INTERNATIONAL COMMISSION ON NON-IONIZING RADIATION PROTECTION

ICNIRP GUIDELINES

ON LIMITS OF EXPOSURE TO LASER RADIATION OF WAVELENGTHS BETWEEN 180 nm AND 1,000 µm

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International Commission on Non-Ionizing Radiation Protection*

Abstract—**Since the publication of the ICNIRP Revision of the Guidelines on Limits of Exposure to Laser Radiation (ICNIRP 1996, 2000), further research supports amending the retinal thermal exposure limits in terms of spot size dependence, pulse duration dependence for short pulses and wavelength dependence between 1,200 nm and 1,400 nm. A detailed discussion of the rational for the changes is presented in the Appendix of these Guidelines (Rationale for updating the Guidelines).**
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INTRODUCTION

THE PRESENT guideline is a revision of the previous ICNIRP guidelines (ICNIRP 1996 and 2000). The guidelines for broadband incoherent optical radiation in the visible and infrared wavelength range were revised (ICNIRP 2013) in parallel with the guidelines for laser radiation. The exposure limits were derived on the basis of current knowledge on damage thresholds and in accordance with the ICNIRP principles (ICNIRP 2002).

PURPOSE AND SCOPE

The purpose of these guidelines is to establish the maximum levels of exposure to laser radiation which are not expected to cause adverse biological effects to the eyes and the skin. The guidelines assist with the development of principles of protection against laser radiation hazards. Separate guidelines are defined for exposure to non-laser optical radiation (ICNIRP 1997, 2004, 2013).

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 listed apply to wavelengths from 180 nm–1 mm and to exposure durations between 100 fs and 30 ks (about 8 h). The guidelines apply to all human exposure to optical radiation emitted by lasers. The exposure limits do not apply to deliberate exposure as an integral part of medical treatment. Due to the assumptions regarding pupil diameter and eye movements for deriving the retinal exposure limits, special considerations related to diagnostic exposures should be considered.

The guidelines apply to exposures to laser radiation producing acute onset of observable biological responses. In general there is a lack of knowledge regarding the injury threshold for effects from long term chronic exposure.

Injury thresholds are well defined for the effects that are in the scope of these guidelines. Therefore, in contrast to the ICNIRP guidelines for electromagnetic fields with wavelengths greater than 1 mm, the guidelines for optical radiation in general do not differentiate between workers and the general public.

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 AND UNITS

Exposure limits for optical radiation are expressed using the following quantities and units (Table 1).

Irradiance, E (W m^{-2}), and *radiant exposure, H* (J m^{-2}), are used in describing the concepts of surface exposure dose rate and surface exposure dose from direct exposure to laser radiation. *Radiance*, L (W m^{-2} sr^{-1}) is used to describe the "brightness" of an extended source that gives rise to an image on the retina and this is integrated over time to obtain *time-integrated radiance* or *radiance dose*, D (J m^{-2} sr^{-1}). Other radiometric quantities such as *fluence rate* and *fluence*, although similarly expressed in W m^{-2} and J m^{-2}, respectively, should not be

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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 $m^{-2}sr^{-1}$

used. The fundamental definitions are different as fluence includes the radiation scattered through the unit area (CIE 2011). For a more detailed discussion see (Sliney and Wolbarsht 1980; Schulmeister 2001; Henderson and Schulmeister 2004).

SOURCES

Lasers are used in a wide variety of industrial, consumer, scientific, and medical applications, including optical fiber communication, compact disc players, alignment, welding, cutting, drilling, heat treatment, distance measurement, entertainment, advertisement, optical computing, and surgery. In most industrial applications the laser radiation is totally enclosed, and even partial enclosures effectively preclude direct human exposure. In some applications exposure to potentially hazardous laser radiation is possible, e.g., lasers used in research laboratories, for medical treatment, in entertainment displays, and for alignment procedures. In recent years, laser use in consumer products has increased. For consumer products it is important that potential exposure of the eye and skin is safe. Often these applications employ low-intensity diode or solid-state lasers emitting at wavelengths ranging between 532 and 910 nm (visible and near-infrared radiation). Examples are laser pointers, projectors, distance measurement devices (range finders), supermarket scanners, optical communications, facsimile and printing equipment, computer game controllers and guidance devices for visually impaired.

BIOLOGICAL EFFECTS

Adverse health effects of exposure to laser radiation are theoretically possible across the entire optical spectrum from 180 nm in the ultraviolet (UV) to 10^3 μm in the far infrared (IR), but the risk of retinal injury due to radiation in the visible and near infrared regions (400–1,400 nm) is of particular concern. Injury thresholds vary enormously across the optical spectrum because of variations in biological effects and the different structures of the eye that are potentially at risk (UNEP et al. 1982). The biological effects induced by optical radiation are essentially the same for both coherent and incoherent sources for any given wavelength, exposure site, area, and duration.

Mechanisms of interaction with biological tissue

Laser biological effects are the result of one or more competing biophysical interaction mechanisms: photochemical, thermal, thermo-acoustic and optoelectric breakdown, which vary depending upon spectral region and exposure duration. For example, in the 400–1,400 nm band, thermal injury to the retina resulting from temperature elevation in the pigmented epithelium is the principal effect for exposure durations less than 10 s, and thermal injury to the cornea and skin occurs at wavelengths greater than 1,400 nm. For exposure duration less than about 10 μs, superheating of melanin granules causing microcavitation dominates the injury mechanism (Kelly and Lin 1997; Lin et al. 1999; Brinkmann et al. 2000; Roegener et al. 2004; Schuele et al. 2005; Lee et al. 2007). Optical breakdown and plasma formation occur from sub-nanosecond exposures (Cain et al. 2005; Roach et al. 2004) and the delayed (24 h) appearance of retinal lesions from picosecond exposures may result from secondary effects produced by reactive oxygen species (Glickman 2002). Photochemical injury predominates in the ultraviolet spectral region and is also the principal type of retinal injury resulting from lengthy exposures (10 s or more) to short-wavelength visible radiation (principally "blue light") (Ham Jr. 1989; Lund et al. 2006).

Effects of ultraviolet radiation

Short-wavelength ultraviolet radiation (UVR) is absorbed within the cornea and conjunctiva, whereas long-wavelength UVR is absorbed largely in the lens (UNEP et al. 1982). Exposure to short wavelength UV laser radiation may produce acute photochemical effects: erythema (reddening of the skin), photokeratitis (corneal inflammation), conjunctivitis (conjunctival inflammation) and cataract (clouding of the lens). Typically, 1,000-fold greater exposures of long wavelength UVR are required to produce photokeratitis and erythema compared to short wavelength UVR exposure.

Thermal injury to the skin or the lens and cornea from near UVR exposure has been demonstrated for short pulse durations but has not been demonstrated experimentally for near UVR exposure durations greater than 1 ms (UNEP et al. 1982). With longer exposures, photochemical effects dominate. For photokeratitis, peak sensitivity is around 270 nm, with a decrease in the action spectrum in each direction (Pitts 1973; Schulmeister et al. 2008b). The peak sensitivity of erythema of the skin varies from 200 to 300 nm depending upon the definition of the degree of severity and the delay of appearance of the effect. In the short wavelength UVR region, the cornea is not substantially more sensitive

to injury than un-tanned lightly pigmented skin, but corneal damage is much more disabling (and painful). Repeated exposure of the skin results in tanning and thickening of the stratum corneum, which provides increased natural protection. The same is not true of the cornea. There is evidence that cortical cataract formation is primarily due to excessive exposure to UVR in the 280–315 nm wavelength range (Merriam et al. 2000). In the aphakic eye, UVR wavelengths greater than 300 nm reach the retina and can cause photochemical injury (Ham et al. 1982).

Effects of visible and near infrared radiation

The primary effect on the eye of visible and near infrared radiation (400–1,400 nm) is damage to the retina. Because of the transparency of the ocular media and, in particular, the inherent focusing properties of the eye, the retina is much more susceptible to damage by radiation in this spectral region than any other part of the body. For a point source of light, the increase in irradiance from the cornea to the retina is approximately 100,000. Most of the radiation that reaches the retina is absorbed by the pigmented epithelium and the underlying choroid, which supplies blood to much of the retina (Geeraets and Berry 1968; Vassiliadis 1971; Birngruber 1978; Gabel et al. 1978). The photoreceptors absorb only a small fraction of the incident radiation—less than 15%.

Photochemical, rather than thermal, effects predominate only in the wavelength region from 400 nm to approximately 550 nm for lengthy exposure times (more than 10 s). Photochemical injury is related to absorption by the retinal-pigmented epithelium and choroid of short-wavelength light in the 400–520 nm region (Ham et al. 1976; Lund et al. 2006). This is usually referred to as the blue-light hazard (Sliney and Wolbarsht 1980) but also as Type II photochemically induced retinal damage (Mellerio 1994). Small temperature rises in the retina (of the order of 2–3°C) appear to be synergistic with the photochemical process so that absorption by melanin over a broad wavelength band will also play a role, albeit secondary (Komarova et al. 1978).

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), also referred to as Type I retinal photochemically induced damage. This type of injury has been suggested to be linked to direct damage of the photoreceptors due to bleaching of the photoreceptor pigments.

Shorter-wavelength visible radiation has been suggested to accelerate retinal aging (Marshall 1984; Young 1988; Remé 2005).

Injury to the skin in this spectral region results from temperature rises exceeding 45°C. Photosensitization of the skin for visible light can happen but is extremely rare.

At threshold levels, different mechanisms leading to damage dominate depending on the exposure duration. For exposures from ~0.1 ms to a few seconds, the damage is due to bulk thermal injury. At threshold, pulses with durations less than about 3–10 μs induce damage by microcavitation around melanosomes in the retinal pigment epithelium (RPE), at levels lower than thermally induced damage of the RPE (Schuele et al. 2005; Lee et al. 2007). At suprathreshold levels, Q-switched pulses lasting of the order of 10 ns will also cause thermo-mechanical disruption of the retina, inducing hemorrhage. Within the transition temporal regime between 3–10 μs localized sub-cellular thermal damage around the hot melanosomes probably dominates the cellular damage mechanism. At near infrared wavelengths where the photochemical effect apparently disappears, thermal effects still dominate for exposure times in excess of 10 s.

Thresholds of damage to the retina are known to be a function of the retinal image size, and are also affected by eye movements. The image size dependence trends also depend on the specific damage mechanism.

The thermal mechanisms of retinal injury as a function of retinal image size, i.e. both for viewing minimal-spot-size ("point-sources"), and for extended sources, in the wavelength region 400–1,400 nm are understood through mathematical models of heat transfer. Radial heat flow produces a strong dependence of retinal injury threshold on retinal image size (Lund et al. 2007; Schulmeister et al. 2008a). Damage thresholds are relatively independent of image size for pulses shorter than 1–10 μs in terms of radiant exposure to the retina. For photochemical injury (exposure durations greater than 10 s) the total retinal radiant exposure determines the effect. The injury threshold does not depend on the retinal spot size. However, if the angular subtense of the retinal spot is small as compared to the angular extent of the eye movements, eye movements reduce the effective radiant exposure (Sliney 1988, 1989; Lund 2006).

Effects of mid and far infrared radiation

In the mid and far infrared regions of the spectrum (wavelengths greater than 1.4 μm), the ocular media are opaque because of absorption of the radiation by water. Thus, in these infrared regions, radiation causes damage primarily to the cornea, although lens damage has also been attributed to wavelengths below 3 μm. The Infrared Radiation (IRR) damage mechanism appears to be thermal, at least for exposure durations greater than 1 μs; for pulses of shorter duration, the mechanism at threshold may be thermomechanical. The CO_2 laser (10.6 μm), the Nd:YAG laser (1.06 μm), and the thulium and holmium lasers (~2 μm) that are now used in surgical applications are typical of IRR sources that cause thermal injury to tissue. In the far infrared region (wavelengths > 3 μm), as

in the UVR region, the exposure threshold for damage to the skin is comparable with that for damage to the cornea (McCally et al. 1992, 2004, 2007; McCally and Bargeron 2001, 2003). However, damage to the cornea is likely to be of greater concern because of the adverse impact on vision.

If exposures approached 1000 W m^{-2} for a second or two, there would be an almost immediate sense of heating of the cornea leading to blinking and rotation of the eye. The infrared corneal aversion response requires further study before user safety requirements are relaxed, but the extreme rarity of infrared laser corneal injuries in the workplace clearly suggests that the corneal aversion response may provide significant protection.

