in Medicine and Biology 56:7449–7471; 2011.

Laakso I, Morimoto R, Heinonen J, Jokela K, Hirata A. Human exposure to pulsed fields in the frequency range from 6 to 100 GHz. Phys Med Biol 62:6980–6992; 2017.

Lee A-K, Choi H-D. Determining the influence of Korean population variation on whole-body average SAR. Phys Med Biol 57:2709–2725; 2012.

Li K, Sasaki K, Watanabe S, Shirai H. Relationship between power density and surface temperature elevation for human skin exposure to electromagnetic waves with oblique incidence angle from 6 GHz to 1 THz. Phys Med Biol 64:065016; 2019.

McIntosh RL, Anderson V. SAR versus VAR, and the size and shape that provide the most appropriate RF exposure metric in the range of 0.5–6 GHz. Bioelectromagnetics 32:312–321; 2011.

Morimoto R, Hirata A, Laakso I, Ziskin MC, Foster KR. Time constants for temperature elevation in human models exposed to dipole antennas and beams in the frequency range from 1 to 30 GHz. Phys Med Biol 62:1676–1699; 2017.

Morimoto R, Laakso I, De Santis V, Hirata A. Relationship between peak spatial-averaged specific absorption rate and peak temperature elevation in human head in frequency range of 1–30 GHz. Phys Med Biol 61:5406–5425; 2016.

Nagaoka T, Niwa T, Watanabe S. Specific absorption rate in mothers and fetuses in the second and third trimesters of pregnancy. Int J Microwave Opt Tech 9:34–38; 2014.

Nagaoka T, Togashi T, Saito K, Takahashi M, Ito K, Watanabe S. An anatomically realistic whole-body pregnant-woman model and specific absorption rates for pregnant-woman exposure to electromagnetic plane waves from 10 MHz to 2 GHz. Phys Med Biol 52:6731–6745; 2007.

Nagaoka T, Watanabe S, Sakurai K, Kunieda E, Taki M, Yamanaka Y. Development of realistic high-resolution whole-body voxel models of Japanese adult males and females of average height and weight, and application of models to radio-frequency electromagnetic-field dosimetry. Phys Med Biol 49:1–15; 2004.

Nagaoka T, Watanabe S. Development of voxel models adjusted to ICRP reference children and their whole-body averaged SARs for whole-body exposure to electromagnetic fields from 10 MHz to 6 GHz. IEEE Access 7:135909–135916; 2019.

Nelson DA, Curran AR, Nyberg HA, Marttila EA, Mason PA, Ziriax JM. High-resolution simulations of the thermophysiological effects of human exposure to 100 MHz RF energy. Phys Med Biol 58:1947–1968; 2013.

Oizumi T, Laakso I, Hirata A, Fujiwara O, Watanabe S, Taki M, Kojima M, Sasaki H, Sasaki K. FDTD analysis of temperature elevation in the lens of human and rabbit models due to near-field and far-field exposures at 2.45 GHz. Radiat Protect Dosim 155:284–291; 2013.

Razmadze A, Shoshiashvili L, Kakulia D, Zaridze R, Bit-Babik G, Faraone A. Influence of specific absorption rate averaging schemes on correlation between mass-averaged specific absorption rate and temperature rise. Electromagnetics 29:77–90; 2009.

Samaras T, Kalampaliki E, Sahalos JN. Influence of thermophysiological parameters on the calculations of temperature rise in the head of mobile phone users. IEEE Trans Electromag Compat 49:936–939; 2007.

Sasaki K, Mizuno M, Wake K, Watanabe S. Monte Carlo simulations of skin exposure to electromagnetic field from 10 GHz to 1 THz. Phys Med Biol 62:6993–7010; 2017.

Sasaki K, Wake K, Watanabe S. Measurement of the dielectric properties of the epidermis and dermis at frequencies from 0.5 GHz to 110 GHz. Phys Med Biol 59:4739; 2014.

Taguchi K, Laakso I, Aga K, Hirata A, Diao Y, Chakarothai J, Kashiwa T. Relationship of external field strength with local and whole-body averaged specific absorption rates in anatomical human models. IEEE Access 6:70186–70196; 2018.

Takei R, Nagaoka T, Nishino K, Saito K, Watanabe S, Takahashi M. Specific absorption rate and temperature increase in pregnant women at 13, 18, and 26 weeks of gestation due to electromagnetic wave radiation from a smartphone. IEICE Comm Exp: 2018XBL0026; 7(6):212–217; 2018.

Tateno A, Akimoto S, Nagaoka T, Saito K, Watanabe S, Takahashi M, Ito K. Specific absorption rates and temperature elevations due to wireless radio terminals in proximity to a fetus at gestational ages of 13, 18, and 26 weeks. IEICE Trans Comm 97:2175–2183; 2014.

Uusitupa T, Laakso I, Ilvonen S, Nikoskinen K. SAR variation study from 300 to 5000 MHz for 15 voxel models including different postures. Phys Med Biol 55:1157–1176; 2010.

Van Leeuwen GM, Lagendijk JJ, Van Leersum BJ, Zwamborn AP, Hornsleth SN, Kotte AN. Calculation of change in brain temperatures due to exposure to a mobile phone. Phys Med Biol 44:2367–2379; 1999.

