ICNIRP STATEMENT ON FAR INFRARED RADIATION EXPOSURE

The International Commission on Non-Ionizing Radiation Protection*

INTRODUCTION

THE INTERNATIONAL Commission on Non Ionizing Radiation Protection (ICNIRP) currently provides guidelines to limit human exposure to intense, broadband infrared radiation (ICNIRP 1997). The guidelines that pertained to infrared radiation (IR) were developed initially with an aim to provide guidance for protecting against hazards from high-intensity artificial sources and to protect workers in hot industries. Detailed guidance for exposure to longer far-infrared wavelengths (referred to as IR-C radiation) was not provided because the energy at longer wavelengths from most lamps and industrial infrared sources of concern actually contribute only a small fraction of the total radiant heat energy and did not require measurement. Based upon the total optical emission of industrial sources and high intensity lamps, the limited IR-C contribution was incorporated into the derivation of the limits. Furthermore, when the limits were developed, the glass or quartz windows used with most portable thermal radiometers blocked most energy beyond 3–4 μm; thus, it was reasoned that the appropriate field measurements of IR sources could best be accomplished by limiting the measurements for risk assessments to wavelengths less than 3 μm. Therefore, ICNIRP previously did not provide specific guidance for IR-C (3 μm–1,000 μm) radiation; although, it was recommended that IR-C radiation should be included in measurements if this was of concern.

Industrial workers in very hot environments, such as in the glass, steel, and aluminum industries have traditionally had to deal with excessive infrared exposure. Industrial protective measures have evolved to counter the health hazards associated with these hot environments. Heat strain and discomfort (thermal pain) normally limit skin exposure to infrared radiation levels below the threshold for skin-thermal injury, and this is particularly true for sources that emit largely IR-C. Furthermore, limits for lengthy infrared exposures would have to consider ambient temperatures. For example, an infrared irradiance of 1 kW m^{-2} (100 mW cm^{-2}) at an ambient temperature of 5°C can be comfortably warming, but at an ambient temperature of 30°C this irradiance would be painful and produce severe heat strain. Therefore, ICNIRP provided guidelines to limit skin exposure to pulsed sources and very brief exposures where thermal injury could take place faster than the pain response time and where environmental temperature and the irradiated skin area were minor factors. Current exposure limits for the skin are to protect against thermal burns within exposure durations less than 10 s only, i.e., no exposure limits are provided for exposure durations longer than 10 s. For exposure durations longer than 10 s, potential health risks other than burning, such as heat strain resulting from excessive heat stress (core body temperature elevation), may become relevant, for which currently no exposure limits can be recommended.

Limits for greater exposure durations exist for ocular exposure, but the ambient temperature must be considered.

The primary impetus for this statement arose from conditions of human exposure where individuals personally elect to be exposed to relatively intense IR radiation and in some cases to induce mild hyperthermia for perceived health benefit. In recent years, relatively new types of infrared heating appliances (e.g., radiant warmers and infrared warming cabins, sometimes referred to as "infrared saunas") have been introduced for home and spa use. IR-C is frequently the main spectral emission encountered in infrared warming cabins, but there are also types which peak in the IR-A or IR-B wavelength range. There has been little or no physiological and medical research on the health effects of IR warming

* ICNIRP, c/o BfS, Gunde Ziegelberger, Ingolstaedter Landstr. 1, 85764 Oberschleissheim, Germany.
For correspondence contact G. Ziegelberger at the above address or email at g.ziegelberger@icnirp.org.
(*Manuscript accepted* 24 *July* 2006)
0017-9078/06/0

cabins, despite the existence of many claims of health benefits (Meffert and Piazena 2002).

This statement was prepared to provide guidance for the special conditions where lengthy periods of far-infrared skin exposure can occur despite the limitations normally imposed by hyperthermia and skin discomfort. This statement examines the potential risks of repeated human exposure to infrared radiant energy and provides expanded guidance for assessing the health risk from IR-C exposure.

BACKGROUND

Infrared radiation spectral regions

Optical radiation in the wavelength range of ~780 nm to 1 mm is known as infrared (IR) radiation. As noted above, the infrared region is often subdivided into IR-A (0.78–1.4 μm), IR-B (1.4–3 μm), and IR-C (3 μm–1 mm). The wavelength range of visible radiation (VIS) is usually considered to lie within the wavelength range of 380 nm to 780 nm. These spectral bands, defined by the International Commission on Illumination (CIE 1987), can be useful as "short hand" notations in discussions of the photobiological effects of optical radiation, although the predominant effects have less sharply defined spectral limits. The three infrared spectral bands roughly distinguish between different penetration depths into tissue, which are strongly dependent upon water absorption. IR-A radiation penetrates several millimeters into tissue. IR-B penetrates less than 1 mm, and at the penetration depth ($1/e$) is least (approximately 1 μm) at wavelengths near 3 μm, where water has its highest absorption peak. IR-C does not penetrate beyond the uppermost layer of the dead skin cells, the stratum corneum.

Most high-intensity broad-band sources (e.g., arc and incandescent sources) produce negligible levels of IR-C compared with the emissions at shorter optical wavelengths, and IR-C radiation can thus be largely ignored in risk assessments of these types of optical sources. Therefore, ICNIRP guidelines do not contain explicit recommended exposure limits for IR-C radiation. Only lasers pose serious potential hazards in this spectral region and current ICNIRP guidance extends to 1 mm for protecting the skin and eyes from laser irradiation (ICNIRP 1996, 2000).

Current guidance (ICNIRP 1997) assumes that all of the IR energy from IR-A and IR-B (780–3,000 nm) poses a risk to the human eye, whereas the contribution from IR-C in a white-light source would be small, or at least more or less equivalent for different sources. There is no strong wavelength dependence for absorption at wavelengths greater than 3 μm. IR radiant energy is nearly totally absorbed and only a very small fraction is reflected, thus a CO_2 laser emitting 10.6 μm radiation requires the least irradiance to heat up the skin surface.

Optical radiation from artificial sources is used in a wide variety of industrial, consumer, scientific, and medical applications. In most instances the visible and infrared energy emitted is not hazardous. In certain unusual situations, however, potentially hazardous levels are accessible, and excessive infrared radiation is typically filtered or baffled to reduce discomfort. Where sufficient visible radiation is present, the natural aversion response of the eye to bright light will substantially reduce potentially hazardous ocular exposure. Moreover, if the total irradiance is sufficiently high, the thermal discomfort sensed by the skin and cornea will also produce an aversion response that will limit the exposure time to a few seconds or less. Nevertheless, certain exposures remain potentially hazardous. Examples include those that may result from molten metals and infrared lamps for surveillance and heating. A variety of electrically heated metallic and ceramic rods and plates are used for radiant heating and drying in industry and in many medical, consumer, and office appliances. Where there is a need for comfort, these sources are frequently enclosed or baffled and seldom pose an actual hazard. Conventional lamps, as well as light-emitting diodes (LEDs), emit relatively little IR-C radiation. Emissions of current infrared LEDs are limited to near-infrared wavelengths.

The optical properties of laser radiation differ significantly from those of conventional, broadband optical radiation. Therefore, the exposure limits for broadband optical sources necessarily differ from those applicable to lasers. In addition, laser guidelines incorporate assumptions of exposure that may not apply to conventional optical sources (ICNIRP 1996, 2000). Most lasers emit radiation over one or more extremely narrow wavelength bands, and no detailed knowledge of the spectral output is required for purposes of hazard evaluation. By contrast, evaluation of the potential hazards of broadband conventional light sources requires spectroradiometric data for application of several different photobiological action spectra for UV and visible hazard evaluation, as well as knowledge of the exposure geometry. Photobiological action spectra are not relevant in the infrared, and spectroradiometric measurements are not required to apply the exposure guidelines for risk assessment (Heath Council of Netherlands Committee on Optical Radiation 1993).

In research studies spectroradiometric data are useful to evaluate the relative effectiveness of different broadband infrared radiation sources—particularly in the short- and mid-wavelength range (IR-A and IR-B). This

is due to the strong dependence of the spectral absorption coefficient on wavelength in the tissue. Furthermore, the geometry of the exposure has to be considered because the thermal effects in the tissue depend on both the irradiated area and the irradiance.

Thermal radiation

All bodies emit a broad spectrum of optical radiation which depends on their absolute temperature expressed in Kelvin (K) and on their spectral emissivity $\varepsilon(\lambda)$. (Note: The temperature in Kelvin is the temperature expressed in degrees Celsius + 273.15.)

Based upon spectral emissivity, which in general depends on wavelength (λ) and on temperature, all bodies can be divided into three types. These are perfect blackbodies with a constant emissivity of $\varepsilon(\lambda) = 1$ for all temperatures of the emitting body; "grey bodies" with a smaller but also spectrally flat emissivity (which may depend on the surface temperature); and spectrally selective radiators showing a smaller and spectrally different emission than perfect blackbodies at a given temperature [$\varepsilon(\lambda) = f(\lambda,T) < 1$].

For perfect blackbodies the spectral radiant exitance W_λ (in units of W m^{-2} nm^{-1}) is described by Planck's Law of Radiation (eqn 1):

$$W_\lambda = (2\,c^2h/\lambda^5) \times (e^{hc/\lambda kT} - 1)^{-1}, \quad (1)$$

where $c = 2.997925 \times 10^8$ m s^{-1} is the vacuum speed of light, $h = 6.6256 \times 10^{-34}$ Js is Planck's Constant, $k = 1.380662 \times 10^{-23}$ JK^{-1} is Boltzmann's constant and where the wavelength λ is expressed in meters (m).

Fig. 1, from Plank's Law, provides the theoretical spectral radiant exitance for ideal blackbodies with different temperatures in the range between 273.15 K (i.e., 0°C) and about 1,000 K (typical of a red-hot electric heater coil).

The peak spectral emission of blackbodies and grey-bodies continues to shift to shorter wavelengths for higher temperatures. This is known as Wien's Displacement Law (eqn 2):

$$\lambda_{max} = 2898/T, \quad (2)$$

where λ_{max} is the wavelength of maximum radiant exitance in units of μm, and T is the temperature in units of K. At the typical environmental background temperature of 300 K, the peak emission is at about 9.7 μm (IR-C), and at the temperature of molten steel (~1,800 K), the peak has shifted to ~1.6 μm (IR-B), but at the surface temperature of the sun (~6,000 K), the peak has shifted to about 0.48 μm (VIS).

