Draft for external consultation – February 2011

GUIDELINES ON LIMITS OF EXPOSURE TO LASER RADIATION OF WAVELENGTHS BETWEEN 180 nm AND 1000 µm

INTRODUCTION

Since the publication of the ICNIRP Revision of the Guidelines on Limits of Exposure to Laser Radiation (ICNIRP 1996, ICNIRP 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 1200 and 1400 nm. A detailed discussion of the rational for the changes is presented in the Appendix of these Guidelines (Rationale for updating the Guidelines).

The present guideline is a revision of the previous ICNIRP guidelines (ICNIRP 1996) and (ICNIRP 2000).

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 basic principles of protection against optical radiation emitted by lasers. Separate guidelines are defined for exposure to non-laser optical radiation (ICNIRP 1997).

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 to 10^3 µm (1 mm) and to exposure duration between 100 fs and 30 ks (about 8 hours). 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.

The guidelines apply to exposures to optical 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; Wolbarsht 1980, UNEP; WHO; IRPA; WHO 1982, McCluney 1984, CIE; ICNIRP 1998, Schulmeister 2001, Henderson; Schulmeister 2004)

QUANTITIES AND UNITS

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

Table 1 Quantities symbols and units

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

Irradiance, *E* ($W \cdot m^{-2}$), and *radiant exposure, H* ($J \cdot m^{-2}$) are used in describing the concepts of surface exposure dose rate and surface exposure dose from direct exposure to laser radiation. *Radiance*, *L* ($W \cdot m^{-2} \cdot sr^{-1}$) 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* ($J \cdot m^{-2} \cdot sr^{-1}$). Other radiometric quantities such as *fluence rate* and *fluence*, although similarly expressed in $W \cdot m^{-2}$ and $J \cdot m^{-2}$, respectively, should not be used. The fundamental definitions are different as fluence includes the radiation scattered through the unit area (CIE 1987). For a more detailed discussion see (Sliney; Wolbarsht 1980, Schulmeister 2001, Henderson; 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, however, for example in research laboratories, medical treatment, laser entertainment displays, and alignment procedures, exposure to potentially hazardous laser radiation is possible. 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, distance measurement devices (range finders), supermarket scanners, optical communications, facsimile and printing equipment, computer game controllers and guidance devices for the blind.

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 (400–1400 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; WHO; IRPA; WHO 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 – 1400 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 1400 nm. For exposure duration less than about 10 µs, superheating of melanin granules causing microcavitation dominates the injury mechanism (Kelly; Lin 1997, Lin; Kelly; Sibayan; Latina; Anderson 1999, Brinkmann; Huttmann; Rogener; Roider; Birngruber; Lin 2000, Roegener; Brinkmann; Lin 2004, Schuele; Rumohr; Huettmann; Brinkmann 2005, Lee; Alt; Pitsillides; Lin 2007). Optical breakdown and plasma formation occur from sub-nanosecond exposures (Cain; Toth; Noojin; Stolarski; Thomas; Rockwell 2002, Roach; Cain; Narayan; Noojin; Boppart; Birngruber; Fujimoto; Toth 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; Stuck; Edsall 2006).

Effects of ultraviolet radiation

Short-wavelength ultraviolet radiation (UVR) radiation is absorbed within the cornea and conjunctiva, whereas long wavelength UVR is absorbed largely in the lens (UNEP; WHO; IRPA; WHO 1982). Exposure to short wavelength UVR, laser radiation may produce acute photochemical effects; erythema (reddening of the skin), photokeratitis (corneal inflammation), and conjunctivitis (conjunctival inflammation) and cataract (clouding of the lens). Typically, 1000-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; WHO; IRPA; WHO 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; Sliney; Mellerio; Lund; Stuck; Zuclich 2008). The peak sensitivity of erythemal 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 cataract formation is

primarily due to excessive exposure to UVR in the 280-315 nm wavelength range (Merriam; Löfgren; Michael; Söderberg; Dillon; Zheng; Ayala 2000). In the aphakic eye, UVR wavelengths greater than 300 nm reach the retina and can cause photochemical injury (Ham; Mueller; Ruffolo; Guerry; Guerry 1982).

Effects of visible and near infrared radiation

The primary effect on the eye of visible and near infrared radiation (400–1400 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; Berry 1968, Vassiliadis 1971, Birngruber 1978, Gabel VP 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 550–700 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; Mueller; Sliney 1976, Lund; Stuck; Edsall 2006). This is usually referred to as blue-light hazard (Sliney; 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; Motzerenkov; Skatskaia; Chemny; Pivovarov 1978).

Animal studies demonstrated that continued exposures over several days to very bright light led to retinal injury (Noell; Walker; Kang; Berman 1966, Mellerio 1994, Rozanowska; 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.

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

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

Data are available that show different thresholds for different biological effects. At threshold levels, different mechanisms leading to damage dominate depending on the exposure duration. For pulsed 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 RPE, at levels lower than thermally induced damage of the RPE (Schuele; Rumohr; Huettmann; Brinkmann 2005, Lee; Alt; Pitsillides; Lin 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 are also determined by 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 400–1400 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; Edsall; Stuck; Schulmeister 2007, Schulmeister; Husinsky; Seiser; Edthofer; Fekete; Farmer; Lund 2008). 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 and therefore, eye movements become an important factor, as well as retinal spot size (Sliney 1988, Sliney 1989).

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; Farrell; Bargeron 1992, McCally; Bargeron 2001, McCally; Bargeron 2003, McCally; Bonney-Ray; Bargeron 2004, McCally; Bonney-Ray; de la Cruz; Green 2007). However, damage to the cornea is likely to be of greater concern because of the adverse impact on vision.

If exposures approached 1000 $W \cdot m^{-2}$ 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; Löfgren; Marcelo; Söderberg 2007). Further, some additivity was observed for corneal and retinal effects within 1 – 4 days (Zuclich 1980, Griess; Blankenstein 1981, 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 required for thermal diffusion to take place, depends upon the volume of tissue heated. For melanin granules, the thermal confinement period is approximately 0.5-1 µs (Neumann; 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 (Zuclich1988). Retinal and cutaneous thermal models employing the Arrhenius integral for first-order rate processes (Lukashev; Denker; Pashinin; Sverchkov 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; Hillenkampf; Flotte; Birngruber 1993, Brinkmann; Huttmann; Rogener; Roider; Birngruber; Lin 2000, Roegener; Brinkmann; Lin 2004). In vivo studies of retinal injury for minimal retinal irradiance diameters report substantial additivity (Stuck; Lund; Beatrice 1978, Lund; Stuck; Beatrice 1981). Lund (Lund; Lund; Edsall 2009) and Griess (Griess; 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 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 the $EL(\lambda)$ can be expressed as the EL for the wavelength where the EL is lowest, EL_{Min}, multiplied with a spectrally correction factor (Eq. 1).

$$EL(\lambda) = EL_{\min} \; Spectral\ correction\ \ factor \qquad Eq.\ 1$$

Separate ('dual') exposure limits are specified related to thermal and photochemical retinal injury with different wavelength, pulse duration and spot size dependencies. 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 with a spectral correction factor for photochemical injury, $C_B(\lambda)$ (Fig. 1) (Eq. 2).