Vermeeren G, Joseph W, Olivier C, Martens L. Statistical multipath exposure of a human in a realistic electromagnetic environment. Health Phys 94:345–354; 2008.

Wainwright P. Computational modeling of temperature rises in the eye in the near field of radiofrequency sources at 380, 900 and 1800 MHz. Phys Med Biol 52:3335–3350; 2007.

Wang J, Fujiwara O. FDTD computation of temperature rise in the human head for portable telephones. IEEE Trans Microwave Theory Tech 47:1528–1534; 1999.

Ziskin MC, Morrissey J. Thermal thresholds for teratogenicity, reproduction, and development. Int J Hypertherm 27:374–387; 2011.

APPENDIX B: HEALTH RISK ASSESSMENT LITERATURE

Introduction

The World Health Organization (WHO) has undertaken an in-depth review of the literature on radiofrequency electromagnetic fields (EMFs) and health, which was released as a Public Consultation Environmental Health Criteria Document in 2014. This independent review is the most comprehensive and thorough appraisal of the adverse effects of radiofrequency EMFs on health. Further, the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR), a European Commission initiative, also produced a report on potential health effects of exposure to electromagnetic fields (SCENIHR 2015), and the Swedish Radiation Safety Authority (SSM) have produced several international reports regarding this issue (SSM 2015, 2016, 2018). Accordingly, the present guidelines have used these literature reviews as the basis for the health risk assessment associated with exposure to radiofrequency EMFs rather than providing another review of the individual studies. However, for completeness, ICNIRP considered more recent research published after the reviews from WHO, SCENIHR and SSM in the development of the current guidelines (cut-off date September 1st, 2019). The discussion of ICNIRP's appraisal of the radiofrequency health literature below provides a brief overview of the literature, a limited number of examples to help explain the overview, and the conclusions reached by ICNIRP.

The summary of the research on biological and health effects of radiofrequency EMFs presented below considers effects on body systems, processes or specific diseases. This

research feeds into the determination of thresholds for adverse human health effects. Research domains considered are experimental tests on cells, animals and humans, and human observational studies assessing relationships between radiofrequency EMFs and a range of potentially health-related outcomes. The experimental studies have the advantages of being able to control a large number of potential confounders and to manipulate radiofrequency EMF exposure. However, they are also limited in terms of making comparisons to realistic exposure environments, employing exposure durations sufficient to assess many disease processes, and, in the case of in vitro and animal research, relating the results to humans can also be difficult. Epidemiological research more closely relates to actual health within the community, but it is mostly observational and, thus, depending on the type of studies, various types of error and bias are of concern. These include confounding, selection bias, information bias, reverse causality, and exposure misclassification; in general, prospective cohort studies are least affected by bias but large sample sizes are needed for rare diseases. Therefore, it is important to consider research across a range of study types in order to arrive at useful conclusions concerning the relation between radiofrequency EMF exposure and adverse health effects.

It is important to note that ICNIRP bases its guidelines on substantiated[8] adverse health effects. This makes the difference between a biological and an adverse health effect an important distinction, where only adverse health effects require restrictions for the protection of humans. Research on the health effects of radiofrequency EMFs has tended to concentrate on a few areas of particular interest and concern, with some other areas receiving little or no attention. There is not sufficient research addressing potential relations between radiofrequency EMFs and the skeletal, muscular, respiratory, digestive, and excretory systems, and so these are not considered further. This review considers the potential for different types of radiofrequency EMF exposure to adversely affect health, including sinusoidal (e.g., continuous wave) and non-sinusoidal (e.g., pulsed) EMFs, and both acute and chronic exposures.

BRAIN PHYSIOLOGY AND FUNCTION

Brain Electrical Activity and Cognitive Performance

Human research addressing higher cognitive function has primarily been conducted within the ICNIRP (1998) basic restriction values. This has mainly been assessed via performance measures and derivations of the electroencephalogram (EEG) and cerebral blood flow (CBF) measures (sensitive measures of brain electrical activity and blood flow/metabolism, respectively). Most double-blind human experimental studies on cognitive performance, CBF or event-related potential (a derivative of the EEG) measures of cognitive function, did not report an association with radiofrequency EMF exposure. A number of sporadic findings have been reported, but these do not show a consistent or meaningful pattern. This may be a result of the large number of statistical comparisons and occasional chance findings. There are therefore no substantiated reports of radiofrequency EMFs adversely affecting performance, CBF, or event-related potential measures of cognitive function. Studies analyzing frequency components of the EEG have reliably shown that the 8–13 Hz alpha band in waking EEG and the 10–14 Hz "sleep spindle" frequency range in sleep EEG, are affected by radiofrequency EMF exposure with specific energy absorption rates (SAR) <2 W kg^{-1}, but there is no evidence that these relate to adverse health effects (e.g., Loughran et al. 2012).

