Laser Eye Safety Research at APL

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The focus of this article is a multidisciplinary research program that addresses corneal injury from exposure to infrared laser radiation. Our general methodology combines experimental physical and biological measurements with theoretical analyses to understand corneal damage. Damage thresholds are determined under carefully controlled exposure conditions, and mathematical damage models are developed to correlate and predict damage conditions for other exposure conditions. The information gained supports the evaluation of current permissible exposure limits promulgated by the American National Standards Institute Z136 Laser Safety Standards.

INTRODUCTION

Laser eye safety research has a long history at APL. The work has focused mainly on the effects of infrared (IR) exposures to the cornea (viz., the transparent front part of the eye). This research has been sponsored primarily by the Army Medical Research and Materiel Command (AMRMC) and supports their mission to assess the health effects and hazards of nonionizing electromagnetic radiation from laser systems. The data obtained in this research also support the evaluation of current permissible exposure limits promulgated by the American National Standards Institute (ANSI) Z136 Laser Safety Standards.

Our earlier research on CO_2 laser exposures (wavelength, 10.6 μm) having durations $\geq$1 ms was discussed in two previous *Technical Digest* articles.[1,2] IR radiation at wavelengths beyond 1.4 μm is absorbed to varying degrees by water in the tissue. The absorbed radiation is rapidly converted to heat, thus raising the temperature in the absorption volume. The heat is subsequently conducted to deeper layers of the absorbing medium, causing the temperature to rise throughout the exposed tissue as a result of both heat conduction and direct radiation. If the temperature increase is sufficiently high, it causes thermal damage to the tissue. The cornea is particularly susceptible because of its relatively high water content, 78%, and its accessibility. Because absorption at these wavelengths is sufficient to prevent the radiation from reaching the sensory retina, this spectral region has been erroneously labeled as "eye-safe." Eye-safe in this context is a very misleading term because painful and visually disabling corneal injuries are possible from overexposure to these wavelengths.

The cornea is the transparent portion of the wall of the eye. Its transparency and smooth curved surface,

which provides ≈75% of the eye's focusing power, are both essential for proper image formation and maintenance of good visual acuity. Figure 1 presents a diagram of the eye indicating the cornea's location as well as a light micrograph showing a cross-sectional view of the human cornea.

In addition to the tear layer, which is about 7 μm thick, the human cornea has five other layers. From anterior (front) to posterior (back), they are the epithelium, Bowman's layer, the stroma, Descemet's membrane, and the endothelium.

The epithelium, which is about 50 μm thick, is composed of several layers of cells. It is attached to a basement membrane that separates it from Bowman's layer. Bowman's layer is an approximately 10-μm-thick acellular layer of thin collagen fibrils that are randomly aligned and embedded in a ground substance. The stroma accounts for 90% of the corneal thickness, which averages 520 μm in humans. It is composed of layers of parallel collagen fibrils that are embedded in a ground substance consisting of glycosaminoglycans, salts, and water. Thin cells called keratocytes are interspersed between layers. The ultrastructural arrangement of the collagen fibrils is important in determining the cornea's optical properties, especially its transparency. Corneal transparency results from destructive interference effects in light scattered from the fibrils. These effects are due to short-ranged ordering in the positions of the fibrils about one another. Understanding the ultrastructural bases of the cornea's optical properties has been a major thrust of the APL Biomedical Program since its beginning and has been the subject of several *Technical Digest* articles.[1,3–5] Descemet's membrane, which is composed of a highly ordered network of very thin collagen filaments, is about 10 μm thick in adults and is the basement membrane of the corneal endothelium. The endothelium is a single cell layer about 3 μm thick that actively pumps fluids into the anterior chamber to maintain corneal hydration at its physiologic value. Maintenance of proper hydration is essential for corneal transparency. Swelling of the cornea disrupts the ordering of the fibrils about one another, which causes increased light scattering and a concomitant loss in transparency. Therefore, because human endothelium does not regenerate readily when injured, it is important that its health be maintained.[6]