$$EL_B = EL_{B:Min} \cdot C_B(\lambda) \qquad Eq.\ 2$$

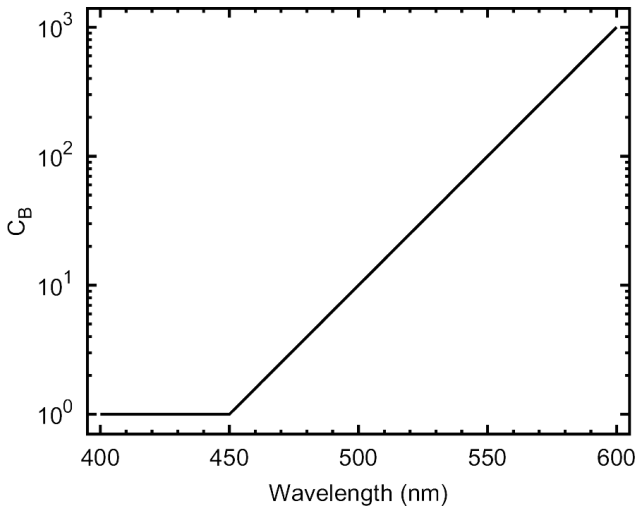

Fig. 1 Exposure limit correction factor, C_B, reflecting the wavelength dependence of photochemically induced retinal injury applicable to exposures of durations greater than 10 s in the visible wavelength range.

For thermal retinal injury in the wavelength range between 700 and 1400 nm, the exposure limit, EL_{Th}, is expressed as the minimum exposure limit $EL_{Th:Min}$ in that wavelength range (which is the EL for the visible wavelength range), multiplied with a combined correction factor, $C_A(\lambda) \cdot C_C(\lambda)$ (Eq. 3).

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

$C_A(\lambda)$ is related to retinal pigment epithelium absorption and defined for $700 < \lambda < 1400$ nm (Fig. 2) and $C_C(\lambda)$, is related to pre-retinal absorption and defined for $1050 < \lambda < 1400$ nm (Fig. 2).

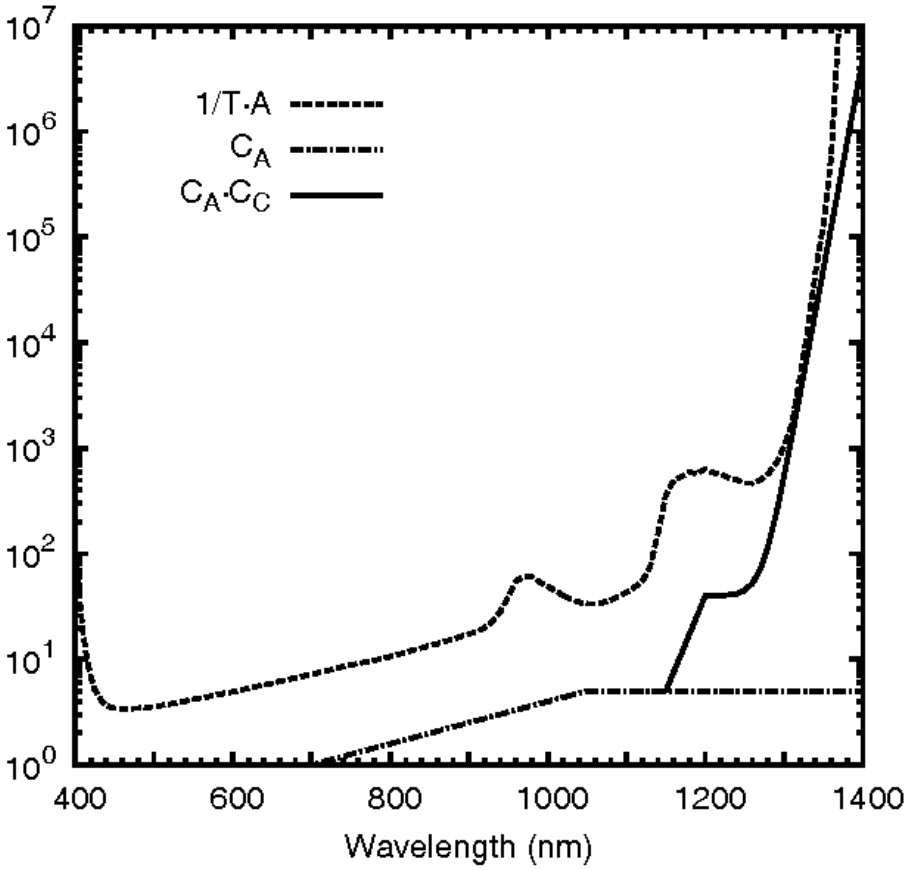

Fig. 2 –The dashed line is the inverse of the product of the absorption in the RPE and the transmittance of the pre-retinal media, representative of the energy absorbed in the RPE relative to the exposure of the cornea. The spectral correction factor C_A (dash-dot) approximates the reciprocal of the absorbance, A, of the RPE., The factor C_C approximates the reciprocal of the spectral transmittance of the pre-retinal ocular media, T (Lund; Edsall; Stuck 2008).

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 nm to 1400 nm produces a minimum spot size on the retina. 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 and exposure to radiation from extended sources is accounted for by a correction factor C_E which depends on the angular

subtense of the apparent source, α (see Eq. 4 which applies to the wavelength range above 1050 nm).

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

RETINAL IMAGE SIZE

For wavelengths between 400 nm and 1400 nm, the "retinal hazard region," the ocular exposure limit for retinal thermal damage depends upon the angle subtended by the apparent source.

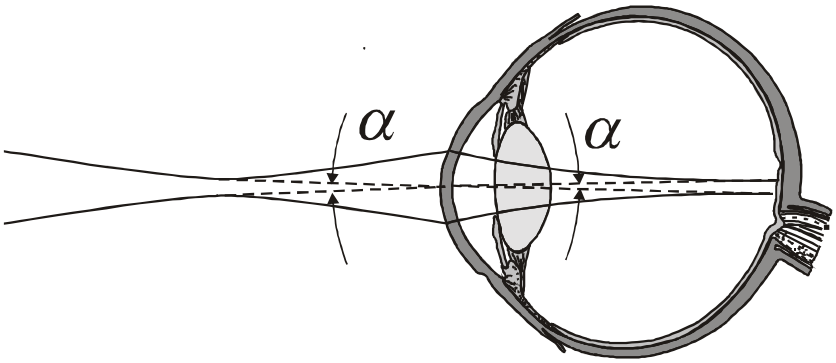

Fig. 3 The parameter α, for a given position of the eye in the beam is the angle subtended by the apparent source that produces the minimal retinal beam profile that can be achieved by accommodation of the eye. The figure is simplified assuming an air-filled eye. Adapted from Henderson et al (Henderson; Schulmeister 2004).

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 that is incident on the eye is the location of the apparent source, and the beam diameter at that position can be considered as the source diameter and 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. It might therefore 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).

Therefore, 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 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 extended sources* and *large extended 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; Wolbarsht 1980).