Repetitive pulses and repeated exposures

For photochemical interaction mechanisms (i.e., in the ultraviolet and blue spectral region) where radiant exposure–exposure time reciprocity (the Bunsen-Roscoe law) holds, the effect depends on the total dose. In biological tissues with low metabolic rates such as the crystalline lens, additivity has been demonstrated over longer periods of time (e.g., a week) (Dong et al. 2007). Further, some additivity was observed for corneal and retinal effects within 1 – 4 days (Griess and Blankenstein 1981; Zuclich and Blankenstein 1988; Ham Jr. 1989). For thermal effects, the duration of exposures and heat dissipation play major roles in injury processes. The thermal confinement time, the time elapsed before thermal diffusion has an effect, depends upon the volume of tissue heated. For melanin granules, the thermal confinement period is approximately 0.5–1 μs (Neumann and Brinkmann 2005), whereas the confinement time for a typical retinal pigmented epithelial (RPE) cell is on the order of 20–25 μs. Independent of the temporal separation between pulses, some additivity from multiple thermal exposures can occur in the absence of a prolonged temperature rise (Zuclich 1988). Retinal and cutaneous thermal models employing the Arrhenius integral for first-order rate processes (Lukashev et al. 1996; Schulmeister 2007), provide good predictions of the additivity of pulses observed in experimental models. These apply only in exposure duration regimes in which purely thermal damage mechanisms are observed.

Microcavitation mechanisms are responsible for retinal injury for exposure durations less than 1–10 μs. In vitro studies of damage to retinal tissues show very limited additivity (Roider et al. 1993; Brinkmann et al. 2000; Roegener et al. 2004). In vivo studies of retinal injury for minimal retinal irradiance diameters report apparent substantial additivity (Stuck et al. 1978; Lund et al. 1981). Lund (Lund et al. 2009) and Griess (Griess and Blankenstein 1981) have reported that larger retinal image diameters demonstrate reduced additive effects for pulses with microcavitation mechanisms for damage.

Empirical evidence implicate that the threshold expressed as radiant exposure per pulse has a trend that can be expressed by $n^{-1/4}$ or shallower dependence, where n is the number of pulses (Sliney and Lund 2009; Lund 2007). This is also supported by a statistical model (Menendez et al. 1993).

STRUCTURE OF EXPOSURE LIMITS

The exposure limits depend on wavelength, exposure duration (pulse duration), and in some cases on irradiance diameter (spot size).

The tabulation of exposure limit as a function of wavelength, $EL(\lambda)$, can be expressed as the exposure limit for the wavelength where the exposure limit is lowest, EL_{Min}, multiplied with a spectral correction factor:

$$EL(\lambda) = EL_{Min} \times Spectral\ correction\ factor \quad (1)$$

Separate ("dual") exposure limits are specified related to thermal and photochemical retinal injury with different wavelength, pulse duration and spot size dependencies. When applying these exposure limits, a given exposure to visible laser radiation has to be below both limits.

For the photochemical retinal limit, EL_B, at a certain wavelength, λ, is the minimum exposure limit for photochemical injury, $EL_{B:Min}$, multiplied by a spectral correction factor for photochemical injury, $C_B(\lambda)$ (Fig. 1):

$$EL_B = EL_{B:Min} \times C_B(\lambda) \quad (2)$$

For thermal retinal injury in the wavelength range between 700 and 1,400 nm, the exposure limit, EL_{Th}, is expressed as the minimum exposure limit $EL_{Th:Min}$ in

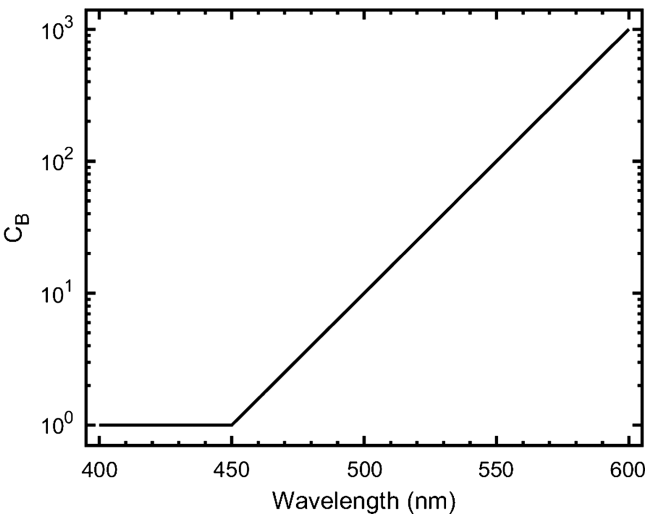

Fig. 1. Exposure limit correction factor, C_B, reflecting the wavelength dependence of photochemically induced retinal injury applicable to exposures of durations greater than 10 s in the visible wavelength range.

that wavelength range (which is the exposure limit for the visible wavelength range), multiplied with a combined correction factor, $C_A(\lambda)\cdot C_C(\lambda)$:

$$EL_{Th} = EL_{Th:Min} \times C_A(\lambda) \times C_C(\lambda) \qquad (3)$$

$C_A(\lambda)$ is related to retinal pigment epithelium absorption and defined for 400 nm < λ < 1,400 nm (Fig. 2); and $C_C(\lambda)$, is related to pre-retinal absorption, and defined for 700 nm < λ < 1,400 nm (Fig. 2).

The minimum thermal exposure limit, $EL_{Th:Min}$, also depends on pulse duration. Exposure limit tables explicitly provide this time dependence.

Exposure to collimated laser beams in the wavelength range of 400 to 1,400 nm produces a minimal spot size on the retina (a point source). For a given power, this exposure condition results in the lowest damage threshold. The exposure limits for retinal thermal injury are therefore expressed for this default condition of a minimum source. Exposure to radiation from extended sources is accounted for by a correction factor C_E. The spot size dependence, C_E depends on the angular subtense of the apparent source, α (see eqn 4, which applies to the wavelength range above 1,050 nm):

$$EL_{Th} = EL_{Th:Min} \times C_{A\,or\,C}(\lambda) \times C_E(\alpha) \qquad (4)$$

RETINAL IMAGE SIZE

For wavelengths between 400 and 1,400 nm, the "retinal hazard region," the ocular exposure limit for retinal thermal damage depends upon the angle subtended by the apparent source.

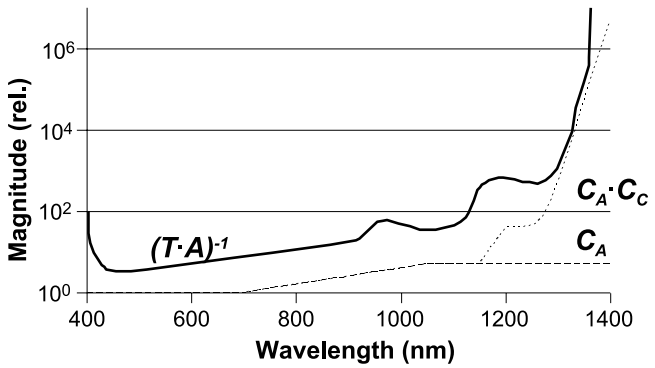

Fig. 2. Comparison of the spectral dependence of the correction factors C_A and C_C with the relative effective spectral absorbance in the RPE. The inverse of the product of the absorption in the RPE and the transmittance of the pre-retinal media, $(T{\cdot}A)^{-1}$ (bold line) is representative of the energy absorbed in the PRE relative to the energy that enters the eye. The spectral correction factor, C_A (dash), approximates the reciprocal of the absorbance, A, of the RPE. The product of the spectral correction factor C_A and the spectral correction factor C_C is plotted as dotted line. The spectral correction factor C_C approximates the reciprocal of the spectral transmittance of the pre-retinal ocular media, T. The correction factor C_C relaxes the corneal exposure limit in the wavelength range 1,150–1,400 nm where the ocular media become increasingly attenuating (Lund et al. 2008).

The parameter α is the plane angle subtended by the apparent source at a given position of the eye in the beam (Fig. 3). The angular subtense of the apparent source is equal to the angle subtended by the smallest retinal image that can be produced considering accommodation of the eye (the accommodation range in laser safety is assumed to be from 10 cm to infinity).

For Gaussian beams (TEM_{00}), it can be shown (Galbiati 2001) that the center of curvature of the wavefront incident on the eye is the location of the apparent source. At this position, the beam diameter can be considered as the source diameter and it determines the angle, α, for the respective exposure position of the eye in the beam.

Since the curvature of the wavefront varies depending on the position in the beam, so does the location of the apparent source. Therefore, it might not be possible to associate a certain apparent source with a given beam, but the location and diameter of the apparent source may depend on the location of determination (Schulmeister 2005).

For low divergence beams, the location of the apparent source is at infinity and α is equal to the beam divergence. However, the angle α should not be confused with the beam divergence. The angular subtense of the apparent source for a laser beam incident on the eye can never be greater than the laser beam divergence, but it can be smaller (Fig. 3). In optics, it is customary to distinguish between a *point source* and an *extended source*. In the context of laser safety, extended sources are subdivided into *intermediate,* and *large sources*.

Point sources

The optical properties of the eye limit the minimum source angle that the eye can resolve. In the context of laser safety, a *point source* is a source subtending an angle less than 1.5 mrad, α_{min}. Sources subtending an angle greater than α_{min} are *extended sources* (Sliney and Wolbarsht 1980).

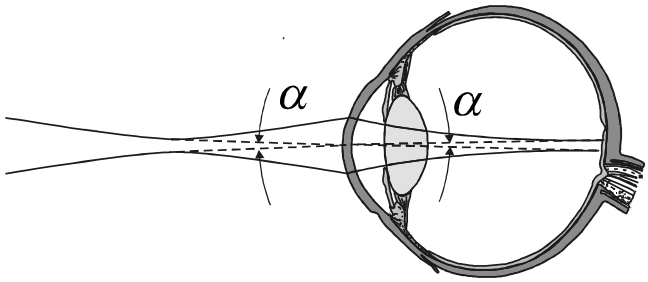

Fig. 3. The parameter α, for a given position of the eye in the beam is the angle subtended by the apparent source that produces the minimal retinal beam profile that can be achieved by accommodation of the eye. The figure is simplified assuming an air-filled eye and that the eye can accommodate to a distance very close to the eye.

Most laser sources are effectively point sources, i.e., they will not produce an extended image on the retina. In a few cases, however, as when viewing a diffuse reflection, some laser diode arrays, or a diffused laser source, extended-source conditions prevail.

The quantities of irradiance (W m^{-2}) and radiant exposure (J m^{-2}) are used for point-source exposure limits. The exposure limits for visible and near infrared radiation can also be expressed as power and energy values where the exposure is the power or energy passing through a 7 mm aperture.

Extended sources

For the purpose of setting exposure limits, it is necessary to treat extended sources in two categories, *intermediate sources* and *large sources*. Retinal injury thresholds for intermediate sources are spot size dependent. When the spot size becomes large enough, the spot size dependence becomes insignificant. This corresponds to an angular subtense, α_{max}. Apparent sources that subtend an angle larger than α_{max} are referred to as large sources.

Intermediate sources

Apparent sources that, at the position of determination, subtend an angle between α_{min} and α_{max} are referred to as *intermediate sources.* For intermediate sources, the retinal injury threshold, due to radial heat flow, is a function of retinal spot size. If the retinal image diameter becomes larger than a critical value, α_{max}, the radial heat flow does not affect the damage threshold when it is given as retinal radiant exposure (Schulmeister et al. 2008a, 2011). Since the extent of radial heat flow depends on time, α_{max} also depends on pulse duration and increases from the value of 5 mrad (0.3°) that is applicable for short pulses to a value of 100 mrad (5.7°) for cw exposure (Fig. 4).

The quantities irradiance (W m^{-2}) and radiant exposure (J m^{-2}) are used for intermediate source exposure limits. The limits can also be expressed in power or energy, the exposure being determined as passing through a 7 mm aperture, and with some rules regarding the angle of acceptance (see section on measurements).

The correction factor C_E (Table 2) is introduced to account for the variation of retinal injury threshold with spot size, which is characterized by the angular subtense of the apparent source (Fig. 3).

The exposure limits are expressed as the product of C_E and the point source exposure limits (i.e., "default" or worst case condition for viewing a laser source).

