Time-temperature Thresholds and Safety Factors for Thermal Hazards from Radiofrequency Energy above 6 GHz

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Abstract—Two major sets of exposure limits for radiofrequency (RF) radiation, those of the International Commission on Nonionizing Radiation Protection (ICNIRP 2020) and the Institute of Electrical and Electronics Engineers (IEEE C95.1–2019), have recently been revised and updated with significant changes in limits above 6 GHz through the millimeter wave (mm-wave) band (30–300 GHz). This review compares available data on thermal damage and pain from exposure to RF energy above 6 GHz with corresponding data from infrared energy and other heat sources and estimates safety factors that are incorporated in the IEEE and ICNIRP RF exposure limits. The benchmarks for damage are the same as used in ICNIRP IR limits: minimal epithelial damage to cornea and first-degree burn (erythema in skin observable within 48 h after exposure). The data suggest that limiting thermal hazard to skin is cutaneous pain for exposure durations less than \( \approx 20 \) min and thermal damage for longer exposures. Limitations on available data and thermal models are noted. However, data on RF and IR thermal damage and pain thresholds show that exposures far above current ICNIRP and IEEE limits would be required to produce thermally hazardous effects. This review focuses exclusively on thermal hazards from RF exposures above 6 GHz to skin and the cornea, which are the most exposed tissues in the considered frequency range.

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INTRODUCTION

As wireless communications develops, there has been a rapidly increasing use of radiofrequency (RF) energy at frequencies above 6 GHz, including by some 5G cellular telephone systems and, in the near future, many IoT (Internet of Things) devices. Consequently, the safety of RF energy in previously little-used (by consumer devices) bands has become a matter of increased public and regulatory attention. Recently, two major international exposure limits, ICNIRP (2020) and IEEE C95.1–2019, have been updated and revised at frequencies above 6 GHz (Table 1).

This review considers thresholds for two identified hazards: (1) thermal damage to skin and ocular tissues and thermal pain and (2) estimates safety factors for IEEE and ICNIRP exposure limits in this frequency range. To avoid misunderstanding, reported “nonthermal” effects of RF energy are important to consider but are beyond the scope of the present review.

The limits are complex, and readers are referred to the guideline documents. Above 6 GHz and particularly in the millimeter wave (mm-wave) range (30–300 GHz), incident RF energy is absorbed close to the surface of the body (Table 2), and the anticipated potential hazards are associated with excessive heating of skin and cornea (Alekseev et al. 2019). The limits distinguish (using different terminology) between exposures measured in air outside the body (limits are called reference levels in ICNIRP and Exposure Reference Levels or ERLs in IEEE) and limits on power absorbed in the body (called basic restrictions in ICNIRP and Dosimetric Reference levels or DRLs in the IEEE standards). Table 1 compares reference levels/ERLs for the limits but avoids a number of qualifications and notes, for which the reader is referred to the guideline/standard documents themselves.

Extensive information is available about thresholds for thermal damage and pain from many studies going back a half century or more. However, there is a paucity of data on thermal hazards from RF exposure in the considered frequency range. Consequently, both IEEE and ICNIRP limits are largely based on electromagnetic and thermal modeling studies.

Thermal hazards, including pain and tissue damage, are characterized in terms of tissue temperature vs. time relations. ICNIRP (2020) states, “The present guidelines treat radiofrequency EMF [electromagnetic field] exposure that results in local temperatures of 41 °C or greater as potentially dangerous.”
harmful” and defines “operational adverse effects thresholds” (i.e., assumed thresholds for hazards) of 5 °C (type 1) or 2 °C (type 2) tissues. These tissues are distinguished, apart from anatomical location, by different normothermal temperatures, typically <33–36 °C (Type 1) and <38.5 °C (Type 2), respectively. Consequently, ICNIRP (2020) is implicitly designed to limit tissue temperature increases to 2°C or 5°C, to maintain tissue temperatures below 41°C. IEEE C95.1–2019 (p. 75) designed RF exposure limits above 6 GHz to provide similar safety factors as in the standard at lower frequencies without specifying maximum temperature increases. In contrast to ICNIRP limits for infrared energy (which are defined for limited exposure durations), ICNIRP and IEEE limits for RF energy are stated without limitation on exposure duration, environmental conditions, or work intensity.

This review compares available data on thermal damage and pain from exposure to RF energy above 6 GHz with corresponding data from other heat sources and estimates safety factors that are incorporated in the IEEE and ICNIRP RF exposure limits. The benchmarks for damage are the same as used in ICNIRP IR limits: (1) minimal epithelial damage to cornea and (2) first-degree burn (erythema in skin observable within 48 h after exposure). The present review extends thermal models for skin from Foster et al. (2016, 2018, 2020).

**ARRHENIUS MODEL FOR THERMAL HAZARDS**

The standard model for thermal damage to tissues derives from studies in the late 1940s by Henriques (1947) and colleagues. These authors adapted the theory developed by Svante Arrhenius in 1889 for the temperature dependence of chemical rates and used it to describe empirically the rates of thermal damage to biological materials (Pearce 2011, 2013).

This model defines a thermal damage index $\Omega$ as the integral of an exponential function (representing the rate of damage) over the duration of exposure

\[ \Omega(T, t) = \frac{1}{\Delta E A} \int_0^t \Delta T dt, \]  

where $\tau$ is time, and the integral is evaluated for all times at which the tissue temperature is above baseline (pre-exposure) levels. $T(t)$ is the tissue temperature ($^\circ$K) at the point of evaluation, $R$ is the ideal gas constant, and $A$ (s$^{-1}$) and $\Delta E$ (J/mol) are empirically determined from thermal damage data, choosing $A$ so that $\Omega = 1$ corresponds to a benchmark for injury (termed isoeffect in the discussion below). Thermal damage data can be summarized by plotting temperature vs. duration of exposure that results in the benchmark damage (isoeffect). For constant-temperature exposure, eqn (1) results in a straight line when plotted on a semilogarithmic scale.