Both rodents and non-human primates have shown a decrease in food-reinforced memory performance with exposures to radiofrequency EMFs at a whole-body average SAR >5 W kg^{-1} for rats, and a whole-body average SAR >4 W kg^{-1} for non-human primates, exposures which correspond to increases in body core temperatures of approximately 1°C. However, there is no indication that these changes were due to reduced cognitive ability, rather than the normal temperature-induced reduction of motivation (hunger). Such changes in motivation are considered normal and reversible thermoregulatory responses, and do not in themselves represent adverse health effects. Similarly, although not considered an adverse health effect, behavioral changes to reduce body temperature have also been observed in non-human primates at whole-body average SARs of 1 W kg^{-1}, with the threshold the same for acute, repeated exposures and for long-term exposures.

There is limited epidemiological research on higher cognitive function. There have been reports of subtle changes to performance measures with radiofrequency EMFs, but findings have been contradictory, as there is no evidence that the reported changes are related to radiofrequency EMF exposure and alternative explanations for observed effects are plausible.

In summary, there is no substantiated experimental or epidemiological evidence that exposure to radiofrequency EMFs affects higher cognitive functions relevant to health.

Symptoms and Wellbeing

There is research addressing the potential for radiofrequency EMFs to influence mood, behavior characteristics, and symptoms.

A number of human experimental studies testing for acute changes to wellbeing or symptoms are available, and these have failed to identify any substantiated effects of exposure. A small portion of the population attributes non-specific symptoms to various types of radiofrequency EMF exposure; this is referred to as Idiopathic Environmental Intolerance attributed to EMF (IEI-EMF). Double-blind experimental

[8]Further details concerning the term substantiated can be found in the main guidelines document.

studies have consistently failed to identify a relation between radiofrequency EMF exposure and such symptoms in the IEI-EMF population, as well as in healthy population samples. These experimental studies provide evidence that "belief about exposure" (e.g., the so-called "nocebo" effect), and not exposure itself, is the relevant symptom determinant (e.g., Eltiti et al. 2018; Verrender et al. 2018).

Epidemiological research has addressed potential long-term effects of radiofrequency EMF exposure from fixed-site transmitters and devices used close to the body on both symptoms and well-being, but with a few exceptions these are cross-sectional studies with self-reported information about symptoms and exposure. Selection bias, reporting bias, poor exposure assessment, and nocebo effects are of concern in these studies. In studies on transmitters, no consistent associations between exposure and symptoms or well-being have been observed when objective measurements of exposure were made or when exposure information was collected prospectively. In studies on mobile phone use, associations with symptoms and problematic behavior have been observed. However, these studies can generally not differentiate between potential effects from radiofrequency EMF exposure and other consequences of mobile phone use, such as sleep deprivation when using the mobile phone at night. Overall, the epidemiological research does not provide evidence of a causal effect of radiofrequency EMF exposure on symptoms or well-being.

However, there is evidence that radiofrequency EMFs, at sufficiently high levels, can cause pain. Walters et al. (2000) reported a pain threshold of 12.5 kW m^{-2} for 94 GHz, 3-s exposure to the back, which raised temperature from 34°C to 43.9°C (at a rate of 3.3°C per second). This absolute temperature threshold is consistent with Torbjork et al. (1984), who observed a median threshold for pain at 43°C, which was in compliance with simultaneously measured response thresholds of nociceptors (41°C and 43°C).

Another instance of pain induced by radiofrequency EMFs is due to *indirect* exposure via contact currents, where radiofrequency EMFs in the environment are redirected via a conducting object to a person, and the resultant current flow, dependent on frequency, can stimulate nerves, cause pain, and/or damage tissue. Induced current thresholds resulting from contact currents are very difficult to determine, with the best estimates of thresholds for health effects being for pain, which is approximately 10 and 20 mA for children and adults, respectively (extrapolated from Chatterjee et al. 1986).

In summary, no reports of adverse effects of radiofrequency EMF exposures on symptoms and wellbeing have been substantiated, except for pain, which is related to elevated temperature at high exposure levels (from both direct and indirect radiofrequency EMF exposure). Thresholds for direct effects on pain are in the vicinity of 12.5 kW m^{-2} for 94 GHz exposures to the back, which is consistent with thermal physiology knowledge. Thresholds for indirect effects (contact currents) are within the vicinity of 10 and 20 mA, for EMFs between 100 kHz and 110 MHz, for children and adults respectively.

Other Brain Physiology and Related Functions

A number of studies of potential adverse effects of radiofrequency EMFs on physiological functions that could adversely affect health have been conducted, primarily using in vitro techniques. These have included multiple cell lines and assessed functions such as intra- and intercellular signaling, membrane ion channel currents and input resistance, Ca^{2+} dynamics, signal transduction pathways, cytokine expression, biomarkers of neurodegeneration, heat shock proteins, and oxidative stress-related processes. There have been some reports of morphological changes to cells, but these have not been verified, and their relevance to health has also not been demonstrated. There have also been reports of radiofrequency EMFs inducing leakage of albumin across the blood-brain barrier in rats (e.g., Nittby et al., 2009), but due to methodological limitations of the studies and failed attempts to independently verify the results, there remains no evidence of an effect. Some studies also tested for effects of co-exposure of radiofrequency EMFs with known toxins, but there is currently no demonstration that this affects the above conclusions.

