INTERNATIONAL COMMISSION ON NON-IONIZING RADIATION PROTECTION

ICNIRP GUIDELINES

ON LIMITS OF EXPOSURE TO INCOHERENT VISIBLE AND INFRARED RADIATION

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ICNIRP GUIDELINES ON LIMITS OF EXPOSURE TO INCOHERENT VISIBLE AND INFRARED RADIATION

International Commission on Non-Ionizing Radiation Protection*

Abstract—**Guidelines for exposure to visible and infrared radiation were first proposed by ICNIRP in 1997. Related guidelines on limits of exposure to ultraviolet radiation (UVR) and laser radiation have been published. This document presents a revision of the guidelines for broadband incoherent radiation.**
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INTRODUCTION

SINCE IT is necessary to consider contributions of UVR to retinal hazard assessment for incoherent broadband radiation, the action spectra recommended in these guidelines for the retina extend to 300 nm. Thus, there is an overlap in terms of wavelength range of these guidelines with the guidelines for ultraviolet radiation that only consider skin, cornea and lens (ICNIRP 2004).

Since the publication of the ICNIRP Guidelines on Limits of Exposure to Broad-Band Incoherent Optical Radiation (0.38 to 3 μm) in 1997 (ICNIRP 1997), further research on thermally induced injury of the retina has led to the need to revise the guidance given so far. In particular, the exposure limit dependence upon source size is now a function of the exposure duration. Further, the retinal thermal hazard function has been revised. The specific rationale for these changes is provided in Appendix A.

PURPOSE AND SCOPE

The purpose of these guidelines is to establish the maximum levels of exposure to incoherent optical radiation from artificial and natural sources with the exception of lasers. Exposure below these maximum levels is not expected to cause adverse effects.

The guidelines assist the development of principles of protection to the eyes and the skin against optical radiation hazards. Separate guidelines are defined for exposure to laser radiation (ICNIRP 1996; ICNIRP 2000). The guidelines are intended for use by the various experts and national and international bodies who are responsible for developing regulations, recommendations, or codes of practice to protect workers and the general public from the potentially adverse effects of optical radiation.

The exposure limits given are for wavelengths from 380 nm to 1 mm. However, the action spectrum for photochemically induced photoretinopathy extends to 300 nm in the ultraviolet radiation (UVR) wavelength range and should be applied for the evaluation of broad-band sources which may also emit UVR. Separate guidelines apply for the exposure of the skin and the anterior parts of the eye to UVR (ICNIRP 2004).

Injury thresholds are well defined for the effects that are in the scope of these guidelines. Therefore, the guidelines for optical radiation in general do not differentiate between exposure to professionals and exposure to the general public. The only exception concerns the action spectrum for photochemically induced photoretinopathy, where for children below 2 years of age the aphakic hazard function should be applied.

The exposure limits apply to the full wavelength range of up to 1 mm even though the determination of the exposure level can be limited to the wavelength range below 3,000 nm. Non-laser sources do not emit enough power in the wavelength region above 3,000 nm to cause a health hazard other than the possibility of heat stress (ICNIRP 2006).

There is paucity of threshold data for long term chronic exposure. Therefore, the guidelines are based on the dataset of threshold data for short delay (up to 48 h) onset of damage. However, current knowledge suggests that there are no effects of chronic exposure to infrared radiation (IRR) below the exposure limits provided.

These guidelines do not apply to deliberate exposure for medical (diagnostic or treatment) purposes.

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Detailed measurement procedures and calculation methods are beyond the scope of this document and are provided elsewhere (Sliney and Wolbarsht 1980; UNEP et al. 1982; McCluney 1984; CIE and ICNIRP 1998; Schulmeister 2001; Henderson and Schulmeister 2004).

QUANTITIES, SYMBOLS AND UNITS

Electromagnetic radiation in the wavelength range between 100 nm and 1 mm is widely termed "optical radiation." A subdivision of this spectral band is defined by the International Commission on Illumination (CIE 2011) and can be useful in discussions of the photobiological effects of optical radiation, although the predominant effects have less sharply defined spectral limits. The optical radiation waveband consists of ultraviolet, visible, and infrared radiation. According to the CIE, ultraviolet radiation (UVR) ranges between 100–400 nm (CIE 2011). A precise border between UVR and visible radiation cannot be defined because visual sensation at wavelengths shorter than 400 nm is noted for very bright sources. Similarly, a precise border between visible and infrared radiation cannot be defined because visual sensation at wavelengths greater than 780 nm is noted for very bright sources. The infrared region is often subdivided into IR-A (780–1,400 nm), IR-B (1,400–3,000 nm), and IR-C (3,000 nm–1 mm).

Exposure limits for optical radiation are expressed in radiometric quantities, depending on the tissue and damage mechanism (Table 1). A complete definition of symbols used is found in Appendix B.

Exposure limits that relate to the skin, the anterior part of the eye or to potential retinal photochemical injury under the "small source" condition are expressed in the radiometric quantities of *irradiance, E* (W m^{-2}) and *radiant exposure*, *H* (J m^{-2}). The quantity of radiant exposure is also sometimes referred to as dose, and irradiance can be understood as dose-rate. *Radiance, L* (W m^{-2} sr^{-1}), and *radiance dose D* (J m^{-2} sr^{-1}) also referred to as time-integrated radiance, are used for exposure limits that relate to retinal injury from extended sources, i.e. sources that result in an image on the retina. Radiance is convenient since it is directly related to the irradiance at the retina (Sliney and Wolbarsht 1980; Henderson and Schulmeister 2004) For completeness it is noted that in the CIE International Lighting Vocabulary (CIE 2011), the symbol for time integrated radiance is L_t and the symbol *D* is reserved for the detectivity of a detector. Since the ICNIRP guidelines make use of additional subscripts, a dedicated single letter symbol was deemed advantageous for the radiance dose, *D*.

Table 1. Radiometric quantities.

Quantity	Symbol	Unit
Power	Φ	W
Energy	Q	J
Irradiance	E	W m^{-2}
Radiant exposure	H	J m^{-2}
Radiance	L	W m^{-2} sr^{-1}
Radiance dose/Time-integrated radiance	D	J m^{-2} sr^{-1}

Depending on the type of interaction, the effectiveness to produce an adverse effect can be strongly wavelength dependent. This wavelength dependence is accounted for by an action spectrum that is used to spectrally weigh the respective exposure quantities. These weighted exposure quantities are then referred to as "effective" quantities, for instance, effective irradiance.

SOURCES AND EXPOSURE CONDITIONS

Optical radiation from artificial sources is used in a wide variety of industrial, consumer, scientific and medical applications, and in most cases the light and infrared energy emitted is not hazardous to the general public. However, people with certain medical conditions may be at risk from exposures that are otherwise innocuous. In certain unusual exposure conditions, potentially hazardous exposures are accessible. Examples include; arc welding, use of some arc lamps in research laboratories, very high intensity flash lamps in photography, infrared lamps for surveillance and heating, a number of medical diagnostic applications, and even printing and photocopying. Excessive light and IRR are typically filtered or baffled to reduce discomfort. Where sufficient visible light is present, the natural and active aversion response of the eye to bright light will substantially reduce potentially hazardous exposure. Moreover, if the total irradiance is sufficient, the thermal discomfort, sensed by the skin and cornea, usually triggers an aversion response and tends to limit exposure to a few seconds or less (ICNIRP 2006).

Many intense optical sources also produce significant amounts of UVR, which may be hazardous to the eye and skin. This hazard should be separately assessed, using UVR guidelines (ICNIRP 2004). It should be noted, however, that the UVR guidelines do not provide exposure limits for photoretinopathy. The risk for photochemically induced photoretinopathy may be evaluated by applying the blue-light photochemical exposure limits given in these guidelines.

Lamps are used in many consumer and office appliances, but because of the need for visual comfort, these sources rarely pose a real hazard. The properties of laser emissions generally differ significantly from those of broad-band incoherent optical sources, and so the exposure limits for broad-band optical sources are necessarily expressed differently from those applicable to lasers. In addition, laser guidelines and product safety standards

(ICNIRP 1996, 2000; IEC 2007) incorporate assumptions of exposure that may not apply to conventional optical sources. 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 apart from the wavelength of the laser. By contrast, evaluation of the potential hazards of broadband conventional light sources requires spectroradiometric data to apply several different photobiological action spectra, as well as knowledge of the exposure geometry. The action spectrum expresses the spectral relative biological efficiency of optical radiation delivered from outside the eye, thus depending on the attenuation of optical radiation before the target tissue and the relative sensitivity of the target tissue. The action spectra are specific to different ocular structures.

BIOLOGICAL EFFECTS

The eye and skin are the organs most susceptible to damage by optical radiation. The type of effect, injury thresholds, and damage mechanisms vary significantly with wavelength (Fig. 1).

The effects may overlap and must be evaluated independently. Action spectra exist for each effect.

Overview of types of damage

At least nine separate types of damage to the eye and skin may be caused by visible and infrared optical radiation:

Eye.

1. Thermal damage of the cornea, approximately 1,400 nm–1 mm;
2. Thermal damage of the iris, approximately 380 nm–1,400 nm;
3. Near-infrared thermal damage of the crystalline lens, approximately 800–3,000 nm;
4. Thermal damage of the retina (380–1,400 nm);
5. "Blue-light" photochemical damage of the retina, principally 380–550 nm; 300–550 nm for the aphakic eye (Ham et al. 1976; Ham Jr 1989; Lund et al. 2006). This is also referred to as Type II photochemical retinal damage; and
6. Photochemical retinal damage from chronic exposure to bright light, Type I photochemical retinal damage (Noell et al. 1966; Williams and Howell 1983; Mellerio 1994).

Ocular damage threshold from optical radiation before adulthood is modified by a different spectral transmittance and a different sensitivity (Söderberg 2011).

Skin.

1. Thermal damage of the skin, burns, approximately 380 nm–1 mm;
2. Damage to the skin by photosensitization. This is generally more typical of UVR wavelengths (less than 380 nm), although such photosensitized reactions can extend to approximately 700 nm, possibly as a side-effect of certain medications (Fitzpatrick et al. 1974; Magnus 1976; Diffey 1982); and
3. Photoallergic reactions in which an antigen, activated by exposure to optical radiation, preferentially UVR, causes an immune reaction (Darvay et al. 2001)

Ultraviolet radiation poses the primary known environmental risk factor for skin cancer. Visible radiation does not contribute to skin cancer risk (IARC 1992; UNEP et al. 1994; ICNIRP 2004, 2007).

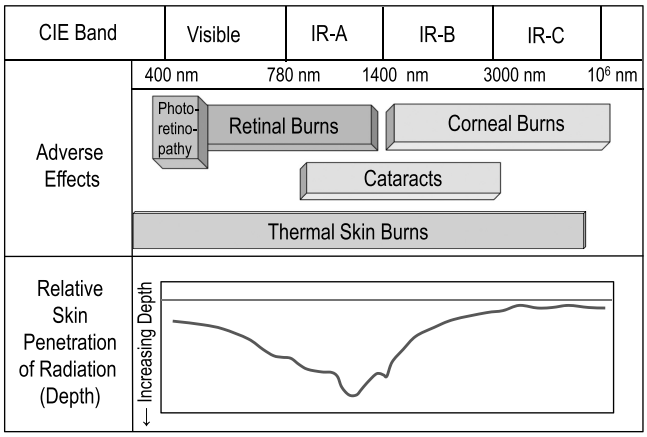

Fig. 1. Potential adverse biological effects and penetration depth of visible and infrared radiation (adapted from Sliney Wolbarsht 1980).