The benchmark measure of damage is typically taken as the $ED_{50}$ (the incident or absorbed power density resulting in 50% probability of a specific damage endpoint), but in some studies, it may be the lowest temperature below which no

### Table 1. ICNIRP (2020) and IEEE C95.1–2019 and limits compared.a

| Frequency range, GHz | Incident power density, W m$^{-2}$ | Averaging area, cm$^2$ | Averaging time, min | Fluence limit for pulses, kJ m$^{-2}$ |
|---------------------|-------------------------------|---------------------|-------------------|---------------------------------|
| FCC                 |                               |                     |                   |                                 |
| ICNIRP (2020)       |                               |                     |                   |                                 |
| Whole body exposure | 1.5–100                       | 2                  |                   |                                 |
| Local exposure      | 2–300                         | 50                 | (whole body)      | 30 (varies with pulse width)    |
| 6–300               | Decrease from 200 at 6 GHz to 100 at 300 GHz$^2$ | 4                | 6                 | (varies with pulse width)       |
| 30–300              | Decrease from 400 at 6 GHz to 200 at 300 GHz | 1              | 6                 |                                 |
| IEEE C95.1 (2019)   | Whole body exposure           |                     |                   |                                 |
| Local exposure      | 2–300                         | 50                 | (whole body)      | 30                              |
| 6–300               | Decrease from 200 at 6 GHz to 100 at 300 GHz$^2$ | 4                | 6                 | $t_{p}^{1/2}$ (30–300 GHz)      |
| 30–300              | Decrease from 400 at 6 GHz to 200 at 300 GHz | 1              | 6                 |                                 |

*aOccupational limits (ICNIRP terminology) or limits for restricted environments (IEEE terminology). Limits for the general public (ICNIRP) or for unrestricted environments (IEEE) are 20% of occupational limits. $T_p$ is pulse width in s.

### Table 2. Power penetration depth calculated for dry skin.a

| Frequency (GHz) | $L$ (mm) dry skin |
|----------------|-------------------|
| 6              | 4.09              |
| 10             | 1.9               |
| 30             | 0.43              |
| 94             | 0.19              |
| 300            | 0.14              |

*aAdapted from Foster et al. (2017, 2020).*
damage is observed. In tissue damage studies, the parameter $\Delta E_a$ is frequently referred to as the “activation energy” as used in the original Arrhenius reaction rate theory. However, thermal damage in tissues may involve multiple reactions that vary in relative contribution at different temperatures, and $\Delta E_a$ is an empirical value representing this mix of reactions.

Equation (1) can be restated by substituting $\tau' = \tau/t_0$, where $t_0$ is a scaling constant (presently taken as 1 s). Then eqn (1) has the form

$$
\Omega(T, t) = \int_0^{t_0} Ae^{-\frac{\Delta E}{RT}} dt' = \int_0^{t_0} Ae^{-\frac{\Delta E}{RT}(t_0)} t_0 d\tau'.
$$

(1a)

It is possible to express the exponent in a Taylor series about a “critical temperature” $T_{crit}$ at which the exponent vanishes. The derivative of (Eq. 1a) with respect to $t_0$ is:

$$
\frac{d\Omega(T, \tau)}{d(\tau)} \bigg|_{t_0} = At_0 e^{-\frac{\Delta E}{RT}} = e^{\ln A t_0 - \frac{\Delta E}{RT}}.
$$

Setting

$$
\frac{d\Omega(T, \tau)}{d(\tau)} \bigg|_{t_0} = 1
$$

for $T_{crit}$ and taking its logarithm, the following relation is found:

$$
T_{crit} = \frac{\Delta E_a}{R \ln A t_0}.
$$

Using this last equation, $d\Omega(T, t)/dt$ can be cast in the form:

$$
\frac{d\Omega(T, t)}{dt} = e^{\alpha(T(t) - T_{ref})}
$$

(2)

with

$$
T_{crit} = \frac{\Delta E_a}{R \ln A t_0} - 273.16 \, (^{oC})
$$

(3)

as the critical temperature and

$$
\alpha = \frac{\ln(A t_0)}{T_{ref} + 273.16K} \, (K^{-1})
$$

(4)

as a slope factor. The exponent in eqn (2) departs from $(\ln(A t_0) \Delta E/RT)$ by less than 5% between 40–70 °C. For constant temperature exposures, the thermal damage rate becomes 1 s$^{-1}$ at $T_{crit}$ and a 1 degree increase in temperature results in an increase in damage rate by a factor of $\exp(\alpha)$.

Dewhirst et al. (2003) note that the apparent activation energy $\Delta E_a$ found in tissue damage studies is not constant with temperature but often shows a change at about 47 °C for human tissues and at about 42.5 °C for murine tissues. Thromborelase (a protective response) sets in after a few minutes’ exposure to temperatures above about 41–43 °C, reducing its susceptibility to thermal damage (Sapareto 1987).

### Constant Temperature vs. Constant Power Exposures

Two paradigm cases of exposure are constant temperature and constant power exposures.

#### Constant temperature

The temperature of the target tissue is assumed to be instantly and uniformly raised to a final temperature $T_o$ and held constant for time $\Delta t$. For constant temperature exposures at $T_o$, the time $\Delta t$ to reach the isoeffect ($\Omega = 1$) follows from eqn (2):

$$
\Delta t = A^{-1} e^{\frac{\Delta E_a}{R t_o}}.
$$

(5)

#### Constant power

In this case, the power input (i.e., RF exposure) is constant, and $T(t)$ varies during exposure. To evaluate eqn (1) or (2) for this case, $T(t)$ must be found, either experimentally or by solution of an appropriate thermal model for the tissue.