Fig. 2 provides the spectral peak shifts of some representative blackbody temperatures.

The spectral and semi-spatial integration of the spectral radiant exitance results in the specific radiant exitance (i.e., the surface radiant flux density), which is related to the absolute temperature T in the Stefan-Boltzmann Law according to eqn (3):

$$W = \varepsilon \times \sigma \times T^4, \quad (3)$$

where $\sigma = 5.6687 \times 10^{-8}$ W m^{-2} K^{-4} is the Stefan-Boltzmann constant, and ε is the total emissivity of the material (with a maximal value of 1.0 for a perfect blackbody). Note that the emitted thermal radiant energy increases very rapidly with temperature.

Fig. 3 provides the specific radiant exitance for a perfect blackbody in comparison with data for bodies of

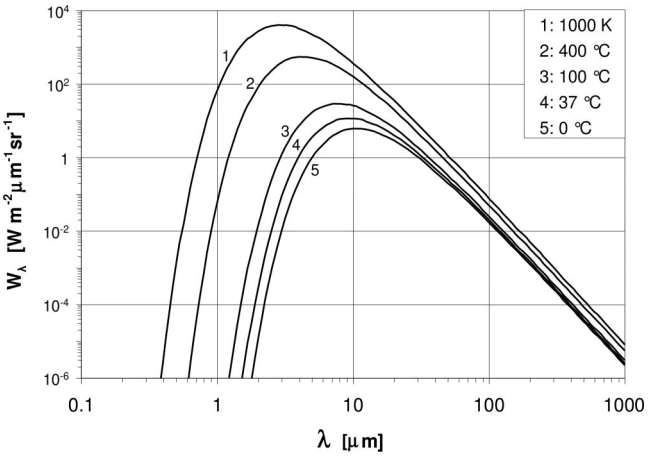

Fig. 1. Dependence of spectral radiant exitance W_λ (Planck's Law) for theoretically perfect blackbodies with temperatures between 273 K (~ 1°C) and 1,000 K (Planck's Law) as a function of wavelength λ in μm.

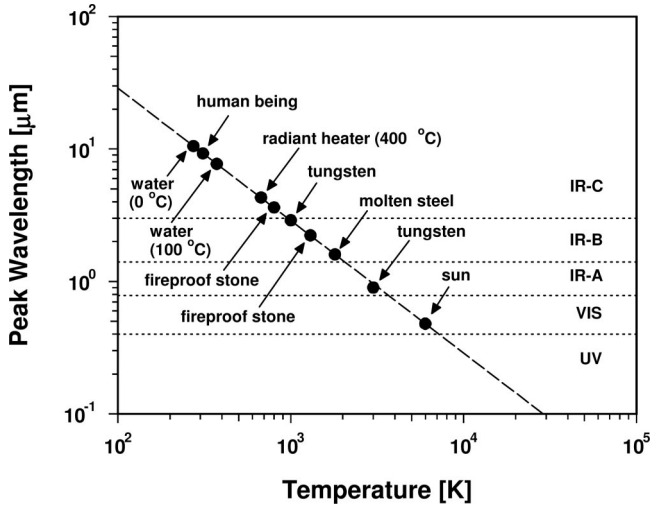

Fig. 2. The wavelength of maximal radiant exitance λ_{max} as a function of the absolute temperature for a perfect blackbody (dashed line) and for different bodies with temperatures between 273.15 K (0°C) and 6,000 K (Wien's Displacement Law).

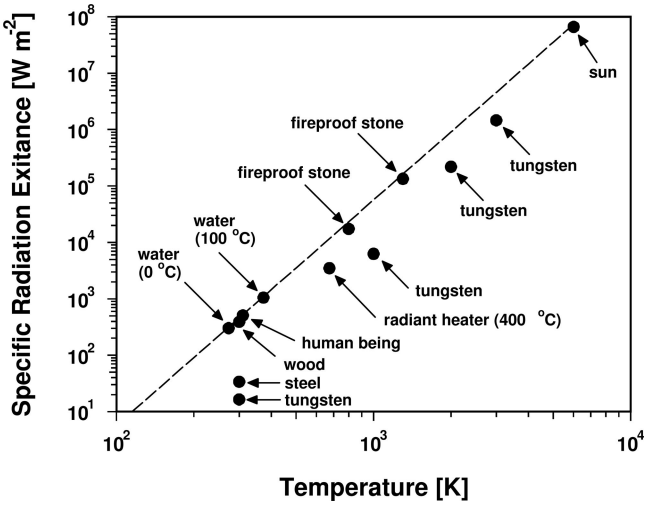

Fig. 3. Stefan-Boltzmann Law. The specific radiation exitance (W m^{-2}) as a function of the absolute temperature for a perfect blackbody (dashed line) and for bodies of different temperature and total emissivities (ε). Water and human skin ($\varepsilon \cong 0.96-0.97$), wood ($\varepsilon \cong 0.8-0.9$ at 300 K), steel ($\varepsilon \cong 0.065$ at 300 K), tungsten ($\varepsilon \cong 0.03$ at 300 K; $\varepsilon \cong 0.111$ at 1,000 K; $\varepsilon \cong 0.242$ at 2,000 K, and $\varepsilon \cong 0.318$ at 3,000 K), fireproof stone ($\varepsilon \cong 0.65-0.85$ at 800 K and $\varepsilon \cong 0.75-0.90$ at 1,300 K), a radiant heater ($\varepsilon \cong 0.3$ at 400°C), and the surface of the sun ($\varepsilon \cong 1.0$ at about 6,000 K).

different total emissivities at temperatures from 300 K to about 3,000 K. For example, the total radiant exitance for the surface of the sun, which can be approximated as a blackbody at 6000 K, is about 7.348×10^7 W m^{-2}. For a blackbody surface at 600 K the Stefan-Boltzmann Law predicts 7.348×10^3 W m^{-2}.

SPECIFIC APPLICATIONS

Medical applications

The medical use of infrared radiation has a long history. It has been widely used in physical medicine for treatment of sports injuries, muscle aches, pain, and some chronic diseases (Licht 1967; Vaupel and Krüger 1992, 1995). In recent years there has been an interest in the use of IR-A sources for hyperthermic treatment of cancers (von Ardenne 1994, 1997; Vaupel and Krüger 1995; Wehner et al. 2001). Because of the deeper penetration of IR-A, this is used almost exclusively, and water filtering of IR to achieve pure IR-A has been recommended in therapeutics. There are also special IR-A therapeutic apparatuses that have been used for hyperthermic treatment of cancers and Raynaud's syndrome (i.e., white finger disease). The typical treatment irradiance of several therapeutic IR devices fall in the range of 800 W m^{-2}. A German standard (DIN 5031-10; DIN 2000) limits IR-A to 1,200 W m^{-2} in therapeutic equipment. Monitoring rectal temperature is the most basic indicator of core body temperature. Therefore in cases of extreme heat environments, workers are sometimes medically monitored in this way to determine the onset of heat strain (ACGIH 2005a, 2005b). This is also used for patients undergoing IR-A therapy.

IR-C is not widely used in therapeutic applications, although it has been suggested for use as a "detoxifying" method following cancer chemotherapy.

Warming cabins

Elective hyperthermia—the classical being wet sauna and hot baths—dates back millennia. There are approximately 26 million regular users of sauna in Germany alone (Meffert and Piazena 2002), and there is an increasing number of far-infrared warming cabins being used. There are a variety of infrared warming cabins that are constructed somewhat like a sauna, except that electrically heated ceramic or metal radiators are placed typically to the back and front of the cabin, such that the seated individual is exposed from the rear and the front. The heating elements are typically heated to about 300° to 400°C when designed to emit primarily IR-C. It is not always recognized that the skin heating and the resulting hyperthermia are produced differently in an IR-C warming cabin compared to a conventional sauna. The heat exchange between the body and the environment is almost purely radiative in the case of far-infrared warming cabins. There are at least three varieties of infrared warming cabins to consider:

1. Cabins with large area low temperature heaters (basically the whole wall becomes hot) which have much lower surface temperatures (about 50 to 70°C) where the irradiance of the skin is relatively low and the emission is purely in the IR-C (with peak wavelengths at ~ 7 μm);
2. Cabins with IR-C heaters that are typically ceramic or metallic rods with a length of about 50 cm or plates of the order of 5 cm by 20 cm. The surface temperature is generally in the range of 300–400°C and the back of the radiator housing forms a metallic reflector. The radiators that are positioned in the back are recessed with respect to the backrest by typically 10–15 cm. The distance of the front-side heaters to the irradiated person is usually of the order of 50 cm to 1 m. The fraction of IR-B that is emitted by these radiators is negligible and the emission lies mainly in the IR-C region; and
3. In some IR warming cabins, filtered filament lamps are used that produce emissions in the IR-B or in the IR-A. The lamps are basically quartz-tungsten lamps with an elongated filament, where the visible component is strongly decreased by filters. However, these lamps also emit a small amount of visible light so that

they can be easily distinguished from the lower temperature IR-C radiators that do not emit any visible radiation. The red light is more obvious from radiators with a maximum emission in the IR-A than the radiators with a maximum emission in the IR-B. As filament temperatures need to be high to produce main emission in the IR-A, cooling of the radiator becomes an issue to prevent exposure levels which are uncomfortable or produce a pain response.