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 ($W \cdot m^{-2}$) and radiant exposure ($J \cdot m^{-2}$) are used for point-source exposure limits. The exposure limits 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* or *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, radial heat flow does not affect the damage threshold when it is given as retinal radiant exposure. This critical image diameter is the biophysical basis of the parameter α_{max} (Schulmeister; Husinsky; Seiser; Edthofer; Fekete; Farmer; Lund 2008, Schulmeister; Stuck; Lund; Sliney 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).

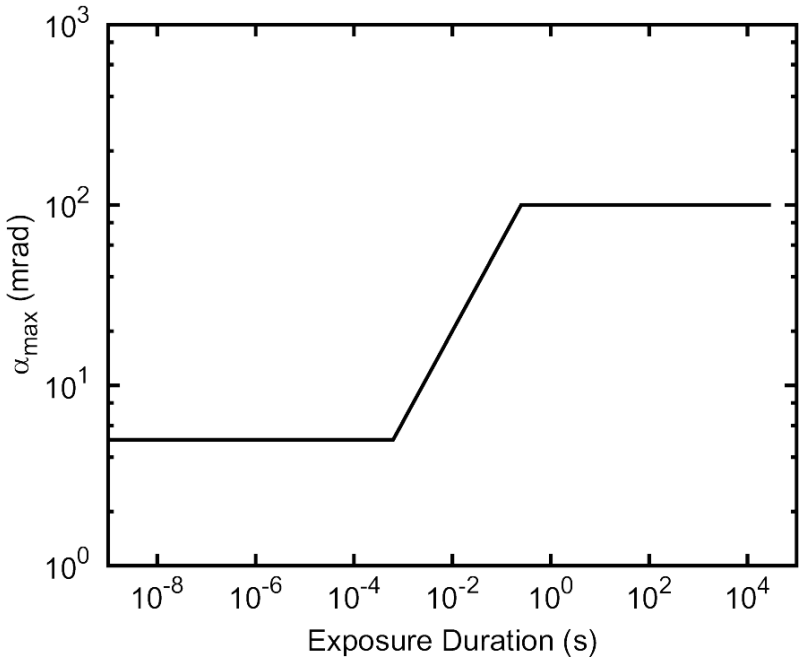

Fig. 4 Exposure duration dependence of the critical angular subtense for intermediate sources, α_{max}.

The quantities irradiance ($W \cdot m^{-2}$) and radiant exposure ($J \cdot m^{-2}$) are used for intermediate sources exposure limits. for intermediate sources, but 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 is introduced to account for the variation of retinal injury threshold with spot size (Table 2).

Table 2 Correction factor to account for the effect of source size

For sources subtending an angle α (mrad)				
C_E	=	1.0	for	$\alpha < \alpha_{min}$
		α / α_{min}	for	$\alpha_{min} \leq \alpha \leq \alpha_{max}$
		$\alpha_{max} / \alpha_{min}$	for	$\alpha > \alpha_{max}$ (for $\gamma = \alpha_{max}$)*
where, for exposure duration t (s)				
α_{max}	=	5 mrad	for	$t < 625$ µs
		200 $t^{0.5}$ mrad	for	625 µs $\leq t \leq$ 0.25 s
		100 mrad	for	$t >$ 0.25 s
And				
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	$\alpha > \alpha_{min}$

*Note: Exposure limits can be expressed in terms of radiance for $\alpha > \alpha_{max}$

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 independent of spot size.

Exposure limits for large sources at angles greater than α_{max} can be described with different units, i.e. as radiance ($W \cdot m^{-2} \cdot sr^{-1}$) and time-integrated radiance (radiance dose) with units of $J \cdot m^{-2} \cdot sr^{-1}$. Thermal model calculations (Freund; Sliney 1999) and experimental data (Lund; Edsall; Stuck; Schulmeister 2007, Schulmeister; Husinsky; Seiser; Edthofer; Fekete; Farmer; Lund 2008) were used to justify the dependence of retinal injury thresholds and ELs for larger image sizes where α exceeds α_{max}.

RATIONAL FOR THE EXPOSURE LIMITS

The assumptions 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. It is therefore preferable to recommend different exposure limits for lasers and non-laser sources such as the sun, tungsten filaments, xenon lamps or LEDs.

Laser radiation is produced by 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.

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; Mellerio; Gabel; Schulmeister 2002). These data are supported with 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 all 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, ICNIRP assigned reduction factors (a fraction of the known dose to produce an adverse effects for a given exposure condition) 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, Dong; Löfgren; Marcelo; Söderberg 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 ophthalmic 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 (Finney 1971).

Light and electron microscopy examination of tissue revealed evidence of injury at exposures below the ED-50 derived by ophthalmic examination. However, microscopic effects usually were found to appear at doses not lower than 50 % of the ED-50. Therefore, most threshold data are based on ophthalmic examination. The fact that microscopic damage may occur below ED-50, derived by ophthalmic examination, was compensated when setting the exposure limit by adding a sufficient reduction factor.

Spectral considerations, ultraviolet radiation

The exposure limits for UVR emitting lasers are very similar to those for

non-laser UVR, and are based on the same biological data (Schulmeister; Sliney; Mellerio; Lund; Stuck; Zuclich 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 far-UVR range, the reduction factor relative to the thresholds for photokeratitis is up to 100 (Sliney 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 1050 nm increase with wavelength by a factor C_A (Fig. 2). C_A increases from 1 to 5 as the wavelength increases from 700 to 1050 nm as shown in Fig. 2.

Between 1050 and 1400 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; WHO; IRPA; WHO 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 1100 nm (Zuclich; Lund; Stuck 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 550 nm, and longer, 550–700 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 far infrared

Exposure limits for the middle and far-infrared region 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; Berezin; Gudakovskii; Muratov; Murzin; Fromzel 1978, Wolbarsht 1978, Stuck; Lund; Beatrice 1981, McCally; Farrell; Bargeron 1992, McCally; Bargeron 2001, McCally; Bargeron 2003, McCally; Bonney-Ray; Bargeron 2004, McCally; Bonney-Ray; de la Cruz; Green 2007). This variation is only 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; Lund; Beatrice 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 1400 to 3000 nm are based on biological threshold data that vary markedly with wavelength for pulsed, but not continuous wave, lasers (Lund; Stuck; Beatrice 1981, Stuck; Lund; Beatrice 1981, Schulmeister; Jean 2011b).

Although 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, this would have required substantially more calculations on the part of 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 guideline in 2000 (ICNIRP 2000), additional biological effects research has described corneal, lens and retinal thresholds for wavelengths near 1.3 µm (Zuclich; Lund; Stuck 2007, Vincelette; Rockwell; Oliver; Kumru; Thomas; Schuster; Noojin; Shingledecker; Stolarski; Welch 2009). In this spectral region, threshold injury to the eye transitions from the cornea, 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 to 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.