In either case, $T(t)$ must be determined at the point at which damage is assessed to establish the parameters $A$ and $\Delta E_a$. Correction for the time dependence of $T(t)$ is important when comparing results of different methods of thermal exposure; e.g., heating of the skin with radiant energy vs. heated water flowing across the skin surface vs. a heated surface placed in contact with skin.

### Reference Models

In the discussion below, thermal damage data will be compared to thermal damage models for skin, cornea, and retina developed by Jean et al. (2013, 2021) and Jean and Schulmeister (2017). These studies were done to inform development of ICNIRP exposure limits for pulsed infrared (IR) radiation (ICNIRP 2013a and b), in part to address potentially serious hazards from pulsed IR lasers. For this purpose, Jean et al. analyzed large amounts of thermal damage data to develop predictive models in the parameters $A$ and $\Delta E_a$. The benchmark for damage in each case corresponded to reversible thermal damage (erythema for skin observable at various times, usually 24 h, after exposure and minimally visible ocular damage for cornea and retina). These became the benchmarks for damage used to develop ICNIRP guidelines for IR exposure.

This model development involved three stages: quantifying energy absorbed in the target tissue from an incident beam (dosimetry); determining the resulting time-dependent temperature $T(t)$ at the site of injury (thermal modeling); and fitting the observed damage rate to eqn (1). Jean et al. synthesized data from many different individual experiments in multiple...
The thermal damage models resulting from this work predicted experimentally observed thresholds to within a factor of about 1.5 to 2.

The three studies are:

- Jean et al. (2013) fitted 93 sets of thermal damage data measured in vivo from pulsed IR energy to pig and human skin over a wide variety of exposure parameters and wavelengths. The exposure durations ranged from 350 μs to 70 s and wavelengths from 500 nm to 10.6 μm. The isoeffect (Ω = 1) was the ED₅₀ for mild erythema, i.e., a minimal level of damage to the dermis. The authors state that their analysis was “optimized in order to fit all available thermally-induced threshold injury data and validated so that it can be used for quantitative risk analysis.”

- Jean and Schulmeister (2017) fitted “all consistent experimental results published over the past 50 years” for thermal damage to retinas of nonhuman primates from pulsed IR energy, with the benchmark of “minimal visible lesion.” The wavelength ranged from 413 to 1,338 nm (a range in which the outer ocular media are relatively transparent and most of the incident radiation is absorbed in the retina), and exposure durations ranged between 100 μs and 50 min. The model was fitted to 253 ED₅₀ values from 31 studies, spanning a range of exposure durations from 100 μs through 3,000 s. The predicted threshold exposures for thermal damage over this wide range of exposure parameters agreed with experimental data with a standard deviation of 31%, a level of accuracy that authors considered “cannot be improved much further due to experimental uncertainties and intersubject variability.”

- Jean et al. (2021) fitted 169 sets of in vivo data on corneal damage in rabbits and rhesus monkeys exposed to pulsed IR radiation, with pulse duration from 1.7 ns to 100 s and wavelengths between 1,050 nm to 10.6 μm (where most incident energy is absorbed close to the surface of the eye). The benchmark for damage (ED₅₀) was “minimal visible lesion with a slit lamp.”

Table 3 summarizes the parameters of these three models. The differences among these models may reflect in part intrinsic differences in thermal sensitivity of these tissues and in part different benchmarks used to assess damage thresholds.

**EXPERIMENTAL THERMAL PAIN AND BURN THRESHOLDS**

Only scant thermal damage data are available from RF exposures to tissue in the considered frequency range. However, a wealth of other thermal damage data is available from tissue damage studies involving exposure to infrared energy [summarized in models by Jean et al. (2013, 2021), Jean and Schulmeister (2017)] as well as from other thermal damage studies.

**OCULAR TISSUES**

Chalfin et al. (2002) exposed corneas of rhesus monkeys to 1–4 s pulses of RF energy at 94 GHz and reported a threshold (ED₅₀) pulse fluence of approximately 50 kJ m⁻² for minimally detectable thermal lesions. The corresponding peak corneal temperature at the ED₅₀ for minimally detectable damage (Fig. 6 of Chalfin et al. 2002) was approximately 60 °C, which is approximately 4 °C higher than predicted by Jean’s Arrhenius model for cornea. The reported temperature increases were maximum values measured by infrared thermography at any location on the surface of the cornea, as opposed to the average temperature increase measured across the corneal surface. In these experiments, the RF-induced temperature increases varied across the corneal surface due to wave reflections from the ocular adnexa including eyelids (Foster et al. 2021), leading to considerable variability in peak temperature increases as related to the incident power density on the cornea.

Kojima et al. (2009, 2015, 2018, 2020) measured thresholds for “slight ocular damage” to the corneal epithelium in groups of 3–15 rabbits exposed to RF energy at 18–162 GHz for 6- or 30-min periods. Corneal temperatures were measured thermographically before and shortly after exposure. After 6 min of exposure, the peak temperatures at isoeffect (ED₅₀ for minimal damage) ranged from 40.3–43.1 °C, as estimated from Fig. 6 in Kojima et al. (2020) and previous papers by those authors. After 30 min of exposure, the lowest peak corneal surface temperature resulting in a lesion was 37.1 °C. These thresholds (ED₅₀ values) are a few degrees below thresholds reported for lens by Guy et al. (1975) and below the expected temperatures from the Arrhenius corneal model.

The reasons for these unexpectedly low thresholds are not clear. In Kojima’s studies, the animals were prevented from blinking by anesthesia during the 6- or 30-min exposures, and some level of corneal desiccation may have occurred despite irrigation of the eyes with saline during exposure. In any event, the RF exposures ranged from several kW m⁻² for 6 min to >0.5 kW m⁻² for 30 min, which far exceed IEEE or ICNIRP exposure limits, and the exposure levels were sufficient to cause facial burns in addition to corneal damage.