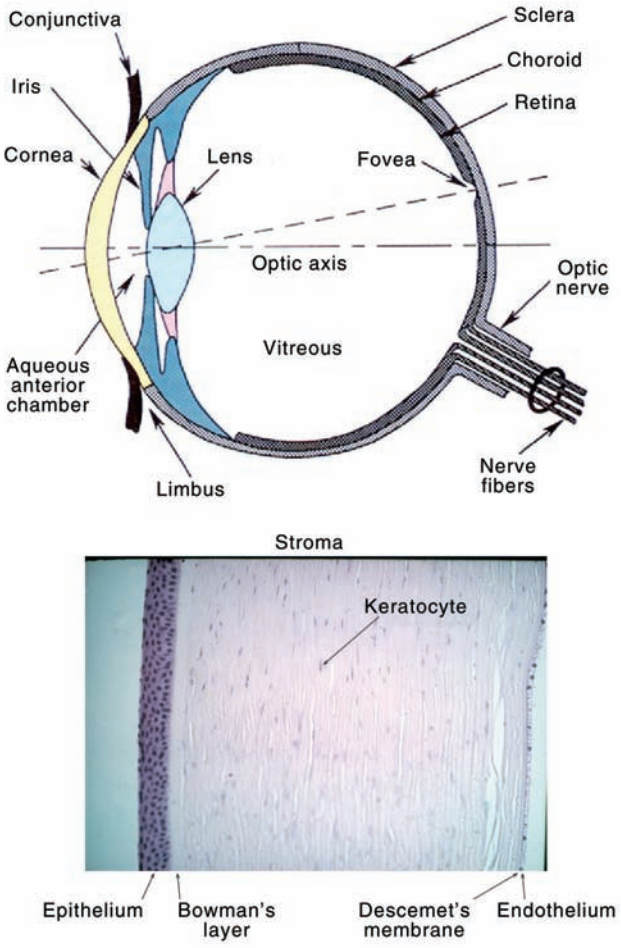

Figure 1. A diagram of the eye showing the location of the transparent cornea and a light micrograph showing a cross section of a human cornea. The various layers from anterior (front) to posterior (back) are the epithelium, showing dark-stained cell nuclei; the acellular Bowman's layer; the stroma, showing keratocytes between its layers; Descemet's membrane; and the endothelium.

The absorption of radiation in a material is described by Beer's law, which is given by

$$I(z) = I_0 \exp(-\alpha z) , \qquad (1)$$

where $I(z)$ is the irradiance at a depth z into the material, I_0 is the incident irradiance, and α is the absorption coefficient. The absorption coefficient depends on the material and the wavelength of the radiation. In the case of the cornea, it is standard practice to use the absorption coefficient of water to approximate that of the cornea.[7–12] Figure 2 shows plots of $I(z)/I_0$ as a function of distance into the cornea for laser wavelengths of 1.54 μm (Er, or erbium, fiber), 2.02 μm (Tm:YAG), and 10.6 μm (CO_2), which have been the focus of our research. A schematic representation of the human cornea (thickness, 520 μm) is also shown for comparison. Note that 10.6-μm radiation (α = 950 cm^{-1}) is almost entirely absorbed in the epithelium and 2.02-μm radiation (α = 55 cm^{-1}) is almost entirely absorbed within the cornea, but only ≈40% of the 1.54-μm laser radiation (α = 12.3 cm^{-1}) is absorbed in the entire depth of the cornea.