Multiple wavelengths

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

For the case that for different wavelengths, 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 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; Sliney 1974, Lyon 1985). Because of the non-linearity of thermally induced injury, if thermal mechanisms are involved, this assumption should be conservative (Schulmeister; 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; Noojin; Hammer; Thomas; Rockwell 1997, Toth; Narayan; Cain; Noojin; Winter; Rockwell; Roach 1997, Roach; Johnson; Rockwell 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; Thompson; Jacques; Rogers 1996, Cain; Noojin; Hammer; Thomas; Rockwell 1997, Hammer; Jansen; Frenz; Noojin; Thomas; Noack; Vogel; Rockwell; Welch 1997, Rockwell; Hammer; Hopkins; Payne; Toth; Roach; Druessel; Kennedy; Amnotte; Eilert; Phillips; Noojin; Stolarski; Cain 1997, Cain; Toth; Noojin; Carothers; Stolarski; Rockwell 1999, Cain; Toth; Noojin; Stolarski; Cora; Rockwell 2001, Cain; Thomas; Noojin; Stolarski; Kennedy; Buffington; Rockwell 2005). 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. (see Rationale for updating the Guidelines).

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; Toth; Noojin; Stolarski; Thomas; Rockwell 2002). 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; White; Tips; Wilson 1970, Schulmeister; Stuck; Lund; Sliney 2007).

For longer duration repetitive exposures (e.g. greater than a second), behavioral factors (tissue movement, aversion) reduce the exposure at a given site. Repeated or intermittent exposures are largely of concern for UVR radiation 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 1 ms. Several different formulations have been applied in the past. However, in recent reviews of the large biological data base for repetitive pulses (Lund 2007, Sliney; Lund 2009), 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.

Delayed Effects

Chronic exposure to laser radiation is usually rare. The accumulated experience of lasers in use has not shown any evidence for delayed effects. There is not enough scientific data available to derive guidelines for chronic exposure.

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 studies and retinal thermal injury studies (Ness; H.; Stuck; Lund; Lund; Molchany; Sliney 2000, Lund 2006) and models (Ness; H.; Stuck; Lund; Lund; Molchany; Sliney 2000, 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).

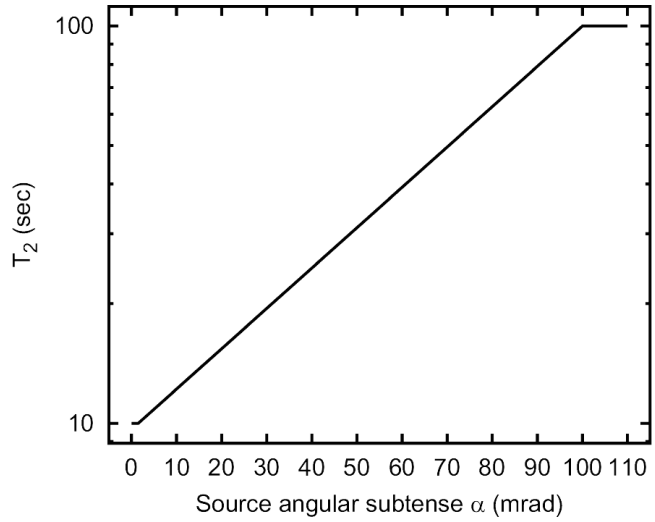

Fig. 5 The time T_2 indicates the transition between the exposure duration dependent exposure limit for extended sources and constant irradiance for exposure durations greater than T_2.

Because the thermal injury threshold expressed as radiant power entering the eye decreases as the exposure duration, *t*, raised to the -0.25-power, i.e., a reduction of only 44 % per tenfold increase in duration, only moderate increases in the exposed retinal area will compensate for the increased risk for longer viewing times. The ever increasing retinal area of irradiation resulting from greater eye movements (Velichowsky; Pomplun; Rieser 1996, Klein; Klein; Moss 2000) with increased viewing time takes longer to compensate for the reduced impact of thermal diffusion in larger extended sources. 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 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; 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), clearly showing the influence of eye movements during fixation; 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 that 100 s. For this reason, the limiting angle off 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 actually different, as the angle γ_{ph} is a linear plane for averaging radiance, and when the exposure limit is expressed as irradiance this acceptance angle 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 α < 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 photochemical injury, eye movements are of the angular extent of 11 mrad incorporated into the visible laser limits 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

Radial heat flow away from an absorbing area and strong scattering in the stratum corneum and epidermis also influence skin injury thresholds, and this, along with a smaller reduction factor for very small spots, is taken into account with the aperture size 3.5 mm used for measurement of exposures. Concerns about heat stress impose restrictions on exposure of large skin surfaces. At wavelengths greater than 1.4 µm, 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}.

Exposure limits for the skin also increase by the spectral correction factor, C_A, for wavelengths between 700 and 1400 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. Likewise, the varying penetration depths in the mid-infrared region allow for the same variations in limits for pulsed lasers as apply for the eye.

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 in the following.

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 5 was chosen for skin exposure.

EXPOSURE LIMITS

Correction factors

Some of the exposure limits are specified with correction factors (see 0).

Spectral dependence

The correction factor, C_B (Fig. 1, Table 3) is related to wavelength dependence of photochemically induced injury to the retina.

Table 3. Factors used in exposure limits and for determining exposure levels

C_A	=	1.0	for	400 nm ≤ λ < 700 nm
		$10^{0.02(\lambda-700)}$	for	700 nm ≤ λ < 1050 nm
		5.0	for	1050 nm ≤ λ < 1400 nm
C_B	=	1.0	for	400 nm ≤ λ < 450 nm

		$10^{0.02(\lambda-450)}$	for	450 nm ≤ λ < 600 nm
C_C	=	1.0	for	700 nm ≤ λ < 1150 nm
		$10^{0.018(\lambda-1150)}$	for	1150 nm ≤ λ < 1200 nm
		$8 + 10^{0.04(\lambda-1250)}$	for	1200 nm ≤ λ < 1400 nm
T_1	=	$10 \times 10^{0.02(\lambda-550)}$ s	for	550 nm ≤ λ < 700 nm
T_2	=	10 s	for	α < 1.5 mrad
		$10 \times 10^{(\alpha-1.5)/98.5}$ s	for	1.5 mrad ≤ α ≤ 100 mrad
		100 s	for	α > 100 mrad
T_i	=	5 µs	for	400 nm to 1050 nm
		13 µs	for	1051 n m to 1400 nm
γ_{ph}	=	11 mrad	for	t ≤ 100 s
		$1.1\ t^{0.5}$ mrad	for	100 s < t < 10 ks
		110 mrad	for	t ≥ 10 ks

The correction factor C_A (Fig. 2, Table 3), defined for 700 < λ ≤ 1400 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_C (Fig. 2, Table 3), defined for 700 < λ ≤ 1400 nm, is based on the wavelength dependence of the absorption 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 1400 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 1.3 for values.

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

The factor T_2 (Fig. 5, Table 3) 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 4, Table 5, and Table 6. Special rules apply for repetitive laser exposure (see 1.3 below). Clarifications to symbols in the tables are given in Table 3.

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 1400 nm, the exposure limits and the exposure can be expressed either as irradiance (or radiant exposure) where the exposure is averaged over a 7 mm aperture, or as power (or energy) where the exposure is determined as power passing through a 7 mm limiting aperture (Schulmeister 2010). For homogeneous extended sources, the exposure limits can also be expressed as radiance (or radiance dose).