Fig. 1 also shows thresholds for thermal damage in rabbit lenses from exposure to 2.45 GHz radiation (Guy et al. 1975). The exposure assessment in that study was exceptionally complete even by present-day standards, and this was one of several eye-damage studies evaluated in developing earlier editions of the C95.1 exposure limit (see Elder...
2003 and Sec. C.6.4.1 of IEEE C95.1–2019). The exposure levels used in that study (1–3 kW m\(^{-2}\) for up to an hour) far exceed present and earlier IEEE and ICNIRP limits and were sufficient to cause facial burns as well. Van Rhoon (2013) cited the study as showing the adverse effect of heat in a tissue reported at the lowest thermal dose.

### Table 3. Arrhenius models for thermal damage to skin, cornea, and retinas.

| Model/ range of data fitted | \(A(s^{-1})\) | \(\Delta E/R\) (K) | \(\alpha\), K\(^{-1}\) | \(T_{crit}\), oC | Factor increase in damage rate \(d\Omega/dt\) for 1 oC increase in temperature (eqn 2) |
|-----------------------------|----------------|-----------------|----------------|----------------|-----------------------------------|
| Skin                        |                |                 |                 |                |                                   |
| (Jean et al. 2013)          |                |                 |                 |                |                                   |
| Visually assessed           |                |                 |                 |                |                                   |
| minor injury (erythema)      |                |                 |                 |                |                                   |
| to pig and human skin       |                |                 |                 |                |                                   |
| without blister, chiefly    |                |                 |                 |                |                                   |
| from pulsed IR laser radiation. | 8.8 \(\times 10^{32}\)  | 63,000          | 0.58            | 56.7           | 1.8                               |
| Cornea                      |                |                 |                 |                |                                   |
| (Jean et al. 2021)          |                |                 |                 |                |                                   |
| Minimal visible damage to   |                |                 |                 |                |                                   |
| cornea of nonhuman primates|                |                 |                 |                |                                   |
| pulsed IR laser radiation.  |                |                 |                 |                |                                   |
| 1.7 ns-100 s                | 1.8 \(\times 10^{37}\) | 59,000          | 0.54            | 58.5           | 1.7                               |
| Retina                      |                |                 |                 |                |                                   |
| (Jean and Schulmeister, 2017)|                |                 |                 |                |                                   |
| Minimal visible damage to   |                |                 |                 |                |                                   |
| retina of nonhuman primates |                |                 |                 |                |                                   |
| pulsed IR laser radiation.  |                |                 |                 |                |                                   |
| 350 \(\mu\)s-50 min         | 1.1 \(\times 10^{35}\) | 72,000          | 0.66            | 55.9           | 1.9                               |

Fig. 1. Temperature-time relations for reversible damage to ocular tissues from RF energy. Studies are Guy et al. (1975; 2.45 GHz, visible cataract, single observations), Chalfin et al. (2002), corneal surface temperatures from single 3-s exposure at 94 GHz; Kojima et al. (2009, 2015, 2018, 2020), \(ED_{50}\) for minimal corneal epithelial damage from 6-min exposures at 40, 75, 95, 162 GHz and from 30-min exposures at 75 GHz. Also shown are thresholds for corneal thermal pain measured in four human subjects by Beuerman and Tanelian (1979). Dotted/dashed lines represent exposures in Arrhenius models for \(ED_{50}\) for minimal visible damage to retina and cornea from infrared energy (Jean and Schulmeister, 2017, Jean et al., 2021). The upper exposure durations considered in developing the Arrhenius models are indicated by the short vertical bars on the Arrhenius curves.
Fig. 1 also shows thresholds for corneal pain (Beuerman and Tanelian 1979). In that study, 2-s jets of temperature-controlled saline were directed from a distance of 2–3 mm against the corneas in six human subjects. The investigators measured the temperature of the saline incident on the corneas, not the corneal temperatures themselves. The thresholds for “irritating” and “painful” sensation reported by the subjects were 3–6 °C and 9–11 °C above the baseline corneal temperature of 33 °C. These pain thresholds are roughly 10 °C below thermal damage thresholds as inferred from the Arrhenius model for the cornea (Jean et al. 2021).

**SKIN**

Very few data could be located for thermal damage to skin from RF energy in the presently considered frequency range.

Parker et al. (2016) exposed six subjects on their lower backs/buttocks to a single 3-s pulse of 94 GHz at an incident power density of 40 or 60 kW m$^{-2}$. Two subjects developed first-degree burns (superficial burns that affect the epidermis only), with peak induced skin temperatures of 50.9 and 59.3 °C (19.9 or 25 °C above baseline skin temperatures, which varied somewhat between subjects). A subsequent case report by Gibbons (2017) described burns to a single male subject who had received a total of 16 exposures (!) to 1- to 1.3-s pulses of 95 GHz radiation at an incident power density of 30–120 kW m$^{-2}$. That subject developed heat urticaria, which the investigators described as “raised, erythematous, nonpruritic, nonpainful areas” at “six exposure sites where the skin temperature exceeded 54 °C.”

Figs. 1 and 2 allow a rough comparison of the thermal damage thresholds from exposure to RF energy with the respective Arrhenius models. The damage thresholds (rabbit lens, cornea) reported by Guy et al. (1975) and by Kojima et al. (2009, 2015, 2018, 2020) are somewhat below predictions of the Arrhenius models for cornea and retina (Fig. 1). The damage thresholds (rabbit lens) were unavailable. The data from Parker et al. (2016) and Gibbons (2017) for skin damage agree roughly with the skin Arrhenius model. However, these studies were very limited (each point in Fig. 2 represents one data point from a single human subject).