Intense pulsed low frequency electric fields (with radiofrequency components) can cause cell membranes to become permeable, allowing exchange of intra- and extra-cellular materials (Joshi and Schoenbach 2010); this is referred to as electroporation. Exposure to an unmodulated 18 GHz field has also been reported to cause a similar effect (Nguyen et al. 2017). Both exposures require very high field strengths [e.g., 10 kV m^{-1} (peak) in tissue in the case of low frequency electric fields, and 5 kW kg^{-1} at 18 GHz]. These levels have not been shown to adversely affect health in realistic exposure scenarios in humans and, given their very high thresholds, are protected against by restrictions based on effects with lower thresholds. Accordingly, electroporation is not discussed further.

In summary, there is no evidence of effects of radiofrequency EMFs on physiological processes that impair human health.

AUDITORY, VESTIBULAR, AND OCULAR FUNCTION

A number of animal and some human studies have tested for potential effects of radiofrequency EMFs on function and pathology of the auditory, vestibular, and ocular systems.

Sub-millisecond pulses of radiofrequency EMF can result in audible sound. Specifically, within the 200–3000

MHz EMF range, *microwave hearing* can result from brief (approximately 35–100 μs) radiofrequency pulses to the head, which cause thermoelastic expansion that is detected by sensory cells in the cochlea via the same processes involved in normal hearing. This phenomenon is perceived as a brief low-level noise, often described as a "click" or "buzzing." For example, Röschmann (1991) applied 10- and 20-μs pulses at 2.45 GHz that caused a specific energy absorption (SA) of 4.5 mJ kg^{-1} per pulse, and which was estimated to result in a temperature rise of approximately 0.00001°C per pulse. These pulses were barely audible, suggesting that this corresponded to a sound at the hearing threshold. Although higher intensity SA pulses may result in more pronounced effects, there is no evidence that microwave hearing in any realistic exposure scenarios can affect health, and so the present Guidelines do not provide a restriction to specifically account for microwave hearing.

Experimental and observational studies have also been conducted to test for adverse effects of EMF exposure from mobile phones. A few studies have investigated effects on auditory function and cellular structure in animal models. However, these results are inconsistent.

Beyond the behavioral and electrophysiological indices of sensory processing described above, a number of studies have tested for acute effects of radiofrequency EMF exposure on auditory, vestibular and ocular functioning in humans. These have largely been conducted using mobile phone-like signals at exposure levels below the ICNIRP (1998) basic restriction levels. Although there are some reports of effects, the results are highly variable with the larger and more methodologically rigorous studies failing to find such effects.

There is very little epidemiological research addressing sensory effects of devices that emit radiofrequency EMFs. The available research has focused on mobile phone use and does not provide evidence that this is associated with increased risk of tinnitus, hearing impairment, or vestibular or ocular function.

Animal studies have also reported that the heating that results from radiofrequency EMF exposure may lead to the formation of cataracts in rabbits. In order for this to occur, very high local SAR levels (100–140 W kg^{-1}) at low frequencies (< 6 GHz) are needed with temperature increases of several °C maintained for several hours. However, the rabbit model is more susceptible to cataract formation than in primates (with primates more relevant to human health), and cataracts have not been found in primates exposed to radiofrequency fields. No substantiated effects on other deep structures of the eye have been found (e.g., retina or iris). However, rabbits can be a good model for damage to superficial structures of the eye (e.g., the cornea) at higher frequencies (30–300 GHz). The baseline temperature of the cornea is relatively low compared with the posterior portion of the eye, and so very high exposure levels are required to cause harm superficially. For example, Kojima et al. (2018) reported that adverse health effects to the cornea can occur at incident power densities higher than 1.4 kW m^{-2} across frequencies from 40 to 95 GHz; no effects were found below 500 W m^{-2}. The authors concluded that the blink rates in humans (ranging from once every 3 to 10 s, as opposed to once every 5 to 20 min in rabbits) would preclude such effects in humans.

In summary, no reported effects on auditory, vestibular, or ocular function or pathology relevant to human health have been substantiated. Some evidence of superficial eye damage has been shown in rabbits at exposures of at least 1.4 kW m^{-2}, although the relevance of this to humans has not been demonstrated.

NEUROENDOCRINE SYSTEM

A small number of human studies have tested whether indices of endocrine system function are affected by radiofrequency EMF exposure. Several hormones, including melatonin, growth hormone, luteinizing hormone, cortisol, epinephrine, and norepinephrine have been assessed, but no consistent evidence of effects of exposure has been observed.

In animal studies, substantiated changes have only been reported from acute exposures with whole-body SARs in the order of 4 W kg^{-1}, which result in core temperature rises of 1°C or more. However, there is no evidence that this corresponds to an impact on health. Although there have been a few studies reporting field-dependent changes in some neuroendocrine measures, these have also not been substantiated. The literature, as a whole, reports that repeated, daily exposure to mobile phone signals does not impact on plasma levels of melatonin or on melatonin metabolism, oestrogen or testosterone, or on corticosterone or adrenocorticotropin in rodents under a variety of conditions.

Epidemiological studies on potential effects of exposure to radiofrequency EMFs on melatonin levels have reported conflicting results and suffer methodological limitations. For other hormonal endpoints, no epidemiological studies of sufficient scientific quality have been identified.