Large sources

Sources, that at the position of determination subtend an angle α larger than α_{max}, are referred to as large sources. For large sources, retinal injury thresholds when expressed as retinal radiant exposure are essentially independent of spot size. The correction factor C_E becomes equal to $\alpha_{max}/\alpha_{min}$ when the field of view of $\gamma = \alpha_{max}$ is used to determine the exposure level. For a homogeneous and circular source, the exposure level can be determined with an open field of view and then the correction factor, C_E, is as defined in eqn (5):

$$C_E = \frac{\alpha^2}{\alpha_{min} \times \alpha_{max}} \quad (5)$$

Exposure limits for large sources subtending angles greater than α_{max} can be described with different units, i.e., as radiance (W m^{-2} sr^{-1}) and time-integrated radiance

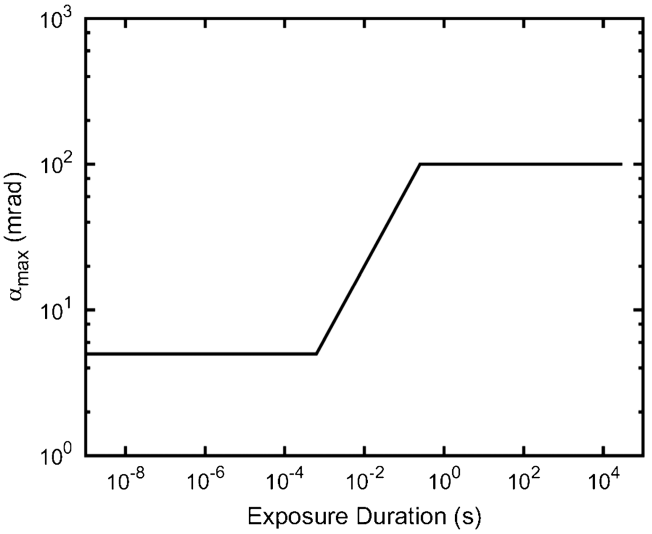

Fig. 4. Exposure duration dependence of the critical angular subtense for intermediate sources, α_{max}.

Table 2. Correction factors to account for the effect of source size.

For sources subtending an angle α (mrad)				
C_E	=	1.0	for	$\alpha \le \alpha_{min}$
		α / α_{min}	for	$\alpha_{min} \le \alpha \le \alpha_{max}$
		α_{max} / α_{min}	for	$\alpha \ge \alpha_{max}$ (with $\gamma = \alpha_{max}$)[a]
where, for exposure duration t (s)				
α_{max}	=	5 mrad	for	$t < 625$ μs[b]
		200 $t^{0.5}$ mrad where t is the exposure time expressed in seconds without the unit	for	625 μs $\le t \le$ 0.25 s
		100 mrad	for	$t >$ 0.25 s
α_{min}	=	1.5 mrad		
For $t > T_2$, the retinal thermal EL is given as constant irradiance				
T_2	=	$10 \times 10^{(\alpha - 1.5)/98.5}$	for	$\alpha_{min} < \alpha \le$ 100 mrad

[a]Note: Exposure limits can be expressed in terms of radiance for $\alpha > \alpha_{max}$. The symbol γ refers to the measurement field of view (angle of acceptance).
[b]Where t is the exposure time in seconds without the unit.

(radiance dose) with units of J m^{-2} sr^{-1}. Thermal model calculations (Freund and Sliney 1999) and experimental data (Lund et al. 2007; Schulmeister et al. 2008a) were used to justify the dependence of retinal injury thresholds and ELs for larger image sizes where α exceeds α_{max}.

Non-circular sources

For a non-circular source, α is the arithmetic mean of the shortest and longest dimension of the image profile. When determining the arithmetic mean, both dimensions have to be limited to α_{min} and to α_{max} (see also Measurement section).

RATIONALE FOR THE EXPOSURE LIMITS

The conditions that result in the most conservative limits for laser and non-laser sources are different. Further, a number of simplifying assumptions are possible for deriving laser exposure limits. Therefore, it is preferable to recommend different exposure limits for lasers and for non-laser sources such as the sun, tungsten filaments, xenon lamps, and LEDs.

Laser radiation is produced by controlled stimulated emission of photons. Stimulated emission typically produces monochromatic radiation, although ultrashort pulses have a broadened spectral bandwidth. Due to the resonant cavity, the laser beam is typically well collimated, but short cavities, e.g. laser diodes, can result in divergent beams. Multimode resonators also produce less well collimated beams, associated with a decreased spatial coherence. The combination of power with collimation is unachievable with non-lasers sources, because conventional sources are limited in radiance.

The exposure limits for lasers were derived on the basis of current knowledge on damage thresholds and in accordance with the ICNIRP principles (ICNIRP 2002). There is a robust set of experimental damage threshold data describing the dose-response relationships for the biological effects of laser radiation on the eye and skin. These damage threshold doses depend on the wavelength, exposure duration and spot size. Most of the threshold data are derived from animal models with response criteria ranging from direct observation of a "minimal visible lesion" (e.g., an ophthalmoscopically visible retinal lesion or a minimal erythema observed in the skin) to assessments of the cellular response by microscopy, histocytochemistry or the function of the system (Sliney et al. 2002). These data are supported by clinical experience with the use of lasers in humans and to some extent analysis of human exposures from both controlled intentional exposures and laser accident cases. Most laser-tissue interactions are supported by application of biophysical models, which assist in understanding the mechanism of injury and estimation of thresholds for exposure conditions not investigated experimentally.

The derivation of exposure limits for laser radiation required a careful analysis of the dependence of the damage thresholds on exposure conditions, assessments of the uncertainty in the experimental data, differences in species and individual susceptibility, the understanding of the underlying interaction mechanism, the implication of the biological effect on the biological system, and the potential for an aversion response to mitigate or limit the exposure for some exposure conditions. Based upon these considerations, reduction factors (a fraction of the known dose to produce an adverse effects for a given exposure condition) were applied to determine the condition-dependent exposure limit.

In view of uncertainties inherent in the damage thresholds, a reduction factor of at least two has been applied in deriving the exposure limits. Simplification of wavelength, exposure duration and/or spot size dependence of the exposure limits compared to the respective trends of the injury thresholds has in many cases implicated higher reduction factors, occasionally as high as approximately two orders of magnitude.

Experimental studies indicate that some additivity exists even beyond the maximum integration duration specified for the exposure limits (such as 30,000 s in the UV wavelength range) (Zuclich 1980; Kremers and van Norren 1988; Ham Jr. 1989; Dong et al. 2007). This was considered in the reduction factors.

Experimentally determined thresholds of injury

For experimental injury threshold determination, incrementing individual retinal exposures are each evaluated by ophthalmoscopy or other methods of examination and rated on a binary scale as damage or not damage. The probability for damage as a function of dose is fitted assuming a normal distribution (Finney 1971). Threshold dose for injury is then referred to as the dose corresponding to a 50% probability for injury, ED-50.

The dose that corresponds to damage at threshold depends on the time interval between the exposure and the examination (the lesion takes some time to biologically develop into detectable change), the method of examination (ophthalmoscopically 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 for thermally induced injury 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.

For determination of the threshold of the cornea and the lens, slitlamp biomicroscopy 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 factor.

Spectral considerations, ultraviolet radiation

The ocular exposure limits for UVR emitting lasers are very similar to those for non-laser UVR sources, and are based on the same biological data (Schulmeister et al. 2008). Most of the experimental threshold data was obtained with lamps spectrally limited to bandwidths of 10 nm or more, but some threshold studies used lasers and these confirm the non-laser data. Because of the extremely strong dependence of the photo-keratitis threshold on wavelength in the range between 300 and 315 nm, slightly more conservative exposure limits were necessary for lasers. For non-laser sources this was not necessary due to averaging over broader wavelength ranges. In the short wavelength range UVR, the reduction factor relative to the thresholds for photokeratitis is up to 100 (Sliney and Marshall 1991). However, the low exposure limit is required in the nanosecond pulse duration range where photoablation is possible at levels lower than the photokeratitis threshold. For a more detailed discussion of UVR health hazards the reader is referred to the rationale for the ICNIRP Guidelines on Limits of Exposure to Ultraviolet Radiation (ICNIRP 2004).

Spectral considerations, visible and near infrared

Injury thresholds for both the cornea and the retina vary considerably with wavelength, and it is therefore necessary to consider the precision required to track this variation. As noted earlier, it was thought acceptable to adjust the exposure limits for different wavelengths, but in a simpler manner than the biological data might indicate. Exposure limits for wavelengths between 700 and 1,050 nm increase with wavelength by a factor C_A (Fig. 2), which increases from 1 to 5 (Fig. 2).

Between 1,050 and 1,400 nm, exposure limits for both eye and skin include a constant spectral correction factor C_A of 5 (incorporated directly into the expressions for the limits) and, for ocular exposure to ultra-short pulses, an additional factor of 2 until non-linear spectral-broadening effects in the 0.1–1.0 ps time domain erase much of the spectral dependence. The reciprocal of the retinal absorption relative to corneal irradiances shown in Fig. 2 is an indication of the relative effectiveness of different wavelengths in causing retinal injury (UNEP et al. 1982).

The correction factor C_C (Fig. 2) adjusts for specific absorption in the ocular media and the factor accounts for the greatly decreased retinal hazard at wavelengths greater than 1,100 nm (Zuclich et al. 2007). The curve in Fig. 2 does not consider the relative hazard to the lens of the eye in the near IR region of the spectrum, which had to be taken into account before limits at this end of the near infrared spectral region were relaxed.

At ocular exposure durations exceeding 10 s, short-wavelength visible radiation can cause photochemical retinal injury. The difference between the ocular exposure limits for short, less than 450 nm, and longer, 450–600 nm, visible wavelengths therefore increases with greater exposure durations. Another wavelength correction factor, C_B, is used to adjust for this change in retinal sensitivity with wavelength. Values of C_B are given in Fig. 1.

Spectral considerations, middle and long wavelength infrared radiation

Exposure limits for wavelengths longer than 1,400 nm were based on an understanding of the possible thermal effects on the cornea and knowledge of exposures that have caused no adverse ocular effects. Because of the lack of accurate data available in much of the far infrared spectral region, worst-case exposure conditions were assumed. Specifically, because of far less variation in spectral absorption and the limited penetration depth of these wavelengths, absorption occurs only in a very thin layer at the anterior surface of the cornea. This condition is epitomized by exposure to laser radiation at 3 μm and at 10.6 μm (CO_2 lasers), and data from studies at the 10.6 μm wavelength were also applied to exposures of the eye for any wavelength beyond approximately 3 μm. At wavelengths less than 3 μm the radiation penetrates more deeply into the cornea in several spectral bands, and significant absorption may take place in the aqueous humour and even the lens (Avdeev et al. 1978; Wolbarsht 1978; Stuck et al. 1981; McCally et al. 1992, 2004, 2007; McCally and Bargeron

2001, 2003). This variation is approximated by spectral divisions at 1.5, 1.8, and 2.6 μm for pulsed lasers.

Spectral correction factors for wavelengths between 1.4 and 3 μm are built into the ocular exposure limits for infrared laser radiation, based on the varying depth of penetration into the cornea and aqueous humour (Stuck et al. 1981). Insufficient data are available, compared with the extensive database at 10.6 μm, to allow highly refined additional wavelength corrections to be defined over the entire IRR range. The exposure limits in the wavelength range between 1,400 and 3,000 nm are based on biological threshold data that vary markedly with wavelength for pulsed, but not for continuous wave, lasers (Lund et al. 1981; Stuck et al. 1981; Schulmeister and Jean 2011b).

It has been suggested that it would be desirable to have smooth transitions in the 1.3–1.5 μm band and around 1.8 μm and beyond. However, this would have required substantially more calculations by the user of the exposure limits. In the past, there have been objections to this approach in other spectral bands. The Commission was reluctant to continue the practice of step functions but considered it to be more important to retain a simple set of values that could be read from a table.

Since the revision of the laser guidelines in 2000 (ICNIRP 2000), additional biological effects research has described corneal, lens and retinal thresholds for wavelengths near 1.3 μm (Zuclich et al. 2007; Vincelette et al. 2009). In this spectral region, the location of the injury at threshold level changes within the eye from the cornea to the lens and to the retina depending on the wavelength and exposure duration. An analysis of the threshold data supports an increase of the EL in the 1.15–1.4 μm spectral region by the spectral correction factor C_c. This significant increase of the limit for retinal thermal injury necessitates a dual-limit to protect the anterior segment of the eye from thermal injury. The dual limit also protects the iris in the visible and infrared spectral region.

Multiple wavelengths

The following applies for exposure to laser radiation that consists of more than one wavelength, such as from combination of beams.

For different wavelengths, if the absorption site is the same, e.g., cornea or retina, and the injury mechanism is the same, e.g., either thermal, thermomechanical or photochemical, the effects are considered spectrally additive. For exposure to wavelengths that are mainly absorbed in different tissues, e.g., one in the cornea and the other in the retina, the exposures have to be considered independently.

In case that the absorption site is the same but the injury mechanisms are different, e.g. when the pulse durations are in different time regimes and/or spot sizes vary, present theories cannot reliably predict the effects of interaction for the various possible combinations. It would be surprising if there were no interaction and if each injury mechanism acted independently of the others. For practical purposes, and in the absence of data, the exposures are considered to be additive where the same tissue is the site of absorption for multiple wavelengths (Wolbarsht and Sliney 1974; Lyon 1985). Because of the non-linearity of thermally induced injury, if thermal mechanisms are involved, this assumption should be conservative (Schulmeister and Jean 2011a).