**CUTANEOUS THERMAL PAIN**

Bakkers et al. (2013) provided a systematic review of thermal pain testing; Zhang and Hedge (2017) reviewed thermal pain thresholds from contact with warm surfaces of electronic devices; and Filingeri (2011) reviewed the neurophysiology of skin thermal sensations.

Pain sensations are experienced when excess heat or pressure impinges on a nociceptor, which is a terminal ending of a sensory nerve that transduces the noxious stimulus into an electrical signal that is transmitted by a sensory neuron to the brain. Nociceptors are found in many parts of the body including the cornea; sensory nerves extend into the epidermal layer of the skin, which is the layer of skin with the highest absorbed energy per volume of tissue. The density of nociceptors varies greatly with location on the body leading to a variation in pain sensitivity across the body surface, particularly from thermal exposures to small skin areas.

Two types of pain can be distinguished: sharp acute pain that is carried by A$\delta$ neurons and dull throbbing pain that is carried by C neurons. The former neurons are insulated with myelin and have a relatively high conduction velocity of 4–30 m s$^{-1}$. They have a lower threshold to pain than C neurons and react rapidly to high-risk situations, such as touching a hot stove. C neurons are unmyelinated, have much lower conduction velocities of 0.4–1.8 m s$^{-1}$, and are best able to respond to slow continuing situations that do not require early detection or a rapid response.

Reported pain thresholds depend on a host of factors, including (a) exposure duration, (b) the location on the body to which heat is applied, (c) experimental protocols (which vary in sensitivity to the response time of the subjects), and (d) sex and age of the subjects (Harju 2002). For example, Defrin et al. (2006) studied the variation in threshold for cutaneous thermal pain in 20 human subjects from a temperature-controlled thermal probe applied to the skin surface in different regions on the body. The investigators compared thresholds measured using two different techniques. One method, which was sensitive to response times of the subjects, yielded thermal pain thresholds ranging from a mean of 44.5 °C in the foot to 42 °C in the chest. The second method, which was not sensitive to response time, yielded a threshold of 42.0 °C independent of body region. To complicate matters further, Hardy and Stolwijk (1966) reported transient thermal pain in subjects who were asked to suddenly immerse their backs in warm water at 37–41 °C. Some authors have reported that the threshold for thermal pain varies with the exposed body area, while others found no pronounced differences (Zhang and Hedge 2017). Neisser (1959) noted that untrained subjects tended to report lower pain thresholds (from infrared energy incident on different areas of the body) than trained subjects, and that subjects varied considerably in their criteria of judgment of pain threshold. In addition, the depth of the nociceptors relative to the skin surface can introduce a time dependence in thresholds due to heat conduction effects—a factor that not all pain threshold studies have considered.

Table 4 summarizes several studies on thresholds for cutaneous pain sensation in human subjects from heat sources other than infrared and RF energy. These include pain from heated liquids flowing across the skin, from exposure to infrared radiation, and to heat applied from a hot surface (thermode) placed against the skin. For short exposures...
Fig. 2. Thresholds for minimal thermal damage to skin and for cutaneous thermal pain. Parker et al. (2016) (single measurements on each of two subjects exposed to 94 GHz RF energy). The thresholds for cutaneous thermal pain sensations are from studies listed in Table 4. Thermal damage data from Moritz and Henriques (1947) for thresholds for erythema in humans from hot water applied to skin, and Suzuki et al. (1991) for superficial skin burns from heat applied via a 7 cm² heated surface to shaved abdomen of rats. The upper exposure duration considered in developing the Arrhenius model for skin is indicated by the short bar.

Table 4. Studies on thermal pain threshold not involving RF exposure (data summarized in Fig. 5).

| Study                  | Subjects/Exposure Details                                                                 | Findings                                                                 |
|------------------------|------------------------------------------------------------------------------------------|--------------------------------------------------------------------------|
| Hardy (1953)           | 6 subjects, IR to forehead. Step power input (exposure levels ranged from 1–30 kW m⁻², pulsewidth from 0.25–300 s) | “Pain was elicited when the skin temperature reached 45.7°C with a standard deviation of +/-1.7°C regardless of the time of exposure and intensity of stimulus.” |
| Stoll and Greene (1959)| 3 subjects exposed on forearm to IR pulses and from touching hot surfaces.               | Thresholds for pain and burn show similar time dependence. Later work by Stoll was the basis of current touch temperature standards for electronic equipment. |
| Nielsen and            | Heat applied via thermode with different contact areas                                     | “The heat pain threshold is influenced by the peak stimulus duration, and not by the rate of temperature change.” |
| Arendt-Nielsen (1998)  |                                                                                          |                                                                          |
| Helme et al. (2004)    | Heat applied by thermode, thresholds measured for “young” (30 y mean age) and “older” (78.9 y mean age) subjects, exposure duration 1–100 s. Body location where heat was applied was not specified. | “Older people have an increased threshold for thermal and electrically induced pain if the stimulus duration is kept short.” |
| Defrin et al. (2006)   | 20 subjects, skin heating from 9 cm² heated surface at 2 °C s⁻¹. thresholds assessed using two methods (one sensitive to response time, other not) | Threshold ranged from 44.5°C (foot) to 42.9 °C (chest) using a protocol sensitive to subject’s response time, constant 42.0 °C using a protocol not sensitive to response time. |
(seconds), pain thresholds exhibit similar exposure-duration curves as for skin damage (Stoll and Greene 1959). For longer exposure times (tens of minutes), pain thresholds level off to a constant value due to accommodation. Hardy et al. (1951) reported that pain thresholds from infrared exposure to the forehead and hands of human subjects varied linearly with skin temperature before exposure, resulting in an approximately constant threshold for cutaneous pain of 45.7 °C. Finally, the distribution of thermal pain sensory endings in the skin introduces variability in thermal pain thresholds for small-area exposures to skin (ICNIRP 2006).