The following discussion mainly refers to IR-C warming cabins with heating element temperatures of about 300–400°C. The air temperature of conventional saunas is maintained typically at approximately 85–90°C. However, the IR-C cabin air temperature may be of the order of ~40°C or less, and the primary heat transfer is by radiant heating. The IR-C skin irradiance in a conventional wet sauna is of the order of 110–200 W m^{-2} from the hot walls, and at least half of the total body heating occurs from convection. The heating of the skin with IR-C warming cabins is faster than the older, conventional sauna, but higher irradiances of the skin must be applied in order to produce noticeable sweating (normally desired by the user) because of the absence of humidity normally present in a traditional sauna. However, in an IR warming cabin, there is negligible or little heat exchange by convective heating, thus the IR-C irradiance values must be higher to achieve a similar whole-body hyperthermia. Since the walls in an IR warming cabin are at a lower temperature than in a conventional wet sauna, the irradiance pattern on the body is far less uniform (except for large area, low temperature heating plates). To achieve the same degree of hyperthermia in these cabins, the average IR-C irradiance at the skin surface measures about 200–400 W m^{-2}, with hot-spots of the order of 1 kW m^{-2}. Many sauna enthusiasts actually have two units—both the original sauna and now an infrared cabin for a more rapid session (Piazena and Meffert 2001).

BIOLOGICAL EFFECTS OF INFRARED RADIATION

Optical radiation is generally absorbed superficially in skin and ocular tissues, but with varying penetration depths depending on wavelength. In the eye, the cornea, lens and retina can be at risk, depending upon spectral band. Following absorption of the radiant energy, the interaction with tissue can be either thermal or photochemical (where individual photons interact directly with individual molecules to directly induce chemical changes). Indirect effects are also possible, ranging from whole-body heat stress (hyperthermia) to cellular effects, where elevated cell temperatures can also interfere with DNA repair. Chronic, repeated elevated skin temperature can also result in pigmentary changes that are referred to as erythema *ab igne*.

General

Mechanisms of interaction with biological tissue. The eye and skin are adapted to protect the body against optical radiation from the natural environment. Humans have learned to use appropriate additional protective devices. The aversion response normally limits the duration of exposure to less than 0.25–10 s. This protects the eyes and skin against thermal injury from sources such as the sun, incandescent lamps, and radiation emitted by hot objects. Although the aversion response to bright light is formalized in eye-safety standards at 0.25 s, there is no such formalism for the thermal aversion response due to the skin's thermal receptors. Large areas of the skin will be more readily heated to higher temperatures than smaller areas (as from a laser beam) for the same irradiance (Sliney and Wolbarsht 1980; UNEP/WHO/IRPA 1982). Because of the distribution of thermal pain sensory endings in the skin, the sensation of heat is more certain when the irradiated skin area is larger (Blick et al. 1997; Cook 1952; Hardy et al. 1953).

Tissue interactions in the ultraviolet and visible portions of the optical spectrum are either thermally or photochemically initiated. However, for infrared optical sources, thermal injury of skin and ocular tissues is the dominant interaction mechanism. Skin cancer caused by optical radiation sources in the absence of ultraviolet radiation is not considered to be a significant risk (UNEP/WHO/IRPA 1982, 1994; IRPA 1985; IARC 1992) and is discussed in more detail in the section below. In recent years there has also been research showing photochemical interactions in the red and IR-A spectral region that can affect cellular activity (Karu 1987, 1998). However for IR-B and IR-C interactions with biological tissues, only thermal interaction mechanisms are known.

IR-A penetrates deeper (to several mm) into tissue than IR-B and IR-C (Hardy et al. 1956). The penetration of IR-C in tissue is only superficial. The *penetration depth* expressed as $1/e$ (37%) of the incident irradiance is of the order of 0.1 mm or less. At approximately 0.25 mm, 95% of the incident radiation has been attenuated by absorption and scattering. For this reason, IR-A is more frequently applied medically for delivering thermal energy to the vasculature and to muscle tissue. Fig. 4 shows the skin penetration depth in mm at different IR wavelengths.

Characteristics of thermal interaction mechanisms. Unlike photochemical injury, thermal injury does

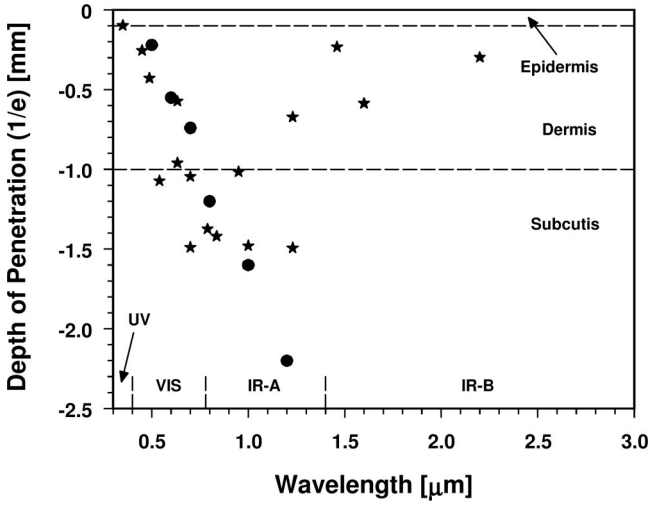

Fig. 4. Skin penetration depths of optical radiation to 37% (1/*e*) of the incident surface irradiance in the spectral range 0.3–3.0 μm into fair human skin as a function of wavelength, and compared with the order of thickness of the epidermis (0.1 mm) and of the dermis (1 mm). The calculated values by using spectral data of the absorption coefficient and of the reduced scattering coefficient published by different authors are shown as stars. These are compared with the data of Anderson and Parrish (1982) (circles).

not show reciprocity between irradiance and exposure duration. Thermal injury is strongly dependent upon heat conduction from the irradiated tissue (Cunningham 1970; Davis 1963). It requires an intense exposure within seconds to cause tissue coagulation. If the exposure is less intense, surrounding tissue will adequately conduct heat away from the exposed site. Thresholds for acute thermal injury of both cornea and retina in experimental animals have been corroborated for the human eye by flash burn accident data (Sliney and Wolbarsht 1980). The time-temperature history for brief exposures has been studied largely with laser pulses and detailed data were obtained for porcine skin and for small images on the retina (Brownell et al. 1969; Priebe and Welch 1978; Allen and Polhamus 1989). The irradiance required to achieve these temperatures depends upon the ambient tissue temperature and the exposed area or irradiated spot size.

Because of the more efficient cooling of small spots, injury of a small spot requires higher irradiances than injury of a large spot. This more rapid cooling of small areas also limits the duration of elevated temperature after the cessation of optical exposure, therefore influencing the critically important time-temperature history of the exposed tissue. Thus there is no single critical temperature for a given exposure duration. The spot size must be specified for describing ocular or skin exposures.

Effects on the skin

Acute infrared exposure and skin burns. Normally a temperature of at least 45°C is necessary to produce a thermal burn (Greene and Hardy 1962; Hardy and Oppel 1937; Hardy et al. 1962; Hardy 1968, 1978; Moritz and Henriques 1947; Derksen et al. 1963; Hendler and Hardy 1960; Hendler et al. 1963; Stolwijk 1980). Higher temperatures (Henriques and Moritz 1947; Henriques 1948) are required for thermal injury to result from exposures of shorter duration (e.g., about 47°C for 10 s or 57°C for 1 ms). There has long been a debate about the dose-response curve for the production of infrared induced thermal skin burns, which also varies with the spectral distribution. Some reports refer to damage studies with white light sources, others to laser damage, but in all cases the assumption is that the injury mechanism is purely thermal. Skin reflectance plays a major role in reducing risk from visible and near-infrared sources (Hardy et al. 1956).

It is critically important to distinguish IR-C (and IR-B) from IR-A, where the infrared penetrates well into the dermis and even deeper into the sub-cutis. The dermis is approximately 1 mm thick and contains most of the nerve endings, whereas the epidermis is at least 100 μm thick and has no blood vessels. IR-B is absorbed in the epidermis and dermis, but is not absorbed as deeply as is IR-A radiation. IR-C is totally absorbed in the stratum corneum and superficial epidermis. Thus, deep heating by IR-C is only achieved by heat transfer, i.e., heat conduction in the tissue and by blood flow. The resulting epidermal temperature is higher when the skin is irradiated with IR-C than similar thermal loads from shorter wavelengths. The prolonged erythemal response produced by IR-C exposure is probably the result of increased epidermal temperatures with this type of radiation.

Histological analyses show detectable early changes even in skin previously exposed to infrared radiation at levels below thermal pain. These include vasodilatation and perivascular accumulation of degranulated mast cells (signs of cellular stress as can be observed following an exposure to UV radiation, but not related to DNA damages). Brief, single infrared exposures also induce the expression of the prostaglandins D_2, E_2, and F_2 (Juhlin et al. 1983; Schulze et al. 1985).

Delayed effects. The only widely reported delayed effect from infrared irradiation is erythema *ab igne*. Erythema *ab igne* appears at the site of chronically repeated episodes of elevated skin temperatures not sufficient to burn the skin (Edwards et al. 1999). The appearance is reticular, macular (or slightly infiltrated),

and reddish to brown color (initial signs are red, then later darker). Initial effects of repeated elevated skin temperature are epidermal atrophy, a dermal mixed-cell infiltrate and pigmentation caused by melanophages, free-lying melanin granules, and hemosiderin. Later, basophilic degeneration of connective tissue, including the alteration of elastic fibers and hyperkeratosis with and without dyskeratosis, are observed (Shahrad and Marks 1977; Hurwitz and Tisserand 1987; Findlayson et al. 1966).

Before the advent of central heating, thermal keratoses and erythema *ab igne* was once found frequently on the naked shins of fireplace owners. Today erythema *ab igne* is rare, although it has been reported to result from chronic use of hot water bottles on the abdomen (Fitzpatrick 1999). It has various other names and was once common in actors and actresses from unfiltered lamps used in 18th century theaters and amongst blacksmiths.

It is not clear if erythema *ab igne* is more likely to occur from far-infrared than IR-A, since the latter delivers radiant energy more deeply. Since erythema *ab igne* is also observed in cases where the skin heating occurred from contact with hot surfaces without exposure to infrared radiation, the induction of erythema *ab igne* is generally linked to repeated episodes of elevated skin temperatures, by whatever means the heat is transferred.