Ultrashort exposure durations

The development of ELs in the sub-ns time domain considered different interaction mechanisms of laser radiation with biological tissues (Cain et al. 1997; Toth et al. 1997; Roach et al. 1999). The non-linear damage mechanisms do not scale in the same way with wavelength, pulse duration, and retinal image size as do thermal and thermo-acoustic damage mechanisms (Gerstman et al. 1996; Cain et al. 1997, 1999, 2001, 2005; Hammer et al. 1997; Rockwell et al. 1997). A review of retinal threshold data in the ultrashort pulse regime was the basis for recommending a simplification of the pulse duration dependence of the ultrashort pulse limits in the visible wavelength range.

Repetitive-pulse exposure

The additive effects of repetitive pulses or multiple exposures depend upon the mechanism of tissue damage. Photochemical effects depend on the total cumulative dose in the absorbing tissue. For thermal injury, when the energy is delivered during the thermal confinement time, e.g., in a duration where there is no significant heat dissipation during the exposure, the total cumulative dose also determines the thermally induced biological effect. For ultrashort pulses where non-linear effects dominate, little additivity would be expected beyond that anticipated by the heating of the tissue (Cain et al. 2005). For repetitive pulse exposures for durations longer than the thermal confinement time, mathematical models predict the additive effects observed in the experimental biological effects data (Mainster et al. 1970; Schulmeister et al. 2007; Clark et al. 2013).

For longer duration repetitive exposures (e.g., greater than a second), behavioral factors (tissue movement, aversion response) reduce the exposure at a given site. Repeated or intermittent exposures are largely of concern for UVR where photochemical effects and repair processes compete.

One of the most difficult problems in developing the exposure limits concerns repetitive-pulse exposure where the individual pulse duration is less than about 10 μs. Several different formulations have been applied in the past. However, in recent reviews of the large biological database for repetitive pulses (Lund 2007; Sliney and

Lund 2009), it was shown that some of the apparent additivity resulted from the statistical treatment of the data. Hence, the rules for determining the exposure limit for repetitive pulse exposures have been simplified.

Effects of chronic exposure

Chronic exposure to laser radiation is usually rare. The accumulated experience of lasers in use has not shown any evidence for effects after chronic exposure. There is not enough scientific data available to derive guidelines for chronic exposure. However, there is no expectation of unique hazards related to chronic exposure from laser radiation as compared with ambient incoherent exposure. Limits for lengthy exposure to UV-B and UV-C lasers are effectively the same or more conservative than those applicable to non-laser sources.

Impact of eye movement on injury thresholds

Eye movements were only considered in the derivation of the limits for exposure durations exceeding 10 s. Only the thermal injury mechanism exists at durations less than 10 s. Within the 0.1 to 10 s time regime physiological eye movements reduce the effective exposure duration of a given point on the retina, adding additional safety. The data from eye-movement and retinal thermal injury studies (Ness et al. 2000; Lund et al. 2008) and models (Lund 2006) were combined to derive a break-point in viewing time, T_2, at which eye movements compensated for the increased theoretical risk of thermal injury for increased retinal exposure durations if the eye were immobilized (Fig. 5).

The thermal injury threshold expressed as radiant power (W) entering the eye decreases approximately as a function of the exposure duration, $t^{-0.25}$, i.e., a reduction of only 44 % per tenfold increase in duration. If a small spot is projected on the retina, the retinal area exposed increases with increasing viewing time due to eye movements (Velichowsky et al. 1996; Klein et al. 2000), and thus the irradiance (W m^{-2}), and therefore the power absorbed per area unit, decreases. If a large spot is projected on the retina, eye movements will only barely increase the retinal area exposed and threshold exposure will be limited by thermal diffusion, independent of the eye movements. Thus, the retinal area exposed if a small spot is projected towards the retina increases with the viewing time, due to the eye movements. Therefore, the corresponding size of a large spot for which only thermal diffusion is limiting depends on the viewing time. The viewing time breakpoint, T_2, corresponding to the maximum spot size for which eye movements are limiting the threshold exposure, is provided in Fig. 5. Thus, for increasing angular subtense α, the break-point T_2 (Fig. 5) increases from 10 s for small sources to 100 s for larger sources. Beyond 100 s there is no further increase in risk

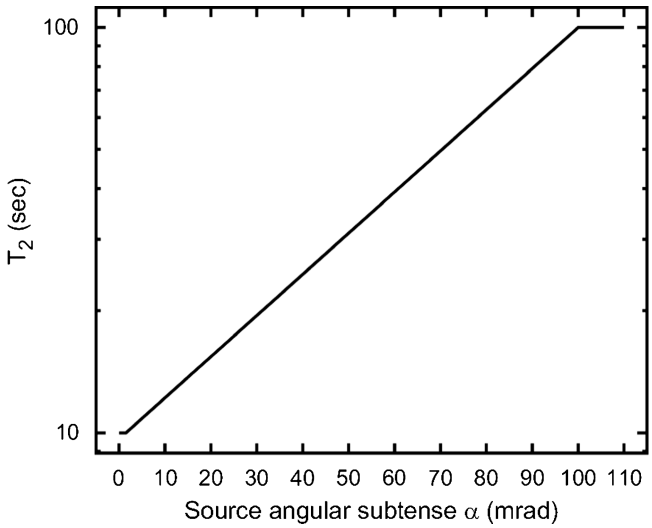

Fig. 5. The time T_2 indicates the transition between the exposure duration dependent exposure limit for extended sources and constant irradiance for exposure durations greater than T_2.

of thermal injury for small and intermediate size images. The specification of limits and measuring conditions attempt to follow these variables with some simplification leading to a conservative determination of exposure.

For photochemically induced retinal injury there is no spot size dependence for a stabilized image. Unlike thermal injury mechanism, the thresholds for photochemical injury are highly wavelength dependent as well as exposure dose dependent, i.e., the thresholds decrease inversely with the lengthening of exposure time. Studies of photochemical retinal injury from welding arcs (Naidoff and Sliney 1974), subtending angles of the order of 1–1.5 mrad, showed typical lesion sizes of the order of 185–200 μm (corresponding to visual angles of 11–12 mrad). These and other studies of eye-movements during fixation led to the derivation of ELs to protect against photochemical retinal injury. These studies also led to the ELs for sources with an angular subtense α less than 11 mrad to be treated equally with "point-type" sources for exposure durations between 10 and 100 s. A field of view, $\gamma_{ph,}$ of 11 mrad should be used to measure the irradiance of all sources subtending an angle greater than 11 mrad. For viewing times in excess of approximately 30–60 s, the saccadic eye motion during fixation is generally overtaken by behavioral movements determined by visual task, and it is quite unreasonable to assume that a light source would be imaged solely in the fovea for durations longer than 100 s. For this reason, the limiting angle of acceptance, γ_{ph}, is increased linearly with the square-root of the exposure duration, t. The minimal angular subtense α_{min} remains at the reference angle of 1.5 mrad for all exposure durations used in thermal retinal hazard evaluation. However, for photochemical retinal hazard assessment, the concept is different, as the angle γ_{ph}

is a linear plane angle for averaging radiance, see below *Angle of acceptance*. When the exposure limit is expressed as corneal irradiance, the angle γ_{ph} is important to apply for extended sources greater than approximately 11 mrad (Schulmeister 2001).

The impact of eye movements for minimal retinal spot sizes, together with the influence of blood flow and the general dependence of retinal thermal injury on exposure duration, permits a leveling of the thermal EL for $\alpha < 1.5$ mrad to a constant irradiance of 10 W m^{-2} in the visible spectrum (400–700 nm) for $t > 10$ s. However, as would be expected, there is only a small impact for a source size of 100 mrad, and the plateau of no further risk of retinal injury due to eye movements does not occur until 100 s. For the photochemical retinal limit, eye movements of the angular extent of 11 mrad are incorporated for exposure durations between 10 and 100 s. Beyond 100 s, it is probably unreasonable to assume that fixation could realistically take place. Conservative limits are recommended by assuming eye movements that increase in terms of angular extent from 11 mrad to 110 mrad with a square root dependence on viewing duration. This results in a constant exposure limit of 1 W m^{-2} for exposure durations longer than 100 s with correspondingly increasing limiting angle of acceptance values γ_{ph}.

Skin exposure

In principle, cutaneous injury thresholds exhibit dependencies on wavelength, exposure duration, and spot size comparable to the eye (Jean et al. 2013).

The exposure limits for the skin are based on injury thresholds for relatively large beam diameters at the skin (spot size greater than 5 mm) (Chen et al. 2005). For smaller beam diameters, the thresholds are higher. Hence, in order to simplify an evaluation, the exposure limits for the skin do not depend on spot size. Guidance is given for use of measurement apertures for very small spots in the section "Measurement."

Exposure limits for the skin also increase by the spectral correction factor, C_A, for wavelengths between 700 and 1,400 nm (Fig. 2). This should not imply that skin exposure limits were derived from ocular exposure data, but since both retinal and skin thresholds vary inversely with melanin absorption in this spectral region, the same correction factor C_A can be used.

In the ultraviolet wavelength range (180 nm to 400 nm), the exposure limits for the skin are set equal to the exposure limits for the eye, which is conservative for the skin regarding immediate onset effects.

In the mid-infrared region where absorption is predominantly influenced by water content, penetration depths for the skin and the cornea are comparable and equal limits apply.

Concerns about heat stress impose restrictions on exposure of large skin surfaces. At wavelengths greater than 1,400 nm, for beam cross-sectional areas of 0.01–0.1 m^2, the exposure limit for durations exceeding 10 s is $10/A_s$ W m^{-2}, where A_s is the area of the exposed skin in m^2. For exposed skin areas exceeding 0.1 m^2, the exposure limit is 100 W m^{-2}.

Reduction factors

The purpose of incorporating a reduction factor into exposure limits is to preclude acute injury or minor effects that could potentially give rise to delayed effects (ICNIRP 2002). Reduction factors were generally largest where uncertainties were greatest or where the fewest experimental data were available. Examples are given as follows.

For the cornea, a minimum reduction factor of approximately 2 was chosen for corneal exposure in the UVR band.

For the retina, generally, an order of magnitude reduction factor was required between the ED-50 for minimal spot size lesions and the exposure limit where some uncertainty regarding the actual retinal spot size exists. Where there is less uncertainty, for example in extended source experiments where spot size is well quantified and probit analysis shows a decreased uncertainty in threshold, a reduction factor of two is thought to be sufficient. This was considered to provide an adequate margin of protection against significant or subjectively-detectable acute injury.

For the visible and infrared wavelength range, a minimum reduction factor of approximately 3 was chosen for skin exposure.

The dependencies of the exposure limits on the relevant parameters of variables were derived from experiments by fitting threshold data to the variables. Often, a linear dependence when plotted on a double logarithmic scale was observed. 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, e.g., t is divided by a factor, t_{ref}, that is equal to 1 × unit such as 1 s. An example is shown in eqn (6) for the retinal thermal limit:

$$H_{EL} = 18\, C_E\, (t\, t_{ref}^{-1})^{0.75} \text{J m}^{-2} \qquad (6)$$

where $t_{ref} = 1$ s.

However, for ease of use, in the guidelines, the dimension-factors were omitted and then it is important that the variables are inserted into the formula in the correct order of magnitude, s and not, e.g., μs.

EXPOSURE LIMITS

Some of the exposure limits are specified with correction factors (Table 3).

In the wavelength range 1,150 nm to 1,400 nm both retinal damage and damage to the anterior segment has to be considered. For wavelengths above 1,400 nm, exposure limits are determined by threshold exposure for damage to the anterior segment of the eye.

Additional parameters used for determination of exposure levels

Some additional parameters used for determination of exposure levels are specified in Table 4.

Spectral dependence

The correction factor C_A (Fig. 2, Table 3), defined for 400 nm $\leq \lambda \leq$ 1,400 nm, is related to the wavelength dependence of the pigment epithelium absorption in the retina, and is also used for skin ELs.

The correction factor C_B (Fig. 1, Table 3), defined for 400 nm $\leq \lambda \leq$ 600 nm, is related to wavelength dependence of photochemically induced injury to the retina.

The correction factor C_C (Fig. 2, Table 3), defined for 700 nm $\leq \lambda \leq$ 1,400 nm, is based on the wavelength dependence of the transmittance of the pre-retinal ocular media.

Spot size dependence

The correction factor C_E (Table 2) applies to extended-source viewing conditions, e.g., diffuse reflection, in the wavelength range of 400 nm to 1,400 nm and implies that the ELs can be increased, provided that the angular subtense of the source, determined at the viewer's eye, is greater than α_{min}, where α_{min} is 1.5 mrad.

Multiple and repetitive pulse dependence

C_p is a correction factor to account for the additivity of multiple pulses for thermally induced injury, see section "Repetitive pulse exposures" for values.

Table 3. Correction factors used in exposure limits in the visible and near infrared waveband.