In summary, the “pain threshold” varies considerably depending on the source of heat, method of assessment, and intersubject and intrasubject variations. For purposes of discussion, a useful range of “thresholds” for cutaneous thermal pain sensation in humans is 42–45 °C, but some measured thresholds for cutaneous thermal pain have varied outside of this range.

Cutaneous pain elicited by RF exposure

Walters et al. (2000) measured cutaneous pain thresholds in 10 subjects exposed on their backs to 3-s pulses of mm-waves at 94 GHz. The mean pulse fluence for “prickling pain” was 37.5 kJ m^-2 (6 times higher than ICNIRP occupational exposure limits for partial body exposure), which produced a peak skin temperature of 43.9 °C (9.9 °C above the baseline skin temperature of 34.0 °C). This is consistent with pain thresholds shown in Fig. 2.

In the only animal study that could be located on thermal pain from mm-wave exposures, Xie et al. (2011) inferred thresholds for thermal pain from EEG responses in anesthetized rats exposed to 35 GHz RF energy at power densities between 5–75 kW m^-2 for up to 30 s, with exposed areas of skin of 1.8 and 3.6 cm². These authors reported a threshold temperature for thermal pain of 44 °C, corresponding to an increase in skin temperature of 10 °C, which is similar to results of Walters et al. (2000) for humans.

Other studies on thermal damage to tissue from mm-waves

Beginning in the mid 1990s, the US Department of Defense sponsored studies on bioeffects of intense mm-waves for safety evaluations for the Active Denial nonlethal weapons system (ADS). The ADS is designed to elicit painful but not hazardous cutaneous pain sensations by beaming brief, high fluence pulses of mm-wave energy (94 GHz) at subjects. Many studies in this program (including Parker et al. 2016) were published only as technical reports, while other studies undoubtedly remain classified. Several of these studies reported catastrophic thermal effects in exposed animals at very high exposure levels. The exposure levels used in those studies far exceeded IEEE and ICNIRP limits, which would not apply to RF exposures from weapons in any event. For reviews of ADS bioeffects research, see Seaman (2009) and Wright et al. (2013).

CHRONIC EXPOSURE TO HEAT

Hardy (1953) and later studies have shown that, for short exposures, thresholds for cutaneous thermal pain are generally below those for thermal damage, and the resulting aversion response is an important protective mechanism against burns. For longer exposures, thermal damage can occur at exposures below the pain threshold. From Fig. 2, the crossover occurs, very roughly, at a constant-temperature exposure of 20 min where skin temperature is about 43 °C. Thermal pain aversion is clearly not sufficient to protect against tissue damage for constant-temperature exposures for times exceeding this crossover time.

Erythema ab igne (EAI) is a rare condition characterized by reddening of the skin and damage to skin blood capillaries, resulting from prolonged exposures to heat below thermally painful levels (ICNIRP 2006). In former times, the condition was reported chiefly in individuals working close to fireplaces and wood burning stoves, where it became known as the “toasted skin” syndrome. More recently a number of cases of EAI have been associated with extended use of portable electronics held against the body, including extended use of laptop computers due to their case temperature (Riahi and Cohen 2012). One case report describes EAI experienced by a woman from repeated use of a heated car seat (for 1 h d⁻¹ over a winter) (Brodell and Mostow, 2012). Product safety standards limit the temperature of heated car seats to 43 °C, but temperature of the car seat used by the individual was not reported.

Almost no quantitative data exist for temperature-time thresholds for EAI, but a few studies suggest thresholds for thermal burns from extended exposures of the skin to heat (Table 5). Suzuki et al. (1991) exposed the shaved abdomens of rats for extended times to a heated surface of 7 cm² area. The investigators reported erythema and “very sparse perivascular infiltration of neutrophils” from 7-h exposures at 40 °C. Greenhalgh et al. (2004) exposed 18 human subjects (patients awaiting elective surgery involving removal of excess skin) for 8 h to light emitting diodes (LEDs) used to simulate pulse oximeters resulting in skin temperatures of 43–44 °C. The investigators noted minor thermal injuries (erythema or a superficial blister) in one of the subjects exposed at 43 °C. These exposures are somewhat higher than reported by Suzuki et al. (1991), indicating lower thresholds for reversible thermal damage in rat vs. human skin.

The results of Greenhalgh et al. from humans (2004) line up well with much earlier results of Moritz and Henriques (1947), while data from Law et al. (1978) and Suzuki et al. (1991) from rats are likewise consistent.
Based on these limited data, it is concluded that the thresholds for minor skin thermal effects (erythema or superficial burns) from 8-h exposures are 40 °C (rats) or 43 °C (humans), with some uncertainty due to the small number of subjects in the latter study. Greenhalgh’s results have informed touch temperature limits, which are designed to prevent burns to users from contact with hot surfaces. The most conservative of touch temperature limits apply to medical devices that can contact patients’ bodies. For example, current IEC touch temperature limits are 43 °C for medical devices that can contact a patient’s body for more than 10 s (IEC 60601-1). Higher touch temperature limits apply to other devices and are intended to allow an individual sufficient time to withdraw from contact with a hot surface before sustaining a burn.

The slope of the damage function (Table 3) suggests that reducing skin temperature by one degree will approximately double the exposure time for a given level of damage. This suggests that damage thresholds for constant-temperature exposures lasting longer than 8 h may be somewhat below 43 °C. However, exposures even slightly below this level would require unrealistically long times to result in injury. No data exist to support such extrapolations, and extended exposures of the skin at constant temperatures for such extended times seems implausible in any event because of body movements or (for patients) occasional repositioning of monitoring devices on the body.