In summary, the lowest level at which an effect of radiofrequency EMFs on the neuroendocrine system has been observed is 4 W kg^{-1} (in rodents and primates), but there is no evidence that this translates to humans or is relevant to human health. No other reported effects have been substantiated.

NEURODEGENERATIVE DISEASES

No human experimental studies exist for adverse effects on neurodegenerative diseases.

Although it has been reported that exposure to pulsed radiofrequency EMFs increased neuronal death in rats, which could potentially contribute to an increased risk of

neurodegenerative disease, other studies have failed to confirm these results. Some other effects have been reported (e.g., changes to neurotransmitter release in the cortex of the brain, protein expression in the hippocampus, and autophagy in the absence of apoptosis in neurons), but such changes have not been shown to lead to neurodegenerative disease. Other studies investigating effects on neurodegeneration are not informative due to methodological or other shortcomings.

A Danish epidemiological cohort study has investigated potential effects of mobile phone use on neurodegenerative disorders and reported reduced risk estimates for Alzheimer disease, vascular and other dementia, and Parkinson disease (Schüz et al. 2009). These findings are likely to be the result of reverse causation, as prodromal symptoms of the disease may prevent persons with early symptoms to start using a mobile phone. Results from studies on multiple sclerosis are inconsistent, with no effect observed among men, and a borderline increased risk in women, but with no consistent exposure-response pattern.

In summary, no adverse effects on neurodegenerative diseases have been substantiated.

CARDIOVASCULAR SYSTEM, AUTONOMIC NERVOUS SYSTEM, AND THERMOREGULATION

As described above, radiofrequency EMFs can induce heating in the body. Although humans have a very efficient thermoregulatory system, too much heating puts the cardiovascular system under stress and may lead to adverse health effects.

Numerous human studies have investigated indices of cardiovascular, autonomic nervous system, and thermoregulatory function, including measures of heart rate and heart rate variability, blood pressure, body, skin and finger temperatures, and skin conductance. Most studies indicate that there are no effects on endpoints regulated by the autonomic nervous system. The relatively few reported effects of exposure were small and would not have an impact on health. The reported changes were also inconsistent and may be due to methodological limitations or chance. With exposures at higher intensities, up to a whole-body SAR of about 1 W kg^{-1} (Adair et al. 2001), sweating and cardiovascular responses have been reported that are similar to that observed under increased heat load from other sources. The body core temperature increase was generally less than 0.2°C.

The situation is different for animal research, in that far higher exposure levels have been used, often to the point where thermoregulation is overwhelmed, and temperature increases to the point where death occurs. For example, Frei et al. (1995) exposed rats to 35 GHz fields at 13 W kg^{-1} whole-body exposure, which raised body core temperature by 8°C (to 45°C), resulting in death. Similarly, Jauchem and Frei (1997) exposed rats to 350 MHz fields at 13.2 W kg^{-1} whole-body exposure and reported that thermal breakdown (i.e., where the thermoregulatory system can no longer cope with the increased body core temperature) occurred at approximately 42°C. It is difficult to relate these animal findings directly to humans, as humans are more-efficient thermoregulators than rodents. Taberski et al. (2014) reported that in Djungarian hamsters no body core temperature elevation was seen after whole-body exposure to 900 MHz fields at 4 W kg^{-1} with the only detectable effect a reduction of food intake (which is consistent with reduced eating in humans when body core temperature is elevated).

Few epidemiological studies on cardiovascular, autonomic nervous system, or thermoregulation outcomes are available. Those that are have not demonstrated a link between radiofrequency EMF exposure and measures of cardiovascular health.

In summary, no effects on the cardiovascular system, autonomic nervous system, or thermoregulation that compromise human health have been substantiated for exposures with whole-body average SARs below approximately 4 W kg^{-1}, with harm only found in animals exposed to whole-body average SARs substantially higher than 4 W kg^{-1}.

IMMUNE SYSTEM AND HAEMATOLOGY

There have been inconsistent reports of transient changes in immune function and haematology following radiofrequency EMF exposures. These have primarily been from in vitro studies, although some animal studies have also been conducted. These reports have not been substantiated.

The few human studies that have been conducted have not provided any evidence that radiofrequency EMFs affect health in humans via the immune system or haematology.

FERTILITY, REPRODUCTION, AND CHILDHOOD DEVELOPMENT

There is very little human experimental research addressing possible effects of radiofrequency EMF exposure on reproduction and development. What is available has focused on hormones that are relevant to reproduction and development, and as described in the Neuroendocrine System section above, there is no evidence that they are affected by radiofrequency EMF exposure. Other research has addressed this issue by looking at different stages of development (for endpoints such as cognition and brain electrical activity), in order to determine whether there may be greater sensitivity to radiofrequency fields as a function of age. There is currently no evidence that developmental phase is relevant to this issue.

Numerous animal studies have shown that exposure to radiofrequency EMFs associated with a significant temperature increase can cause effects on reproduction and development. These include increased embryo and fetal

losses, increased fetal malformations and anomalies, and reduced fetal weight at term. Such exposures can also cause a reduction in male fertility. However, extensive, well-performed studies have failed to identify developmental effects at whole-body average SAR levels up to 4 W kg^{-1}. In particular, a large four-generation study in mice on fertility and development using whole-body SAR levels up to 2.34 W kg^{-1} found no evidence of adverse effects (Sommer et al. 2009). Some studies have reported effects on male fertility at exposure levels below this value, but these studies have had methodological limitations and reported effects have not been substantiated.