Characteristics of photochemical interaction mechanisms

The threshold radiant exposure is subject to the principle of *reciprocity,* the Bunsen–Roscoe Law of Photobiology, stating that the effect depends only on the dose (the radiant exposure;, i.e., the product of irradiance and exposure duration). Thus, blue-light retinal injury (photochemically induced photoretinopathy) can result from viewing either an extremely bright light for a short duration or a less bright light for a longer duration. The observation of reciprocity helps to distinguish these effects from thermal injuries (see below). For photochemical injury of the retina, the sensitivity peaks at approximately 440 nm for the eye with an intact crystalline lens, a phakic eye (Ham et al. 1976).

Characteristics of thermal interaction mechanisms

Thermal injury, unlike photochemical injury, does not show reciprocity between irradiance and exposure

duration. Thermal injury is strongly dependent upon heat conduction from the irradiated tissue. It requires an intense exposure to cause tissue coagulation. When exposure is less intense, surrounding tissue conducts 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. Normally, a temperature of at least 45°C is necessary to produce a thermal burn. Higher temperatures are required for thermal injury to result from exposures of shorter duration, e.g., about 55°C for 10 s or 69°C for 1 ms (Fig. 2) (Priebe and Welch 1978; Allen and Polhamus 1989; Schulmeister and Jean 2011).

For small images and exposure durations longer than about 10 ms, the steady state temperature is reached during the exposure. Due to the exponential dependence of the degree of thermal injury on temperature, the cooling phase after the cessation of the pulse has no influence on the critical temperature (Schulmeister and Jean 2011). Consequently, the critical temperature is lower than for larger images, for which the peak temperature is reached at a later point of time.

The irradiance required to achieve these temperatures depends upon the ambient tissue temperature and the exposure spot size. Because of the more efficient cooling of small spots, injury of small spots requires higher irradiances than injury of large spots.

Retinal injury

The principal retinal hazard from viewing bright light sources is photochemically induced photoretinopathy, e.g. solar retinitis (Vos and van Norren 2001) with an accompanying scotoma from staring at the sun ("eclipse blindness") or from staring into a welding arc without proper eye protection (Uniat et al. 1986; Choi et al. 2006). Laboratory studies demonstrated that photochemical injury from exposures of the order of ~10 s to 1–2 h duration is related to absorption of 380 nm to 520 nm short-wavelength light by the retinal pigment epithelium and the choroid (Ham et al. 1976). This is usually referred to as blue-light hazard (Sliney and Wolbarsht 1980) but also as Type II photochemically induced retinal damage (Mellerio 1994). Small temperature rises in the retina may be synergistic with the photochemical process.

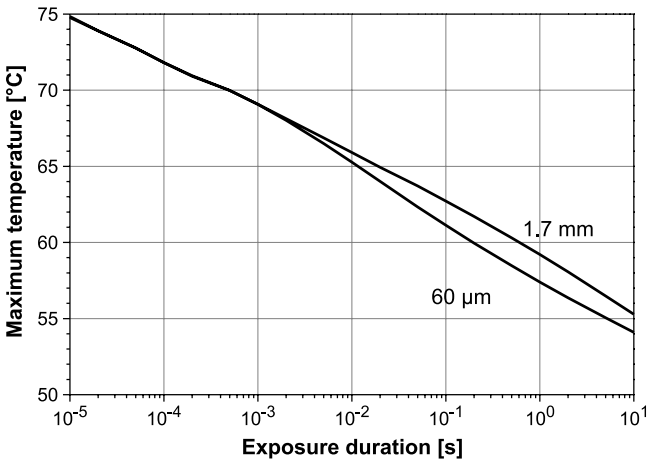

Fig. 2. The calculated peak temperature (at the lesion radius) required for retinal thermal injury in a 60 μm and 1.7 mm retinal image as a function of duration of light exposure (Schulmeister and Jean 2011).

Animal studies demonstrated that continued exposures over several days to very bright light led to retinal injury (Noell et al. 1966; Mellerio 1994; Rozanowska and Sarna 2005; Organisciak and Vaughan 2010) also referred to as Type I retinal photochemically induced damage. This type of damage has been suggested to be linked to damage of the photoreceptors as a result of prolonged bleaching of rhodopsin.

Shorter-wavelength visible radiation has been suggested to accelerate retinal aging (Marshall 1983; Young 1988).

Only sources with a very high radiance such as a xenon-arc flash lamp, a nuclear flash, or a laser are capable of producing *thermal* injury of the retina.

The mechanisms involved in thermal retinal injury as a function of retinal image size in the wavelength region 380–1,400 nm are well understood and supported with experimental threshold data, retinal explant data and models of thermal retinal injury (Mainster et al. 1970; Birngruber et al. 1985; Freund et al. 1996; Lund et al. 2007; Schulmeister et al. 2008).

Although there are no clear boundaries between injury mechanisms, certain mechanisms dominate depending on the spectral region and the exposure duration. Photochemical, rather than thermal, effects dominate in the wavelength region below approximately 600 nm for exposure times in excess of approximately 10 s. For short-duration, less than a few seconds, the damage is due to thermal injury. At infrared wavelengths, where photochemical effects have not been detected, thermal effects still dominate for exposure times longer than 10 s. Radial heat flow produces a strong dependence of retinal injury threshold on retinal image size. For small and medium retinal image sizes, eye movements affect the retinal photochemical injury thresholds by distributing the retinal radiant exposure over a larger area (Yarbus 1967; Naidoff and Sliney 1974; Sliney 1988; Ness et al. 1999). See also further discussion below (the effect of eye movements).

Anterior segment injury

In the anterior segment of the eye, opacification of the cornea or the lens are the major effects of concern after exposure to IRR. However, damage of the cornea is only possible if intense radiation is focused onto the cornea. For

the lens, chronic exposure to high levels of IRR potentially causes cataract. For exposure to intense pulsed light sources at close proximity to the eye, thermally induced damage of the iris is a concern (Sutter and Landau 2003; Javey et al. 2010; Lee et al. 2011).

Threshold exposures for lenticular changes caused by IR-A are of the order of 50 MJ m^{-2} (Pitts and Cullen 1981). Similar levels have been reported using an Nd:YAG laser operating at 1064 nm (Wolbarsht 1978, 1991). The temperature rise in the lens was estimated to be several degrees (Scott 1988a and b). Glass and steel workers exposed in hot environments to infrared irradiances of the order of 800–4,000 W m^{-2} daily for 10–15 y have reportedly developed lenticular opacities (Lydahl 1984).

Circadian rhythm regulation

Light has a profound impact upon circadian regulation of the human neural endocrine system (Brainard et al. 2001; Berson et al. 2002; Turner and Mainster 2008). A non-visual photoreceptor in the human retina (photoreceptive ganglion cell) mediates this response. The secretion of melatonin is suppressed by retinal exposure to short wavelength visible radiation. The implications for adverse health impacts from de-regulation of the circadian rhythm remain speculative and are therefore not considered in the current guidelines.

Visual disturbance

Temporary visual disturbances such as disability glare, discomfort glare, after-images and "flash blindness" may be caused by brief exposures to bright light sources at levels below the exposure limits (Chisum 1973), and precautions should be taken against secondary safety hazards resulting from temporary visual impairment (Reidenbach 2009).

Skin injury

Photosensitized injury of the skin by visible light is possible as a result of the presence of both endogenous and exogenous photosensitizers such as bilirubin and phenothiazine. Although this effect is far less likely to be caused by light than by UVR, it may occur with topically applied substances as well as after ingestion of certain photosensitizing compounds in food or medicines (Fitzpatrick et al. 1974; Magnus 1976; Diffey 1982). For example, the action spectrum for porphyria frequently has secondary peaks at about 400 nm and 500 nm (Diffey 1982).

Thermal injury thresholds of the skin are highly dependent upon the size of the exposed area, perfusion, pigmentation and the initial skin temperature, which is usually 22–25°C compared with 37°C for the retina. Very high irradiances are needed to produce thermal injury within the pain reaction time, <1 s (Hardy and Oppel 1937; Moritz and Henriques 1947; Stolwijk 1980). Higher temperatures (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). In typical industrial situations, whole-body heat stress tends to limit the duration of exposure to optical radiation, keeping it below the threshold for thermal damage to the skin. Hence, only pulsed, or very brief, exposures to very high irradiances pose a thermal hazard to the skin. Chronic elevation of skin temperature (not only caused by optical radiation) is known to induce a fixed reddening of the skin, erythema ab igne (ICNIRP 2006).

Heat stress

Long-term whole body exposure below the thresholds for thermal damage to the eye and skin can overload the body's temperature regulating capacity and result in heat stress (ICNIRP 2006; ACGIH 2010).

BIOPHYSICAL BASIS FOR THE EXPOSURE LIMITS

Adverse effects of radiation are theoretically possible across the entire optical spectrum, but in the context of these guidelines there is particular concern about the visible and near-infrared regions, 380–1,400 nm where radiation can cause retinal injury. The photobiological hazards on the skin and eye vary widely with wavelength. Exposure to broadband (non-laser) optical sources that emit light and infrared radiant energy must be evaluated by applying several specific action spectra.

In developing exposure limits for broad-band optical sources, action spectra were specified. The action spectra were used to spectrally weight the exposure to derive a "biologically effective radiance or irradiance." This provides the most accurate hazard assessment. Exposure limits can then, independently of the source spectrum, be specified in terms of exposure duration and other relevant parameters, so that all sources are evaluated with the same risk criteria. For a given exposure, produced by a given source at the eye and the skin, several action spectra may need to be applied. Also, for the determination of the effective radiance relevant for the blue light hazard, due to averaging of the radiance over a certain solid angle, the effective radiance may be lower than the actual physical radiance. The effective exposure levels are subsequently compared with the corresponding exposure limits.

This dosimetric concept reflects that for exposure to radiation from a given source, there may be more than one hazard, e.g., photochemically induced photoretinopathy *and* thermal damage to the lens.

Retina

Retinal image size and source size. In the wavelength range where the pre-retinal media of the eye are

transparent (mainly 380 nm to 1,400 nm), optical radiation is imaged onto the retina as shown schematically in Fig. 3.

The focal length of the optical system of the human adult eye (cornea and lens) if immersed in air would for the case of focusing at infinity equal 17 mm (Gullstrand 1909). The planar angular subtense of the source, α (Fig. 3), is the angle subtended by the actual source at the position of the eye. The planar angular subtense in units of radians is obtained by dividing the source extent by the distance. For the simplified assumption of the optical power of the eye immersed in air, the angle subtended by the source is equal to the angle subtended by the image (Sliney and Wolbarsht 1980). For retinal images resulting from sources of small planar angular subtense the retinal image dimension, d_r (mm), is directly related to the source dimension, d_s (mm), the effective focal length of the eye in air, f (i.e. 17 mm), and the viewing distance from the source, r (mm) (eqn 1) (Sliney and Wolbarsht 1980):

$$d_r = d_s \times \frac{f}{r} \quad (1)$$

Note that optical elements such as lenses or diffusers optically transform the radiation emitted from the source. The angular subtense, α, characterizes the angular subtense of the apparent source (Henderson and Schulmeister 2004) that produces the smallest retinal image that can be achieved by accommodation. The angular subtense of a source should not be confused with the beam spread (divergence) of a source, such as a searchlight, although under certain conditions (for a well collimated beam and when the source is projected to infinity by optics in front of the lamp) it is equivalent.