**REDUCTION AND SAFETY FACTORS IN ICNIRP AND IEEE LIMITS**

IEEE C95.1–2019 defines the safety factor as: “A divisor (≥1) applied to the exposure level that causes an adverse effect, used to establish a dosimetric reference limit (DRL) that includes inter-subject biological variability, uncertainties concerning threshold effects due to pathological conditions or drug treatment, uncertainties in computational models, uncertainties in dosimetry, and variations in temperature and humidity.”

ICNIRP defines a “reduction factor” [in earlier ICNIRP usage, “safety factor”] “to 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” (ICNIRP 2002).

As applied to thermal hazards, assessments of safety factors and setting of reduction factors require knowledge of the induced temperature increases and temperature-time relations for thermal hazards. The following comments compare safety factors estimated from thermal damage/pain data and from modeling studies, and comments on major uncertainties in assessing safety factors.

**Safety factors from thermal damage data**

Table 6 compares measured thresholds for thermal damage or cutaneous thermal pain from the experiments summarized above, in comparison with ICNIRP (2020) and IEEE C95.1–2019 limits. These studies involved exposures to RF energy that exceed the occupational exposure limits by factors ranging from ≈10–40. With one exception (Walters et al., 2000), these results are based on very limited experimental data, in many cases single data points from single human subjects.

**Safety factors from thermal modeling studies**

For purposes of discussion, the threshold for adverse effects (thermal damage or cutaneous thermal pain) is
assumed to be 43 °C (9 °C above the normal skin temperature of 34 °C).

Two exposure situations are considered below: (1) transient heating from RF pulses at the maximum fluence allowed by ICNIRP, IEEE, or Federal Communications Commission (FCC) limits, and (2) steady-state heating at the maximum exposure at 300 GHz allowed by ICNIRP or IEEE limits summarized in Table 1. Exposures are assumed to consist of plane wave RF energy normally incident on tissue, and safety factors are estimated with respect to limits for partial body exposures to areas >4 cm². Two cases are considered: exposures to brief, high-fluence pulses and continuous exposures. The first case is characterized by transient heating of tissue and the second by steady-state heating. All comparisons below are with respect to occupational exposure limits.

### Transient temperature increases from maximum-fluence pulses

Both IEEE C95.1–2019 and ICNIRP (2020) explicitly limit the fluence (incident energy density time pulse duration) of mm-wave pulses. The US Federal Communications Commission (FCC; 2010) guidelines do not explicitly limit pulse fluence, but the combination of the maximum permissible exposure and the averaging time implicitly limits pulse fluence to 18 kW m⁻² for both occupational and general public limits.

Laakso et al. (2017) computed the transient temperature increases in a high-resolution image-based model for the human head exposed to plane wave RF energy at 100 GHz. From that paper, the calculated peak temperature increases from maximum-fluence pulses of 5-s range from 0.6–0.8 °C (IEEE limits for restricted environments, which are comparable to occupational limits), 2.6–4.3 °C (ICNIRP occupational limits), and 5.0–24 °C (FCC occupational limits) (Fig. 3). These studies suggest that safety factors for these pulsed exposures, relative to cutaneous pain thresholds, range from 2 to 16. The FCC guidelines would permit RF pulses with sufficient fluence to cause painful and even damaging heating, but such exposures are far more extreme than would be produced by real-world technologies apart from some military-directed energy weapons.

### Steady-state temperature increases from continuous thermal exposures

Three modeling studies have estimated steady-state temperature increases from continuous RF exposures to the body at frequencies above 6 GHz (for a review, see Hirata et al., 2021).

- Sasaki et al. (2017) calculated temperature increases from RF exposures at frequencies up to 1,000 GHz using a multilayered planar model of the body (epidermis, dermis, subcutaneous tissue, muscle) exposed to plane waves at 10 W m⁻². The investigators measured the thicknesses...
of the tissue layers in 30 subjects, both sexes and ages 20–37 y, and conducted a Monte Carlo analysis involving 10^5 different combinations of tissue thickness. Adjusting their results to an incident power density of 100 W m^{-2}, their model predicts a steady-state temperature increase of the skin ranging from 0.9 to 2.4 °C and 1 THz, respectively, with a relative standard deviation of 6 to 9%.

- Recently, a similar analysis was reported by Sacco et al. (2021). The investigators determined the increase in surface temperature in a multilayer planar model of tissue subject to RF exposure at 26 and 60 GHz. The investigators carried out a Monte Carlo simulation with 10^6 iterations, considering the variation in skin blood flow, tissue thickness, and dielectric properties with age from 5 to 70 y. The maximum increase in steady-state temperature at the skin was found for models with parameters for 70-y-old individuals and (after adjusting to an incident power density of 100 W m^{-2}) were 1.7 °C to 2.0 °C at 10 GHz and 1 THz, respectively, with a relative standard deviation of 6 to 9%.

- Christ et al. (2020) calculated temperature increases in a planar multilayered model of skin, fat, and muscle at frequencies from 6–100 GHz, considering a wide range of model parameters. The maximum calculated increase in steady-state temperature at ICNIRP reference levels (occupational) was in the range of 1–4 °C at 100 GHz depending on the assumed boundary conditions on the skin and the thickness of different tissue layers. The highest computed increases in surface temperature assumed unrealistic exposure conditions (no heat exchange with the surrounding environment) and a thick stratum corneum characteristic of the palms and soles of the feet.