Epidemiological studies have investigated various aspects of male and female infertility and pregnancy outcomes in relation to radiofrequency EMF exposure. Some epidemiological studies reported associations between radiofrequency EMFs and sperm quality or male infertility, but, taken together, the available studies do not provide evidence for an association with radiofrequency EMF exposure as they all suffer from limitations in study design or exposure assessment. A few epidemiological studies are available on maternal mobile phone use during pregnancy and potential effects on child neurodevelopment. There is no substantiated evidence that radiofrequency EMF exposure from maternal mobile phone use affects child cognitive or psychomotor development, or causes developmental milestone delays.

In summary, no adverse effects of radiofrequency EMF exposure on fertility, reproduction, or development relevant to human health have been substantiated.

CANCER

There is a large body of literature concerning cellular and molecular processes that are of particular relevance to cancer. This includes studies of cell proliferation, differentiation and apoptosis-related processes, proto-oncogene expression, genotoxicity, increased oxidative stress, and DNA strand breaks. Although there are reports of effects of radiofrequency EMFs on a number of these endpoints, there is no substantiated evidence of health-relevant effects (Vijayalaxmi and Prihoda 2019).

A few animal studies on the effect of radiofrequency EMF exposure on carcinogenesis have reported positive effects, but, in general, these studies either have shortcomings in methodology or dosimetry, or the results have not been verified in independent studies. Indeed, the great majority of studies have reported a lack of carcinogenic effects in a variety of animal models. A replication of a study in which exposure to radiofrequency EMFs increased the incidence of liver and lung tumors in an animal model with prenatal exposure to the carcinogen ENU (ethylnitrosourea) indicates a possible promoting effect (Lerchl et al. 2015; Tillmann et al. 2010). The lack of a dose-response relationship, as well as the use of an untested mouse model for liver and lung tumors whose relevance to humans is uncertain (Nesslany et al. 2015), makes interpretation of these results and their applicability to human health difficult, and, therefore, there is a need for further research to better understand these results.

Two recent animal studies investigating the carcinogenic potential of long-term exposure to radiofrequency EMFs associated with mobile phones and mobile phone base stations have also been released: one by the U.S. National Toxicology Program (NTP 2018a and b) and the other from the Ramazzini Institute (Falcioni et al. 2018). Although both studies used large numbers of animals, best laboratory practice, and exposed animals for the whole of their lives, they also have inconsistencies and important limitations that affect the usefulness of their results for setting exposure guidelines. Of particular importance is that the statistical methods employed were not sufficient to differentiate between radiofrequency-related and chance differences between treatment conditions; interpretation of the data is difficult due to the high body core temperature changes that resulted from the very high exposure levels used; and no consistency was seen across these two studies. Thus, when considered either in isolation (e.g., ICNIRP 2019) or within the context of other animal and human carcinogenicity research (HCN 2014, 2016), their findings do not provide evidence that radiofrequency EMFs are carcinogenic.

A large number of epidemiological studies of mobile phone use and cancer risk have also been performed. Most have focused on brain tumors, acoustic neuroma and parotid gland tumors, as these occur in close proximity to the typical exposure source from mobile phones (Röösli et al. 2019). However, some studies have also been conducted on other types of tumors, such as leukaemia, lymphoma, uveal melanoma, pituitary gland tumors, testicular cancer, and malignant melanoma. With a few exceptions, the studies have used a case-control design and have relied on retrospectively collected self-reported information about mobile phone use history. Only two cohort studies with prospective exposure information are available. Several studies have had follow-ups that were too short to allow assessment of a potential effect of long-term exposure, and results from case-control studies with longer follow-up are not consistent.

The large Interphone study, coordinated by the International Association for Research on Cancer, did not provide evidence of a raised risk of brain tumors, acoustic neuroma, or parotid gland tumors among regular mobile phone users, and the risk estimates did not increase with longer time since first mobile phone use (Interphone 2010, 2011). It should be noted that although somewhat elevated odds ratios were observed at the highest level of cumulative call time for acoustic neuroma and glioma, there were no trends observed for any of the lower cumulative call

time groups, with among the lowest risk estimates in the penultimate exposure category. This, combined with the inherent recall bias of such studies, does not provide evidence of an increased risk. Similar results were observed in a Swedish case-control study of acoustic neuroma (Pettersson et al. 2014). Contrary to this, a set of case-control studies from the Hardell group in Sweden report significantly increased risks of both acoustic neuroma and malignant brain tumors already after less than five years since the start of mobile phone use, and at quite low levels of cumulative call time. However, they are not consistent with trends in brain cancer incidence rates from a large number of countries or regions, which have not found any increase in the incidence since mobile phones were introduced.

Furthermore, no cohort studies (which unlike case-control studies are not affected by recall or selection bias) report a higher risk of glioma, meningioma, or acoustic neuroma among mobile phone subscribers or when estimating mobile phone use through prospectively collected questionnaires. Studies of other types of tumors have also not provided evidence of an increased tumor risk in relation to mobile phone use. Only one study is available on mobile phone use in children and brain tumor risk (Aydin et al. 2011). No increased risk of brain tumors was observed.