From knowledge of the optical parameters of the human eye and the radiometric parameters of a light source, it is possible to calculate irradiances at the retina, as shown below (eqn 2). In physiological optics, it is necessary to distinguish between a "point source" and an "extended source." In these guidelines, a source is considered a point source if the angular subtense is less than or equal to α_{min}, where α_{min} equals 1.5 mrad, which corresponds to a retinal spot size of 25 μm. As an extended source is viewed at ever-increasing distance, it begins to behave as a point source and eqn (1) becomes invalid for image dimensions less than approximately 25–50 μm (Sliney and Wolbarsht 1980; Schulmeister 2013). For ease of application, the blue-light hazard exposure limits for extended sources and small (or "point") sources are expressed in different quantities. Radiance (W m^{-2} sr^{-1}) and radiance dose (time integrated radiance, J m^{-2} sr^{-1}) are used for exposure limits derived to protect the retina. Irradiance (W m^{-2}) and radiant exposure (J m^{-2}) are generally used for exposure limits derived to protect the skin, the cornea, and the lens, and may also be used to express limits for the retina where minimal image sizes apply.

The angle of acceptance, γ_{meas} , is the planar angle limited by the circular aperture in front of the detector over which the radiance is averaged. Adopting the angle of acceptance, the retinal exposure rate can be expressed as radiance or irradiance. Radiance can be converted to an equivalent irradiance by multiplying by the solid angle Ω corresponding to γ_{meas}, i.e., by $(\pi \times \gamma_{meas}^2)/4$ (Henderson and Schulmeister 2004). The measurement conditions are discussed below.

Calculating retinal exposure. Retinal irradiance E_r (W m^{-2}) is related to source radiance, L_s (W m^{-2} sr^{-1}), ("brightness"), the transmittance of the ocular media, τ (rel.), the pupil diameter, d_p (mm), and inversely related to the effective focal length of the eye, according to the Gullstrand eye (Gullstrand 1909) $f = 17$ mm (eqn 2) (Sliney and Wolbarsht 1980; Henderson and Schulmeister 2004).

$$E_r = \frac{\pi \times L_s \times \tau \times {d_p}^2}{4 \times f^2}$$
or
$$E_r = a \times L_s \times \tau \times {d_p}^2, \quad (2)$$

where a = 2,700 m^{-2}. Radiance is *not* directly related to corneal irradiance.

For the visible spectrum, the transmittance in the ocular media for younger people and most animals is as high as 0.9 (Geeraets and Berry 1968). The retinal irradiance is given by eqn (2), where d_p is in m.

In albino individuals, the iris is not very effective and some scattered light reaches the retina. Nevertheless,

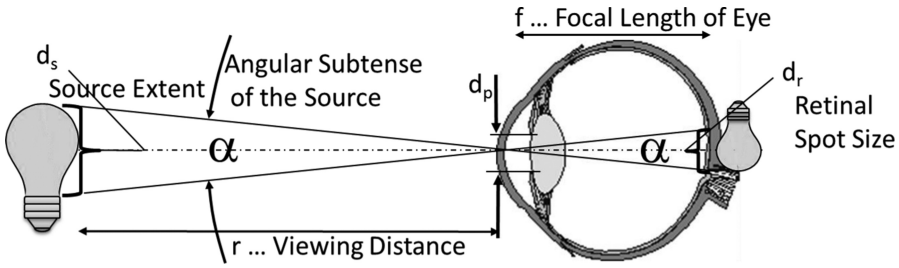

Fig. 3. Imaging of a broad band source on the retina showing the planar angular subtense, α, of the source.

imaging of the light source still occurs. Therefore, eqn (2) is valid if the contribution of scattered light, which falls over the entire retina, is added.

Injury to retinal tissues depends not only on the retinal irradiance but also very strongly on radial heat flow and eye movements.

The effect of retinal heat flow. For retinal thermal injury, the thresholds vary with retinal image size. This is because of the impact of radial heat flow on the temperature in the center of the image on the retina during the exposure. For very small and very large spots, the impact of radial heat flow on the exposed area during the exposure is negligible. Therefore, for very small and large retinal spot sizes, a constant radiance threshold applies. But mathematical models and experimentally determined thresholds for retinal thermal injury show that due to the radial heat flow, threshold retinal irradiance varies approximately inversely as the image diameter for image diameters from approximately 25 μm (1 mrad) to approximately 2,000 μm (0.1 rad) for exposure durations on the order of 1 s (Sliney and Wolbarsht 1980; Allen and Polhamus 1989; Courant et al. 1989). Since this effect depends on heat flow during the exposure, this effect is greater for long duration exposures and is nearly non-existent for short-duration exposures of the order of 1 μs (Freund et al. 1996; Framme et al. 2004; Schuele et al. 2005; Schulmeister et al. 2008).

The effect of eye movements. Continuous eye movements are of major significance in the derivation of the exposure guidelines, particularly for photochemically induced injury, and have smaller impact on thermally induced injury (Lund 2006). Eye movements effectively enlarge the irradiated retinal area and increase the angular distribution of the energy on the retina. The extent of eye movements depends upon the viewing duration. For brief exposures, involuntary eye movements dominate (Ness et al. 2000). For very long exposures of 1,000 s or more, task-determined eye movements dominate and the angular extent of the eye movements is at least 100 mrad (Yarbus 1967; Walker-Smith et al. 1977). Hence, the angle required for spatially averaging the radiance of a small source for comparison with the photochemical retinal limit is related to the angular extent of the eye movements. An averaging angle of 11 mrad is recommended for determination of the exposure level for photochemical retinal effects and exposure durations less than 100 s. An averaging angle of 11 mrad represents minimal eye movements that can be associated with staring (fixating) a certain point. Continued fixation of a point for longer durations than 10 s will usually occur only when concentration on a specific target is necessary, e.g., during welding. Thus, the averaging angle of 11 mrad is a conservative value.

For longer exposure durations, the extent of eye movements is not generally characterized and strongly depends on the task and behavior. As a very conservative value, an averaging angle of 110 mrad is specified for exposure durations of 10,000 s (the maximum integration duration for the blue light hazard). If the visual task and the behavior can be characterized, a safety analysis can account for more realistic eye movements and a larger averaging angle can be used. The average fields of view of 11 mrad and 110 mrad correspond to a retinal image of 190 μm and 1.9 mm, respectively.

Infrared exposure of the retina with low visual stimulus. A special exposure limit is required to protect the retina against thermal injury while viewing infrared LEDs or other specialized infrared illuminators in which visible light has been removed by filters. The lack of visible stimulus results in loss of the aversion response implicating that pupil constriction cannot be assumed.

This exposure limit for low visual stimulus was therefore derived assuming a 7-mm pupil and extending the trend of the exposure limit for pulses beyond 0.25 s. The long-term exposure duration limit is based largely on studies of thermal injury in infrared spectral bands (Ham et al. 1973).

Anterior structures of the eye

For many sources, assessment of potential thermal hazard to the anterior segment of the eye is essential in addition to evaluating the retinal hazards. Contributions of IR-A (780–1,400 nm) and IR-B (1.4–3.0 μm) must be considered. Data on which to base exposure limits for chronic exposure of the anterior portion of the eye to IRR are limited. Sliney and Freasier (1973) stated that the average corneal exposure from IRR in sunlight is of the order of 10 W m^{-2}.

In ultraviolet and short-wavelength light exposure, wavelength dependent photochemical action spectra are characteristic, implicating that the spectrum of the source must be considered in exposure limits. However, because the effects of IRR are thought to be largely thermal, the biological response to chronic IRR exposure of the cornea and lens is not believed to have strong spectral dependence (Barthelmess and Borneff 1959; Sliney 1986). Radiant energy absorbed in the cornea, aqueous humour, and iris is dissipated through thermal conduction, and some heating will occur in the lens regardless of the optical penetration depth. Penetration depth strongly varies between 1.2 and 3 μm. However, this should have no considerable effect on the final temperature rise resulting from exposure to a continuous-wave source once thermal equilibrium is achieved.

The final temperature of the lens also depends on the ambient temperature (Sliney 1986; Okuno 1991). For each degree that ambient temperature falls below 37°C, an added irradiance of at least 6 W m^{-2} would be required to maintain the temperature of the lens (Stolwijk and Hardy 1977). 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. However, 1 kW m^{-2} on the face would be painfully warm and not tolerated in a warm environment.

Synergistic effects

The synergism between thermal and photochemical effects in the lens and retina has been studied in some experiments. Thermal enhancement of photochemical reaction has been experimentally demonstrated (Pitts and Cullen 1981; Allen and Polhamus 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 reduction factor (see below).

Skin exposure

A realistic risk of thermal injury to the skin exists only in environments where a very high irradiance can be delivered from a pulsed source. ICNIRP provides guidance only for exposures lasting less than 10 s (based on empirical conservative assumptions).

For lengthier exposures, heat stress guidelines must be consulted. Most guidelines for control of heat stress are designed to limit deep-body temperature to 38°C, and require consideration of air flow, ambient temperature, and humidity (ICNIRP 2006; ACGIH 2010).

Aversion responses

The eye is adapted to protect itself against excessive optical radiation from the natural environment, and humans have learned to use additional protective devices if adverse effects occur. Natural aversion response for exposure to bright light includes, pupillary constriction, eye movements, squinting and in some cases blinking.

Gerathewohl and Strughold (1953) studied the blink reflex for full field of view exposure to flash lamps and determined that the shortest time from onset of the flash to complete lid closure was 180 ms. However, a reflexive blink is highly variable from person to person (Reidenbach 2005) and can be behaviorally suppressed. The potential hazard from longer exposure durations is mitigated by involuntary eye movements which distribute the light energy over a much greater area of the retina. In addition, behavioral reactions such as movement of the head (Fender 1964; Yarbus 1967) also reduce exposure to a given retinal area. For thermally induced injury, another factor is the weak dependence of the injury thresholds, given in irradiance, with longer exposure durations (Schulmeister and Jean 2011). Reduction of the pupil size due to exposure to bright light dynamically reduces the retinal exposure for long-duration exposure (Fig. 4) (Stamper et al. 2002).

The effect of eye movements on time-averaged retinal irradiance was illustrated by (Ness et al. 1999) and (Lund 2006).

Elevated temperatures of the skin and cornea produce an avoidance response within a few seconds. Temperatures that induce pain sensation (Randolph and Stuck 1976) are below temperatures that lead to thermal injury (Henriques 1947). Pain sensations usually induce an avoidance response within a few seconds that prevent a burn, with the exception of very high irradiances that rapidly heat the skin (Randolph and Stuck 1976). Under the influence of alcohol and some medications, pain sensation might be reduced and thus avoidance is not induced.