C_A	=	1.0	for	400 nm $\leq \lambda <$ 700 nm
		$10^{0.002(\lambda/1\ \mathrm{nm} - 700)}$	for	700 nm $\leq \lambda <$ 1,050 nm
		5.0	for	1,050 nm $\leq \lambda \leq$ 1,400 nm
C_B	=	1.0	for	400 nm $\leq \lambda <$ 450 nm
		$10^{0.02(\lambda/1\ \mathrm{nm} - 450)}$	for	450 nm $\leq \lambda \leq$ 600 nm
C_C	=	1.0	for	700 nm $\leq \lambda <$ 1,150 nm
		$10^{0.018(\lambda/1\ \mathrm{nm} - 1150)}$	for	1,150 nm $\leq \lambda <$ 1,200 nm
		$8 + 10^{0.04(\lambda/1\ \mathrm{nm} - 1250)}$[a]	for	1,200 nm $\leq \lambda \leq$ 1,400 nm

[a] C_C becomes large as the wavelength approaches 1,400 nm. However, the calculated exposure limit from Table 5 must then be compared with the skin exposure limit or 2× the skin exposure limit in accordance with note C of Table 5. The lower of the two limits applies.

Table 4. Additional parameters used for determination of exposure limits and exposure levels.

T_1		10 s	for	$\lambda <$ 450 nm
	=	$10 \times 10^{0.02(\lambda/1\ \mathrm{nm} - 450)}$ s	for	450 nm $\leq \lambda <$ 500 nm
		100 s	for	$\lambda \geq$ 500 nm
T_2	=	10 s	for	$\alpha <$ 1.5 mrad
		$10 \times 10^{(\alpha/1\ \mathrm{mrad} - 1.5)/98.5}$ s	for	1.5 mrad $\leq \alpha \leq$ 100 mrad
		100 s	for	$\alpha >$ 100 mrad
T_i	=	5 μs	for	400 nm $\leq \lambda <$ 1,050 nm
		13 μs	for	1,051 nm $\leq \lambda <$ 1,400 nm
γ_{ph}	=	11 mrad	for	$t \leq$ 100 s
		1.1 $t^{0.5}$ mrad	for	100 s $< t <$ 10 ks
		110 mrad	for	$t \geq$ 10 ks

Critical exposure time for transition between exposure duration dependent and constant irradiance exposure limit

The parameter T_2 (Fig. 5, Table 4) indicates the spot size dependent transition between the exposure duration dependent exposure limit for extended sources and constant irradiance for exposure durations greater than T_2. It is also derived from the time-dependence of eye movements.

Limits

The exposure limits for eye and skin are provided in Table 5, Table 6, and Table 7. Special rules apply for repetitive laser exposure (see "Repetitive-pulse exposure" below). Specification of parameters used in the Table 5, Table 6, and Table 7 are given in Table 3 and Table 4. The aperture over which the irradiance or radiant exposure is to be averaged is also given in the respective tables.

Exposure limits for the eye are always specified in relation to the corneal plane perpendicular to the optical axis of the eye. For skin, exposure limits are specified at the skin surface.

In the retinal hazard wavelength range, 400 to 1,400 nm, the EL and exposure dose can be expressed as the corneal radiant exposure, H (J m^{-2}). The corneal radiant exposure can be determined by measuring the total energy through a 7 mm aperture, the effective "total intraocular energy," and dividing by the area defined by that aperture. The EL can be expressed as this total intraocular energy (Table 5 and Table 6) (Schulmeister 2010). Alternatively, for a homogenous extended source, the exposure limits can also be expressed in terms of the radiance dose, D_R (J m^{-2} sr^{-1}).

For exposure durations shorter than those defined in Table 5 and Table 6 (such as less than 1 ns in the ultraviolet wavelength range), the exposure should be limited to the irradiance value that is calculated from the exposure limit given as radiant exposure for the lower range of exposure durations (such as 1 ns).

The time T_1 (Table 4, Fig. 6) applies for small sources, $\alpha \leq \alpha_{\min}$ ($C_E = 1$), and is the critical exposure time below

Table 5. Laser exposure limits for the eye, expressed as irradiance or radiant exposure at the cornea; for the retinal limits, also expressed as power or energy, where the exposure is to be determined as power or energy through a 7 mm aperture.[a,b]

	Exposure duration		Exposure limit ($W\ m^{-2}$ or $J\ m^{-2}$)	Exposure limit (W or J)	Restrictions
Wavelength (nm)	Lower limit	Upper limit			
Ultraviolet					Aperture sizes[b]: 1 mm for $t < 0.35$ s; 1.5 $t^{0.375}$ mm for 0.35 s $< t <$ 10 s; 3.5 mm for $t \geq 10$ s
$180 \leq \lambda < 302$	1 ns	30 ks	30 J m^{-2}		
$302 \leq \lambda < 303$	1 ns	30 ks	40 J m^{-2}		
$303 \leq \lambda < 304$	1 ns	30 ks	60 J m^{-2}		
$304 \leq \lambda < 305$	1 ns	30 ks	100 J m^{-2}		
$305 \leq \lambda < 306$	1 ns	30 ks	160 J m^{-2}		
$306 \leq \lambda < 307$	1 ns	30 ks	250 J m^{-2}		
$307 \leq \lambda < 308$	1 ns	30 ks	400 J m^{-2}		
$308 \leq \lambda < 309$	1 ns	30 ks	630 J m^{-2}		
$309 \leq \lambda < 310$	1 ns	30 ks	1.0 kJ m^{-2}		
$310 \leq \lambda < 311$	1 ns	30 ks	1.6 kJ m^{-2}		
$311 \leq \lambda < 312$	1 ns	30 ks	2.5 kJ m^{-2}		
$312 \leq \lambda < 313$	1 ns	30 ks	4.0 kJ m^{-2}		
$313 \leq \lambda < 315$	1 ns	30 ks	6.3 kJ m^{-2}		
$315 \leq \lambda < 400$	1 ns	10 s	5.6 $t^{0.25}$ kJ m^{-2}		
$315 \leq \lambda < 400$	10 s	30 ks	10 kJ m^{-2}		
Also not to exceed					
$180 \leq \lambda < 315$	1 ns	10 s	5.6 $t^{0.25}$ kJ m^{-2}		
Visible[c]					All for 7 mm limiting aperture
$400 \leq \lambda < 700$	100 fs	10 ps	1.0 C_E mJ m^{-2}	3.8×10^{-8} C_E J	
$400 \leq \lambda < 700$	10 ps	5 μs	2.0 C_E mJ m^{-2}	7.7×10^{-8} C_E J	
$400 \leq \lambda < 700$	5 μs	10 s	18 $C_E t^{0.75}$ J m^{-2}	7×10^{-4} $C_E t^{0.75}$ J	
Dual limits for 400–600 nm visible laser exposure at t>10 s					
Photochemical[c]					
$400 \leq \lambda < 600$	10 s	100 s	100 C_B J m^{-2}	3.9×10^{-3} C_B J	1) For $\alpha > \gamma_{ph}$ use $\gamma = \gamma_{ph}$ mrad
$400 \leq \lambda < 600$	100 s	30 ks	1.0 C_B W m^{-2}	39 C_B μW	2) For $\alpha \leq \gamma_{ph}$, γ not restricted
Thermal[c]					
$400 \leq \lambda < 700$	10 s	30 ks	10 W m^{-2}	0.39 mW	For $\alpha \leq 1.5$ mrad
$400 \leq \lambda < 700$	10 s	T_2 s	18 $C_E t^{0.75}$ J m^{-2}	7.0×10^{-4} $C_E t^{0.75}$ J	For $\alpha > 1.5$ mrad
$400 \leq \lambda < 700$	T_2 s	30 ks	18 $C_E T_2^{-0.25}$ W m^{-2}	7.0×10^{-4} $C_E T_2^{-0.25}$ W	For $\alpha > 1.5$ mrad
Short wavelength IRR[d]					
$700 \leq \lambda < 1,050$	100 fs	10 ps	1.0 C_E mJ m^{-2}	3.8×10^{-8} C_E J	For 7 mm aperture
$700 \leq \lambda < 1,050$	10 ps	5 μs	2.0 $C_A C_E$ mJ m^{-2}	7.7×10^{-8} $C_A C_E$ J	
$700 \leq \lambda < 1,050$	5 μs	10 s	18 $C_A C_E t^{0.75}$ J m^{-2}	7.0×10^{-4} $C_A C_E t^{0.75}$ J	
$1,050 \leq \lambda < 1,400$	100 fs	10 ps	1.0 $C_C C_E$ mJ m^{-2}	3.8×10^{-8} $C_C C_E$ J	
$1,050 \leq \lambda < 1,400$	10 ps	13 μs	20 $C_C C_E$ mJ m^{-2}	7.7×10^{-7} $C_C C_E$ J	
$1,050 \leq \lambda < 1,400$	13 μs	10 s	90 $C_C C_E t^{0.75}$ J m^{-2}	3.5×10^{-3} $C_C C_E t^{0.75}$ J	
$700 \leq \lambda < 1,400$	10 s	30 ks	10 $C_A C_C$ W m^{-2}	3.9×10^{-4} $C_A C_C$ W	For $\alpha \leq 1.5$ mrad
$700 \leq \lambda < 1,400$	10 s	T_2 s	18 $C_A C_C C_E t^{0.75}$ J m^{-2}	7.0×10^{-4} $C_A C_C C_E t^{0.75}$ J	For $\alpha > 1.5$ mrad
$700 \leq \lambda < 1,400$	T_2 s	30 ks	18 $C_A C_C C_E T_2^{-0.25}$ W m^{-2}	$7.0\times10^{-4} C_A C_C C_E T_2^{-0.25}$ W	For $\alpha > 1.5$ mrad
Mid and long wavelength IRR					Aperture sizes[b]: 1 mm for $t < 0.35$ s; 1.5 $t^{0.375}$ mm for 0.35 s $< t <$ 10 s; 3.5 mm for $t \geq 10$ s
$1\,400 \leq \lambda < 1\,500$	1 ns	1 ms	1 kJ m^{-2}		

(Continued on next page)

Table 5. *(Continued)*

Wavelength (nm)	Exposure duration Lower limit	Exposure duration Upper limit	Exposure limit (W m^{-2} or J m^{-2})	Exposure limit (W or J)	Restrictions
1 400 ≤ λ < 1 500	1 ms	10 s	5.6 $t^{0.25}$ kJ m^{-2}		
1 500 ≤ λ < 1 800	1 ns	10 s	10 kJ m^{-2}		
1 800 ≤ λ < 2 600	1 ns	1 ms	1.0 kJ m^{-2}		
1 800 ≤ λ < 2 600	1 ms	10 s	5.6 $t^{0.25}$ kJ m^{-2}		
2 600 ≤ λ < 1 mm	1 ns	100 ns	100 J m^{-2}		
2 600 ≤ λ < 1 mm	100 ns	10 s	5.6 $t^{0.25}$ kJ m^{-2}		
1 400 ≤ λ < 1 mm	10 s	30 ks	1.0 kW m^{-2}		

[a]t in seconds.
[b]For exposure duration t, the "lower limit" ≤ t < "the upper limit". For example when the exposure duration lower limit is 100 fs and the upper limit of the exposure limit is 10 ps, then 100 fs ≤ t < 10 ps. Likewise for a lower limit and upper limit of 10 ps and 5 μs respectively, then 10 ps ≤ t ≤ 5 μs.
[c]For beam diameters less than 1mm and pulse durations less than 0.35s, the actual radiant exposure, i.e. not averaged over of the limiting aperture of 1 mm, should be compared to the exposure limit.
[d]In the visible wavelength range for large retinal spot sizes, the retinal thermal exposure limit for the eye given in terms of the corneal radiant exposure may exceed the exposure limit of the skin. In that case, the skin exposure limit also applies to the exposure of the eye to protect the anterior parts of the eye. For exposures of the eye only in the infrared wavelength range, two times the skin exposure limit should be applied. For general safety analysis, both the skin and the eye exposure limit would have to be considered. Therefore, this additional restriction for the exposure of the eye (using the skin exposure limit as dual limit) is relevant only for situations where only the eye is exposed.
[e]For small sources subtending an angle of 1.5 mrad or less, the visible dual exposure limits from 400 nm to 600 nm, for times greater than 10 s, reduce to the thermal limits for times less than T_1 and to photochemical limits for longer times (Table 2, Table 3 and Table 4).