If the heating practice is discontinued, erythema *ab igne* disappears after some years. However, there have been anecdotal reports that chronic inflammation can be the site of development of skin cancer. Indeed, this was termed "turf-fire cancer" or "Irish farmers' wives cancer," or "cangri cancer." The associated cancer is nearly always a squamous cell carcinoma and rarely a basal cell carcinoma. There is no evidence to support the hypothesis that infrared radiation is a carcinogen. There are only a few publications that have suggested infrared effects upon DNA. For example, in studies of infrared photoprotection by the introduction of mineral reflecting substances (TiO_2, zinc) in sunscreen preparations (Pujol and Lecha 1992/1993), there have been concerns that infrared and heat may act synergistically with UV radiation to produce cutaneous DNA denaturation (Roth and London 1977) and may also be potentially photocarcinogenic (Freeman and Knox 1964). Others have argued that near infrared radiation may protect normal human dermal fibroblasts from solar UV phototoxicity (Menezes et al. 1998). However, there is also evidence that increased temperature can reduce the repair efficiency of existing DNA damage and it is not unreasonable that repeated heating of the epidermis may promote (accelerate) skin cancer formation induced by other agents (such as ultraviolet radiation). Edwards et al. (1999) reported increasing thickness of epidermis in IR irradiated skin as well as cytochemical changes in Glucose-6-phosphate dehydrogenase which are similar to changes observed in solar keratoses and in preneoplastic and neoplastic tissue caused by UV exposures. Perhaps because of these observations, Dover et al. (1989) suggested a photocarcinogenic potential of IR radiation, but these tissue changes produced by increased temperatures do not confirm direct thermally induced DNA damage in the exposed tissue. A review of the literature supports the conclusion that heat alone is not expected to produce DNA damage (Dewhirst 2003b) in normal cells. However, excessive epidermal temperature could act as a co-carcinogen (promoter) by reducing the efficiency of DNA repair from damage initiated by UV or chemical exposures. It is known that elevated temperatures alter ultraviolet-induced erythema thresholds. Elevated temperatures can genomically destabilize an already transformed cell, leading to cancer progression (Boukamp et al. 1999). Temperature can be considered as a co-carcinogenic factor. However, there is experimental evidence that UV irradiation between 280 and 340 nm was less effective than the full spectrum of a powerful medium pressure mercury arc, and it is suspected that elevated skin temperatures from VIS and IR might be responsible (Bain and Rusch 1943; Bain et al. 1943). They demonstrated that at room temperature between 35–38°C, the UV (280–340 nm) carcinogenesis evolved at a much enhanced rate in comparison to normal room temperature (around 23°C, which showed little difference with 3–5°C). Freeman and Knox (1964) confirmed these latter results on room temperatures (32 vs. 24°C). Overall result indicates a 3–7% higher efficacy in UV dosages per degree C (van der Leun and de Gruijl 2002). Van der Leun and de Gruijl postulated that climate change could have an unexpected impact on skin cancer incidences. For this reason, it is important to warn users to avoid the use of warming cabins for at least 24 h following substantial sun exposures or tanning with artificial sources.

Studies of erythema *ab igne* (Findlayson et al. 1966; Hurwitz and Tisserand 1987; Sharad and Marks 1977) could be of some value; however, as this is no longer widely observed, there are no recent scientific studies and much evidence remains anecdotal (Fitzpatrick et al. 1974; Fitzpatrick 1999). There have been attempts to produce erythema *ab igne* in animals (Walsder and Hargis 2002) with some success. Meffert and Piazena (unpublished[†]) measured the time course of elevated skin temperatures for IR-C irradiation of 100 W m^{-2} and 1,000 W m^{-2}. They noted the development of erythema

[†] Meffert H, Piazena H. Personal Communication. Berlin: Dermatology Hospital; June 2003.

after a 15-min exposure to 1,000 W m^{-2} (i.e., 100 mW cm^{-2} leading to a radiant exposure of 90 J cm^{-2}). There was no pain reported by most of the subjects (indicating a skin temperature less than 45°C). Initially the erythema appears uniform, but after some minutes becomes reticular, corresponding to the locations of small dermal blood vessels. This may be an early step in the development of a long persistent erythema, i.e., erythema *ab igne*. The lasting erythema may result more from an inflammatory response of the epidermis than from the direct dilatation of blood vessels that is characteristic of the transient erythema induced by IR-A exposures below burn threshold. First-degree skin burns are homogeneous and appear rapidly. There is leakage of small vessels and a persistent redness in a burn. The distinction between a clear case of first-degree burn and IR-C persistent erythema may be ill defined.

Erythema *ab igne* on its own is not a serious health problem, but more a cosmetic problem. However, it indicates a thermal damage of skin which may increase the risk of skin cancer development in the presence of carcinogenic chemicals or UVR exposure. It is important to note that the thresholds doses to induce erythema *ab igne* may be below the thresholds of thermal pain. Currently available data (Meffert and Piazena unpublished[†]) indicate the existence of a threshold exposure dose and irradiance to cause abnormally persisting erythema of the skin. Further studies are needed to provide a definitive threshold.

However, a practical recommendation is to consider that if the acute erythema is persistent (i.e., it lasts for at least an hour or two), this is abnormal, and should be considered as a warning sign of abuse. Likewise, if a reticulated pattern appears, this is a clear signal to limit future exposures. When exposures are not repeated, the erythema disappears after some time, i.e., if exposure leading to persistent erythema is not often repeated, there should not be a delayed effect. It should be noted, however, that with many infrared warming cabin designs the exposure is largely to the back and the dermal response may not be readily observed by the user. The absence of pain is not an indication of lack of an adverse effect. Persistent erythema has appeared even when the subject does not experience pain at the time of exposure (Helm et al. 1997).

Effects on the eye

Any calculation of potential thermal hazards from intense incoherent optical sources normally includes a consideration of the contributions of IR-A and IR-B, but IR-C is seldom considered for most light sources, including the sun or molten metals, since the contribution of IR-C is marginal. Different ocular structures are affected by different infrared spectral bands: for wavelengths up to 1,350–1,400 nm, the ocular media transmit energy to the retina. At longer wavelengths, the anterior segment of the eye absorbs incident energy. Only very intense sources such as xenon-arc lamps and nuclear fireballs can produce retinal exposures that can produce thermal burns. The infrared radiation that is absorbed by the anterior segment (the cornea, aqueous, and lens) can produce clouding of the cornea and lens when the corresponding thresholds are exceeded. Exposure limits are set to protect both against acute as well as chronic exposure.

Data on which to base exposure limits for chronic exposure of the anterior portion of the eye to infrared radiation are very limited. Sliney and Freasier (1973) stated that the average corneal exposure from infrared radiation in sunlight was of the order of 1 mW cm^{-2}, considering that the eyes are seldom directed toward the sun except at sunrise and sunset. Glass and steel workers exposed in hot environments to infrared irradiances of the order of ~80–400 mW cm^{-2} daily for 10–15 y have reportedly developed lenticular opacities (Sliney and Wolbarsht 1980; Lydahl 1984). The corneal and lenticular exposures are affected by the relative position of the source and the degree of lid closure.

Pitts and Cullen (1981) showed that the threshold exposures for acute lenticular changes caused by IR-A were of the order of 50 MJ m^{-2} (5 kJ cm^{-2}) for exposure durations of the order of an hour or longer. Threshold irradiances for damage were at least 40 kW m^{-2} (4 W cm^{-2}). Wolbarsht (1978, 1992) showed somewhat similar levels using a Nd:YAG laser operating at 1064 nm, and Scott (1988a, 1988b) and Okuno (1991, 1994) showed that the calculated temperature rise was several degrees. Although Vos and van Norren (1994) argued that an irradiance of 1 kW m^{-2} would not increase the temperature of the anterior segment of the eye by more than 1°C, and that this level would be acceptable (as with collimated laser beams), such an irradiance over the entire head or much of the body from an incoherent source would not be acceptable for extended periods. The Commission therefore recommended that, for very warm environments (>35°C), the ocular irradiance should not exceed 100 W m^{-2} for lengthy exposures. However, higher irradiances could be safely sustained for shorter periods. Higher irradiances are permitted in cold environments provided that the lenticular temperature is maintained below 37°C. For radiant warming in outdoor environments in winter, irradiances of the order of 300 W m^{-2} are routinely used. Radiant energy absorbed in the cornea, aqueous humour, and lens is transported by conduction, and some heating will occur in the lens regardless of the optical penetration depth. Penetration

depth strongly varies in the IR-A and IR-B spectral bands, but these variations—between 1.2 and 3 μm—have only a minor effect on the final temperature rise resulting from exposure to a continuous source once thermal equilibrium is achieved. The final temperature of the lens also depends on the ambient temperature (Sliney 1986). For each degree that ambient temperature falls below 37°C, an added radiant exposure of at least 6 W m^{-2} (0.6 mW cm^{-2}) would be required to maintain the temperature of the lens (Stolwijk and Hardy 1977).

There are no comprehensive epidemiological studies that were directed toward environmental cataract and temperature. Miranda (1979, 1980) and Weale (1981) noted the differences in the incidence of cataract and the time of onset of cataract and presbyopia in different geographical regions. It was clear that the time of onset of both was earlier in hotter environments. More recently, a latitudinal variation in the type of cataract was shown by Sasaki et al. (1999). This and laboratory studies (Kojima et al. 2002) may suggest a thermal component—particularly with regard to the incidence and time of onset of nuclear cataract (Sliney 2002).

As previously noted, rapidly changing photochemical action spectra are characteristic of ultraviolet and short-wavelength light exposure. Spectral data are therefore particularly important in that wavelength range. Because the infrared effects are thought to be largely thermal, chronic infrared exposures of the cornea and lens are not believed to involve rapid changes in spectral sensitivity (Barthelmess and Borneff 1959; Sliney 1986, 2002).