Threshold injury to the cornea from IR laser radiation is usually confined to the corneal epithelium because this cellular layer experiences the highest temperature increase. Threshold injury is characterized by the presence of a superficial gray-white spot that develops within 0.5 h after exposure and is barely visible with a slit-lamp biomicroscope.[13] Such minimal lesions heal completely

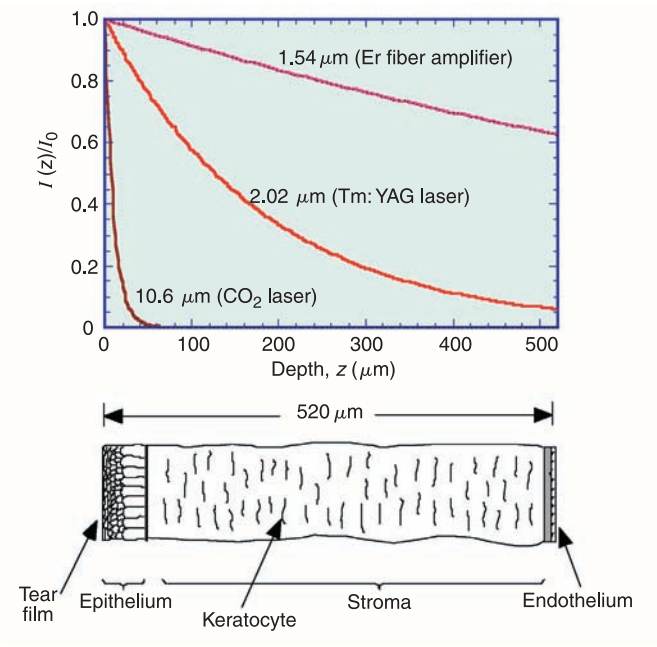

Figure 2. Beer's law of absorption of IR radiation in the cornea. The plots of $I(z)/I_0$ show absorption for the three lasers discussed in the text; a schematic of the cornea on the same scale is also provided.

within 24 to 48 h. Threshold exposures are determined by bracketing exposures above and below the threshold; the bracket is narrowed until there is only about a 10% difference in irradiance (or radiant exposure) between an exposure that produces a minimal lesion and one that does not. The injury threshold is taken to be at the center of the bracket. Threshold measurements are made using a rabbit model. (A rabbit cornea is similar to a human cornea in structure and composition but is thinner.) In making threshold measurements, the lasers are operated in the mode that has a Gaussian irradiance profile (TEM_{00} mode) given by

$$I(r) = I_0 \exp(-r/r_{1/e})^2 . \qquad (2)$$

Here the peak irradiance I_0 is related to the total laser power P by $I_0 = P/A_{1/e}$, where $A_{1/e}$ is the area within the $1/e$ radius, $r_{1/e}$. Obviously a similar relationship holds for radiant exposure $H(r)$, defined as the surface density of the radiant energy incident on the tissue expressed in units of J/cm^2. Use of the fundamental TEM_{00} mode facilitates comparisons between experimental results as well as comparisons with thermal models.

The material in this article is based on our research and on publications subsequent to our earlier *Technical Digest* articles that reviewed laser ocular effects.[1,2] We review our research on the effects of very short pulses (80 ns) of highly absorbed CO_2 radiation, discuss injury thresholds for single- and multiple-pulse exposures to moderately penetrating radiation from a Tm:YAG laser, and examine injury thresholds for highly penetrating radiation from an Er fiber laser. The threshold exposures are discussed in terms of a critical temperature damage model.

LASER EXPOSURES

CO_2

Injury thresholds for CO_2 laser exposures having durations ≥1 ms have been extensively documented by us[1,2,10,14] and others.[15–17] We also have determined thresholds for sequences of pulses having individual pulse durations ≥1 ms.[2,10] Damage in this regime is thermal, and thresholds are correlated by a modified critical temperature model in which the "damage temperature" is a weak function of exposure duration, with shorter exposures requiring higher temperatures than long exposures for damage.[2,10] Before 1990, there were very few studies of injury thresholds for pulse durations less than 1 ms and no data for exposures to sequences of such pulses.

The military uses many laser systems (e.g., training devices, rangefinders, target designators, communications devices) that emit either short pulses or sequences of short pulses. Because personnel risk exposure to these devices, a need existed to broaden the database of damage thresholds for both single- and multiple-pulse exposures to short pulses. A more complete understanding of the mechanisms responsible for damage caused by very short pulses also was needed. Such information provides a rational basis for setting exposure limits to prevent ocular injury.