Table 6. Laser exposure limits for the eye for λ = 400−1400 nm expressed as radiance or radiance dose.[a]

Wavelength (nm)	Exposure duration Lower limit	Exposure duration Upper limit	Exposure limit	Restrictions
Visible				
for t ≤ 10 s				
and α ≥ α_{max}				
400 ≤ λ < 700	100 fs	10 ps	0.17 kJ m^{-2} sr^{-1}	All only for large sources with constant radiance
400 ≤ λ < 700	10 ps	5.0 μs	0.34 kJ m^{-2} sr^{-1}	
400 ≤ λ < 700	5.0 μs	0.625 ms	3.1 $t^{0.75}$ MJ m^{-2} sr^{-1}	
400 ≤ λ < 700	0.625 ms	0.25 s	76 $t^{0.25}$ kJ m^{-2} sr^{-1}	
400 ≤ λ < 700	0.25 s	10 s	0.15 $t^{0.75}$ MJ m^{-2} sr^{-1}	
For t > 10 s; dual limits				
Photochemical				Photochemical radiance EL valid for all α, but averaging of exposure level over γ_{ph}
400 ≤ λ < 600	10 s	10 ks	1.0 C_B MJ m^{-2} sr^{-1}	
400 ≤ λ < 600	10 ks	30 ks	100 C_B W m^{-2} sr^{-1}	
Thermal for α ≥ 100 mrad				
400 ≤ λ < 700	10 s	100 s	0.15 $t^{0.75}$ MJ m^{-2} sr^{-1}	
400 ≤ λ < 700	100 s	30 ks	47 kW m^{-2} sr^{-1}	
Short wavelength IRR				
for α ≥ α_{max}				
700 ≤ λ < 1 400	100 fs	10 ps	0.17 kJ m^{-2} sr^{-1}	
700 ≤ λ < 1 400	10 ps	5.0 μs	0.34 C_A C_C kJ m^{-2} sr^{-1}	
700 ≤ λ < 1 400	5.0 μs	0.625 ms	3.1 $t^{0.75}$ C_A C_C MJ m^{-2} sr^{-1}	
700 ≤ λ < 1 400	0.625 ms	0.25 s	76 $t^{0.25}$ C_A C_C kJ m^{-2} sr^{-1}	
700 ≤ λ < 1 400	0.25 s	10 s	0.15$t^{0.75}$ C_A C_C MJ m^{-2} sr^{-1}	
700 ≤ λ < 1 400	10 s	30 ks	10 C_A C_C W m^{-2}	
700 ≤ λ < 1 400	10 s	100 s	0.15$t^{0.75}$ C_A C_C MJ m^{-2} sr^{-1}	
700 ≤ λ < 1 400	100 s	30 ks	47 C_A C_C kW m^{-2} sr^{-1}	

[a]t in seconds.

Table 7. Laser radiation exposure limits for the skin.[a]

Wavelength (nm)	Exposure duration		Exposure limit	Restrictions
	Lower limit	Upper limit		
Ultraviolet				
$180 \leq \lambda < 400$	1 ns	30 ks	Same as EL for the eye See Table 5	3.5 mm limiting aperture[b]
Visible and short wavelength IRR				3.5 mm limiting aperture[b]
$400 \leq \lambda < 1{,}400$	1 ns	100 ns	200 C_A J m^{-2}	
$400 \leq \lambda < 1{,}400$	100 ns	10 s	11 C_A $t^{0.25}$ kJ m^{-2}	
$400 \leq \lambda < 1{,}400$	10 s	30 ks	2.0 C_A kW m^{-2}	
Mid and long wavelength IRR[c]				
$1{,}400 \leq \lambda < 1$ mm	1 ns	30 ks	Same as EL for the eye See Table 5	3.5 mm limiting aperture[b]

[a] t in seconds.
[b] For beam diameters less than 1 mm, the actual radiant exposure, i.e., not averaged over of the limiting aperture of 3.5 mm, should be compared to the exposure limit.
[c] For wavelengths above 1,400 nm, exposure durations longer than 10 s and exposed skin areas greater than 0.1 m^2, the exposure limit is reduced to 100 W m^{-2}. For exposed areas between 0.01 m^2 (where the limit is 1,000 W m^{-2}) and 0.1 m^2 (where it is 100 W m^{-2}), the exposure limit is adjusted proportionally to the inverse of the exposed area.

which the retinal thermal EL is lower than the photochemical EL.

At exposure times exceeding 10 s, photochemical injury predominates in the ultraviolet and the short wavelength visible part of the spectrum (Ham Jr. 1989; Lund et al. 2006).

Exposure limits

In Table 5, the EL for thermally and photochemically induced retinal injury are expressed as irradiance or radiant exposure limiting the exposure at the corneal level which is averaged over an aperture of 7 mm diameter, as well as an alternative way to express the exposure limits that apply to the retina, in terms of power or energy passing through an aperture of 7 mm.

Some ocular ELs as a function of exposure durations and some selected wavelengths are shown in Fig. 7.

Since laser beams usually are point sources, producing a minimal retinal spot size, measurements and analysis are simplified by providing exposure limits as irradiance or radiant exposure and, alternatively, as power or energy through an aperture. It is possible to express the retinal thermal and photochemical ELs also in units of radiance or radiance dose, which results in equivalent analysis provided that correct averaging field of views are used for the determination of the exposure level. Table 6 lists these alternative radiance or radiance dose ELs for retinal thermal ELs for the case of large sources ($\alpha > \alpha_{max}$) and

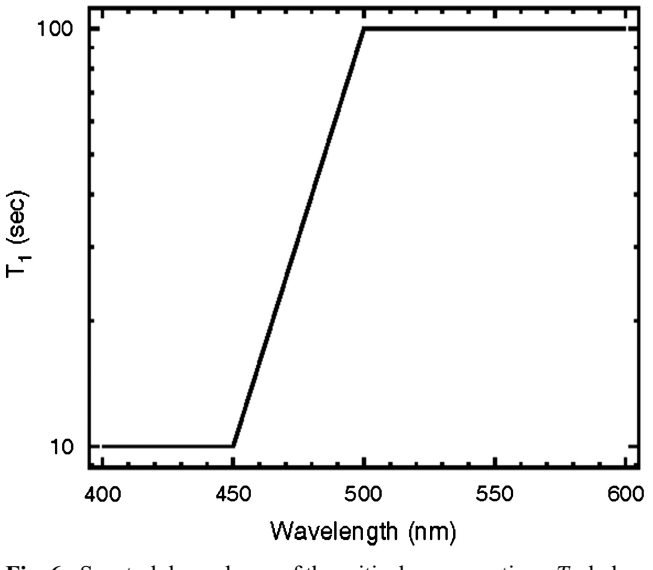

Fig. 6. Spectral dependence of the critical exposure time, T_1, below which the retinal thermal EL is lower than the photochemical EL, for the case of small sources (α <1.5 mrad) (obtained by equating the two exposure limits for the case of small sources).

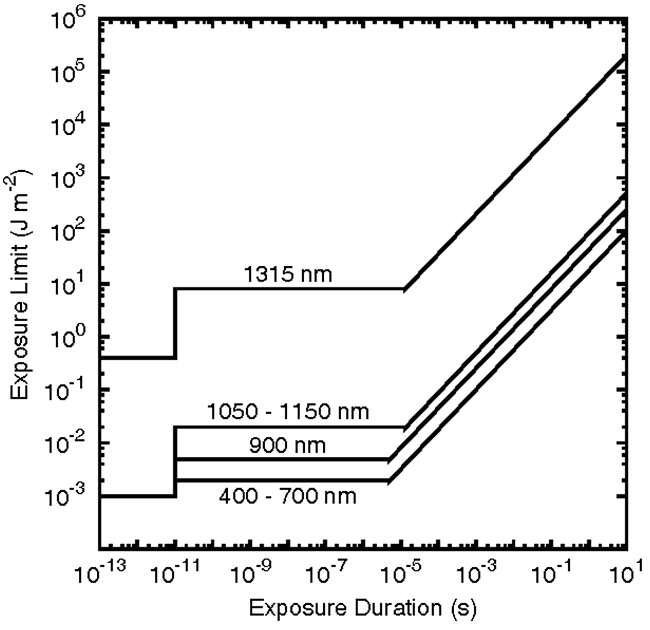

Fig. 7. Exposure limits for point-source viewing of pulsed laser radiation for selected wavelengths in the range 400–1400 nm.

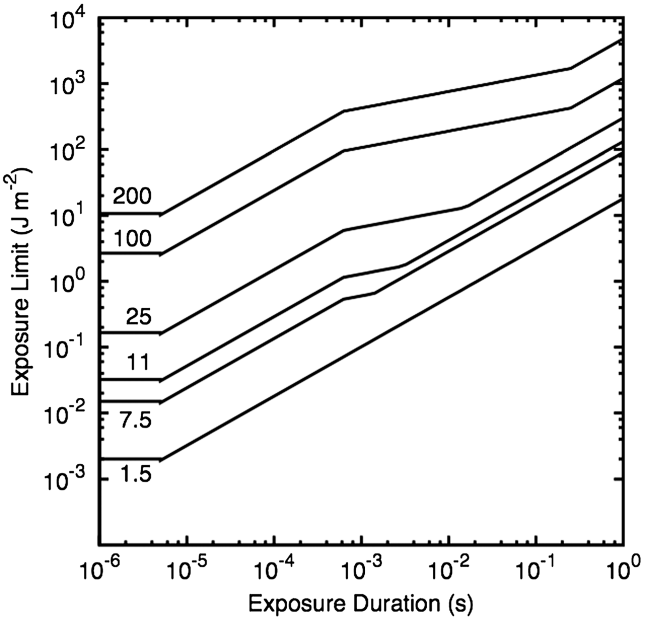

Fig. 8. Exposure duration dependence of the retinal thermal limits for a number of angular subtenses of the source, for the wavelength range 400–700 nm.

for retinal photochemical exposure limits, which are applicable to all source sizes.

The exposure duration dependences of the retinal thermal limits for a number of angular subtenses of the apparent source are plotted in Fig. 8.

The exposure limits for continuous-wave laser radiation for point sources in the wavelength range of 400–1,400 nm are shown in Fig. 9.

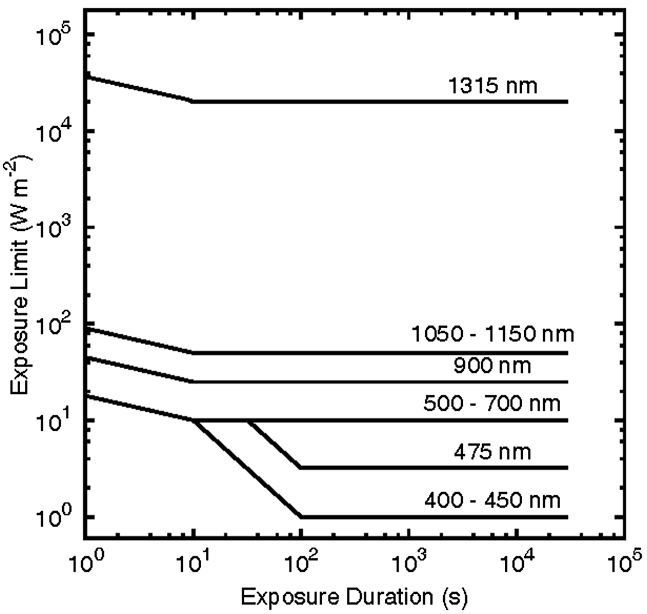

Fig. 9. Exposure limits for point-source viewing of continuous-wave laser radiation for selected wavelengths in the range 400–1,400 nm.

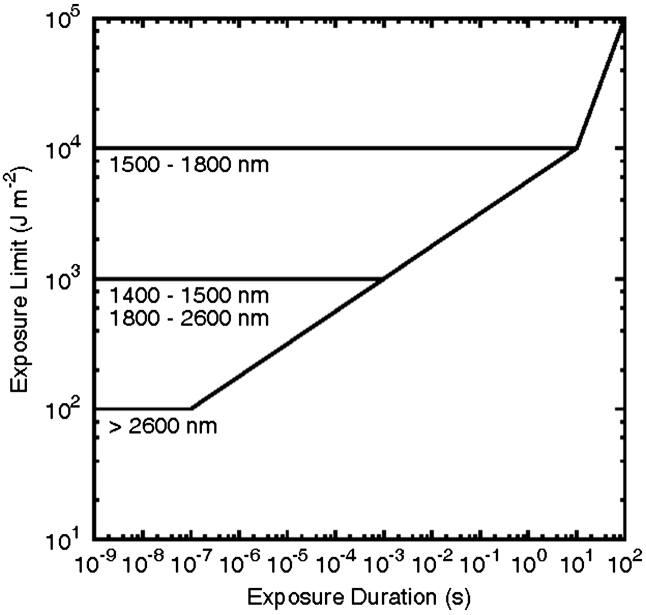

Fig. 10. Exposure limits for ocular and skin exposure to middle and far-infrared laser radiation.

The exposure limits for ocular exposure to middle and far-infrared laser radiation are given in Fig. 10.

Exposure duration

Determining the exposure limit applicable for a specific laser exposure requires a determination of the wavelength and the exposure duration. For a single-pulse exposure, this duration is generally taken as full-width half-maximum (FWHM). However, the following criteria should be applied where repeated exposures or lengthy exposures occur.