Table 4 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 7 mm aperture

Wavelength λ (nm)	Exposure duration (t)	Exposure limit (W m^{-2} or J m^{-2})	Exposure limit (W or J)	Restrictions
Ultraviolet				Aperture sizes: 1 mm for t<0.35 s 1.5 $^{0.375}$ mm for 0.35 s<t<10 s 3.5 mm for t>10 s
180.–302	1. ns – 30 ks	30. J·m^{-2}		
303.	1. ns – 30 ks	40. J·m^{-2}		
304.	1. ns – 30 ks	60. J·m^{-2}		
305.	1. ns – 30 ks	100. J·m^{-2}		
306.	1. ns – 30 ks	160. J·m^{-2}		
307.	1. ns – 30 ks	250. J·m^{-2}		
308.	1. ns – 30 ks	400. J·m^{-2}		
309.	1. ns – 30 ks	630. J·m^{-2}		
310.	1. ns – 30 ks	1.0 kJ·m^{-2}		
311.	1. ns – 30 ks	1.6 kJ·m^{-2}		
312.	1. ns – 30 ks	2.5 kJ·m^{-2}		
313.	1. ns – 30 ks	4.0 kJ·m^{-2}		
314.	1. ns – 30 ks	6.3 kJ·m^{-2}		
315. – 400	1. ns – 10 s	5.6 $t^{0.25}$ kJ m^{-2}		
315. – 400	10. s – 30 ks	10. kJ·m^{-2}		
Also not to exceed				
180. – 315	1. ns – 10 s	5.6 $t^{0.25}$ kJ m^{-2}		
Visible				All for 7 mm limiting aperture
400. – 700	100. fs - 10 ps	1. C_E mJ·m^{-2}	$3.8 \cdot 10^{-8}$ C_E J	
400. – 700	10. ps – 5 µs	2. C_E mJ·m^{-2}	$7.7 \cdot 10^{-8}$ C_E J	
400. – 700	5. µs – 10 s	18. $C_E \cdot t^{0.75}$ J·m^{-2}	$7. \cdot 10^{-4}$ $C_E \cdot t^{0.75}$ J	
Also not to exceed 2 times the skin				For special conditions where only the eye is exposed.

exposure limit				
Dual limits for 400-600 nm visible laser exposure at t>10 s				
Photochemical[a]				
400. – 600	10. s - 100 s	100. C_B J·m^{-2}	3.9·10^{-3} C_B J	For $\alpha > 11$ mrad use $\gamma = 11$ mrad
400. – 600	100. s- 30 ks	1 C_B W·m^{-2}	39 C_B µW	For $\alpha \leq \gamma_{ph}$, γ not restricted
Thermal[a]				
400. – 700	10. s – 30 ks	10. W·m^{-2}	0.39 mW	For $\alpha \leq 1.5$ mrad
400. – 700	10. s - T_2 s	18. C_E·t$^{0.75}$ J·m^{-2}	7. 10^{-4} C_E·t$^{0.75}$ J	For $\alpha > 1.5$ mrad
400. – 700	T_2 s - 30 ks	18. C_E·$T_2^{-0.25}$ W·m^{-2}	7. 10^{-4} C_E·$T_2^{-0.25}$ W	For $\alpha > 1.5$ mrad
Near IR				
700. - 1 050	100. fs - 10 ps	1. C_E mJ·m^{-2}	3.8·10^{-8} C_E J	For 7 mm aperture
700. - 1 050	10. ps – 5 µs	2. C_A·C_E mJ·m^{-2}	7.7·10^{-8} C_A·C_E J	
700. - 1 050	5. µs – 10 s	18. C_A·C_E·t$^{0.75}$ J·m^{-2}	7.·10^{-4} C_A·C_E·t$^{0.75}$ J	
1 051. - 1 400	100. fs – 10 ps	1. C_C·C_E mJ·m^{-2}	3.8·10^{-8} C_C·C_E J	
1 051. - 1 400	10. ps -13 µs	20. C_C·C_E mJ·m^{-2}	3.8·10^{-7} C_C·C_E J	
1 051. - 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. - 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. - 1 400	10. s – T_2 s	18 C_A·C_C·C_E·t$^{0.75}$ J·m^{-2}	7.·10^{-4} C_A·C_C·C_E·t$^{0.75}$ J	For $\alpha > 1.5$ mrad
700. - 1 400	T_2 s -30 ks	18 C_A·C_C·C_E·$T_2^{-0.25}$ W·m^{-2}	7.·10^{-4} C_A·C_C·C_E·$T_2^{-0.25}$ W	For $\alpha > 1.5$ mrad
Also not to exceed 2 times the skin exposure limit				For special conditions where only the eye is exposed. Specifically relevant to exposures of 1.15 – 1.4 µm.
Mid and far IR				Aperture sizes: 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 > 10 s
1 400. - 1 500	1. ns – 1 ms	1. $kJ \cdot m^{-2}$
1 400. - 1 500	1. ms - 10 s	$5.6\ t^{0.25}$ $kJ \cdot m^{-2}$
1 500. - 1 800	1. ns – 10 s	10. $kJ \cdot m^{-2}$
1 801. - 2 600	1. ns – 1 ms	1. $kJ \cdot m^{-2}$
1 801. - 2 600	1. ms -10 s	$5.6\ t^{0.25}$ $kJ \cdot m^{-2}$
2 601 nm - 1 mm	1. ns - 100 ns	100. $J \cdot m^{-2}$
2 601 nm - 1 mm	100. ns – 10 s	$5.6\ t^{0.25}$ $kJ \cdot m^{-2}$
1 400 nm - 1 mm	10. s – 30 ks	1. $kW \cdot m^{-2}$

t in seconds, 1 ks = 1000 s

[a] 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 10s, reduce to the thermal limits for times less than T_1 and to photochemical limits for longer times (Table 2, Table 3,).

The time T_1 (Table 3, Fig. 6) applies for small sources, $\alpha \leq \alpha_{min}$ ($C_E = 1$), and is the critical exposure time below which the retinal thermal EL is lower than the photochemical EL.

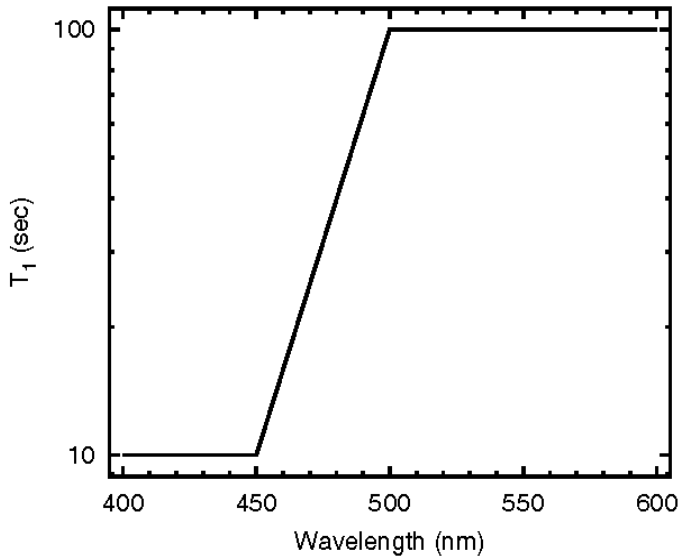

Fig. 6 Spectral dependence of the critical exposure time, T_1, below which the retinal thermal EL is lower than the photochemical EL, for the case of small sources (α <1.5 mrad) (obtained by equating the two exposure limits for the case of small sources).