Studies of exposure to environmental radiofrequency EMFs, for example from radio and television transmitters, have not provided evidence of an increased cancer risk either in children or in adults. Studies of cancer in relation to occupational radiofrequency EMF exposure have suffered substantial methodological limitations and do not provide sufficient information for the assessment of carcinogenicity of radiofrequency EMFs. Taken together, the epidemiological studies do not provide evidence of a carcinogenic effect of radiofrequency EMF exposure at levels encountered in the general population.

In summary, no effects of radiofrequency EMFs on the induction or development of cancer have been substantiated.

SUMMARY

The only substantiated adverse health effects caused by exposure to radiofrequency EMFs are nerve stimulation, changes in the permeability of cell membranes, and effects due to temperature elevation. There is no evidence of adverse health effects at exposure levels below the restriction levels in the ICNIRP (1998) guidelines and no evidence of an interaction mechanism that would predict that adverse health effects could occur due to radiofrequency EMF exposure below those restriction levels.

REFERENCES

Adair ER, Mylacraine KS, Cobb BL. Human exposure to 2450 MHz CW energy at levels outside the IEEE C95.1 standard does not increase core temperature. Bioelectromagnetics 22:429–439; 2001.

Aydin D, Feychting M, Schüz J, Tynes T, Andersen TV, Schmidt LS, Poulsen AH, Johansen C, Prochazka M, Lannering B, Klæboe L, Eggen T, Jenni D, Grotzer M, Von der Weid N, Kuehni CE, Röösli M. Mobile phone use and brain tumors in children and adolescents: a multicenter case-control study. J National Cancer Inst 103:1264–1276; 2011.

Chatterjee I, Wu D, Gandhi OP. Human body impedance and threshold currents for perception and pain for contact hazard analysis in the VLF-MF band. IEEE Trans Biomed Engineer 33:486–494; 1986.

Eltiti S, Wallace D, Russo R, Fox E. Symptom presentation in idiopathic environmental intolerance with attribution to electromagnetic fields: evidence for a nocebo effect based on data re-analyzed from two previous provocation studies. Frontiers Psychol 9:1563; 2018.

Falcioni L, Bua L, Tibaldi E, Lauriola M, De Angelis L, Gnudi F, Mandrioli D, Manservigi M, Manservisi F, Manzoli I, Menghetti I, Montella R, Panzacchi S, Sgargi D, Strollo V, Vornoli A, Belpoggi F. Report of final results regarding brain and heart tumors in Sprague-Dawley rats exposed from prenatal life until natural death to mobile phone radiofrequency field representative of a 1.8 GHz GSM base station environmental emission. Environment Res 165:496–503; 2018.

Frei MR, Ryan KL, Berger RE, Jauchem JR. Sustained 35-GHz radiofrequency irradiation induces circulatory failure. Shock 4:289–293; 1995.

Health Council of the Netherlands. Mobile phones and cancer: part 2. Animal studies on carcinogenesis. The Hague: Health Council of the Netherlands; Publication 22; 2014.

Health Council of the Netherlands. Mobile phones and cancer: part 3. Update and overall conclusions from epidemiological and animal studies. The Hague: Health Council of the Netherlands; Publication 06; 2016.

International Commission on Non-Ionizing Radiation Protection. Guidelines for limiting exposure to time-varying electric, magnetic and electromagnetic fields (up to 300 GHz). Health Phys 74:494–522;1998.

International Commission on Non-Ionizing Radiation Protection. ICNIRP note: critical evaluation of two radiofrequency electromagnetic field animal carcinogenicity studies published in 2018. Health Phys 118(5):525–532; 2020.

Interphone Study Group. Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. International J Epidemiol 39:675–694; 2010.

Interphone Study Group. Acoustic neuroma risk in relation to mobile telephone use: results of the INTERPHONE international case–control study. Cancer Epidemiol 35:453–464; 2011.

Jauchem JR, Frei MR. Body heating induced by sub-resonant (350 MHz) microwave irradiation: cardiovascular and respiratory responses in anesthetized rats. Bioelectromagnetics 18: 335–338; 1997.

Joshi RP, Schoenbach KH. Bioelectric effects of intense ultrashort pulses. Critical Rev Biomed Engineer 38:255–304; 2010.

Kojima M, Susuki Y, Sasaki K, Taki M, Wake K, Watanabe S, Mizuno M, Tasaki T, Sasaki H. Ocular effects of exposure to 40, 75 and 95 GHz Millimeter Waves. J Infrared, Millimeter and Terahertz Waves. 39(9):912–925; 2018.

Lerchl A, Klose M, Grote K, Wilhelm AF, Spathmann O, Fiedler T, Streckert J, Hansen V, Clemens M. Tumor promotion by exposure to radiofrequency electromagnetic fields below exposure limits for humans. Biochem Biophys Res Comm 459: 585–590; 2015.

Loughran SP, McKenzie RJ, Jackson ML, Howard ME, Croft RJ. Individual differences in the effects of mobile phone exposure

on human sleep: rethinking the problem. Bioelectromagnetics 33:86–93; 2012.