For any single-pulse laser exposure, the exposure duration is the pulse duration, t, as defined above. For all skin exposure limits, and for ocular exposure to non-visible or weakly visible wavelengths, i.e., less than 400 nm or greater than 700 nm, the exposure duration for continuous wave lasers is the maximum anticipated time, T_{max}, of direct exposure. For exposure of the eye to any continuous wave laser, the exposure duration is the maximum anticipated time of direct viewing. However, if purposeful staring into a visible, 400–700 nm, beam is not intended or anticipated, an exposure duration of 0.25 s should be used. For ocular exposures in the near-infrared, 700–1,400 nm, a maximum exposure duration of 10 s provides an adequate hazard criterion for unintended viewing conditions. In this case, eye movements will provide a natural exposure limitation and thus eliminate the need to consider exposure durations greater than 10 s, except for unusual conditions. In special applications, such as intentional exposure from medical instrumentation for diagnostic purposes, even longer exposure durations may apply (Sliney et al. 2005).

Because of lack of biological retinal threshold data for pulse durations less than 100 fs it is recommended to limit the peak irradiances to the exposure limit applicable to 100 fs pulses at the wavelength of interest. At present, exposure limits for the skin are not provided for durations less than 1 ns because of a lack of biological data. However, as a conservative interim approach, one could limit exposures to levels less than 10 % of the 1 ns exposure limit. Similarly, ocular exposure limits for wavelengths less than 400 nm and greater than 1400 nm are not provided for pulse durations less than 1 ns, and a similar, conservative interim guideline would be to limit exposure below 10 % of the 1 ns exposure limit.

Repetitive pulse exposures

Within any one day, repeated exposure to laser radiation can be the result of multiple exposures to a beam from a continuous-wave laser or of exposures to repetitively pulsed lasers and some scanning beam lasers. Scanning beams create repetitive-pulse exposures of the eye. Both the individual pulse duration and the total cumulative exposure duration must be determined. Total exposure duration of the train of pulses is determined in the same manner as for continuous wave exposures. That is the elapsed time from the beginning of the exposure (the beginning of the first pulse) to the end of the last pulse including the time between pulses.

Currently available data in the nanosecond pulse duration regime suggest that the threshold expressed as energy per pulse decreases close to $n^{-0.25}$ where n is the number of pulses. However, in these studies, exposure sites were observed 1 h after exposure to determine the ED-50, and those thresholds are not consistent with 24 h single pulse threshold data. Data determined at 24 h after exposure are needed for empirical confirmation of the dependence on pulse number observed for 1 h data. The current guidance, for exposures exceeding 600 pulses, is equal to the previous exposure limits (ICNIRP 2000).

Each of the following three general rules should be applied to all repetitive exposures as occur from repetitively pulsed or scanning laser systems:

1. The exposure from any single pulse in a train of pulses shall not exceed the EL for a single pulse of that pulse duration;
2. The exposure from any group of pulses, or sub-group of pulses in a train, delivered in time T shall not exceed the EL for time T. T is to vary between the pulse duration and the total exposure duration; and
3. For the retinal thermal limits, an additional factor C_p is applied to the single pulse limit with the following conditions. The value of C_p is equal to $n^{-0.25}$ (except as otherwise stated), where n is the number of pulses which occur within an exposure time of T_2 (Table 4).
   a. For $\alpha \leq 5$ mrad with pulse durations exceeding T_i (5 μs for 400–1,050 nm), $C_p = 1.0$;
   b. For $\alpha > 5$ mrad, and individual pulse durations exceeding T_i, then
      if $\alpha \leq \alpha_{max}$, and if $n > 40$, $C_p = 0.4$
      if $\alpha > \alpha_{max}$ and $\alpha < 100$ mrad and if $n > 625$, $C_p = 0.2$
      if $\alpha \geq 100$ mrad $C_p = 1$
   c. For pulse durations less than or equal to T_i, and for exposure durations less than or equal to 0.25 s, $C_p = 1.0$. For an exposure duration (used for the safety assessment as assumed maximum anticipated exposure duration) longer than 0.25 s and more than 600 pulses within exposure duration, $C_p = 5 \times n^{-0.25}$. For the case of visible radiation, this additional restriction only applies for the condition of intentional exposure.

Special precautions

These exposure limits apply to the general population. It should however be recognized that some rare photosensitive individuals may react to UVR laser exposures below these limits. In addition, the exposure limits from 300 nm to 400 nm do not apply to infants or to aphakic individuals (ICNIRP 1997). Such individuals should therefore take more rigorous precautions to avoid exposure to UV laser radiation.

These exposure limits are not intended to limit use of lasers as an integral and essential part of medical treatment. However, for diagnostic exposures, the special considerations related to this exposure condition should be considered (Sliney et al. 2005).

The above ocular exposure limits should preclude injury from non-linear (ultrashort) damage mechanisms, thermal damage mechanisms and photoretinopathy from short-wavelength light as discussed in these guidelines. However with pupils medically dilated and stabilized retinal exposures, another type of photochemical retinal injury can be of potential concern. This holds for wavelengths outside the "blue-light" hazard region but where middle-wave (green) and long-wave (red) cone photoreceptors both strongly absorb, i.e., 500-600 nm (Balaratnasingam et al. 2008). These conditions also require lengthy exposures and apparently result from an oversaturation of the cone opsins (Kremers and van Norren 1988; Mellerio 1994). While this has traditionally been thought of as an unrealistic exposure condition (Sliney and Wolbarsht 1980), it may occur in some specialized ophthalmic instrument exposures and caution must be exercised (Mellerio 1994).

MEASUREMENT

Limiting apertures used for averaging exposure

The exposure limits are expressed as irradiance (or radiant exposure). Depending on the spatial profile of the

beam, the measured irradiance can depend on the diameter of the averaging aperture.

For the retina, an averaging aperture of 7 mm is specified, being based on the diameter of a dilated pupil.

Several difficulties arise from the use of small apertures; more time is required to assess exposure, a more sensitive instrument is required, calibration problems give rise to potential inaccuracies, and calculations may be more difficult (Le Bodo 1976; Rocherolles 1978; Sliney and Wolbarsht 1980).

For the discussion of averaging apertures, small irradiance hot spots within a larger beam have to be distinguished from small beam diameters due to focusing. With consideration of the above, averaging apertures as described below are justified even for the case of irradiance hot-spots as long as the beam diameter is not significantly smaller than the averaging aperture.

A 1 mm aperture is biologically supportable because of scattering in tissue; hence, a 1 mm aperture is recommended for pulsed exposure of the cornea and conjunctiva to UVR and to IRR of wavelength greater than 1.4 μm.

An averaging aperture of 3.5 mm was deemed justifiable for both pulsed and continuous exposure of the skin, where increased scattering takes place. Moreover, for continuous exposure conditions of the eye, as well as the skin, heat flow, body movements, and scattering, tend to eliminate any adverse effects of "hot spots" smaller than about 3.5 mm (Rockwell and Goldman 1974; Sliney and Wolbarsht 1980; McCally et al. 1992; IEC 2007; ANSI 2009). The same arguments hold for continuous exposure of the cornea and conjunctiva to UVR at wavelengths less than 400 nm. Furthermore, two factors that account for localized variations in beam irradiances, atmospherically induced "hot spots" by scintillation and the mode structure in multimode lasers, seldom account for significantly higher localized beam irradiances within areas less than 3.5 mm in diameter.

Another problem appears at far infrared wavelengths greater than 100 μm, at which the aperture size of 1 mm begins to create significant diffraction effects and calibration becomes difficult. Fortunately, "hot spots" must, because of the laws of physics, be generally larger than at shorter wavelengths, and aperture diameters of 11 mm, an area of about 1 cm^2, are therefore specified for wavelengths greater than 100 μm.

The diameters of the apertures used for averaging exposure levels are summarized in Table 8.

No modifications of the exposure limits are permitted for reduced energy entering an assumed pupil size less than 7 mm.

For beams, incident on the cornea or the skin, with a diameter of less than 1 mm, it might be prudent to determine the actual irradiance or radiant exposure. For the case that the beam diameter is significantly less than the averaging aperture, it cannot be excluded that minor thermal injury occurs when the averaged irradiance is approaching the exposure limit. When the beam diameter is less than 1 mm, the actual irradiance or radiant exposure needs to be determined and compared with the exposure limits to avoid injury to the skin. The beam diameter that can be used is, applying a Gaussian approximation, the distance between diametrically opposed points in the beam where the local irradiance is 1/e, 0.37 times peak irradiance.

Angle of acceptance

For the retinal limits, for extended sources, the angle of acceptance of the radiometer can have an impact on the determined exposure.

For point sources, the receptor acceptance angle γ must be at least α_{min}. For extended sources, one has to distinguish between application of photochemical and thermal limits.

For uniform large sources when the exposure limit is expressed as radiance, the acceptance angle can be as large as α, both for the photochemical as well as the thermal limits.

Thermal

For comparison of the exposure of uniform intermediate sources with photothermal limits in terms of irradiance, the acceptance angle γ must be at least as large as α.

If the source is non-uniform, i.e., contains hot spots, an acceptance angle must be chosen so that it is sufficiently small to assess the hot spot but not less than 1.5 mrad nor greater than α_{max}. For each hot spot, or a non-uniform part of the source, assessed by an angle of acceptance, γ, the exposure must be compared with the limit applicable to a source size subtending an angle of α that is set equal to γ. The size and position of the angle of acceptance γ within the apparent source has to be adjusted to produce the most restrictive analysis (i.e., to maximize the ratio of energy determined within γ over α).

Photochemical

For comparison of the exposure from sources smaller than 11 mrad with the photochemical limits, expressed

Table 8. Averaging apertures for applying the exposure limits.

Spectral region (nm)	Exposure duration, t (s)	Eye exposure (mm)	Skin exposure (mm)
$180 \le \lambda < 400$			
	1.0 ns – 0.35 s	1.0	3.5
	0.35 s – 10 s	1.5 $t^{3/8}$	3.5
	10 s – 30 ks	3.5	3.5
$400 \le \lambda < 1\,400$ nm	1.0 ns – 30 ks	7.0	3.5
$1{,}400 \le \lambda < 10^5$ nm			
	1.0 ns – 0.35 s	1.0	3.5
	0.35 s – 10 s	1.5 $t^{3/8}$	3.5
	10 s – 30 ks	3.5	3.5
$10^5 \le \lambda < 10^6$ nm	1.0 ns – 30 ks	11	11

as irradiance, or radiant exposure, and for all exposure durations (10 s–30 ks), any acceptance angle larger than the source size can be used. For sources greater than 11 mrad and exposure durations between 10 and 100 s, use an acceptance angle (γ) that is equal to γ_{ph} = 11 mrad. For exposure durations between 100 s and 10 ks, the angle of acceptance, γ, steadily increases with time and it defines the cone angle over which the irradiance is collected (Schulmeister 2001). Specifically, for exposure durations between 100 s and 10 ks and source sizes $\alpha > \gamma_{ph}$, an acceptance angle of $\gamma_{ph} = 1.1 \times t^{0.5}$ mrad should be used for comparison with the exposure limit expressed in irradiance (or radiant exposure). For sources greater than 110 mrad and exposure durations from 10 ks to 30 ks, the measurement acceptance angle for limits expressed in irradiance should be 110 mrad. A linear cone angle of 11 mrad is approximately equivalent to a solid angle of 10^{-4} sr and a linear cone angle of 110 mrad corresponds to a solid angle of approximately 10^{-2} sr.

Limits and exposure expressed in radiance

For uniform large sources when the retinal thermal exposure limit is expressed as radiance, the acceptance angle can be as large as α and does not have an effect on the determined radiance. When the photochemical radiance limit is applied, the radiance exposure value is averaged over γ_{ph} (Schulmeister 2001).

PROTECTIVE MEASURES

The most effective means of controlling laser hazards to the eye and skin is total enclosure of the laser and all beam paths. For conditions where this is not possible, partial beam enclosure, laser eye protectors, restricted access to beam paths, and administrative controls may be necessary. Laser safety standards and guidelines have been developed worldwide that make use of a hazard classification scheme to permit specification of control measures based on the risk posed by the laser. In some laser operations, control measures are also necessary for electrical and fire hazards, x rays, noise, and airborne contaminants. These are generally encountered only with high power laser systems.

SPECIAL CONSIDERATIONS

These guidelines are considered to be adequate for the general population as well as for occupational exposure. No special assumptions such as adult ocular size, preexposure of skin, thickness of stratum corneum, or body size were made in deriving the limits. Only two exceptions need to be made to the foregoing. Some rare photosensitive or photosensitized individuals may react to UVR irradiances below the specified exposure limits, and such people should take more rigorous precautions to avoid exposure to UVR. The limits for ocular exposure from 300 nm to 400 nm do not adequately protect the retina of infants and aphakic individuals. These groups would require UVR absorbing lenses. Additionally special adjustments of the guidelines may be necessary for some ophthalmic instrument exposure (Sliney et al. 2005).