Table 5 Laser exposure limits for the eye for λ = 400–1400 nm expressed as radiance or radiance dose

Wavelength, λ (nm)	Exposure duration, t	m^{-2}	Restrictions
Visible			
for $t \leq 10$ s and $\alpha \geq \alpha_{max}$			
400. – 700	100. fs – 10 ps	0.17 $kJ \cdot m^{-2} \cdot sr^{-1}$	All only for large sources with costant radiance
400. – 700	10. ps - 5 µs	0.34 $kJ \cdot m^{-2} \cdot sr^{-1}$	
400. – 700	5. µs – 0.625 ms	3.1. $t^{0.75}$ $MJ \cdot m^{-2} \cdot sr^{-1}$	
400. – 700	0.625 ms – 0.25 s	76. $t^{0.25}$ $kJ \cdot m^{-2} \cdot sr^{-1}$	
400. – 700	0.25 s – 10 s	0.15 $t^{0.75}$ $MJ \cdot m^{-2} \cdot sr^{-1}$	
For $t > 10$ s; dual limits			
Photochemical			Photochemical radiance EL valid for all α, but averaging of exposure level over γ_{ph}
400. – 600	10. s – 10 ks	1. C_B $MJ \cdot m^{-2} \cdot sr^{-1}$	
400. – 600	10. ks – 30 ks	100. C_B $W \cdot m^{-2} \cdot sr^{-1}$	
Thermal for $\alpha \geq$ 100 mrad			
400. – 700	10. s - 100 s	0.15 $t^{0.75}$ $MJ \cdot m^{-2} \cdot sr^{-1}$	
400. – 700	100 s - 30 ks	47. $kW \cdot m^{-2} \cdot sr^{-1}$	
IR-A			
700. – 1 400	100. fs – 10 ps	0.17 $kJ \cdot m^{-2} \cdot sr^{-1}$	
700. – 1 400	10. ps - 5 µs	0.34 C_A C_C $kJ \cdot m^{-2} \cdot sr^{-1}$	
700. – 1 400	5. µs – 0.625 ms	3.1. $t^{0.75}$ C_A C_C $MJ \cdot m^{-2} \cdot sr^{-1}$	

700. – 1 400	0.625 ms – 0.25 s	76. $t^{0.25}$ C_A C_C kJ·m^{-2}·sr^{-1}
700. – 1 400	0.25 s – 10 s	0.15$t^{0.75}$ C_A C_C MJ·m^{-2}·sr^{-1}
700. - 1 400	10. s – 30 ks	10. C_A·C_C W·m^{-2}
700. - 1 400	10. s – 100 s	0.15$t^{0.75}$ C_A·C_C MJ·m^{-2}·sr^{-1}
700. - 1 400	100 s -30 ks	47. C_A·C_C kW·m^{-2}·sr^{-1}

t in seconds, 1 ks = 1000 s

Table 6 Laser radiation exposure limits for the skin

Wavelength, λ (nm)	Exposure duration, t (s)	Exposure limit, EL m^{-2}	Restrictions
Ultraviolet			
180. – 400	1. ns – 30 ks	Same as EL for eye	3.5 mm limiting aperture
Visible and IR-A			3.5 mm limiting aperture
400. – 1 400	1. ns – 100 ns	200. C_A J·m^{-2}	
400. – 1 400	100. ns – 10 s	11. C_A $t^{0.25}$ kJ·m^{-2}	
400. – 1 400	10. s – 30 ks	2. C_A kW·m^{-2}	
Far infrared*			
1 400 nm – 1 mm	1 ns – 30 ks	Same as EL for eye	3.5 mm limiting aperture

t in seconds, 1 ks = 1000 s

* For wavelengths above 2600 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 and 0.1 m^2, the exposure limit is inversely proportional to the exposed area..

1.1 Exposure limits presented as radiance

In Table 5, the EL for thermally and photochemically induced retinal injury are expressed as irradiance or radiant exposure (Fig. 7),

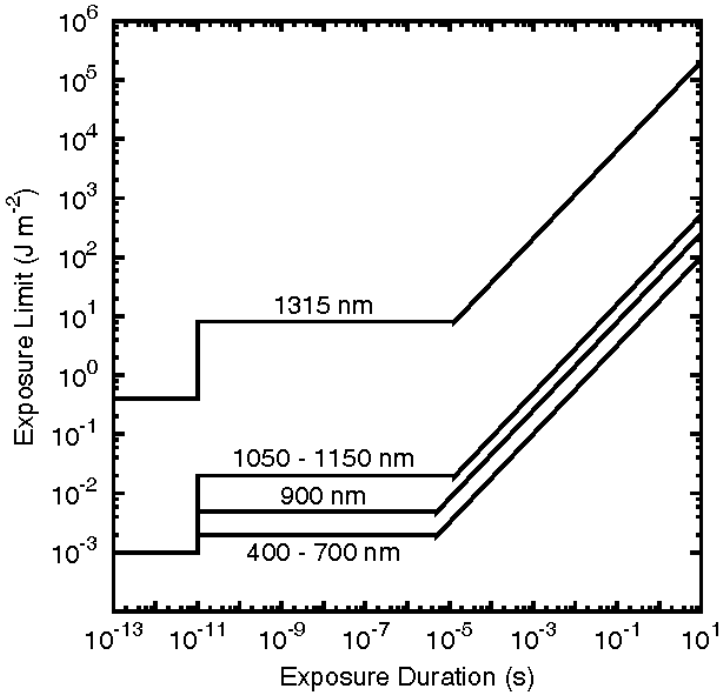

Fig. 7 Exposure limits for point-source viewing of pulsed laser radiation for selected wavelengths in the range of 400 nm to 1400 nm.

limiting the exposure at the corneal level by an averaging aperture of 7 mm diameter. Since laser beams usually represent small sources producing a minimal retinal spot size, point sources, this representation simplifies measurements and analysis. 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 5 lists these alternative radiance or radiance dose ELs for retinal thermal ELs for the case of large sources ($\alpha > \alpha_{max}$) and for retinal photochemical ELs which are applicable to all source sizes (Limiting apertures used for averaging exposure).

The exposure duration dependence of the retinal thermal limits for a

number of angular subtenses of the apparent source is given in Fig. 8.