RATIONALE

The exposure limits were derived on the basis of current knowledge on damage thresholds and in accordance with the ICNIRP principles (ICNIRP 2002). The exposure limits are set to a level below the damage thresholds by applying a reduction factor. In view of uncertainties inherent in the damage thresholds, a reduction factor of at least two has been applied in deriving the exposure limits. In addition, wavelength-, exposure duration-, and/or spot size-dependence of the exposure limits have been simplified with respect to known injury threshold. These simplifications have sometimes imposed higher reduction factors, occasionally as high as approximately two orders of magnitude.

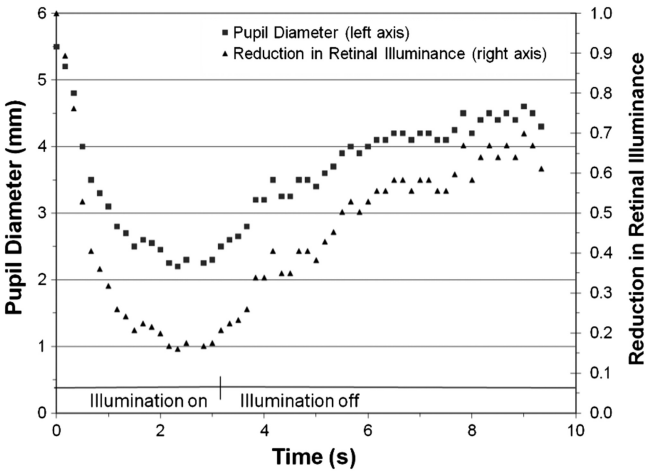

Fig. 4. Dynamics of the pupillary constriction and reopening (squares-left axis) after a 3-s exposure to a bright light and the reduction of the retinal illuminance due to pupil diameter change (triangles-right axis). The initial pupil diameter was 5.5 mm. Adapted from Stamper et al. (2002)

The exposure limits derived for the eye are the most restrictive due to higher sensitivity of the eye than of the skin. The consequences of overexposure of the eye are also generally more serious than those of overexposure of the skin, and safety standards for optical sources, including lasers, have, therefore, emphasized protection of the eye (UNEP et al. 1982; Duchêne et al. 1991; Health Council of the Netherlands 1993; ICNIRP 1996, 1997, 2000, 2010).

The guidelines are based predominantly on injury after experimental exposure of animals. Further, information from human retinal injuries resulting from viewing the sun and welding arcs was considered. The exposure limits for the retina and pulsed exposure of the cornea were predominantly derived from experimental data obtained with laser sources.

For experimental injury threshold determination, incrementing individual exposures are each evaluated by ophthalmoscopy or other methods of examination and rated on a binary scale as lesion or no lesion. The probability for damage as a function of dose is fitted assuming a normal distribution (Finney 1971). The effective dose resulting in a 50% probability for damage, ED-50, is commonly referred to as the threshold dose (Sliney et al. 2002).

The exposure that corresponds to damage at threshold depends on the time interval between the exposure and the examination (the lesion takes some time to develop biologically into detectable change), the method of examination (ophthalmologically visible lesion in vivo, light microscopic change), and the site of exposure (macula, paramacula). Generally, when ophthalmoscopic examination is performed at 24 h after exposure, retinal lesions are observed that were not visible at 1 h after exposure, resulting in an ED-50 for the 24 h endpoint that is lower than the ED-50 determined for the 1 h endpoint. For this reason, recent retinal threshold data are reported for observations at 24 h as well as at 1 h and for macular exposure. Typically, the 24 h ED-50 is a factor of 2 to 3 below the ED-50 determined at 1 h after exposure (Lund et al. 2007; Zuclich et al. 2008). The threshold for photochemically induced retinal injury was reported for a 1 h and 48 h interval after exposure, respectively (Lund et al. 2006). Light and electron microscopy examination of tissue has indicated cellular alterations at exposures in the proximity of the ED-50 derived by ophthalmic examination 24 h after the exposure (Lund et al. 2007; Zuclich et al. 2008). For determination of the threshold of the cornea and the lens, slitlamp microscopy is used to observe radiation induced opacifications. For the lens, the interval between exposure and observation is 24 h to 48 h (Pitts et al. 1977). For thermally induced corneal injury, the threshold lesion is usually observed at 1 h, whereas photochemical threshold effects are observed at 24 h to 48 h after exposure.

For the skin, the criterion for threshold is based on radiation induced erythema determined by direct observation within 48 h after exposure. In some studies, direct observation was supported by histopathology.

The exposure limits and their functional dependence on specific exposure parameters (wavelength, pulse duration, retinal spot size, etc.) are based on threshold data determined by direct observation, i.e., ophthalmoscopy in case of retinal exposures. In setting the exposure limits, ICNIRP incorporated those considerations in the reduction factors.

The exposure limits for visible radiant energy also contain an underlying assumption that most outdoor environmental exposures are indirect or off-axis, normally not hazardous to the eye except in environments producing reflections from surfaces such as snow and sand.

Exposure conditions that in animal experiments led to retinal photochemical damage Type I (Noell et al. 1966) were extreme and far exceed those experienced by humans with broadband sources. Therefore, no special exposure limits are recommended for avoidance of Type I damage.

Environmental illumination influences human health and well being by altering circadian, neuroendocrine, neurobehavioral and melatonin responses. An action spectrum for some of these responses has been described (Brainard et al. 2001) and potential therapeutic applications are being evaluated for treatment of sleep disorders, depression or circadian rhythm disruption. However, side effects of these therapeutic levels of illumination have included photophobia, ocular discomfort, headache, and enhanced glare sensitivity. Just as optical radiation protection levels do not protect against visual disturbances from glare, these guidelines do not preclude potential light-induced modulation of physiological rhythms since the effects are not necessarily adverse responses.

Injury of the skin following photosensitization is highly dependent on the photosensitizer and must be treated according to toxicological criteria, which is out of the scope of these guidelines.

The mechanical disruption of tissue and other effects caused by ultra-short laser pulses does not occur with current non-laser sources and is, therefore, not considered in the derivation of these guidelines.

For all currently known arc and incandescent sources, the contribution made by the far infrared radiation, 3 μm–1,000 μm, is normally of little or no practical concern (ICNIRP 2006). Only lasers pose potential hazards in this spectral region. For thermal radiators with temperatures high enough that an exposure can be hazardous, the radiation in the wavelength range below 3,000 nm is the critical part. Thus, far IRR can be largely ignored when a risk assessment for such sources is made. In addition the exposure limits are set to a level that accounts

for possible contributions from radiation in the wavelength band above 3,000 nm. Normally, considerations of heat stress will dominate the risk assessment for conditions where there is significant IR-C.

Including the IR-C component in the determination of the exposure would constitute a conservative risk analysis when the total IRR exposure is 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.

A more detailed rationale for the changes of the exposure limits compared with previous guidelines (ICNIRP 1997) is given in Appendix A.

Correct application of the exposure limits requires knowledge of the spectral radiance, L_λ, or spectral irradiance, E_λ, and, for determination of the retinal thermal exposure limit, the angular subtense of the source as perceived by the eye.

For a white-light source, such detailed spectral data are generally required only if the luminance exceeds 10^4 cd m^{-2} (ICNIRP 1997). This rule of thumb results in an exclusion of many simple light sources, since these do not exceed the exposure limits for the retina.

In the derivation of the limits to protect against retinal thermal injury, two different pupil diameters were assumed, 7 mm for the dark-adapted eye and approximately 3 mm for bright light conditions. Two limits are defined to protect against retinal thermal injury. There is one exposure limit for the general case of incoherent broad-band sources which emit visible radiation, based on the assumption of a 7-mm pupil for exposure durations up to approximately 0.5 s; and for longer exposures a 3-mm pupil due to pupillary constriction. If there is concern about longer exposures resulting from determined visual effort, the exposure limits for longer durations may apply. There is a second exposure limit for infrared sources without a strong visual stimulus, assuming a 7-mm pupil. The exposure limit for photochemically induced retinal injury was derived with the assumption of a pupil diameter of approximately 3 mm.

For exposure conditions where there is loss of the aversion response because of reduced visual sensitivity or surgical anesthesia, ICNIRP provides recommendations for adjustment of exposure limits (Sliney et al. 2005).

The dependencies of the exposure limits on the relevant parameters for the variables were derived from experiments by fitting threshold data to the variables. Often, a linear dependence was observed when plotting on a double logarithmic scale. For a proper treatment of the dimensions, the threshold and the variables need to be transformed to relative values to make them dimensionless. As a result, a fully dimensionally correct way of specifying the dependence of the exposure limits on the relevant variables can be derived when the variables such as t or α are each divided by a reference factor that is equal to 1 × the unit such as 1 s or 1 rad. An example is shown in eqn (3) for the retinal thermal limit:

$$L_R \le \frac{20,000 \times \alpha_{ref}}{\alpha} (t \times t_{ref}^{-1})^{0.75} J\, m^{-2}\, sr^{-1}, \quad (3)$$

where α_{ref} = 1 rad and t_{ref} = 1 s.

However, to decrease complexity in the formulas in the guidelines, the dimension-factors were omitted. Therefore, the formulas do not appear dimensionally correct and it is important that the numbers of the variables are inserted into the formula in the correct order of magnitude, i.e., α in rad and not for instance mrad.

Biological weighting functions are used to express the wavelength dependence of the effect to protect against retinal injury (Fig. 5, Table 2).

Retinal thermal hazards (380–1,400 nm)

Protection of the human retina from thermal injury requires that the spectrally weighted effective radiance does not exceed the retinal thermal exposure limit. The effective retinal thermal radiance, L_R (W m^{-2} sr^{-1}), is the integration (or summation) of the product of the spectral radiance, L_λ (W m^{-2} sr^{-1} nm^{-1}), and the retinal thermal hazard function $R(\lambda)$ (i.e., the retinal thermal biological weighting function) (Table 2) over the wavelength range λ (nm) from 380 to 1,400 nm (eqn 4):

$$L_R = \sum_{380}^{1400} L_\lambda \times R(\lambda) \times \Delta\lambda \quad (4)$$

The retinal thermal hazard function R(λ) characterizes the spectral efficiency to cause threshold retinal injury and is given in Table 2. The effective radiance, L_R, given by eqn (4) is then compared with the exposure limit in Table 4.

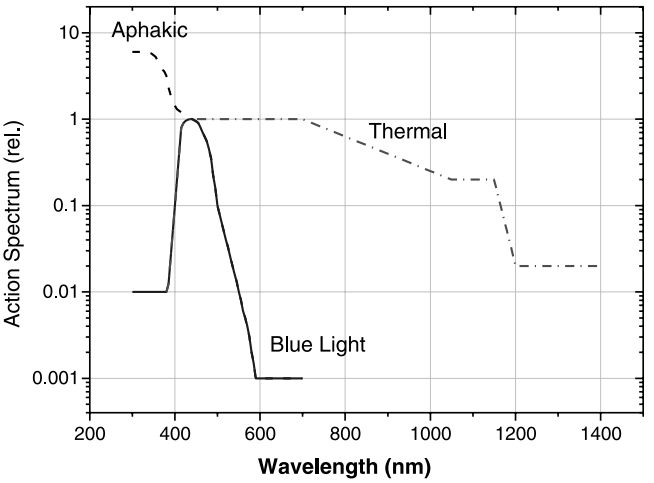

Fig. 5. Action spectra for blue-light photoretinopathy with lens (phakic) B(λ) and without lens (aphakic) A(λ), and for thermally induced photoretinopathy R(λ).