We used a CO_2-TEA laser that emits 80-ns pulses to determine injury thresholds for single- and multiple-pulse exposures at repetition frequencies of 10 and 16 Hz.[18] The damage threshold radiant exposures per pulse are plotted in Fig. 3 as a function of the number of pulses. Least-squares fits to these data show that the thresholds are correlated by an empirical power law of the form

$$H_{th} = CN^{-\beta}, \qquad (3)$$

where N is the number of pulses in the sequence. The empirical constants C and β are determined by a least-squares fit to the experimental data. For the 10-Hz thresholds, C = 291 mJ/cm^2/pulse and $\beta = 0.162$ (R = 0.976); for the 16-Hz thresholds, C = 300 mJ/cm^2/pulse and $\beta = 0.194$ (R = 0.997). It is not possible to discern if the slight difference between the two fits is real; however, both fall within the ±10% accuracy estimated from the bracketing procedure used to determine the thresholds. The values for the constant C differ by less than 5% from the measured damage threshold radiant exposure for a single pulse. This is well within the

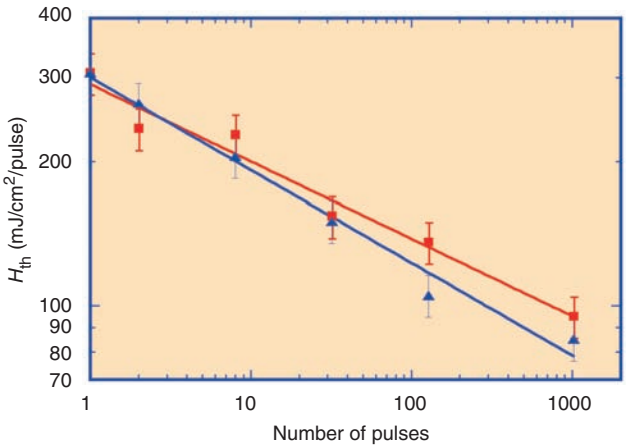

Figure 3. The dependence of the threshold radiant exposure per pulse as a function of the number of pulses from a CO_2-TEA laser at pulse frequencies of 10 and 16 Hz. The lines are least-squares fits to a power law of the form $H_{th} = CN^{-\beta}$. The corresponding values of C and β are given in the text. The error bars are ±10% of the experimental threshold values (squares, 10 Hz; triangles, 16 Hz) and represent the estimated accuracy of the bracketing procedure used to determine the thresholds.[18]

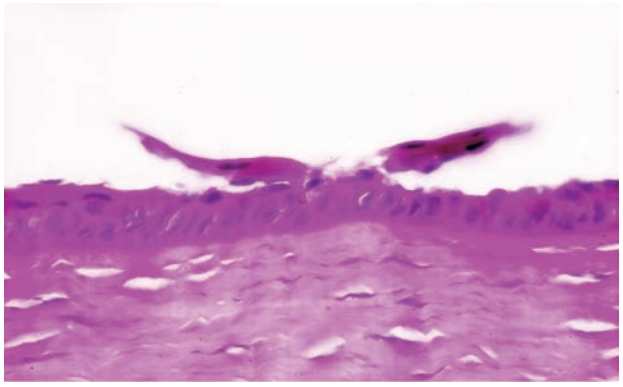

Figure 4. A hematoxylin/eosin-stained section showing the anterior region of a rabbit cornea that was exposed to an 80-ns pulse at 397 mJ/cm^2 from a CO_2-TEA laser. This exposure is 1.29 times the epithelial injury threshold. The damage is primarily confined to the anterior epithelium where the cells are lifted from the surface. The cellular disruption is similar to that expected from the passage of a tensile wave.[21]

estimated accuracy of the procedure used for determining individual thresholds.