There have been several efforts to mathematically model heat transport within the eye and to calculate temperature rises in an IR-exposed eye. Calculations for the human eye (Scott 1988a, b; Okuno 1991, 1994) show that ocular temperatures generally rise rapidly with exposure time for the first two minutes, then gradually level off and reach the maximum within approximately 5 min. Scott (1988a, b) also showed that it takes several minutes for the eye to cool down after an exposure ceases. For IR-B or IR-C exposure of the eye, Okuno (1991, 1994) showed that the temperature rise is the largest at the corneal surface and decreases gradually toward the retina. He also showed that the temperature rise is the quickest at the corneal surface and is slightly delayed at deeper ocular structures. A trial measurement of ocular temperatures in the rabbit eye exposed to infrared radiation (Kojima et al. 2002) exhibits all these trends. Focal radiation in the lens can occur with the use of certain ophthalmic-instrument light sources (Okuno et al. 2005). The cornea is extremely sensitive to thermal stimulus and this will tend to limit hazardous infrared exposure (Dawson 1963; Beuerman and Tanelian 1979).

In addition to the criteria to protect the cornea and lens against thermal damage from infrared exposure, a second criterion is required to protect the retina against thermal injury. Viewing specialized IR-A illuminators with visible radiation removed by filters results in a loss of the aversion response to bright light. This criterion for retinal protection (ICNIRP 1997) is based largely on the studies of Ham et al. (1976), who demonstrated the absence of photochemical effects in the infrared spectral bands.

Synergism between thermal and photochemical effects in the lens and retina has been studied in a number of experiments. Thermal enhancement of photochemical reaction has been experimentally demonstrated (Pitts and Cullen 1981; Ham and Mueller 1989), although the effect is less than a factor of two; this has been taken into account in deriving the exposure limits by introducing a greater margin of safety.

Thermoregulation

The human body maintains itself at a nearly constant temperature of about 37°C in the deep interior; this is referred to as the "core body temperature." The term thermoregulation is normally used to describe the maintenance of this core body temperature within a given range near 37°C (Adair 1983; Adair et al. 1985, 1998; Berglund 1983; Dewhirst et al. 2003a; Gagge et al. 1941; Hardy and Bard 1974; Hardy 1978; Nielsen and Nielsen 1965; Stolwijk and Hardy 1966, 1977; Stolwijk 1980; Walters et al. 2000; Whittow 1971). The actual temperature varies somewhat with the individual and time of day (circadian rhythm), but only within about 1°C. With vigorous exercise or in a disease state (Shimada and Stitt 1983), the core body temperature could vary from a lower extreme of approximately 35.5°C to an upper extreme of about 40°C. The thermal loading also affects the core temperature. Thermal loads come from the alteration in ambient conditions (air temperature, ambient vapor pressure, air velocity, irradiation and clothing) and from changes in heat production within the body (endogenous sources) (Werner and Buse 1988). The Law of Conservation of Energy forms the basis for the study of thermoregulation (Bligh and Johnson 1973). In order to maintain a relatively constant body temperature, heat exchange with the environment must be balanced. The main constituents of heat exchange are:

- metabolic heat generation (~100 W for an adult);
- physical activity—100 W to 400 W for sustained activity;
- radiation exchange—depending upon the temperature difference between the skin and environment (Stefan-Boltzmann Law);

- convective heat exchange—depending upon air temperature, air velocity, exposed skin surface, etc.;
- evaporative cooling—depending upon relative humidity, exposed skin surface, etc.; and
- heat storage within the body—depending upon body mass, etc.

The thermoregulatory system functions as a negative feedback control system, with a reference (or "set") temperature. Thermal sensors are located throughout the body to provide information about the local tissue temperature environment. The thermal sensors located within the skin are the most important, but there are others deeper in the body. Animal studies of *heat stress* and thermoregulation can sometimes be misleading, particularly for small rodents, which have less resilience when thermally stressed to produce *heat strain*.

Sweating plays a major role in environmental heat exchange (Nadel et al. 1971a, 1971b; Wenger 1983). Ogawa (1991) reported that there is little direct effect of IR on sweat glands. The direct effect is small, and is not dependent upon wavelength.

Quantitative approaches to describe heat exchange have taken several forms, depending upon the objective. There are complex equations of heat exchange, several types of heat stress indices (ACGIH 2005a, 2005b; Gagge 1937; Belding and Hatch 1955; ISO 1989a, 1989b, 2004a, 2004b), and effective temperature and thermal comfort indices. Radiant heating tends to play a greater role for indoor work environments, since sunlight provides the principal radiant heat load on the body for outdoor workers, and skin reflects significant solar radiation, which is predominantly visible, and IR-A radiation. By contrast, the IR-B and IR-C radiation that is dominant in hot industrial work environments is not significantly reflected by the skin as shown in Fig. 5 (Clark et al. 1959). Since heat strain depends not only upon the radiant heat load (i.e., upon irradiance levels) but also upon air temperature, humidity, and air movement, it is not possible to set simple irradiance limits to prevent heat stress and the resulting heat strain, unless worst case values were recommended—as was once the approach in developing microwave exposure limits. The latter type of guidance would be too restrictive in many cases for environmental infrared exposure or for radiant heating in cold environments. For the special case of infrared warming cabins or saunas, some level of heat strain is often intended, and limiting heat stress to prevent heat strain would be regularly exceeded. If deep body core temperature is of concern, as it might be for intense IR-A warming cabins, it could be monitored directly by specialized instrumentation. Rapid increase in body heat content may cause a prolonged attenuation in physical performance capacity and even predispose to heat related illnesses (Ilmarinen et al. 2004).

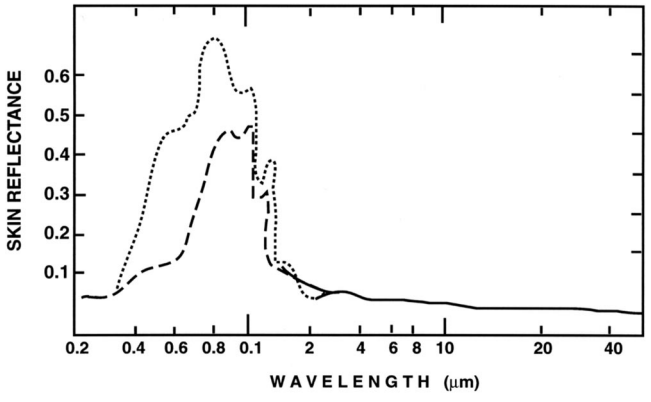

Fig. 5. Spectral reflectance of lightly (dotted line) and darkly (dashed line) pigmented human skin.

EXPOSURE LIMITS

The current ICNIRP (1997) guidelines for infrared radiation and recommendations for their application to IR-C wavelength ranges are as follows.

CORNEA AND LENS

To avoid thermal injury of the cornea and possible delayed effects on the lens of the eye (cataractogenesis), infrared radiation (770 nm $< \lambda <$ 3 μm) should be limited to 100 W m^{-2} (10 mW cm^{-2}) for lengthy exposures ($>$ 1,000 s), and to 1.8 $t^{-3/4}$ W cm^{-2} for shorter exposure durations:
or

$$E_{IR} \leq 18t^{-3/4} \text{ kW m}^{-2} \text{ for } t < 1{,}000 \text{ s}$$
$$E_{IR} \leq 1.8t^{-3/4} \text{ W cm}^{-2} \text{ for } t < 1{,}000 \text{ s} \quad (4a)$$

or

$$E_{IR} \leq 100 \text{ W m}^{-2} \text{ for } t > 1{,}000 \text{ s}$$
$$E_{IR} \leq 10 \text{ mW cm}^{-2} \text{ for } t > 1{,}000 \text{ s}. \quad (4b)$$

Exemption. In cold environments, these limits may be increased to 40 mW cm^{-2} at 0°C and approximately 30 mW cm^{-2} at 10°C for special applications, i.e., where infrared sources are used for radiant heating for reasons of comfort. When these limits are observed, lenticular temperature will not exceed 37°C (Stolwijk and Hardy 1977). If applying the guidelines to the IR-C wavelength range, it is stated that to protect the lens and cornea from excessive heating due to IR-C radiation, the application of the limit can be extended to include IR-C radiation,

i.e., the irradiance E_{IR}, which is compared to the exposure limit and is the total integrated radiant energy.

Retina (IR-A only)

Higher temperature sources may produce significant levels of IR-A radiation. For an infrared heat lamp or any near-IR source that provides no strong visual stimulus, the near-IR, or IR-A (770–1,400 nm), should be limited to:

$$\Sigma L_\lambda \times R(\lambda) \times \Delta\lambda \leq (6{,}000\ \mathrm{W} \times \mathrm{m}^{-2} \times \mathrm{sr}^{-1})/\alpha \quad (\text{for } t > 10\ \mathrm{s}) \quad (5a)$$

or

$$\Sigma L_\lambda \times R(\lambda) \times \Delta\lambda \leq (0.6\ \mathrm{W} \times \mathrm{cm}^{-2} \times \mathrm{sr}^{-1})/\alpha \quad (\text{for } t > 10\ \mathrm{s}), \quad (5b)$$

where L_λ is the spectral radiance, which is not to be averaged over angles of acceptance less than 11 mrad. $R(\lambda)$ is the retinal thermal weighting function defined in ICNIRP (1997) and α is the angular subtense of the source specified in units of milliradians. For very large sources α is limited to 100 mrad and the spectrally weighted radiance reduces to 60 kW cm^{-2} sr^{-1}, or 6 W cm^{-2} sr^{-1}.

For exposure durations less than 10 s, eqn (4) of the ICNIRP (1997) guidelines applies.

Since wavelengths greater than 1,400 nm do not contribute to the retinal hazard, this limit does not relate to IR-C exposure.

Thermal injury to the skin

To protect the skin from thermal injury from optical radiation in the wavelength range of 400 nm to 3 μm, the radiant exposure for durations less than 10 s should be limited to:

$$H = 20{,}000\, t^{1/4}\ \mathrm{J\ m}^{-2} \quad (6a)$$

or

$$H = 2\ t^{1/4}\ \mathrm{J\ cm}^{-2}. \quad (6b)$$

The guidance to address any concern about exposure to IR-C wavelength ranges is conservative.

To protect the skin from thermal injury due to IR-C radiation, the application of the limit can be extended to include IR-C radiation, i.e., the irradiance E_{IR}, which is compared to the exposure limit and is the total integrated visible and thermal radiation.