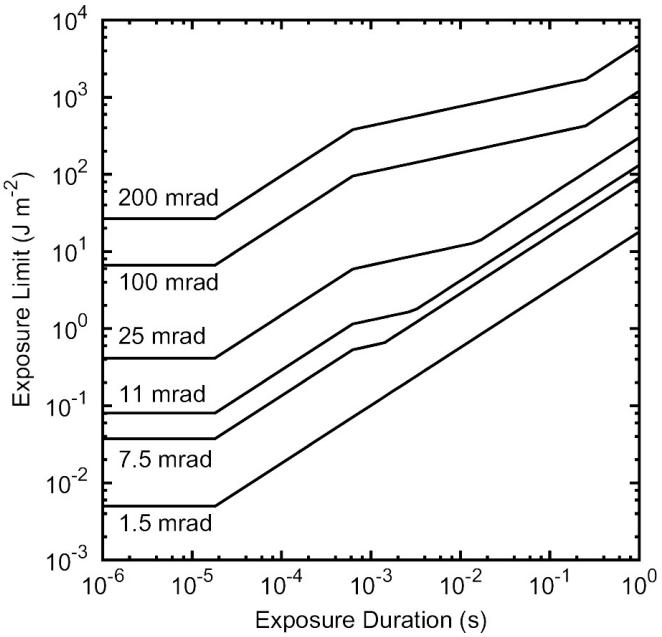

Fig. 8 Exposure duration dependence of the retinal thermal limits for a number of angular subtenses of the source, for the wavelength range of 400 nm to 700 nm.

The exposure limits for continuous-wave laser radiation for point sources in the wavelength range of 400 nm-1400 nm is given in Fig. 9.

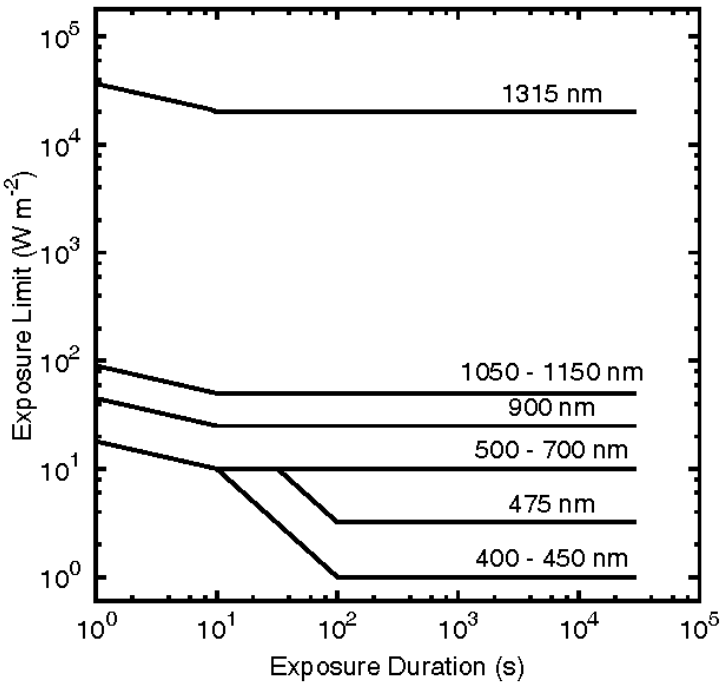

Fig. 9 Exposure limits for point-source viewing of continuous-wave laser radiation for selected wavelengths in the range of 400 nm to 1400 nm .

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

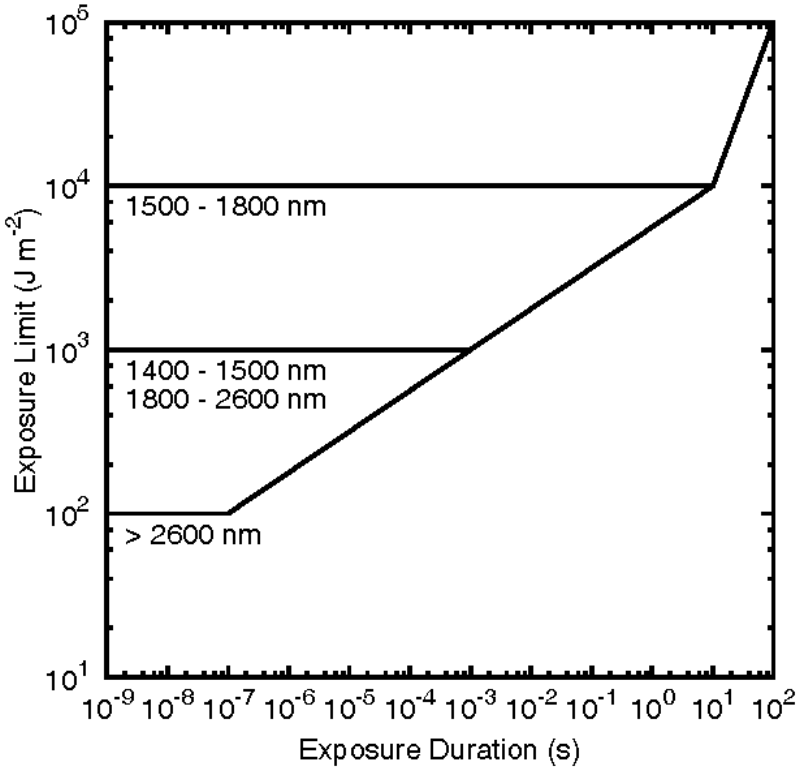

Fig. 10 Exposure limits for ocular and skin exposure to middle and far-infrared laser radiation.

1.2 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, 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 nm-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 nm-1400 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; Aron-Rosa; DeLori; Fankhauser; Landry; Mainster; Marshall; Rassow; Stuck; Trokel; Motz-West; M. 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.

1.3 Repetitive laser 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 in the retinal hazard region, 400 nm-1400 nm. 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.

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 3).

a.) For $\alpha \leq 5$mrad with pulse durations exceeding T_i (5 µs for visible wavelengths), $C_p = 1.0$.

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 if $n > 625$, $C_p = 0.2$.

c) For pulse durations less than or equal to T_i, and for exposure durations less than or equal 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, $C_p = 0.5$. 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. Such individuals should therefore take more rigorous precautions to avoid exposure to UVR laser radiation. In addition, the exposure limits from 300 nm to 400 nm do not apply to infants or to aphakic individuals (ICNIRP 1997).

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; Aron-Rosa; DeLori; Fankhauser; Landry; Mainster; Marshall; Rassow; Stuck; Trokel; Motz-West; M. 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; Morgan; Bass; Cringle; Yu 2008). These conditions also require lengthy exposures and apparently result from an oversaturation of the cone opsins (Kremers; van Norren 1988, Mellerio 1994). While this has traditionally been thought of as an unrealistic exposure condition (Sliney; 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; Wolbarsht 1980).

With consideration of the above, a 1 mm aperture is about the smallest practical size for averaging of irradiance and 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; Goldman 1974, Sliney; Wolbarsht 1980, McCally; Farrell; Bargeron 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 about 11 mm, an area of about 1 cm^2, are therefore specified for wavelengths greater than 100 µm.

The diameters of the limiting apertures used for averaging exposure levels are summarized in Table 7.

Table 7 Limiting apertures for applying the exposure limits

Spectral region (nm)	Exposure duration, t (s)	Eye exposure (mm)	Skin exposure (mm)
180. – 400			
	1. 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. -1 400 nm	1. ns -30 ks	7.0	3.5
1 401. – 10^5 nm			
	1. 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 – 10^6 nm	1.0 ns – 30 ks	11.0	11.0

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

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 photothermal 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 a collection angle, 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.