Table 2. Retinal hazard spectral weighting functions (aphakic and blue light hazard functions are the same as in ICNIRP (1997).

Wavelength (nm)	Aphakic[a] hazard function, $A(\lambda)$	Blue-light[a] hazard function, $B(\lambda)$	Retinal thermal hazard function $R(\lambda)$ (where λ is in nm)
300	6.00	0.01	—
305	6.00	0.01	—
310	6.00	0.01	—
315	6.00	0.01	—
320	6.00	0.01	—
330	6.00	0.01	—
335	6.00	0.01	—
340	5.88	0.01	—
345	5.71	0.01	—
350	5.46	0.01	—
355	5.22	0.01	—
360	4.62	0.01	—
365	4.29	0.01	—
370	3.75	0.01	—
375	3.56	0.01	—
380	3.19	0.01	0.01
385	2.31	0.0125	0.0125
390	1.88	0.025	0.025
395	1.58	0.050	0.05
400	1.43	0.100	0.1
405	1.30	0.200	0.2
410	1.25	0.400	0.4
415	1.20	0.800	0.8
420	1.15	0.900	0.9
425	1.11	0.950	0.95
430	1.07	0.980	0.98
435	1.03	1.000	1.0
440	1.000	1.000	1.0
445	0.970	0.970	1.0
450	0.940	0.940	1.0
455	0.900	0.900	1.0
460	0.800	0.800	1.0
465	0.700	0.700	1.0
470	0.620	0.620	1.0
475	0.550	0.550	1.0
480	0.450	0.450	1.0
485	0.400	0.400	1.0
490	0.220	0.220	1.0
495	0.160	0.160	1.0
500	0.100	0.100	1.0
505	0.079	0.079	1.0
510	0.063	0.063	1.0
515	0.050	0.050	1.0
520	0.040	0.040	1.0
525	0.032	0.032	1.0
530	0.025	0.025	1.0
535	0.020	0.020	1.0
540	0.016	0.016	1.0
545	0.013	0.013	1.0
550	0.010	0.010	1.0
555	0.008	0.008	1.0
560	0.006	0.006	1.0
565	0.005	0.005	1.0
570	0.004	0.004	1.0
575	0.003	0.003	1.0
580	0.002	0.002	1.0
585	0.002	0.002	1.0
590	0.001	0.001	1.0
595	0.001	0.001	1.0
600–700	0.001	0.001	1.0
700–1,050	—	—	$10^{(700-\lambda)/500}$
1,050–1,150	—	—	0.2
1,150–1,200	—	—	$0.2 \cdot 10^{0.02\ (1150-\lambda)}$
1,200–1,400	—	—	0.02

[a]The UVR extension of $A(\lambda)$ and $B(\lambda)$ at wavelengths below 380 nm are provided for the evaluation of optical spectra that may contain UVR. The aphakic hazard function, $A(\lambda)$, is normalized to correlate with the blue-light hazard function, $B(\lambda)$ for wavelengths above 440 nm.

Table 3.

Exposure duration	Limiting angles	
t	α_{min}	α_{max}
Seconds	Radians	Radians
$t < 625 \times 10^{-6}$	1.5×10^{-3}	0.005
$625 \times 10^{-6} \le t < 0.25$	1.5×10^{-3}	$0.2 \cdot t^{0.5a}$
$t \ge 0.25$	1.5×10^{-3}	0.1

[a] t is input in seconds to calculate the correct numeric value of α_{max} in rad.

The retinal thermal effective radiance dose, D_R(J m^{-2} sr^{-1}), is obtained by integrating L_R over the exposure time, $t(s)$. If the radiance is constant over the exposure duration, t, the radiance dose is simply the product of the radiance L_R and the exposure duration ($D_R = L_R \times t$). The width of $\Delta\lambda$ for the determination of the effective retinal thermal radiance should be selected based on the wavelength dependence of the radiance. In regions where the biologically spectral efficiency, $R(\lambda)$, changes rapidly with wavelength, a higher spectral resolution is required. For a source with a radiance that is constant over the exposure duration, the effective radiance dose, D_R, is given in eqn (5).

Angular subtense of the source. The angle, α, subtended by the source at the position of the eye (Fig. 3) is often referred to as the "source size." If the radiation emitting area of a source is circular with a diameter, d_s, and is at a distance, r, from the eye (Fig. 3), then α is the ratio between the diameter of the source and the distance to the source, d_s/r. For a non-circular source, α is the arithmetic mean of the shortest and longest dimension. Before calculating the arithmetic mean, the angular subtense in each dimension shall be limited to α_{min} and α_{max}, respectively. A source that subtends an angle of 1.5 mrad or less is referred to as a "point" source. Such a source produces approximately the smallest retinal image size in a given dimension when ocular diffraction and aberrations are considered. In addition, the retinal thermal injury threshold does not change for sources sizes $\alpha \le \alpha_{min}$. Hence, α_{min} is assigned the value 1.5×10^{-3} radians (i.e., $\alpha_{min} = 1.5 \times 10^{-3}$ radians). Large sources are defined as sources where $\alpha \ge \alpha_{max}$, where α_{max} is the retinal image size where the retinal injury threshold expressed in retinal radiant exposure does not change with increasing size (Schulmeister et al. 2011).

Intermediate sources are those where the source size is between α_{min} and α_{max} or $\alpha_{min} < \alpha \le \alpha_{max}$. For intermediate source sizes, the thermal retinal injury threshold is dependent upon the source size a (i.e., the retinal irradiance diameter) and the exposure (or "pulse") duration, t.

Determination of radiance. When assessing spatially irregular sources or sources with "hot" spots, the radiance should be averaged over an acceptance angle of γ_{th}. This acceptance angle depends on the exposure (or

Table 4. Retinal thermal exposure limits.

			Exposure limit (EL)		
	Exposure duration t (s)	Source size α radians	Radiance L_R^{EL} $W\,m^{-2}sr^{-1}$ (t in s and α in rad)	Radiance dose D_R^{EL} $J\,m^{-2}sr^{-1}$ (t in s and α in rad)	Reference note
Basic exposure limit	$1 \times 10^{-6}\ s \le t < 0.25\ s$	$\alpha_{min} \le \alpha \le \alpha_{max}$	$2.0 \times 10^4 \cdot \alpha^{-1} \cdot t^{-0.25}$	$2.0 \times 10^4 \cdot \alpha^{-1} \cdot t^{0.75}$	1, 2, 3, 4, 5
	$t < 1 \times 10^{-6}\ s$	$\alpha_{min} \le \alpha \le \alpha_{max}$	—	$0.63 \cdot \alpha^{-1}$	1, 2, 3
	$t \ge 0.25\ s$	$\alpha_{min} \le \alpha \le \alpha_{max}$	$2.8 \times 10^4 \cdot \alpha^{-1}$	$0.71 \times 10^4 \cdot \alpha^{-1}$	1, 2, 4
Small sources	$t < 1 \times 10^{-6}\ s$	$\alpha \le \alpha_{min}$	—	420	2,3
	$1 \times 10^{-6}\ s \le t < 0.25\ s$	$\alpha \le \alpha_{min}$	$1.3 \times 10^7 \cdot t^{-0.25}$	$1.3 \times 10^7 \cdot t^{0.75}$	1, 2,3
	$t \ge 0.25\ s$	$\alpha \le \alpha_{min}$	1.9×10^7	—	2,4
Large sources	$t < 1 \times 10^{-6}\ s$	$\alpha \ge \alpha_{max}$	—	130	3, 5
	$1 \times 10^{-6}\ s \le t < 625 \times 10^{-6}\ s$	$\alpha \ge \alpha_{max}$	—	$4.0 \times 10^6 \cdot t^{0.75}$	1, 5, 6
	$625 \times 10^{-6}\ s \le t < 0.25\ s$	$\alpha \ge \alpha_{max}$	—	$10 \times 10^4 \cdot t^{0.25}$	1, 5, 6
	$t \ge 0.25\ s$	$\alpha \ge \alpha_{max}$	28×10^4	—	4, 5

Notes:
1. To calculate exposure limits, L_R^{EL} and D_R^{EL}, must be in seconds and α must be in radians;
2. $\alpha_{min} = 0.0015$ radian. If $\alpha \le \alpha_{min}$, then $\alpha = \alpha_{min}$ for calculating the exposure limit;
3. If $t < 10^{-6}$ s, then $t = 10^{-6}$ s for calculation of the exposure limit in the radiance dose, D_R^{EL};
4. If $t > 0.25$ s, then $t = 0.25$ s for calculation of the exposure limit, L_R^{EL};
5. For 1×10^{-6} s $\le t < 625 \times 10^{-6}$ s, then $\alpha_{max} = 0.005$ radian. If 625×10^{-6} s $\le t < 0.25$ s, then $\alpha_{max} = 0.2 \times t^{0.5}$. If $t \ge 0.25$ s, then $\alpha_{max} = 0.1$ radian; and
6. If 0.1 radian > α_{max}, then $\alpha = \alpha_{max}$.

pulse) duration, t. For a continuous wave (CW) source where the exposure duration is greater than 0.25 s ($t \geq 0.25$ s), $\gamma_{th} = 11 \times 10^{-3}$ radians (11 mrad). For pulses where the exposure duration is less than 0.25 s ($t < 0.25$ s) and where there are radiance hot spots, the radiance should be determined with an angle of acceptance, γ_{th}, equal to 5×10^{-3} radians ($\gamma_{th} = 5$ mrad). If the source size, α, is smaller than the angle of acceptance, γ_{th}, and the radiance is averaged over this angle of acceptance, the value of α used to determine the retinal thermal exposure limit must not be less than the angle of acceptance, γ_{th}.

Basic retinal thermal limit. To protect against thermal injury to the retina, the effective radiance, L_R, or effective radiance dose, D_R, (eqn 5) must not exceed the exposure limit (EL) in radiance, L_R^{EL}, or radiance dose, D_R^{EL}, respectively (eqns 6 and 7). The exposure limits, L_R^{EL} and D_R^{EL}, are empirical expressions that yield the respective exposure limit values in W m^{-2} sr^{-1} and J m^{-2} sr^{-1} only when α is input in radians and t in seconds (Table 3):

$$L_R \leq L_R^{EL} \text{ or } D_R \leq D_R^{EL} \quad (5)$$

$$L_R^{EL} = 2.0 \times 10^4 \times \alpha^{-1} \times t^{-0.25}\, W m^{-2} sr^{-1} \quad (6)$$

$$D_R^{EL} = 2.0 \times 10^4 \times \alpha^{-1} \times t^{0.75}\, J m^{-2} \mathrm{sr}^{-1} \quad (7)$$

Eqns (6) and (7) apply when the angular source size, α, is bounded by α_{min} and α_{max} and the exposure duration, t, by 1 μs and 0.25 s.