No formalized limit is provided for longer exposure durations because of the strong dependence upon ambient thermal environmental conditions. Depending upon initial skin temperature and ambient temperature, normal pain response and avoidance behavior will impose limits on duration of exposure. Much longer exposure durations are dominated by concerns of heat stress and heat strain. For lengthy exposures, the reader is referred to the appropriate guidelines for avoiding heat strain discussed below.

Other standards

There are a number of industrial health guidelines dealing with heat strain. Among these are the ISO (1989a, 2004a), the ASHRAE handbook (ASHRAE 1986), and ACGIH Threshold Limit Values and Biological Exposure Indices (ACGIH 2005b), which make use of the WBGT (Wet-Bulb-Globe-Temperature) index, the Heat Stress Index (HSI), or related formulations.

There is also a detailed consideration of skin exposure in a German national standard, DIN 33403 (part 3; DIN 2001) ("Climate at the workplace and its environment; assessment of the climate which can be tolerated by human beings"). In addition to the classical heat stress factors (air temperature, air velocity, humidity, thermal radiation, work load, clothing insulation factors), the DIN 33403 standard also deals with the isolated effects of thermal radiation. Two conditions are considered.

Heat strain. Maximum long-term irradiance values that can be tolerated and limit heat stress are promulgated in DIN 33403. These values are also dependent on the individual's work load.

Pain response. The borderline between an absence of pain and the onset of pain if the skin is irradiated by thermal radiation is shown as a plot of the irradiance (in kW m^{-2} "effective irradiance") as a function of the duration of exposure (clause 5.2) in Fig. 6. Thus, for a given exposure duration the irradiance that just produces a pain sensation can be determined. Similarly, for a given irradiance, one can determine the exposure duration for the onset of the pain sensation. The borderline is not provided as a fine line, but is shown as a spread of values. This reflects the fact that there are individual variations in the reaction to thermal radiation (Nadel et al. 1973). The figure is valid for acclimatized and non-acclimatized people. It is valid for the unprotected skin, and the values are independent of the size of the irradiated skin area. The wavelength range of the radiation is not specified. Therefore, it can be assumed that it covers all wavelengths that significantly contribute to the radiation exposure. Fig. 6 compares the currently recommended ICNIRP guidelines for limiting skin exposure with the pain thresholds in DIN 33403 (DIN 2001).

Note that "effective irradiance" used in infrared standards is the difference between the radiant flux density that is irradiated on the skin of a human being and

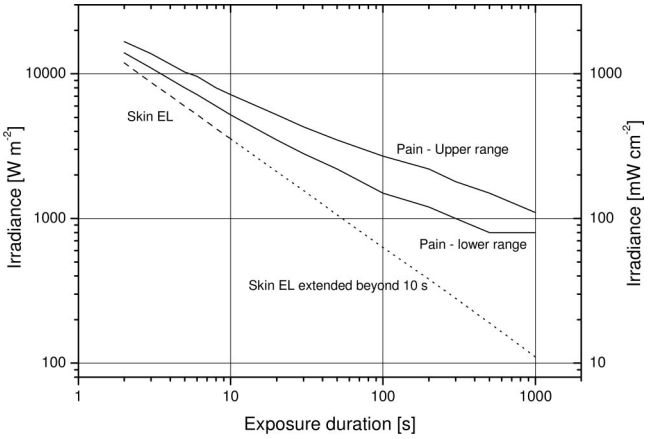

Fig. 6. Current ICNIRP exposure limit (EL) guidelines for IR-A and IR-B skin exposure are defined for exposure durations for up to 10 s. The function for the EL has been extended in the plot to exposure durations beyond 10 s for comparison with the change in slope of pain thresholds (top two curves) as adopted from the standard DIN 33403 (2001).

the radiant flux density that is emitted by the skin. As the skin temperature is assumed to be at 32°C, the radiation emission from the skin plays a minor role if the radiation source's temperature is much higher (e.g., several hundred degrees Celsius).

CONCLUSION

General

As stated in the ICNIRP "Guidelines on Limits of Exposure to Broad-band Incoherent Optical Radiation" (ICNIRP 1997), for all currently known arc and incandescent sources the contribution made by the IR-C spectral region (3–1,000 μm) is normally of no practical concern from a health hazard standpoint. However, there may be situations where substantial IR-C exposure is present and may contribute significantly to heat stress. Because heat stress depends upon other environmental factors such as air movement, temperature, and humidity—as well as the radiant thermal load—IR-C cannot be evaluated as an isolated factor. Heat stress must be evaluated using appropriate guidelines that consider all contributing factors.

Infrared exposure resulting in tissue heating should not be considered as a carcinogen or even a promoter for UV or chemical carcinogenesis. However, based upon some past experimental data, elevated skin temperatures might accelerate the evolution of skin cancer due to carcinogens such as UV radiation or chemical agents (Bain and Rusch 1943; Bain et al. 1943; Freeman and Knox 1964; Goss and Parsons 1976; van der Leun and de Gruijl 2002). These effects are however not limited to infrared radiation exposure, and apply to generally elevated skin temperatures. There is anecdotal evidence from past clinical observations of a higher risk of skin cancer development on skin suffering from erythema *ab igne*. In addition, there is some experimental evidence that tissue heating accelerates ultraviolet-induced photoaging of the skin (Dover et al. 1989; Kligman and Kligman 1984).

The relative contribution of IR-C radiation to temperature elevation of the crystalline lens of the eye, when compared to the heating from shorter wavelength infrared bands, would normally be small when considering incandescent and thermal sources. The infrared industrial heat cataract is recognized as largely the result of IR-A and IR-B exposure (ICNIRP 1997; Lydahl 1984).

As indicated in the ICNIRP guidelines (1997) in the section on IR-C measurement, the IR-C radiation can be included in the determination of the exposure level, which is then compared to the exposure limits for protection of the anterior parts of the eye (cornea and lens) and for protection against thermal injury of the skin. Exposure limits for protection against thermal injury (burns) of the skin for exposure durations greater than 10 s are not provided since ambient temperature would greatly impact the threshold irradiance for thermal injury. Thermal pain is induced by skin temperatures that are lower than the temperatures needed to produce a thermal burn, and this pain would limit the exposure so that a thermal injury is prevented. It should be noted, however, that some medications, drugs and alcohol can decrease the pain sensation, and thus individuals under such influence should not be exposed to significant levels of optical radiation.

If it is necessary to limit exposure to visible and infrared radiation for individuals who would not experience the natural aversion responses (i.e., pain sensation), the values given in DIN 33403 for thermal pain for prolonged exposure durations may be consulted as a reference point to limit exposure. A very conservative irradiance guideline for continuous exposure is provided by the whole-body IR-C laser irradiation limit of 100 W m^{-2}, and this limit, while overly conservative for cooler environments, would preclude delayed effects such as erythema *ab igne.* A general application of this guideline would adversely impact many useful applications of IR-C in industry and in cooler environments. An upper limit where continuous exposure would produce adverse heat stress even in cool environments is 1 kW m^{-2}.

Lamp designs and infrared technology are undergoing continuous evolution, using newer and often more powerful sources of optical radiation. Because of the rapid growth in the use of new types of special-purpose radiant heaters and warmers, the importance of infrared

radiation exposure limits will increase. There is an urgent need for research related to damage mechanisms of infrared cataract and the dose response curve for producing erythema *ab igne*. Research is needed to further study the potential delayed effects (and benefits) of IR-C cabins and for the manufacturers to prevent localized areas of irradiation and to limit the irradiance to that which is necessary, and to properly inform the user that he/she should not overuse infrared warming cabins and perhaps to perform self examination to prevent persistent erythema, which may develop into erythema *ab igne*.

Infrared cabins

Infrared warming cabins represent a special type of exposure situation, as irradiance level of the skin can be relatively high, often combined with elevated air temperatures, and exposure is elective. To date, there have been no reported cases of erythema *ab igne* associated with the continued use of IR cabins. Indeed, it would be highly unlikely for erythema *ab igne* to result from typical use patterns of IR warming cabins. Erythema *ab igne* has been characteristically induced by intense chronic infrared exposure for hours each day. Cabins with large area heaters have much lower surface temperatures (about 50 to 70°C), and the irradiance is much lower than in cabins with higher temperature heating elements.

Because of the long history of sauna use, it has long been presumed that there are not adverse health effects from conventional sauna (Teir et al. 1976). However, there is also an absence of controlled clinical trials or prospective epidemiological studies that clearly show the health effects of the sauna. There are also no controlled studies of the benefit of infrared warming cabins. However, some insight into possible risks of excessive exposure may be drawn from more general studies of the effects of heat upon biological systems.

Recommendations

Further research is needed to determine if routine IR-C exposures that produce heat stress and some degree of heat strain are without risk.

With regard to potential adverse delayed effects as revealed by experimental research, persons should be discouraged from heating of the skin either simultaneously with or within 24 h after UVR exposure from artificial sources and sun-bathing.

Individuals who may be at risk from hyperthermia, such as individuals suffering from cardiovascular disease, should seek medical advice before use of infrared warming cabins.

When persistent erythema (reddening of the skin lasting more than a day) and netlike color changes persist after regular exposure to infrared radiation, exposure should not be repeated and medical advice should be sought to prevent development of erythema *ab igne.*

Persons with compromised heat pain sensation should not use infrared warming cabins, which include persons under the influence of alcohol and tranquilizers.

Acknowledgments—This statement was prepared by ICNIRP Standing Committee SC-IV Task Group on the Far-Infrared: Jean-Pierre Césarini, Maila Hietanen, Hans Meffert, Tsutomo Okuno, Helmut Piazeno, Karl Schulmeister, Harald Siekmann, David Sliney, Per Soderberg, Jan Stolwijk, Bruce Stuck, Frederick Urbach, and Marko Weber.

REFERENCES

Adair ER. Microwaves and thermoregulation. New York: Academic Press; 1983.

Adair ER, Spiers DE, Rawson RO, Adams BW, Sheldon DK, Pivirotto PJ, Akel GM. Thermoregulatory consequences of long-term microwave exposure at controlled ambient temperatures. Bioelectromagnetics 6:339–363; 1985.

Adair ER, Kelleher SA, Mack GW, Morocco TS. Thermophysiological responses of human volunteers during controlled whole-body radio frequency exposure at 450 MHz. Bioelectromagnetics 19:232–245; 1998.