The exposure limits presented here should be used as guidelines for controlling human exposure to laser radiation. They should not be regarded as thresholds of injury or as sharp demarcations between "safe" and "dangerous" exposure levels. Exposure at levels below the exposure limits should not result in adverse health effects. The limits incorporate the collective knowledge generated worldwide by scientific research and experience of laser safety and are based upon the best available published information.

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APPENDIX

Using the exposure limit tables

Example. To find the exposure limit for He-Ne (632.8 nm) laser radiation for a 0.25 s exposure, use Table 5. First use the left-hand column to find the wavelength. Choose the third 400–700 nm entry since the 0.25 s exposure duration falls between 5 μs and 10 s (second column). The exposure limit expressed as radiant exposure, H_{EL}, and as irradiance, E_{EL}, for collimated beam (small source) intrabeam exposure ($C_E = 1$) is then:

$$H_{EL} = 18\, C_E t^{0.75}\ \text{J m}^{-2} (\text{Table 5, Row : Visible, 3}^{\text{rd}}\ \text{entry, column 3})$$
$$= 18\,(0.25)^{0.75}\ \text{J m}^{-2}$$

$$H_{EL} = 6.3\ \text{J m}^{-2} = 6.3\ \text{W s m}^{-2}$$

$$E_{EL} = 6.3\ \text{W s m}^{-2}/(0.25\ \text{s}) = 25\ \text{W m}^{-2}$$

The same exposure limit, expressed as power passing through a 7 mm aperture, equals 1 mW.

Rationale for updating the guidelines

Changes have been made to the recommended guideline exposure limits to provide more accurate hazard criteria for sub-microsecond exposure durations, extended-source ocular exposures to pulses, repetitive-pulse exposures, and certain infrared exposures of the eye (1,150–1,400 nm).

Since the publication of the Revision of the ICNIRP Guidelines for Laser Radiation (ICNIRP 2000) to limit exposures that may pose a retinal thermal hazard, further research has taken place with regard to the spatial, temporal and wavelength dependence of retinal thermal injury, particularly with respect to laser-tissue interaction mechanisms for pulse durations less than 100 μs. In these time domains, bulk thermal denaturation (photocoagulation) is no longer considered to be the damage mechanism for pulsed laser exposures. Localized heating of melanin granules dominates and thermo-acoustic effects determine the damage. The increased understanding of the spatial, spectral and temporal scaling factors made it possible to modify the ELs in this time domain with limited uncertainties. Each of these variables is discussed below.

Spot size dependence. Because of heat flow during the exposure, there is a dependence of the retinal injury threshold on retinal irradiance 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. 2008a). Two domains need to be distinguished in terms of the dependence of the exposure limits on α. For values of α smaller than a critical angle

α_{max}, the exposure limit, expressed as radiance or radiance dose, depends linearly on the inverse of α (Sliney and Wolbarsht 1980; Ham Jr. 1989). This $1/\alpha$ dependence reflects the fact that larger retinal irradiance patterns exhibit reduced radial cooling as compared to smaller ones. For values of α larger than $\alpha_{\max}$, the exposure limit expressed as radiance no longer depends on α. This is because the retinal irradiance pattern is large compared to the heat diffusion distance during the pulse so that the center of the retinal irradiance pattern 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. 2000) 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 $1/\alpha$ spot size dependence in the previous exposure limits was applied up to a critical angle of $\alpha_{\max}$ = 100 mrad.

Recent thermal models and ex-vivo studies (Schulmeister et al. 2008a) 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 $\alpha_{\max}$ to better reflect the retinal irradiance diameter dependence for pulsed sources (ICNIRP 2007). The value of $\alpha_{\max}$ = 100 mrad still applies for exposure to cw sources, i.e., for exposure durations larger than 0.25 s.

For short pulses and very large sources, the reduction of $\alpha_{\max}$ can result in a significantly increased retinal exposure, up to 20-fold increase for sources subtending greater than 100 mrad. For the case of Maxwellian viewing, positioning of the beam such that the beam waist is at the position of the cornea of the eye, or for diffused sources placed at the eye, sources that are safe for the retina may be capable of damaging the iris and/or the cornea. For these types of sources and applications, additionally to the retinal thermal limit, 2 times the skin exposure limit should also be applied to protect the iris.

Pulse duration dependence. The threshold study published by (Zuclich et al. 2000) for a range of spot sizes not only confirmed the expected lack of a spot size dependence for the retinal damage threshold for pulses in the microsecond and nanosecond temporal regime, but it also indicated that there was an insufficient reduction factor for minimal image sizes in this same time domain. The reported damage threshold for a spot size of 80 μm and a pulse duration of 5 ns was less than a factor of 2 above the exposure limit. *Ex-plant ex-vivo* RPE damage threshold studies (Gerstman et al. 1996; Kelly and Lin 1997; Lin et al. 1999; Brinkmann et al. 2000; Clark et al. 2013) led to the conclusion that the damage mechanism underlying these low thresholds was not thermal denaturation, but microcavitations (vapor bubbles) that formed around the melanosomes in the RPE cells. Schuele et al. (2005) and Lee et al. (2007) showed for the wavelength of 532 nm that the microcavity induced damage threshold becomes lower than the thermally induced damage threshold for pulse durations lower than about 10–50 μs, i.e., while the thermally induced damage thresholds remain at a constant level for pulse durations less than about 20 μs, (as also predicted by thermal models), the microcavity induced damage thresholds continue to decrease with shorter pulse durations (Schulmeister et al. 2011). It is therefore not appropriate to base the pulse duration below which the exposure limit assumes a constant dose value on the thermal confinement time of 18 μs. This corresponded to a homogeneous medium heated at minimal retinal spot size, in the case for the previous exposure limits. It was determined that lowering the break time for the visible wavelength range from 18 μs to 5 μs (corresponding to decreased confinement times in melanosomes), in combination with the updated spot size dependence, provided for a consistent reduction factor between the damage threshold and the new exposure limit. The reduction of the break time from 18 μs to 5 μs results in a lowering of the small source exposure limit by a factor of 2.5 when compared to the previous exposure limit in the nanosecond regime. For both the visible and the near infrared radiation retinal hazard, melanin absorption is the basis for the temperature increase. Therefore, the reduction by a factor of 2.5 was also applied to the exposure limit for the near infrared wavelength region.

A review of the damage threshold studies in the pulse duration regime between 100 fs and 1 ns (Gerstman et al. 1996; Kelly and Lin 1997; Lin et al. 1999; Roach et al. 1999; Brinkmann et al. 2000) showed that there is only a limited temporal dependence of the damage thresholds, that makes it possible to retain the new lower exposure limit down to a pulse duration of 10 ps. At 10 ps there is a step of a factor of 2 in the exposure limits for the visible wavelength range. It was believed that a step function, in contrast to the previously defined ramp with a $t^{0.75}$ time dependence, facilitated the application of the ELs since determination of the pulse duration is not necessary in the two temporal regimes above and below the step function, where the exposure limit is a constant radiant exposure value. Also, it was possible to set the EL for pulse durations less than 10 ps at a higher level than in the previous guidelines since the previous reduction factor was overly conservative. In the IR-A range, however, the step at 10 ps is larger than 2, since C_A is not applied to EL for pulse durations less than 10 ps (Table 5). That is, the EL for pulse durations less than 10 ps in the wavelength range between 400 nm and 1050 nm are at the same level. This reflects that the damage thresholds in the femtosecond time

regime, where non-linear optical effects play a role (Rockwell et al. 1997; Roach et al. 1999), exhibit a decreased wavelength dependence and damage thresholds at 1,064 nm are at lower levels than would be expected based on the wavelength of melanin absorption.

Multiple pulses. In the course of the review of the retinal thermal injury, it became clear that the treatment of repetitive exposures, i.e., multiple-pulse exposures, needed revision. As with single-pulse exposures, it was found to be important to distinguish the temporal regime of thermally induced retinal damage from microcavity induced damage, which dominates in the nanosecond pulse duration regime.

In the pulse duration regime where microcavitation dominates, i.e., for pulse durations shorter than ~50 μs, it is apparent that pulse additivity previously reflected in the correction factor C_p greatly over-estimated the actual risk of injury. Both the theoretical understanding of the microcavitation injury mechanism and analysis of the impact of probability summation upon the experimentally determined thresholds applicable to multiple exposures that were statistically independent of each other (Menendez et al. 1993) showed that much of the apparent additivity resulted from limitations of the experimental method for determining the minimal visible lesion threshold (Lund 2007). This was further supported by a follow-on study of large-spot-size repetitive-pulse exposures (Lund et al. 2009). For single-pulse damage thresholds with a steep slope of the probit curve, which is the case for threshold studies in recent years, the probability summation model predicts a very shallow reduction of the damage threshold with number of pulses (Brinkmann et al. 2000; Roegener et al. 2004; Lund 2007). It was argued recently (Sliney and Lund 2009) that this reduced additivity, in comparison to the thermal additivity, is covered by the existing reduction factor of the ELs and that for pulse durations less than T_i it was not necessary to reduce the single pulse EL with a multiple-pulse factor.

In the temporal regime greater than T_i, where thermal injury dominates, there is only limited experimental data for multiple-pulse retinal exposures in the non-human primate model (Zuclich and Blankenstein 1988). In the millisecond duration regime, thermally induced retinal injury for multiple pulses can be modeled well with computer models, which have been validated by experimental studies in retinal explants and where single-pulse thresholds agreed with in vivo single-pulse data (Schulmeister 2013). Computer model calculations show that the additivity of multiple pulse exposures of minimal retinal images does not require the additional correction-factor C_p reduction, but larger image-size exposure requires some reduction factor. The larger spot-size exposure additivity can be accounted for in appropriate manner with the $n^{-0.25}$ factor, which had been previously used for C_p in exposure guideline for small to intermediate retinal spot sizes and intermediate to long pulse durations. The factor $n^{-0.25}$, however, overestimates the pulse additivity for high repetition rates. An analysis of the data presented by Schulmeister (2007) was examined relative to the single-pulse threshold as well as in terms of average power to show that the additivity reduction factor, C_p, does not decrease below 0.2. As a result, the methodology for computing multiple pulse exposure limits was revised (Repetitive-pulse exposure), and most importantly it was found that there was no need for $C_p < 1.0$ for the intrabeam "point-source" ELs.

Wavelength dependence for retinal thermal exposure limit between 1,150 and 1,400 nm. A number of damage-threshold studies with wavelengths between 1,150 nm and 1,400 nm provided for an update of the wavelength dependence of the retinal thermal exposure limits. The precedent exposure limits in the wavelength range 1,150–1,400 nm, for simplicity were held constant, $C_C = 8$, although that was associated with a reduction factor larger than necessary. Recently, a number of damage threshold studies were conducted in the wavelength range 1,150–1,400 nm for different pulse durations (Zuclich et al. 2007; Vincelette et al. 2009). These threshold data are consistent with the decreasing transmission of the pre-retinal ocular media towards longer wavelengths in the range 1,150–1,400 nm. Therefore in the present guideline, a term for exponential increase with increasing wavelength was added to the factor C_C in the wavelength range 1,200–1,400 nm (Table 3), thus increasing the exposure limit towards longer wavelengths.

Dual limit to protect anterior parts of the eye. In the current guidelines, there are two exposure conditions where the ocular exposure limits protecting against retinal thermal injury were increased compared to previous guidelines: 1. Pulsed exposure to radiation from extended

Table A1. Abbreviations used.

Abbreviation	Unabbreviated
CIE	Commission Internationale d'Eclairage
EL	Exposure limit
ICNIRP	International Commission for Non-Ionizing Radiation Protection
IR	Infrared
IRPA	International Radiation Protection Association
IRR	Infrared radiation
UNEP	United Nations Environment Programme
UV	Ultraviolet
UVR	Ultraviolet radiation
WHO	World Health Organization

sources (time dependent α_{max}); 2. Exposure in the wavelength range of 1,150 nm to 1,400 nm (C_c). Consequently, for exposure under special conditions, the exposure limit for the eye expressed as corneal radiant exposure can exceed the level that is necessary to protect the anterior parts of the eye (cornea, iris, lens). For the case of visible wavelengths, where the iris is the tissue at risk, this special condition can only occur for highly divergent beams and at very small exposure distances.

For simplicity, the exposure limit of the skin is used as a dual limit in the visible wavelength range to protect the anterior parts of the eye, while it is justified to use two times the skin exposure limit in the infrared wavelength range. Since in a safety analysis for general exposure scenarios, both the skin exposure and the eye exposure has to be evaluated and has to be below the respective exposure limits, this additional limit is in practice not an additional restriction. Therefore, this is relevant only for a situation where only the eye is exposed.

Table A1 shows abbreviations used.

■■