The histology of corneas exposed somewhat above the damage threshold shows features consistent with both mechanical and thermal damage mechanisms. Mechanical damage could result from large temperature gradients at the anterior surface that produce pressure transients via a thermoelastic process.[18–23] The disruptions of the superficial epithelial cells shown in Fig. 4 are consistent with the type of structural alteration that might be produced by a tensile stress wave. In this regard, it is noteworthy that the thermoelastic stress wave generated by laser absorption at a free surface consists of a compression wave followed by a tensile wave.[20,22,23] However, the vacuoles (voids) and loss of well-defined organelles (cellular components) in the anterior epithelial cells shown in Fig. 5 are characteristic of thermal damage.

The maximum temperature *increases* calculated for the damage threshold exposures are in the range of 25° to 35°C. Temperatures are calculated at a position on the beam axis 10 μm beneath the surface of the tear layer. Thus, assuming that the tear layer is about 7 μm thick,[24] the temperature increases are those that occur just inside the anteriormost epithelial cells. Because of heat conduction, the temperature at this position does not reach its maximum value until about 164 μs after the final pulse. The maximum temperature increases for both the single pulse and pulse sequences are lower than those calculated previously for longer individual pulse durations and are lower than predicted by the modified critical temperature damage model that described thresholds for exposure durations ≥1 ms.[2,10] Nevertheless they are constant to within ±10% of their mean values and independent of the number of pulses in the sequences. This result suggests that the damage mechanism has a substantial thermal component and can be described by a critical temperature damage model.[18]

We tested this suggestion further by measuring damage thresholds in corneas that had been cooled

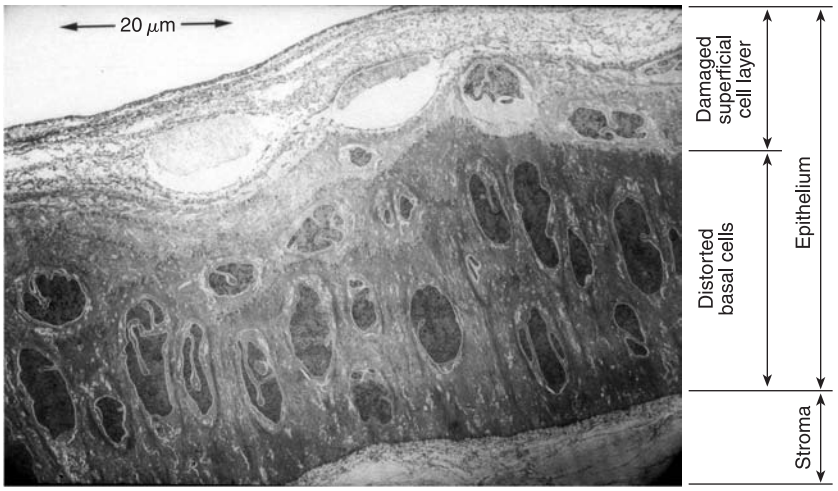

Figure 5. Transmission electron micrograph of the epithelium in the center of a lesion in a rabbit cornea produced by an 80-ns pulse at 755 mJ/cm^2 from a CO_2-TEA laser. This exposure is 2.46 times the epithelial injury threshold. Damage is characterized by a degenerating superficial cell layer that overlies intact, but distorted, basal cells. The degenerating cells show loss of well-defined organelles (cellular components), accumulation of amorphous electron dense material, and vacuolation (voids). This type of damage is characteristic of thermal lesions.[21]

to room temperature. We noted that if a critical temperature model is valid, then damage should occur at the same final critical temperature (not temperature increase).[18] Thus for a cooled cornea, sufficient energy would have to be supplied to raise the temperature, first to the *in vivo* temperature (assumed to be 35°C; Ref. 25), and then to the damage temperature. We found that, in fact, the energy needed to produce threshold damage would raise the calculated temperature to a level that was greater than the level associated with damage in the *in vivo* corneas. This finding provided additional, strong evidence that the damage from 80-ns pulses has a substantial thermal component and led us to suggest that the lower than expected damage temperature for the 80-ns pulses might be a result of thermoelastic stress waves potentiating thermal damage.[18]