Allen RG, Polhamus GD. Ocular thermal injury from intense light. In: Wolbarsht ML, ed. Laser applications in medicine and biology. New York: Plenum Press; 1989.

American Conference of Government Industrial Hygienists. Documentation of the threshold limit values for chemical substances and physical agents in the workroom environment. Cincinnati, OH: ACGIH; 2005a.

American Conference of Government Industrial Hygienists. Threshold limit values for chemical substances and physical agents in the workroom environment. Cincinnati, OH: ACGIH; 2005b.

American Society of Heating, Refrigerating and Air Conditioning Engineers. Physiological principles and thermal comfort. In: ASHRAE handbook 1986 fundamentals. Atlanta, GA: ASHRAE; 1986: 8.1–8.29.

Anderson RR, Parrish JA. Optical properties of human skin. In: Reagan JD, Parrish JA, eds. The science of photomedicine. New York and London: Plenum Press; 1982: 172 (Table 6-2).

Bain JA, Rusch HP. Carcinogenesis with ultraviolet radiation of wavelength 2,800–3,400 A. Cancer Res 3:425–430; 1943.

Bain JA, Rusch HP, Line BE. The effect of temperature upon ultraviolet carcinogenesis with wavelength 2,800–3,400 A. Cancer Res 3:610–612; 1943.

Barthelmess GA, Borneff J. Ueber die genetische Schaedigung der Augenlinse durch Waermestrahlung. Graefes Arch Ophth 160:641–652; 1959 (in German).

Belding HS, Hatch TF. Index for evaluating heat stress in terms of resulting physiological strain. Heating, Piping and Air Conditioning 27:129–136; 1955.

Berglund LG. Characterizing the thermal environment. In: Adair ER, ed. Microwaves and thermoregulation. New York: Academic Press; 1983: 15–31.

Beuerman RW, Tanelian DL. Corneal pain evoked by thermal stimulation. Pain 7:1–14; 1979.

Blick DW, Adair ER, Hurt WD, Sherry CJ, Walters TJ, Merritt JH. Thresholds of microwave-evoked warmth sensation in human skin. Bioelectromagnetics 18:403–409; 1997.

Bligh J, Johnson KG. Glossary of terms for thermal physiology. J Applied Physiol 35:941–961; 1973.

Boukamp P, Popp S, Bleuel K, Tomakidi E, Bürkle A, Fusenig NE. Tumorigenic conversion of immortal human skin keratinocytes (HaCaT) by elevated temperature. Oncogene 18:5638–5645; 1999.

Brownell AS, Parr WH, Hysell DK. Skin and carbon dioxide laser radiation. Arch Environ Health 18:437–442; 1969.

Clark C, Vinegar R, Hardy J. Goniometer spectrometer for the measurement of diffuse reflectance and transmittance of skin in the infrared spectral region. J Opt Soc Am 43:993–998; 1959.

Commission Internationale de l'Eclairage (International Commission on Illumination). International lighting vocabulary. Vienna: CIE; Publication 17.4; 1987.

Cook HF. The pain threshold for microwave and infra-red radiations. J Physiol 118:1–11; 1952.

Cunningham DJ. An evaluation of heat transfer through the skin in extremity. In: Hardy JD, Gagge AP, Stolwijk JAJ, eds. Physiological and behavioral temperature regulation. Springfield, IL: Charles. C. Thomas; 1970: 302–315.

Davis TP. The heating of skin by radiant energy. In: Herzfeld CM, Hardy JD, eds. Temperature, its measurement and control in science and industry. New York: Reinhold Publishing Corp 3; 1963: 149–169.

Dawson W. The thermal excitation of afferent neurons in the mammalian cornea and iris. In: Herzfeld CM, Hardy JD, eds. Temperature, its measurement and control in science and industry. New York: Reinhold Publishing Corp, 3; 1963: 199–210.

Derksen WL, Monahan TI, Delhery GP. The temperature associated with radiant energy skin burns. In: Herzfeld CM, Hardy JD, eds. Temperature, its measurement and control in science and industry. New York: Reinhold Publishing Corp, 3; 1963: 171–175.

Dewhirst DW, Lora-Michiels M, Viglianti BL, Dewey WC, Repacholi M. Carcinogenic effects of hyperthermia. Int J Hyperthermia 19:236–251; 2003a.

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

DIN 5031-10. Strahlungsphysik im optischen Bereich und Lichttechnik—Teil 10: Photobiologisch wirksame Strahlung, Groeβen, Kurzzeichen und Wirkungsspektren. Berlin: Deutsche Institut fuer Normung; 2000.

DIN 33403-3. Klima am Arbeitsplatz und in der Arbeitsumgebung—Teil 3: Beurteilung des Klimas im Warm—und Hitzebereich auf der Grundlage ausgewaehlter Klimasummenmasze. Berlin: Deutsche Institut für Normung; 2001 (in German).

Dover JS, Philips TJ, Kenneth AA. Cutaneous effects and therapeutic uses of heat with emphasis on infrared radiation. J Am Acad Dermatol 20:278–286; 1989 (in German).

Edwards C, Gaskell SA, Hill SA, Heggie R, Pearse AD, Marks R. Effects on human epidermis of chronic suberythermal exposure to pure infrared radiation. Arch Dermatol 135:608–609; 1999.

Findlayson GR, Sams WM, Smith JG. Erythema ab igne. A histopathological study. J Invest Dermatol 46:104–107; 1966.

Fitzpatrick TB, ed. Fitpatrick's dermatology in general medicine. New York: McGraw-Hill; 1999.

Fitzpatrick TB, Pathak MA, Harber LC, Seiji M, Kukita A, eds. Sunlight and man, normal and abnormal photobiologic responses. Tokyo: University of Tokyo Press; 1974.

Freeman RG, Knox JM. Influence of temperature on ultraviolet injury. Arch Dermatol 89:858–864; 1964.

Gagge AP. A new physiological variable associated with sensible and insensible perspiration. Am J Physiol 120:277–287; 1937.

Gagge AP, Burton AC, Bazett HC. A practical system of units for the description of the heat exchange of man with his environment. Science 94:428–430; 1941.

Goss P, Parsons PG. Temperature-sensitive DNA repair of ultraviolet damage in human cell lines. Int J Cancer 17:296–303; 1976.

Greene LC, Hardy JD. Adaptation to thermal pain in the skin. Applied Physiol 17:693–696; 1962.

Ham WT Jr, Mueller HA. The photopathology and nature of the blue light and near-UV retinal lesions produced by laser and other optical sources. In: Wolbarsht ML, ed. Laser applications in medicine and biology. New York: Plenum Publishing Corp; 1989: 191–246.

Ham WT Jr, Mueller HA, Sliney DH. Retinal sensitivity to damage from short wavelength light. Nature 260:153–155; 1976.

Hardy JD. Pain following step increase in skin temperature. In: Kenshato DR, ed. The skin senses. Springfield, IL: Charles C. Thomas; 1968: 444–456.

Hardy JD. Regulation of body temperature in man—an overview. In: Stolwijk JAJ, ed. Energy conservation strategies in buildings. New Haven, CT: Yale University Printing Service; 1978: 14–37.

Hardy JD, Bard P. Body temperature regulation. In: Mountcastle VB, ed. Med Physio. St. Louis, MO: Mosby CV Co; 1974.

Hardy JD, Oppel TW. Studies in temperature sensation III. The sensitivity of the body to heat and the spatial summation of the warmth sense organ responses. J Clinical Investigation 16:533–540; 1937.

Hardy JD, Jacobs I, Meixner MD. Thresholds of pain and reflex contractions as related to noxious stimulation. Applied Physiol 5:725–739; 1953.

Hardy JD, Hammel HT, Murgatroyd D. Special transmittance and reflectance of excised human skin. J Appl Physiol 9:257; 1956.

Hardy JD, Wolff HG, Goodell H. Pain reactions and sensations. New York: Hafner Publishing Co; 1962.

Health Council of the Netherlands Committee on Optical Radiation. Health-based exposure limits for electromagnetic radiation in the wavelength range from 100 nanometer to 1 millimeter. The Hague: Health Council of the Netherlands; Report 1993/09E; 1993.

Helm TN, Spigel GT, Helm KF. Erythema Ab Igne caused by a car heater. Cutis 59: 81–82; 1997.

Hendler E, Hardy JD. Infrared and microwave effects on skin heating and temperature sensation. IRE Transactions on Medical Electronics ME-7:143–152; 1960.

Hendler E, Hardy JD, Murgatroyd D. Skin heating and temperature sensation produced by infrared and microwave irradiation. In: Herzfeld CM, Hardy JD, eds. Temperature, its measurement and control in science and industry. New York: Reinhold Publishing Corp; 1963: 211–230.

Henriques Jr FC. The predictability and the significance of thermally induced rate processes leading to irreversible epidermal injury. Am J Path 23:489–502; 1948.

Henriques Jr FC, Moritz AR. Study of thermal injury: 1. The conduction of heat to and through skin and the temperatures attained therein. A theoretical and an experimental investigation. Am J Path 23:531–549; 1947.

Hurwitz RM, Tisserand ME. Erythema ab igne. Arch Dermatol 123:21–22; 1987.

Ilmarinen R, Lindholm H, Koivistoinen K, Hellsten P. Physiologic evaluation of chemical protective suit systems (CPSS) in hot conditions. Int J Occup Saf Ergon 10:215–226; 2004.

International Agency for Research on Cancer. IARC monographs on the evaluation of carcinogenic risks to humans, solar and ultraviolet radiation. Lyon: International Agency for Research on Cancer; 55; 1992.

International Commission on Non-Ionizing Radiation Protection. Guidelines on limits of exposure to laser radiation of wavelengths between 180 nm and 1,000 μm. Health Phys 71:804–814; 1996.

International Commission on Non-Ionizing Radiation Protection. Guidelines of limits of exposure to broad-band incoherent optical radiation (0.38 to 3 μm). Health Phys 73:539–554; 1997.