When the source size α is less than α_{min}, the EL is calculated for a source size equal to α_{min}. Likewise, when the source size is greater than α_{max}, the EL is calculated for a source size α equal to α_{max}. For exposure duration less than 1 μs, the EL in radiance dose is calculated for an exposure (or pulse) duration equal to 1 μs and applies as a constant radiance dose. If the exposure duration is greater than 0.25 s, the EL in radiance is calculated for an exposure duration, t, equal to 0.25 s (eqn 6) and applies as a constant radiance given in Table 4.

There may be special individual circumstances where the pupil remains dilated (tonic) and exposures extend beyond 0.25 s (e.g., some ophthalmic examination procedures; Sliney et al. 2005). Under these unusual conditions the basic EL given in radiance in eqn (6) applies for exposure duration beyond 0.25 s (Table 4).

Since a 7-mm pupil, i.e., a dark adapted pupil, is assumed for pulsed exposure and for continuous wave (CW) sources with exposure durations up to about 0.5 s, the retinal thermal exposure limit is conservative for a normal, reactive pupil. Under daylight luminance conditions, the diameter of a normal reactive pupil will be less than 7 mm in diameter. Hence, for daylight conditions where small pupil diameters are assured, the exposure limit can be increased accordingly.

Large sources $\alpha > \alpha_{max}$. Large sources are defined as sources with an angular subtense greater than α_{max} (Table 3). The exposure limits for large sources also provide a conservative limit for smaller sources ($\alpha < \alpha_{max}$) and are calculated from the basic limit (eqns 6 and 7) by setting α equal to α_{max} (Table 3). These ELs are also tabulated in Table 4.

For $t \leq 1$μs (pulse duration less than 1 μs), the exposure limit, D_R^{EL}, is a constant radiant dose given in Table 4.

Intermediate and point sources. Intermediate sources are defined as sources with an angular subtense, α, greater than α_{min} and less than α_{max} or $\alpha_{min} \leq \alpha < \alpha_{max}$ (Table 3). For intermediate sources, the retinal thermal injury exposure limit depends on both the exposure duration, t (in seconds), and the angular subtense of the source, α (in radians). Point sources are defined as sources that subtend an angle less than or equal to α_{min} or 1.5×10^{-3} rad (1.5 mrad). When $\alpha \leq \alpha_{min}$, the exposure limit is calculated by using a source angle $\alpha = 1.5 \times 10^{-3}$ rad for the determination of the exposure limit by using eqn (6) or (7) (Table 4).

Retina: weak visual stimulus of a near infrared source. For an infrared heat lamp or any near-IR source that provides no strong visual stimulus, the weighted radiance, L_R, should be limited as given in eqn (8) and eqn (9). For a near infrared exposure with a weak visual stimulus and an exposure duration longer than 0.25 s, the basic radiance and radiance dose given in eqn (8) and eqn (9) apply:

$$L_{WVS}^{EL} = 2.0 \times \alpha^{-1} \times t^{-0.25} \times 10^4\, W m^{-2} sr^{-1} \quad (\text{for } 0.25\, s < t < 100\, s); \quad (8)$$

$$L_{WVS}^{EL} = 0.63 \times \alpha^{-1}\, W \times m^{-2} sr^{-1} \quad (\text{for } t \geq 100\, s). \quad (9)$$

For comparison with the EL, and for the case that the source exhibits hot spots, the radiance should be averaged over $\gamma_{th} = 11$ mrad and the value of α for the determination of the exposure limit in this case should not be less than 11 mrad.

Protection of the anterior segment of the eye. For sources in contact with or in immediate proximity to the eyes, injury to the anterior segment of the eye cannot be

Table 5.

Exposure duration (seconds)	Acceptance averaging angle γ_{ph}(radians)
$t < 100$ s	0.011
$100 \leq t < 10{,}000$ s	$0.0011 \cdot t^{0.5}$
$t > 10{,}000$ s	0.110

Note: t must be input in seconds to calculate γ_{ph} in radians

excluded for short pulses and large sources at exposure levels approaching the thermal retinal exposure limits. However, without additional research, a specific exposure limit cannot be stated. In the absence of specific guidance, the infrared exposure limits for the anterior portion of the eye will provide a conservative guideline when also applied to the visible spectral range.

Blue-light photochemical retinal hazard (300 - 700 nm)

For protection of the retina against acute photochemically-induced photoretinopathy, the blue-light effective radiance or the blue-light effective radiance dose has to be limited. The effective blue-light radiance, L_B (W m^{-2} sr^{-1}), given in eqn (10) is obtained by integrating (or summating) the product of the spectral radiance of the source, L_λ(W m^{-2} sr^{-1} nm^{-1}) and the blue-light hazard weighting function $B(\lambda)$ tabulated in Table 2:

$$L_B = \sum_{380}^{1400} L_\lambda \times B(\lambda) \times \Delta\lambda \qquad (10)$$

For non-constant radiance values, intermittent or pulsed exposure, the effective blue light radiance dose D_B in J m^{-1} sr^{-2} is obtained by integration of L_B over the exposure duration, t. If the radiance is constant over the duration of the exposure, t, the blue-light effective radiance dose, $D_{B,}$ is given in eqn (11):

$$D_B = L_B \times t = \sum_{380}^{1400} L_\lambda \times t \times B(\lambda) \times \Delta\lambda \qquad (11)$$

The effective blue-light weighted radiance or radiance dose must not exceed the respective exposure limit, L_B^{EL} or D_B^{EL}, as given in eqn (12):

$$L_R \le L_B^{EL} \; or \; D_R \le D_B^{EL} \qquad (12)$$

For 0.25 s $\le t \le$ 10,000 s (approx. 2.8 h), the effective radiance dose, D_B, is limited by the blue-light limit, D_B^{EL} in $J\,m^{-2}\,sr^{-1}$, given in eqn (13):

$$D_B^{EL} = 1 \times 10^6 J\,m^{-2} sr^{-1} \qquad (13)$$

For $t >$ 10,000 s, the radiance limit, L_B^{EL}, that limits the blue light weighted effective radiance L_B (i.e., $L_B \le L{\downarrow}B{\uparrow}EL$ to avoid adverse effects) is given in eqn (14):

$$L_B^{EL} = 100\, W m^{-2} sr^{-1} \qquad (14)$$

The radiance, L_B, is spatially averaged over an angle γ_{ph}. The averaging angle of acceptance, γ_{ph}, varies as a function of exposure duration, t, as given in Table 5. The position of the field stop (the aperture that defines the averaging angle of acceptance, or field of view) in the imaging plane should be adjusted to assure determination of maximum exposure.

If the visual task and the behavior can be characterized, a safety analysis can account for more realistic eye movements. In that case, a larger averaging angle can be used, as long as the analysis is done so that any spot on the retina is not exposed to a radiant exposure level higher than is derived from the basic radiance dose limit given in eqn (11) (see also "Determination of exposure level").

For sources where the angular subtense of the apparent source is less than γ_{ph}, the radiance limit can be converted to an equivalent irradiance or radiant exposure limit for a given duration, t. The conversion is done by multiplying the radiance dose limit with the solid angle defined by the plane angle, γ_{ph}. This limit is compared with the effective radiant exposure or irradiance values determined with an "open" field of view (Schulmeister 2001). This is often easier to measure than averaged radiance. The potential hazard may be evaluated by mathematically weighting the spectral irradiance, E_λ, against the blue-light hazard function to obtain the blue-light hazard weighted effective irradiance, E_B. The effective radiant exposure, $H_{B,}$, is obtained by integrating the effective irradiance, $E_{B,}$, over the exposure duration. The exposure limits for the blue-light hazard expressed as the effective radiant exposure and effective irradiance at the cornea are applicable to sources that are smaller than the angle of acceptance, γ_{ph} (Table 5).

The blue-light effective radiant exposure, H_B, or the blue-light effective irradiance, E_B, are compared to the exposure limit in radiant exposure, $H_B^{EL}(J\,m^{-2})$, or irradiance $E_B^{EL}(W\,m^{-2})$, in eqns 15, 16, and 17, respectively:

$$E_B \le E_B^{EL} \text{ or } H_B \le H_B^{EL} \qquad (15)$$

$$H_B^{EL} = 100\, J\,m^{-2} \quad 0.25 \le t < 100\, s \qquad (16)$$

$$E_B^{EL} = 1\, W\,m^{-2} \quad 100 \le t < 30,000\, s \qquad (17)$$

In effect, because of eye movements involved in normal visual tasks, the maximal exposure duration that needs to be considered for small sources is 100 s. Hence, for exposure durations longer than 100 s, the small source limit expressed in irradiance is constant 1 W m^{-2}.

Notice that when the irradiance measurement is performed with the angle of acceptance as specified in Table 5 (which for the case of irradiance is not an averaging angle but a limiting angle of acceptance), the limits specified in eqns (16) and (17) can also be applied to sources larger than the specified angle of acceptance and are fully equivalent to the limits specified as radiance.

Retinal photochemical hazard to the aphakic eye and the infant eye (300–700 nm). At one time, patients treated surgically for cataract did not receive intraocular lens (IOL) implants, although such patients are rare today. However, during the surgical removal of a cataract, and before the IOL has been implanted, a patient is exposed to near-ultraviolet radiant energy of

approximately 300–400 nm from surgical operating lights (Michael and Wegener 2004). Very occasionally, an individual may be born without a crystalline lens. It is under these special conditions that the aphakic photochemical retinal hazard exists. This is a more serious type of blue-light retinal hazard. In case of aphakia, additional ocular UVR protection should be used under UVR exposure conditions. Moreover, UVR transmittance of the crystalline lens is much higher in infants under the age of 2 y (CIE 2012) than in older children and adults. For this reason eye protection should be considered close to water or in snow.

This potential retinal hazard is evaluated by spectrally weighting the radiance against the blue-light hazard function, altered for wavelengths less than 440 nm for the aphakic eye; this altered action spectrum is given the symbol $A(\lambda)$. The approach is to apply $A(\lambda)$ instead of $B(\lambda)$ in Table 2 to eqn (18).

For $t \leq 10{,}000$ s, the effective aphakic hazard radiance, L_{Aphake}, can be calculated from the spectral radiance, L_λ, with the aphakic hazard function, $A(\lambda)$ (Fig. 5, Table 2) (eqn 18):

$$D_{Aphakic} = \sum_{300}^{700} L_\lambda \times A(\lambda) \times \Delta\lambda \leq 1.0\, MJ\ m^{-2}\ sr^{-1} \qquad (18)$$

The lens in infants aged less than 2 y transmits more ultraviolet than the adult lens (WHO 1994) and more protection is needed for the developing retina (CIE 2012). Thus, the A(λ) weighting function should be used for a conservative hazard assessment of light sources to which infants are exposed.