Nagaoka T, Watanabe S. Development of voxel models adjusted to ICRP reference children and their whole-body SARs for whole-body exposure to electromagnetic fields from 10 MHz to 6 GHz. IEEE Access 7:135909–135916; 2019.

Nesslany F, Aurengo A, Bonnet-Belfais M, Lambrozo J. Comment on Lerchl study: "Tumor promotion in mice by exposure to radiofrequency electromagnetic fields still waiting evidence." Biochem Biophys Res Comm 467:101–102; 2015.

Nguyen THP, Pham VTH, Baulin V, Croft RJ, Crawford RJ, Ivanova EP. The effect of a high frequency electromagnetic field in the microwave range on red blood cells. Sci Rep 7:1–10; 2017.

Nittby H, Brun A, Eberhardt J, Malmgren L, Persson BR, Salford LG. Increased blood-brain barrier permeability in mammalian brain 7 days after exposure to the radiation from a GSM-900 mobile phone. Pathophysiol 6:103–112; 2009.

National Toxicology Program. Technical report on the toxicology and carcinogenesis studies in Hsd:Sprague Dawley SD rats exposed to whole-body radio frequency radiation at a frequency (900 MHz) and modulations (GSM and CDMA) used by cell phones. National Toxicology Program. Research Triangle Park, NC: NTP TR 595; 2018a.

National Toxicology Program. Technical report on the toxicology and carcinogenesis studies in B6C3F1/N mice exposed to whole-body radio frequency radiation at a frequency (1900 MHz) and modulations (GSM and CDMA) used by cell phones. National Toxicology Program; NTP TR 596; 2018b.

Pettersson D, Mathiesen T, Prochazka M, Bergenheim T, Florentzson R, Harder H, Feychting M. Long-term mobile phone use and acoustic neuroma risk. Epidemiol 25:233–241; 2014.

Röösli M, Lagorio S, Schoemaker MJ, Schüz J, Feychting M. Brain and salivary gland tumors and mobile phone use: evaluating the evidence from various epidemiological study designs. Annual Rev Public Health 40:221–238; 2019.

Röschmann P. Human auditory system response to pulsed radiofrequency energy in RF coils for magnetic resonance at 2.4 to 170 MHz. Magnetic Resonance Med 21:197–215; 1991.

Scientific Committee on Emerging and Newly Identified Health Risks. Potential health effects of exposure to electromagnetic fields (EMF). Luxembourg: SCENIHR; 2015.

Schüz J, Waldemar G, Olsen JH, Johansen C. Risks for central nervous system diseases among mobile phone subscribers: a Danish retrospective cohort study. PLoS One 4:e4389; 2009. DOI 10.1371/journal.pone.0004389.

Sommer AM, Grote K, Reinhardt T, Streckert J, Hansen V, Lerchl A. Effects of radiofrequency electromagnetic fields (UMTS) on reproduction and development of mice: a multi-generation study. Radiat Res 171:89–95; 2009.

SSM. SSM's Scientific Council on Electromagnetic Fields. Recent research on EMF and health risk—tenth report from SSM's Scientific Council on Electromagnetic Fields. Luxembourg: Publication 19; 2015.

SSM. SSM's Scientific Council on Electromagnetic Fields. Recent research on EMF and health risk—eleventh report from SSM's Scientific Council on Electromagnetic Fields. Stockholm: SSM; Publication 15; 2016.

SSM. SSM's Scientific Council on Electromagnetic Fields. recent Research on EMF and health risk—twelfth report from SSM's Scientific Council on Electromagnetic Fields. Publication 09; 2018.

Taberski K, Klose M, Grote K, El Ouardi A, Streckert J, Hansen VW, Lerchl A. Noninvasive assessment of metabolic effects of exposure to 900 MHz electromagnetic fields on Djungarian Hamsters (Phodopus sungorus). Radiat Res 181:617–622; 2014.

Tillmann T, Ernst H, Streckert J, Zhou Y, Taugner F, Hansen V, Dasenbrock C. Indication of cocarcinogenic potential of chronic UMTS-modulated radiofrequency exposure in an ethylnitrosourea mouse model. International J Radiat Biol 86:529–41; 2010.

Torebjork HE, LaMotte RH, Robinson CJ. Peripheral neural correlates of magnitude of cutaneous pain and hyperalgesia: simultaneous recordings in humans of sensory judgments of pain and evoked responses in nociceptors with C-fibers. J Neurophysiol 51:325–339; 1984.

Verrender A, Loughran SP, Dalecki A, Freudenstien F, Croft RJ. Can explicit suggestions about the harmfulness of EMF exposure exacerbate a nocebo response in healthy controls? Environ Res 166:409–417; 2018.

Vijayalaxmi, Prihoda TJ. Comprehensive review of quality of publications and meta-analysis of genetic damage in mammalian cells exposed to non-ionising radiofrequency fields. Radiat Res 191:20–30; 2019.

Walters TJ, Blick DW, Johnson LR, Adair ER, Foster KR. Heating and pain sensation produced in human skin by millimetre waves: comparison to a simple thermal model. Health Phys 78:259–267; 2000.

World Health Organization. Radiofrequency fields; Public Consultation Document, released October 2014. Geneva: WHO; 2014.

■■