International Commission on Non-Ionizing Radiation Protection. Revision of guidelines on limits of exposure to laser radiation of wavelengths between 400 nm and 1.4 μm. Health Phys 79:804–814; 2000.

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

International Standards Organization. Hot environments—estimation of the heat stress on working man, based on the WBGT-Index (Wet Bulb Globe Temperature). Geneva: ISO 7243; 1989a.

International Standards Organization. Hot environments—analytical determination and interpretation of thermal stress using calculation of required sweat rate. Geneva: ISO 7933; 1989b.

International Standards Organization. Ergonomics of the thermal environment—analytical determination and interpretation of heat stress using calculation of the predicted heat strain. Geneva: ISO 7933; 2004a.

International Standards Organization. Ergonomics of the thermal environment—determination of metabolic rate. Geneva: ISO 8996; 2004b.

Juhlin L, Civier A, Shroot S, Hensby C. Effect of infrared irradiation on the recoverable levels of free arachidonic acid and prostaglandins in human forearm skin. J Invest Dermatol 81:297–300; 1983.

Karu TI. Photobiological fundamentals of low-power laser therapy. IEEE J Quant Electr QE-23:1703–1717; 1987.

Karu TI. Photobiology of low-power laser therapy. London: Gordon and Breach; 1998.

Kligman LH, Kligman AM. Reflections on heat. British J Dermatol 110:369–375; 1984.

Kojima M, Okuno T, Miyakoshi M, Sasaki K, Takahashi N. Environmental temperature and cataract progression in experimental rat cataract models. Dev Ophthalmol 35:125–134; 2002.

Licht S. Therapeutic electricity and ultraviolet radiation. New Haven: New Haven Press; 1967.

Lydahl E. Infrared cataract (with 6 appendices). Acta Ophthalmol Suppl 166:1–63; 1984.

Meffert H, Piazena H. Effects of artificial infrared radiation on human beings. Akt Dermatol 28:187–192; 2002.

Menezes S, Coulomb B, Lebreton C, Dubertret L. Noncoherent near infrared radiation protects normal human dermal fibroblasts from solar ultraviolet toxicity. J Invest Dermatol 111:629–633; 1998.

Miranda MN. The geographic factor in the onset of presbyopia. Tr Am Ophthalmol Soc 77:603–621; 1979.

Miranda MN. Environmental temperature and senile cataract. Tr Am Ophthalmol Soc 78:255–264; 1980.

Moritz AR, Henriques Jr FC. Studies of thermal injury: II. The relative importance of time and surface temperature in the causation of cutaneous burns. Am J Path 23:695–720; 1947.

Nadel ER, Bullard RW, Stolwijk JAJ. Importance of skin temperature in the regulation of sweating. J Applied Physiol 31:80–87; 1971a.

Nadel ER, Mitchell JW, Saltin B, Stolwijk JAJ. Peripheral modifications to the central drive for sweating. J Applied Physiol 31:828–833; 1971b.

Nadel ER, Mitchell JW, Stolwijk JAJ. Differential thermal sensitivity in the human skin. Pflugers Archiv 340:71–76; 1973.

Nielsen B, Nielsen M. Influence of passive and active heating on the temperature regulation of man. Acta Physiologica Scandinavica 64:323–331; 1965.

Ogawa T. Dynamic sweating response of man to infrared radiation in various spectral regions. Int J Biometereol 35:18–23; 1991.

Okuno T. Thermal effect of infra-red radiation on the eye: a study based on a model. Ann Occup Hyg 35:1–12; 1991.

Okuno T. Thermal effect of visible light and infra-red radiation (IR-A, IR-B and IR-C) on the eye: a study of infra-red cataract based on a model. Ann Occup Hyg 38:351–359; 1994.

Okuno T, Kojima M, Hata I, Sliney DH. Temperature rises in the crystalline lens from focal irradiation. Health Phys 88:214–222; 2005.

Piazena H, Meffert H. Therapie mit Infrarotstrahlen—Physikalische Grundlagen und Anwendung in der Sauna und in Infrarotgeraeten. In: Buehring M, Kemper FH, eds. Naturheilverfahren 1. Berlin, Heidelberg, New York: Springer-Verlag; 03-09; 2001: 1–21.

Pitts DG, Cullen AP. Determination of infrared radiation levels for acute ocular cataractogenesis. In: Albrecht von Graefes. Arch Klin Ophthalmol 217:285–297; 1981.

Priebe LA, Welch AJ. Asymptotic rate process calculations of thermal injury to the retina following laser irradiation. F Biomech Eng 100:49–54; 1978.

Pujol JA, Lecha M. Photoprotection in the infrared range. Photoderm Photoimmunol Photomed 9:275–278; 1992/1993.

Roth D, London M. Acirdine probe study into synergistic DNA-denaturing action of heat and ultraviolet light in squamous cells. J Invest Dermatol 69:369–372; 1977.

Sasaki K, Sasaki H, Kojima M, Shui YB, Hockwin O, Jonasson F, Cheng HM, Ono M, Katoh N. Epidemiological studies on UV-related cataract in climatically different countries. J Epidemiol 9:33–38; 1999.

Schulze H-J, Schmidt R, Mahrle G. Das Infrarot-(IR)-Erythem. Z Hautkr 60:940–944; 1985.

Scott JA. A finite model of heat transport in the human eye. Phys Med Biol 33:227–241; 1988a.

Scott JA. The computation of temperature rises in the human eye induced by infrared radiation. Phys Med Biol 33:243–257; 1988b.

Shahrad P, Marks R. The wages of warmth. Changes in erythema ab igne. Br J Dermatol 97:179–186; 1977.

Shimada SG, Stitt JT. Body temperature regulation during euthermia and hyperthermia. In: Adair ER, ed. Microwaves and thermoregulation. New York: Academic Press; 1983: 139–160.

Sliney DH. Physical factors in cataractogenesis: ambient ultraviolet radiation and temperature. Invest Ophthalmol Vis Sci 27:781–790; 1986.
Sliney DH. Geometrical gradients in the distribution of temperature and absorbed ultraviolet radiation in ocular tissues. Dev Ophthalmol 35:40–59; 2002.
Sliney DH, Freasier BC. Evaluation of optical radiation hazards. Applied Optics 12:1–24; 1973.
Sliney DH, Wolbarsht ML. Safety with lasers and other optical radiation sources. New York: Plenum Press; 1980.
Stolwijk JAJ. Mathematical models of thermoregulation. Annals of the New York Academy of Sciences 335:98–106; 1980.
Stolwijk JAJ, Hardy JD. Temperature regulation in man—a theoretical study. Pflugers Archiv 291:129–162; 1966.
Stolwijk JAJ, Hardy JD. Control of body temperature. In: Lee DHK, ed. Handbook of physiology, section 9. Bethesda, MD: American Physiological Society; 1977: 45–68.
Teir H, Collan Y, Valtakari P, eds. Sauna studies: Papers presented at the VI International Sauna Congress, Helsinki 15–17 August 1974. First edition. Helsinki: Vammalan Kirjapaino; 1976.
UNEP/WHO/IRPA (United Nations Environment Programme/World Health Organization/International Radiation Protection Association). Lasers and optical radiation. Geneva: World Health Organization; Environmental Health Criteria 23; 1982.
UNEP/WHO/IRPA (United Nations Environment Programme/World Health Organization/International Radiation Protection Association). Ultraviolet radiation. Geneva: World Health Organization; Environmental Health Criteria; 1994.
van der Leun JC, de Gruijl FR. Climate change and skin cancer. Photochem Photobiol Sci 1:324–326; 2002.
Vaupel P, Krüger W. Waermetherapie mit wassergefilterter Infrarotstrahlung—Grundlagen und Anwendungsmöglichkeiten. Stuttgart: Hippokrates Verlag; 1992 (in German).
Vaupel P, Krüger W. Waermetherapie mit wassergefilterter Infrarotstrahlung—Grundlagen und Anwendungsmöglichkeiten. Stuttgart: Hippokrates Verlag; 1995 (in German).
von Ardenne M. Principles and concept 1993 of the Systemic Cancer Multistep Therapy (SCMT). Extreme whole-body hyperthermia using the infrared-A technique IRATHERM 200—selective thermosensitisation by hyperglycemia—circulatory back-up by adapted hyperoxemia. Strahlenther Onkol 170:581–589; 1994.
von Ardenne M, ed. Systemische Krebs-Mehrschritt-Therapie—Hyperthermie und Hyperglykämie als Therapiebasis. Stuttgart: Hippokrates Verlag; 1997.
Vos JJ, van Norren D. Weighing the relative significance of three heat dissipation mechanisms to produce cataract. Lasers Light Ophthalmol 6:107–114; 1994.
Walsder EJ, Hargis AM. Chronic moderate heat dermatitis (erythema ab igne) in five dogs, three cats and one silvered langur. Vet Derm 13:283; 2002.
Walters TJ, Blick DW, Johnson LR, Adair ER, Foster KR. Heating and pain sensation produced in human skin by millimeter waves: comparison to a simple thermal model. Health Phys 78:259–267; 2000.
Weale R. Human ocular aging and ambient temperature. Br J Ophthalmol 65(12):869; 1981.
Wehner H, von Ardenne A, Kaltofen S. Whole-body hyperthermia with water-filtered infrared radiation: technical-physical aspects and clinical experiences. Int J Hyperthermia 17:19–30; 2001.
Wenger CB. Circulatory and sweating responses during exercise and heat stress. In: Adair ER, ed. Microwaves and thermoregulation. New York: Academic Press; 1983: 251–276.
Werner J, Buse M. Temperature profiles with respect to inhomogeneity and geometry of the human body. J Applied Physiol 65:1110–1118; 1988.
Whittow GC. Comparative physiology of thermoregulation. New York: Academic Press; Vol II; 1971.
Wolbarsht ML. The effects of optical radiation on the anterior structures of the eye. In: Tengroth B, Epstein D, eds. Current concepts in ergophthalmology. Stockholm: Karolinska Institute, Department of Ophthalmology; 1978: 29–46.
Wolbarsht ML. Cataract from infrared lasers: evidence for photochemical mechanisms. Lasers Light Ophthalmol 4:91–96; 1992.

■ ■