Tm:YAG

There is an increasing emphasis in both the military and civilian sectors on laser systems that operate in the 1.3- to 3.0-μm spectral region because, as noted previously, such radiation does not reach the retina. IR lasers operating at these wavelengths are being used in a variety of applications such as communications devices, battlefield training devices, rangefinders, lidars, and therapeutic medical devices. Because of the paucity of injury threshold data for this spectral region prior to the early 1990s, safety standards had been set based on extrapolations from other spectral regions (primarily using CO_2 data at 10.6 μm) with added safety factors. Moreover, there were no threshold data for sequences of pulses. An important issue that policy makers wanted to address, therefore, was whether the standards were overly conservative since damage data were lacking. Because of our experience with CO_2 threshold research, Dr. David Sliney of the U.S. Army Center for Health Promotion and Preventive Medicine (USACHPPM) (then called the U.S. Army Environmental Hygiene Agency) encouraged us to begin research in this area and supported our initial efforts. Subsequent research was supported by the AMRMC.

Our initial investigation of single-pulse exposures used a continuous-wave diode pumped laser that was loaned to us by Coherent Technologies (Boulder, CO).[11] This laser had a maximum power of 1 W and operated in the TEM_{00} mode. Subsequent investigations of single- and multiple-pulse exposures used a continuous-wave diode pumped laser built for us by the Naval Research Laboratory. This laser had a maximum power of 350 mW and also operated in the TEM_{00} mode.[12]

Thresholds were measured for single pulses and for sequences of pulses at repetition frequencies of 1, 10, 20, and 100 Hz. Figure 6 shows a lesion produced by exposure to 999 0.025-s pulses at 20 Hz. This exposure was 70% greater than the epithelial injury threshold. Figure 7 is a light micrograph of a small region in the lesion shown in Fig. 6. Damage is confined to the epithelium, and no obvious stromal abnormalities are present.

The multiple-pulse thresholds are plotted in Fig. 8, where it is evident that they are correlated by the same form of empirical power law that correlated CO_2 laser exposures (cf. Eq. 3). The values of C and β obtained from the fits are, respectively, 10.1 J/cm^2/pulse and 0.287

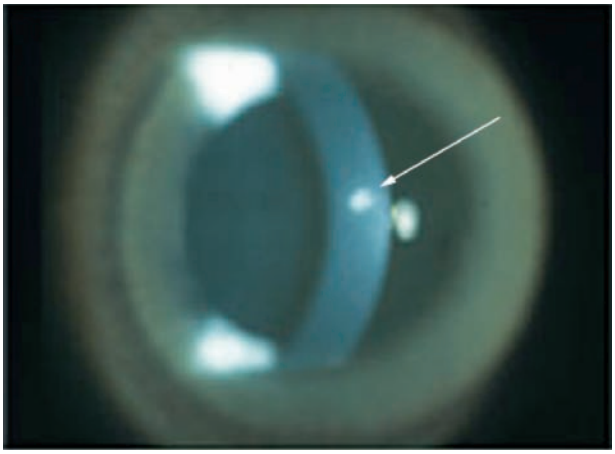

Figure 6. The arrow points to a lesion in a rabbit cornea resulting from an exposure to 999 pulses from the Tm:YAG laser. The pulse repetition frequency was 20 Hz, the individual pulse duration was 0.025 s, and the radiant exposure was 0.472 J/cm^2/pulse, which is approximately 1.7 times the damage threshold for these conditions.[12]