These three simulation studies show that steady-state increases in skin temperature from exposure to 100 GHz RF radiation at 100 W m^{-2} will be in the range of 1–2 °C. Increases from exposures at the same power density at lower frequencies will be lower due to the increased power penetration depths. Since both ICNIRP and IEEE limits allow higher exposures to localized areas on the body of <1 cm^2 subject to area-averaging provisions (Table 1), somewhat higher skin temperatures might result for small area exposures compliant with limits. The corresponding temperature increases allowed under the lower limits for the general public are well below 1 °C and are comparable to or below ordinary diurnal variations of body core temperature (Refinetti and Menaker 1992).
These modeling results for steady-state temperature increases cannot be used directly to estimate safety factors because major thermoregulatory changes will occur as skin temperature increases toward the threshold for pain. In particular, large increases in skin blood flow (Song et al. 1990) will moderate the increases in skin temperatures. The models of Sasaki, Christ, and Sacco might be extended to include such thermoregulatory effects, but so far, they apparently have not been. These thermoregulatory responses will vary with individual and environmental conditions.

Few if any data are available for steady-state temperature increases produced by exposure to RF energy in the considered frequency range. However, Piazena and Kelleher (2010) measured steady-state temperature increases in the backs of human subjects exposed to infrared radiation over areas on their backs of area > 100 cm². The radiation had been filtered to include wavelengths >0.83 μm and had a power penetration depth in tissue in the mm range (similar to that of mm-wave RF energy) (Table 7). At an irradiance of 100 W m⁻², the steady-state increase in skin temperature was ≈1 °C, similar to that calculated from the thermal models for RF energy at that incident power density assuming an energy transmission coefficient of 70% at 100 GHz (Foster et al. 2020). An absorbed irradiance exceeding 1,800 W m⁻² was required to cause the steady-state temperature at the skin surface to reach the pain threshold of 43 °C. This suggests a safety factor of ≈18 relative to the pain threshold for occupational limits.

As a practical matter, in occupational settings, both RF exposure limits and heat stress limits would jointly serve to protect a worker. This is implicitly acknowledged in ICNIRP IR (but not RF) exposure limits. For example, ICNIRP limits for incoherent IR exposure to the skin are provided only for exposure times up to 10 s (ICNIRP 2013a). The guideline states that “…much longer exposure durations are dominated by concerns of heat stress…” and refers to occupational heat stress standards. By contrast, ICNIRP and IEEE limits for RF exposure apply to exposures of any duration without reference to environmental conditions and work intensity. Nevertheless, in very warm environments where the safety factors in RF exposure limits would be reduced (due to increased skin and ocular temperatures), heat stress limits would undoubtedly be the major considerations in protecting workers against thermal hazards.

### COMMENTS ON ADEQUACY OF THE DATA

The thermal damage studies described above vary greatly in size and quality. For short term exposures, the data are quite extensive. Jean et al. (2013, 2021), Jean and Schulmeister (2017) summarized an immense amount of thermal damage data for ocular tissues and skin, from carefully done studies by multiple investigators that were meticulously analyzed. Most of those data derive from laser hazard studies, involve exposure to brief and high intensity pulses of infrared energy, and provide a good understanding of temperature-time relations for thermal damage for short term exposures.

By contrast, very limited data are available for thermal damage thresholds for exposures of duration of more than a few minutes from any source of heat. In preparing this review, only one study could be located that determined thermal damage thresholds in humans from 8-h exposures (Greenhalgh et al. 2004), and that involved a small number of subjects and heat sources (light-emitting diodes placed against the skin) that are quite different from IR or RF radiant energy. However, the reported 8-h threshold exposure for humans from that study, 43 °C, is about 10 °C above normal skin temperature and would require RF exposures far above current exposure limits to achieve.

Several of the human studies were published almost three quarters of a century ago, and those, together with most of the more recent human studies on thermal damage, are extremely small. Given current ethical standards for human and animal experimentation, it seems unlikely that extensive data on human damage thresholds will be added in the future.

Additional uncertainties arise from the questionable internal and external validity of the bioheat equation, which is used for most thermal modeling studies involving RF exposure (Foster et al. 2018). While the bioheat equation, together with standard parameter values found in the literature, yields reasonable predictions in accord with experimental data (Foster et al. 2016), it lacks theoretical rigor (Foster et al. 2018), and the parameters in the model are subject to considerable variation due to individual and environmental factors. Steady-state temperatures, in particular, depend strongly on tissue blood perfusion, which is highly variable depending on individual and environmental factors. More experimental studies on thermal responses of the body to modest levels of RF exposure would help refine the theoretical model, provided that RF exposures and temperature increases were documented with sufficient care. Such studies need not raise ethical issues of

| Skin temp at steady state °C | Absorbed irradiance, W m⁻² |
|-----------------------------|-----------------------------|
| 34.0                        | 0                           |
| 38.1                        | 582                         |
| 39.9                        | 867                         |
| 41.5                        | 1151                        |
| 42.3                        | 1435                        |
| 42.8                        | 1728                        |
| 43 pain threshold          |                             |

*IR-A radiation (wavelength > 0.83 μm) vs. absorbed irradiance. Data extracted from Fig. 10 of Piazena and Kelleher (2010)
the sort potentially raised by thermal damage or pain threshold studies.

COMMENTS ON REDUCTION FACTORS

ICNIRP (2020) calls for very comprehensive data on hazards of RF exposure: “…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” (ICNIRP 2020). Present data for thresholds for thermal damage and cutaneous thermal pain are insufficient to permit such a comprehensive analysis. However, the present analysis suggests that current IEEE and ICNIRP exposure limits are sufficient to protect against the thermal hazards presently considered with very large margins of safety, particulary in limits for the general public. Some uncertainty exists for safety factors from very small area exposure to high RF radiation levels, which are consistent with the 1 cm² spatial average in the limits (e.g., Funahashi et al. 2018). These are being addressed by further studies by several groups.

Finally, it is noted that the few reported severe injuries from exposure to RF energy involve accidents with high-powered RF sources, where an individual receives exposures far above ICNIRP and IEEE safety limits before being able to terminate exposure (Ziskin et al. 2002). Protecting against such accidents is a matter of safe work practices and safe equipment design and is not a problem that can be addressed by exposure limits.

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