Photochemical

For comparison of the exposure from sources smaller than 11 mrad with the photochemical limits, expressed 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 $\alpha > \gamma_{ph}$, then an acceptance angle of $\gamma_{ph} = 1.1 \cdot t^{0.5}$ 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 affect 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 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, pre-exposure 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 aphakic individuals or infants, who would require UVR absorbing lenses. Additionally special adjustments of the guidelines may be necessary for some ophthalmic instrument exposure (Sliney; Aron-Rosa; DeLori; Fankhauser; Landry; Mainster; Marshall; Rassow; Stuck; Trokel; Motz-West; M. 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.

ACKNOWLEDGMENTS

During the preparation of this document, ICNIRP was composed of the following members: P. Vecchia, Chairman, Italy; M. Hietanen, Vice-Chairperson, Finland (until 2008); R. Matthes, Vice-Chairman, Germany; A. Ahlbom, Sweden (until 2008); E. Breitbart, Germany (until 2008); M. Feychting, Sweden; A. Green, Australia; F.R. de Gruijl, The Netherlands (until 2008); K. Jokela, Finland; J.C. Lin, United States of America; A.P. Peralta, The Philippines; K. Schulmeister, Austria; P. Söderberg, Sweden; B.E. Stuck, United States of America; A.J. Swerdlow, United Kingdom; M. Taki, Japan (until 2008); R. Saunders, United Kingdom; B. Veyret, France.
The Task Group which prepared the draft guidelines was composed of the following members: P. Söderberg, Chairman, Sweden; B. Stuck, (Chairman until 2008), USA; R. Greinert, Germany; D. Sliney, USA, K. Schulmeister, Austria; B. Lund, USA; R. Thomas, USA. ICNIRP also gratefully acknowledges the useful comments received from D.J. Lund, USA, J.A: Zuchlich, USA and R. Matthes, Germany, in the drafting phase.

Finally, the support received by ICNIRP during that period from the International Radiation Protection Association, the World Health Organization, the European Commission, and the German Federal Environment Ministry is gratefully acknowledged.

APPENDIX

USING THE EXPOSURE LIMIT TABLES

Example. To find the exposure limit for He-Ne (632.8 nm) laser radiation for a 0.25 second exposure, use Table 4. First use the left-hand column to find the wavelength. Choose the second 400 nm-700 nm entry since the 0.25 second (aversion response) exposure duration falls between 5×10^{-6} and 10 seconds (second column). The exposure limit (EL) for collimated beam (small source) intrabeam exposure ($C_E = 1$) is then:

EL = $18\ C_E\ t^{0.75})$ J m^{-2} (Table 4, Row: Visible, 3rd entry, column 3) =
$18\ (0.25)^{0.75}$ J m^{-2}
= 6.3 J m^{-2} = 6.3 W·s m^{-2}
= (6.3 W·s m^{-2})/(0.25 s) = 25 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 (1150-1400 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 the damage mechanism for pulsed laser exposures. Localized heating of melanin granules dominates and thermo-acoustic effects determine the damage. With this increased understanding of the spatial, spectral and temporal scaling factors, it was 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; Rumohr; Huettmann; Brinkmann 2005, Zuclich; Lund; Stuck 2007, Schulmeister; Husinsky; Seiser;

Edthofer; Fekete; Farmer; Lund 2008, Schulmeister; Sliney; Mellerio; Lund; Stuck; Zuclich 2008) 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; 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 α_{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; Lund; Edsall; Hollins; Smith; Stuck; McLin; Till 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 α_{max} = 100 mrad.

Recent thermal models and ex-vivo studies (Schulmeister; Husinsky; Seiser; Edthofer; Fekete; Farmer; Lund 2008, Schulmeister; Sliney; Mellerio; Lund; Stuck; Zuclich 2008) provided for a more complete understanding of the variation of the spot size dependence of retinal thermal injury with pulse duration. This allows for the formulation of a time dependent α_{max} to better reflect the retinal irradiance diameter dependence for pulsed sources (ICNIRP 2007). The value of α_{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 α_{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. 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; Lund; Edsall; Hollins; Smith; Stuck; McLin; Till 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 only a factor of 2 above the exposure limit. *Ex-plant ex-vivo* RPE damage threshold studies, (Gerstman; Thompson; Jacques; Rogers 1996, Kelly; Lin 1997, Lin; Kelly; Sibayan; Latina; Anderson 1999, Brinkmann; Huttmann; Rogener; Roider; Birngruber; Lin 2000) 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. (Schuele; Rumohr; Huettmann; Brinkmann 2005, Lee; Alt; Pitsillides; Lin 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 µs – 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; Stuck; Lund; Sliney 2007). 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, 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 2.6 when compared to the previous exposure limit in the nanosecond regime.

A review of the damage threshold studies in the pulse duration regime between 100 fs and 1 ns (Gerstman; Thompson; Jacques; Rogers 1996, Kelly; Lin 1997, Lin; Kelly; Sibayan; Latina; Anderson 1999, Roach; Johnson; Rockwell 1999, Brinkmann; Huttmann; Rogener; Roider; Birngruber; Lin 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 100 ps. At 100 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 100 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 100 ps is larger than 2, since C_A is not applied to EL for pulse durations less than 100 ps (Fig. 2). That is, the EL for pulse durations less than 100 ps in the wavelength range between 400 nm and 1150 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; Hammer; Hopkins; Payne; Toth; Roach; Druessel; Kennedy; Amnotte; Eilert; Phillips; Noojin; Stolarski; Cain 1997, Roach; Johnson; Rockwell 1999), exhibit a decreased wavelength dependence and damage thresholds at 1064 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; Cheney; Zuclich; P. 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; Lund; Edsall 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; Huttmann; Rogener; Roider; Birngruber; Lin 2000, Roegener; Brinkmann; Lin 2004, Lund 2007). It was argued recently (Sliney; 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; 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 2007). 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 (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 (section 1.3), and most importantly it was found that there was no need for $C_p < 1.0$ for the intrabeam "point-source" ELs.

Wavelength Dependence between 1150 nm and 1400 nm

A number of damage threshold studies with wavelengths between 1150 nm and 1400 nm provide for an update of the wavelength dependence of the retinal thermal exposure limits. It was recognized before that the reduction factor, in that wavelength range was larger than necessary, but for simplicity, the exposure limits were held constant with wavelength for wavelengths between 1150 nm and 1400 nm, i.e. $C_C = 8$. Recently there was increased interest in that wavelength range and a number of damage threshold studies were conducted for different pulse durations, reviewed by Zuclich et al. (Zuclich; Lund; Stuck 2007, Vincelette; Rockwell; Oliver; Kumru; Thomas; Schuster; Noojin; Shingledecker; Stolarski; Welch 2009). These threshold data confirmed the trend that was expected based on the greatly reduced transmission of the pre-retinal ocular media and resulted in an increase in the exposure limits by an exponential increase with increasing wavelength of the factor C_C. This provided a more realistic wavelength dependence for ELs, based upon existing knowledge of pre-retinal transmittance and absorption properties of the retina.

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