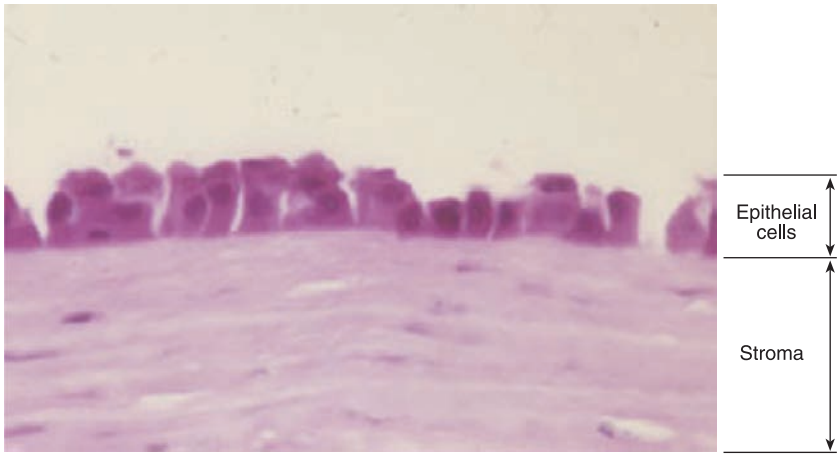

Figure 7. A hematoxylin/eosin-stained section of the lesion shown in Fig. 6 exhibiting the damaged epithelial cells (dark pink). There is loss of superficial epithelium and possible pyknosis (i.e., condensation and reduction of the size of the cells or their nuclei) of the remaining epithelium. No obvious abnormalities in the stroma (light pink region) are present.[12]

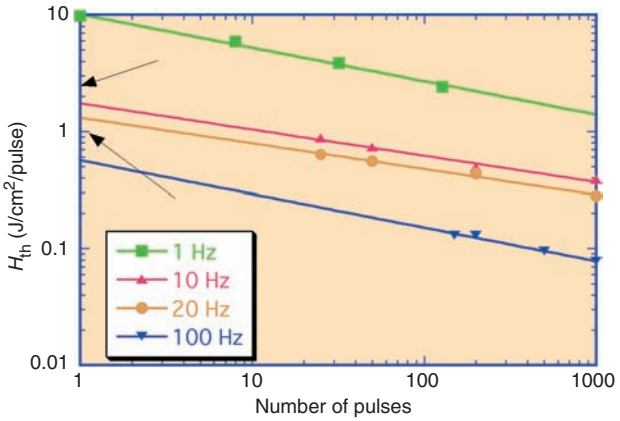

Figure 8. Dependence of the threshold radiant exposure per pulse as a function of the number of pulses from a Tm:YAG laser at pulse repetition frequencies of 1, 10, 20, and 100 Hz. The individual pulse durations were 0.300 s for the 1-Hz exposures, 0.025 s for the 10- and 20-Hz exposures, and 0.005 s for the 100-Hz exposures. The lines are least-squares fits to a power law of the form $H_{th} = CN^{-\beta}$. The corresponding values of C and β are given in the text. The upper and lower arrows show the estimated threshold for a single 0.025-s pulse and a single 0.005-s pulse, respectively.[12]

for the 1-Hz exposures; 1.75 J/cm^2/pulse and 0.22 for the 10-Hz exposures; 1.32 J/cm^2/pulse and 0.22 for the 20-Hz exposures; and 0.58 J/cm^2/pulse and 0.29 for the 100-Hz exposures. The value of C for the 1-Hz exposures is within 4% of 9.75 J/cm^2/pulse, which is the measured threshold for a single 0.30-s pulse. However, it is obvious that the power law breaks down at small numbers of pulses for exposures at 10 and 20 Hz. The fits for these data have the same value for the parameter β (they are parallel on the log-log plot), but they have different values of the parameter C, even though the individual pulse duration is the same. The laser power was insufficient to measure the threshold for a single 0.025-s pulse; however, its value could be estimated from the existing measured data. The upper arrow in Fig. 8 denotes the estimated threshold for a single 0.025-s pulse (≈2.5 J/cm^2). Similarly, the constant C for the 100-Hz exposures with 0.005-s pulses is lower than the estimated threshold for a single 0.005-s pulse (≈0.95 J/cm^2). The lower arrow in Fig. 8 denotes this threshold.

Based on these results, we noted that in terms of setting safety standards, the power law that is valid for large numbers of pulses will in some circumstances underestimate the damage threshold for small numbers of pulses.[12] In such cases this would provide an additional margin of safety for exposures to small numbers of pulses. However, these results also imply that using the measured threshold for a single pulse for the value of C in the power law to predict damage from a sequence of a large number of pulses risks lessening the margin of safety.