Cornea and lens (780 nm–1 mm)

To avoid thermal injury of the cornea and possible delayed effects on the lens of the eye (cataractogenesis), infrared irradiance E_{IR} in the wavelength range of 780 nm–3 μm (eqn 19) should be limited by the exposure limits E_{IR}^{EL} given in eqns (20) and (21):

$$E_{IR} = \sum_{780}^{1000} 0.3\, E_\lambda + \sum_{1000}^{3000} E_\lambda \qquad (19)$$

For simplicity, measurements can be made using a thermal detector, neglecting the action spectrum defined in eqn (19), which results in a conservative exposure value. The action spectrum is intended to be applied for non-thermal radiators such as infrared LEDs, where a spectroradiometer is regularly used to measure the spectral irradiance, E_λ.

$$E_{IR}^{EL} = 18 \times t^{-0.75} \times 10^3\ \mathrm{W}\ m^{-2} \text{ for } t < 1000\, s \qquad (20)$$

$$E_{IR}^{EL} = 100\, W\, m^{-2} \text{ for } t \geq 1000\, s \qquad (21)$$

In cold environments, the long-term exposure limits may be increased to 400 W m^{-2} at 0°C and approximately 300 W m^{-2} at 10°C without the lenticular temperature exceeding 37°C. The relaxation of the limits is based on environmental heat exchange rates for the head (Stolwijk and Hardy 1977), the final temperature of the lens being calculated from ambient temperature.

Visible and infrared thermal injury to the skin

To protect the skin from thermal injury, the radiant exposure, H, determined for wavelengths less than 3,000 nm and durations less than 10 s should be limited as given in eqn (22).

$$H_{skin}^{EL} = 2.0 \times t^{0.25} \times 10^4\, J\, m^{-2} \quad (t \leq 10\, s \text{ and t in seconds}). \qquad (22)$$

No limit is provided for longer exposure durations, as normal avoidance behavior will impose limits on duration of exposure. Much longer exposure durations are dominated by concerns of heat stress, and the reader is referred to the appropriate guidelines (Barry et al. 1997; CEN 2004; ISO 2004a and b; ACGIH 2010).

Thermal pain is induced by skin temperatures greater than ~45°C, which 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 by avoidance reactions.

Repetitive exposure

For exposure to repetitive pulses, intermittent exposure, or non-constant exposure levels, the following applies.

As a basic principle, any exposure within the anticipated maximum exposure duration, T, has to be below the corresponding exposure limit for that duration.

For exposure to a series of pulses, this means that the radiant exposure or radiance dose of each pulse has to be below the exposure limit applicable for the respective pulse duration. From this basic principle, it also follows that the exposure dose, summed up over T, has to be below the exposure limit, expressed as radiant exposure or radiance dose, and calculated for T. This latter requirement is mathematically equivalent to a comparison of the average irradiance or average radiance with the exposure limit calculated for T and expressed as irradiance or radiance. For irregular pulse patterns or non-constant exposure levels, any exposure for exposure durations ("temporal analysis windows") between the pulse duration and T is to be considered, as exposure to a part of the pulse pattern can be more critical than exposure to a single pulse or the exposure averaged over T. For times T_{max} where the exposure limit has reached a constant radiance or irradiance level (such as 10,000 s for photochemically induced retinopathy, 1,000 s for infrared exposure of the anterior parts of the eye, 0.25 s for thermally induced retinopathy), for the case that the anticipated exposure duration T is larger than T_{max}, it is not necessary to consider the added radiant exposure or radiance dose (or averaging irradiance or radiance) over longer exposure durations. In other words, for

exposure durations beyond T_{max} (such as 1,000 s for the anterior parts of the eye), no further additivity of exposures needs to be considered, i.e. the 1,000 s exposure episodes can be treated as independent. In all cases of irregular exposure levels, the analysis time window of a given duration (which is to be varied up to T_{max} or T, whichever is shorter) is to be positioned on the time axis over different positions. The most critical scenario, i.e., the highest ratio of exposure level over exposure limit, limits the exposure.

For repeated exposure of the skin where T_{max} equals 10 s, it is not possible to give guidance on the necessary cooling time between exposures, since the injury threshold will depend on ambient temperature as well as on the area that is exposed. When the skin has sufficiently cooled after a given exposure that lasted up to 10 s, there is little biophysical additivity of repeated exposures and the exposures can be considered as in principle independent. Also, as long as there is no pain response (assuming normal pain reaction), there is no risk for injury. For large areas exposed, heat stress is often the limiting factor.

For the retinal thermal limit, there is an additional requirement for the case that the pulse repetition frequency exceeds 5 Hz and that the source is in the extended source regime where the angular subtense of the apparent source is larger than 5 mrad.

The retinal thermal limit for single pulses in a train of n pulses within the exposure duration has to be reduced as follows:

- When the angular subtense of the source is smaller or equal to α_{max}, the single pulse exposure limit, *EL*, is reduced by $n^{-0.25}$ (for $n < 40$) and 0.4 (for $n \geq 40$);
- When the angular subtense of the apparent source is larger than α_{max} but less than 100 mrad, the single pulse exposure limit, *EL*, is reduced by $n^{-0.25}$ (for $n < 625$) and 0.2 (for $n \geq 625$); and
- When the angular subtense of the apparent source is larger than 100 mrad, no reduction of the single pulse exposure limit, *EL*, is necessary.

The maximum anticipated exposure duration must not exceed 100 s. These factors are simplified and overrestrictive for the case of small number of pulses and can, for a less restrictive analysis, be replaced by a multiplication factor $n^{-0.25}$ that is limited to 1/2.5 for the case of $\alpha \leq \alpha_{max}$ and 1/5 for the case of $\alpha > \alpha_{max}$, where n is the number of pulses within the maximum anticipated exposure duration but not longer than 100 s.

APPLYING THE LIMITS

Exposure distance

For an analysis, the exposure of the eye and skin at the position of exposure is compared with the respective exposure limit.

For analysis of the retinal exposure for small sources, such as a small diameter optical fiber, the closest distance at which the human eye can sharply focus is about 100–200 mm. A viewing distance of 100 mm requires extreme near-point accommodation and really applies only to small children and to very myopic individuals. Therefore, 100 mm viewing distance is generally only applied for worst-case assessment of point-source divergent-beam lasers. For evaluation of both the retinal thermal hazard and the blue-light photochemical hazard, a closest viewing distance of 200 mm from the source can be assumed to represent the worst-case exposure.

At shorter distances, the image of a light source would be out of focus and blurred. In most situations, such short viewing conditions are unrealistic. A 20-cm worst-case assessment distance is realistic for conventional lamp sources (including LEDs).

For analysis of the exposure of the anterior sections of the eye, for special applications, such as contact exposure to xenon flash lamps in the face, closer distances might have to be considered.

Determination of pulse duration

The pulse duration is defined as full-width-half-maximum.

Determination of exposure level

For exposures to inhomogeneous irradiance profiles, ICNIRP recommends an averaging aperture with a diameter of 7 mm. For homogenous irradiance profiles, the measurement aperture can be larger.

For comparison with skin exposure limits, the detector should have a cosine response unless the source is sufficiently small.

For comparison with retinal photochemical exposure limits specified as radiance or radiance dose, the exposure level (determined as radiance or radiance dose) has to be averaged over an appropriate angle of acceptance. The usage of the angle of acceptance provides a spatially averaged radiance that accounts for eye movements (Schulmeister 2001). A specific angle of acceptance (field of view) can be accomplished either by using telescopic receiving optics on the instrument to limit the angle of acceptance to γ_{ph}, or by placing, as close as possible to the light source, an opaque baffle with an aperture that subtends an angle of γ_{ph} as seen by the detector. For example, a circular aperture of 11 mm diameter placed over a lamp source will subtend an angle of 11 mrad at a distance of 1 m. The angle of acceptance, γ_{ph}, varies with the exposure duration and is defined in Table 5.

If for a specific exposure scenario, the eye movements are characterized as a function of time, the exposure scenario can be used for an analysis of the exposure limit.

Then, the blue light effective radiant exposure, i.e., B(λ) weighted, of any given point on the retina (assuming no transmission loss) should not exceed 24 kJ m^{-2}. This retinal radiant exposure is derived from the basic limit 10^6 J m^{-2} sr^{-1}, for a pupil diameter of 3 mm. The transmission loss was excluded so that the exposure level can be determined by measurement or calculation "outside" of the eye. For instance, the effective radiant exposure can be measured with an imaging lens and a detector placed in the imaging plane. The limit applies to each image point and no averaging over an acceptance angle larger than 1.5 mrad should be performed since the eye movements are accounted for directly by the measurement.

For comparison with the retinal thermal exposure limits, the acceptance angle can be important if the source has localized radiance hotspots. For pulsed sources with hot-spots, an angle of acceptance of 5 mrad should be used. When the source is smaller than 5 mrad and an averaging angle of acceptance of 5 mrad is used to determine the exposure level, then the source angle, α, for determination of the exposure limit shall not be less than 5 mrad. For CW sources, the angle of acceptance does not have to be less than 11 mrad. If the source is less than 11 mrad and the averaging angle of acceptance is 11 mrad, the source angle α is set to 11 mrad for the determination of the exposure limit. If no hot spots are present in a source that is larger than 5 mrad for the case of pulsed sources and 11 mrad for the case of CW sources, the averaging angle of acceptance can be larger. The averaging angle of acceptance should, however, never be larger than the source. For the case of hot-spots, the measurement of un-averaged radiance is a conservative but sometimes simpler approach.

For comparison with limits to protect the anterior segment of the eye, radiation outside of an angle of acceptance of 80° does not need to be collected due to protection by the eyelids.

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 or little practical concern. The exposure limits for the skin and the anterior parts of the eye were defined so that only the partial irradiance in the wavelength range below 3,000 nm needs to be compared with the respective exposure limits. The partial irradiance that is in the spectral range above 3,000 nm can contribute to the actual physical exposure and this added exposure is accounted for by setting the exposure limit to a corresponding level. For instance, for a thermal radiator with a surface temperature of 1,000°C, about 50 % of the total irradiance is contained in the wavelength range above 3,000 nm, so that when the part below 3,000 nm is limited to 100 W m^{-2}, the permitted total irradiance equals about 200 W m^{-2}. This is well below levels that have induced cataract in an industrial setting (Lydahl 1984). A restrictive but simplified exposure assessment can be performed by using an unfiltered thermal detector that is also sensitive to radiation with wavelengths above 3,000 nm. In that case, irradiances may be averaged over an aperture of 10–50 mm for lengthy exposures.

PROTECTIVE MEASURES

Protective measures should be considered based on general risk management principles. These guidelines do not address specific protective measures.

If the hazard cannot be mitigated by the selection of the source, the most effective hazard control is by engineering controls such as total enclosure of the light source and its emission. In circumstances where such containment is not possible, partial beam enclosure, administrative controls, and restricted access to intense sources, eye and/or skin protectors may be necessary (Hietanen and Hoikkala 1990; Hietanen 1991).

Safety standards for welding have been developed worldwide (Sliney and Wolbarsht 1980; UNEP et al. 1982; Sutter 1990; CEN 2004; ANSI 2009).

PRODUCT SAFETY STANDARDS

Lamp safety standards have been developed which make use of a risk group classification scheme to permit specification of control measures based upon risk posed by the light source (CEI/IEC 2006; IEC 2007; IESNA/ANSI 2007). The emission limits in the product safety standards are generally derived from the ICNIRP or the ACGIH guidelines (ACGIH 2010). IEC and ISO also issue product safety standards for specific product groups which may contain limitations of the emission of optical radiation (IEC 2009; ISO 2010).

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APPENDIX A

RATIONALE FOR THE CHANGES SINCE THE PREVIOUS GUIDELINES

Since the publication in 1997 of the ICNIRP Guidelines for Broadband Incoherent Optical Radiation (ICNIRP 1997), further research has taken place with regard to the temporal, spatial and wavelength dependence of retinal thermal injury.

Spot size dependence

Because of heat flow during the exposure, there is a dependence of the retinal injury threshold on retinal image diameter ("spot-size"). This effect is greatest for longer duration exposures and is nearly non-existent for short-duration pulses of the order of 1 μs or less (Schuele et al. 2005; Zuclich et al. 2007; Schulmeister et al. 2008). Two domains need to be distinguished in terms of the dependence of the exposure limits on the planar angular subtense of the source, α: For values smaller than a critical angle, the exposure limit expressed as radiance or radiance dose depends linearly on the inverse of the planar angular subtense of the source α (Sliney and Wolbarsht 1980; Ham 1989). This dependence reflects the fact that larger retinal irradiance patterns exhibit reduced radial cooling as compared to smaller ones. For values of the planar angular subtense of the source larger than this critical angle, the exposure limit no longer depends on the planar angular subtense of the apparent source. When the diameter of the irradiated spot is large compared to the heat diffusion distance during the pulse, the center of the irradiated spot in the retina is not affected by radial heat flow during the pulse.

It was known from physical principles and from short pulsed laser threshold studies (Zuclich et al. 2007) that for short pulses (where heat flow is negligible during the pulse) there is no spot size dependence. However, as a conservative simplified approach, the inverse spot size dependence in the previous exposure limits was applied up to a critical angle of 100 mrad irrespective of the exposure duration even for pulses. Recent thermal model and ex-vivo studies (Schulmeister et al. 2008) provided for a more complete understanding of the variation of the spot size dependence of retinal thermal injury with pulse duration. This allows for the formulation of a time dependent critical angle to better reflect the retinal irradiance diameter dependence for pulsed sources (Schulmeister 2007). The value of 100 mrad still applies for exposure to CW sources, i.e., for exposure durations larger than 0.25 s.

With the more complete understanding of the temporal trend of the spot size dependence, it was possible to more accurately define the exposure limits.

Pupillary reaction was not considered in the limits for exposures to pulsed sources. To consider the potential of flash exposure in low ambient light levels, a pupil of 7 mm in diameter was applied. However, for longer exposure durations the closure of the pupil reduces the retinal irradiance as shown in Fig. 4 (Stamper et al. 2002) and therefore reduces the risk of injury. For exposure durations longer than 0.25 s, the closure of the pupil decreases the retinal illumination faster than the damage threshold, expressed as retinal irradiance, is reduced. Eye movements and blood flow (Ness et al. 1999) also reduce the risk of thermal injury.

Revision of the retinal thermal hazard function

The study by (Lund et al. 2006) provided the basis to correct the retinal thermal hazard function $R(\lambda)$. When $R(\lambda)$ was first derived more than two decades ago there was a controversy as to the possible synergistic effects between photochemical and thermal retinal damage mechanisms at wavelengths less than 500 nm (blue light). There were two controversial threshold data points for the wavelength of 441.6 nm (Ham et al. 1976; Ham 1989) for blue wavelengths and exposure durations of 1 s and 16 s. It was expected at that time that further research would soon be conducted to determine whether these thresholds were in fact correct or, as existing theory would predict, were apparently one order of magnitude too low. Lund et al. (2006) showed conclusively that the originally published thresholds were indeed one order-of-magnitude too low. This discrepancy was attributed to an error in the dose calculation performed at that time. The more recent study was far more comprehensive than the initial study that had given rise to the conservative adjustment of the $R(\lambda)$ function to provide values greater than 1.0. It had always been assumed that these values were likely to be a great over-statement of the risk and for that reason the $R(\lambda)$ function was not normalized at the maximum value where the $R(\lambda)$ values were at 10.0 (at 435 and 440 nm). After a review of the original work and the recent study, ICNIRP concluded that the $R(\lambda)$ function values above 1.0 were indeed unjustified. The adjustment to the values for $R(\lambda)$ included setting $R(\lambda) = 1.0$ for wavelengths from 445 nm to 495 nm and multiplying all values of $R(\lambda)$ from 385 nm to 440 nm by 0.10. The revised values for the spectral weighting functions are provided in Table 2. No changes to the Aphakic or Blue-Light Hazard Functions (ICNIRP 1997) were required.

Action spectrum for cornea and lens

The exposure limit to protect the cornea and lens for the case of exposure to radiation in the infrared wavelength range (eqns 20 and 21) was originally developed without considering wavelength dependencies since for thermal radiators and long exposure durations, these have little consequence. For infrared LEDs that emit in a relatively small wavelength band, the wavelength dependence of the absorption of optical radiation in the anterior parts of the eye is, however, relevant. Only a part of the optical radiation that is incident on the cornea in the wavelength range of 780 nm to about 1,000 nm is absorbed in the anterior parts of the eye as the cornea is transparent and the iris is also partially transmitting for infrared radiation. Whereas, for wavelengths greater than 1,400 nm, all of the radiation incident on the cornea is absorbed in the ocular media in front of the iris.

As a simple action spectrum it is proposed to weigh the partial exposure applicable to the wavelength range between 780 nm and 1000 nm with a factor of 0.3, which in effect raises the permitted exposure of the eye for near IR LEDs by a factor of about three. This is justified, because although the iris absorbs well in the visible wavelength range, the absorption coefficient of melanin, as chromophore in the iris epithelium (and depending on the iris color also within the stroma of the iris), has a pronounced wavelength dependence (Gabel et al. 1978). In the wavelength range of 780 nm to 1,000 nm, roughly 30 % of the radiation that is incident on the cornea is transmitted through the iris (where it will be scattered and will be finally absorbed by the retina). Indirect heating of the lens via radiation absorbed by the iris is considered to be a risk factor for the development of cataract (Goldmann 1933). However, for the wavelength range 780 nm to 1,000 nm, the energy absorbed in the cornea and the aqueous humor is less than for longer wavelengths where water starts to absorb.

Another aspect to consider is that the exposure limit of 100 W m^{-2} for exposure durations of 1,000 s and longer was set so that contributions to the exposure from the wavelength range beyond 3,000 nm (which is not included in the measurement of the exposure level) were considered in the exposure limit. For the case of IR LEDs, there is no irradiance above 3,000 nm that would add to the actual irradiance of the eye. For thermal radiators on the other hand, the proposed action spectrum adds only a small fraction of additionally permitted exposure level, i.e., less than 1% for surface temperatures of 1,000°C and 5 % for surface temperatures of 1,500°C.

Comparison with exposure limits for laser radiation

The biological effects induced by all types of optical radiation should be similar for any given exposure site, area, and duration of exposure in the same spectral region. For a given broadband source it is necessary to consider several possible types of injury (with different wavelength, pulse duration and spot size dependencies), while for laser radiation for a given single wavelength, exposure

geometry and exposure duration, the most restrictive injury type is defined.

Because of the very high brightness (radiance) of lasers, many lasers are capable of producing thermal burns of the skin, the cornea, or the retina (depending on the wavelength); whereas it is very rare that a broad-band source poses such a risk.

The degree of quantitative uncertainty in relating biological thresholds (derived from broad-band and narrow-band sources to laser exposure) has frequently necessitated the use of additional reduction factors in deriving the exposure limits for lasers that are unnecessary for broad-band optical sources (ICNIRP 2000).

To the extent possible, the exposure limits for broad-band radiation closely parallel those for lasers. In the UV wavelength range and for retinal hazards, the two sets of exposure limits are essentially equivalent. However, for the convenience of the application of the guidelines, the retinal limits are expressed in different units for the two sets of limits, since the default condition for laser radiation is a small source, while for non-laser sources, only extended sources can constitute a retinal hazard. For lasers, it is also necessary to define specific limits for ultra-short pulses.

■■

APPENDIX B

Definition of symbols.

Symbol	Quantity	Units	Comment
$A(\lambda)$			Aphakic hazard function
$B(\lambda)$			Blue-light hazard function
D	Radiance dose or time integrated radiance	J m^{-2} sr^{-1}	Time integrated radiance
D_R	The effective retinal thermal radiance dose	J m^{-2} sr^{-1}	Time integrated radiance dose spectrally weighted for retinal thermal damage
D_B	The effective retinal blue-light radiance dose	J m^{-2} sr^{-1}	Time integrated radiance dose spectrally weighted for retinal blue-light damage
d_p	Pupil diameter	m	
d_r	Retinal diameter	m	
d_s	Diameter of a circular source	m	
E	Irradiance	W m^{-2}	Radiant flux or power (in watts) per unit area incident on a surface
E_B	Blue-light irradiance	W m^{-2}	Irradiance spectrally weighted for blue-light damage in the retina
E_{IR}	Irradianced in the infrared (IR)	W m^{-2}	Irradiance in the infrared for wavelengths from 780 nm to 3000 nm
E_v	Illuminance	lm m^{-2}	Luminous flux (in lumens) per unit area incident upon a surface
E_r	Retinal irradiance	W m^{-2}	
E_λ	Spectral irradiance	W m^{-2} nm^{-1}	
f	Focal length	m	
H	Radiant exposure	J m^{-2}	
H_v	Light exposure	lm s m^{-2}	Photometric dose
IRR			Infrared radiation
IR-A			Infrared radiation type A
IR-B			Infrared radiation type B
IR-C			Infrared radiation type C
L	Radiance	W m^{-2} sr^{-1}	
L_{Aphake}	Aphakic hazard radiance	W m^{-2} sr^{-1}	Spectrally weighted quantity
L_{IR}	Near infrared radiance	W m^{-2} sr^{-1}	
L_v	Luminance	lm m^{-2} sr^{-1} or cd m^{-2}	
L_R	Effective radiance weighted for thermal retinal injury	W m^{-2} sr^{-1}	Radiance spectrally weighted for thermal injury of the retina
L_B	Effective radiance weighted blue-light	W m^{-2} sr^{-1}	Radiance spectrally weighted for blue-light damage in the retina
L_λ	Spectral radiance	W m^{-2} sr^{-1} nm^{-1}	

(Continued on next page)

APPENDIX B (continued)

Symbol	Quantity	Units	Comment
Q	Energy	J	Radiant energy in joules
$R(\lambda)$			Retinal thermal hazard function
t	Exposure duration	s	
t_{max}	Maximum exposure duration	s	
UVR			Ultraviolet radiation
α	Planar angular subtense (of a source)	rad	
α_{min}	Minimal source planar angular subtense	rad	
α_{max}	Maximal planar angular subtense	rad	
γ	Planar angle of acceptance	rad	
γ_{meas}	Planar measurement angle of acceptance	rad	
γ_{th}	Planar angle of acceptance for assessing thermal hazards	rad	
γ_{ph}	Planar angle of acceptance for assessing photochemical hazards	rad	
λ	Wavelength	nm	
τ	Transmittance		
Φ	Radiant flux or Power	W	
Ω	Solid angle	sr	