As noted previously, epithelial injury thresholds from CO_2 laser radiation were found to be consistent with either a critical temperature damage model or a modified critical temperature model. The calculated temperature increases for the single-pulse thresholds average 45.8 ± 4.2°C (mean ± SD) and are therefore constant to within the experimental uncertainty.[12] They are thus consistent with a critical temperature damage model. However, the pattern of variability in the temperature increases from the multiple-pulse exposures is not consistent with a critical temperature model. The temperature increases for pulse sequences with longer durations tend to decrease.[12] This behavior is shown in Fig. 9, where the peak temperature (*not temperature increase*) is plotted as a function of duration of the pulse sequence given by $D = (N-1)/\mathrm{PRF} + \tau$, where PRF is the frequency and τ is the individual pulse duration (the ambient temperature of the cornea is assumed to be 35°C).[25] The calculated temperatures for the single-pulse exposures are also included in the figure. The critical peak temperatures at the damage threshold ($\mathrm{CPT}_{\mathrm{Tm:YAG}}$) are consistent with a modified critical temperature model given by

$$\mathrm{CPT}_{\mathrm{Tm:YAG}} = 76D^{-0.054}\ ^{\circ}\mathrm{C}\,, \qquad (4)$$

which is similar to the modified critical temperature model we found for single pulses of CO_2 radiation.[2,10]

Er Fiber

In addition to military applications, the 1.5- to 1.7-μm spectral region is being used in the civilian sector for free-space laser communications in urban environments. Because optical aids such as binoculars and telescopes transmit these wavelengths and greatly increase irradiance at the exit pupil, irradiance levels that would

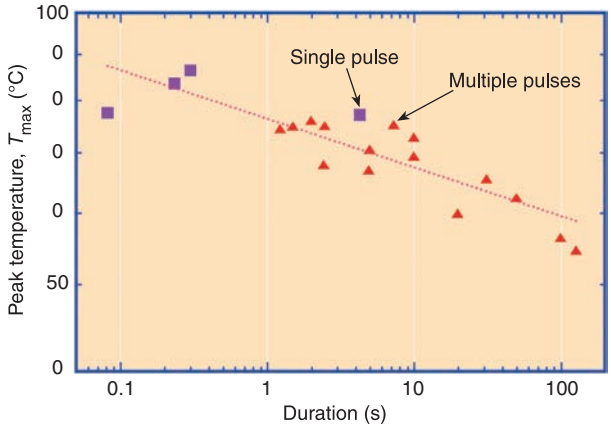

Figure 9. The calculated peak temperature 10 μm beneath the anterior tear surface as a function of total exposure duration. Calculations are shown for both single- and multiple-pulse exposures. The ambient temperature of the cornea was assumed to be 35°C. The line is a least-squares fit and yields the empirical modified critical temperature model given by Eq. 4.[12]

be relatively "safe" to the unaided eye could be amplified to dangerous corneal exposure levels. For example, a 10× sighting telescope would increase corneal irradiance by more than 50-fold. With the large number of fire-control telescopes and binoculars on the modern battlefield, soldiers could readily be exposed to dangerous IR wavelengths from anti-sensor lasers. Depending on the magnifying power and entrance aperture of telescopic optics, the cornea could be exposed to a range of laser spot-sizes. Moreover, because wavelengths in the 1.4- to 1.7-μm range are not as strongly absorbed in the cornea as the other radiation we have studied (cf. Fig. 1), they have a far greater likelihood of damaging the corneal endothelium and thereby may have a greater impact on the likelihood of delayed effects and corneal healing. Because of these considerations we began a comprehensive research program to investigate threshold exposures as a function of exposure duration and laser beam diameter. Terabeam Corporation supported the initial work. Subsequent studies, which are ongoing, are supported by the AMRMC under the Peer-reviewed Medical Research Program.

The following experiments were done using an Er fiber amplifier (IRE Polus Group, Model EAU-9M) driven by a laser diode. Terabeam Corporation loaned this laser system to us. The laser emits mid-IR radiation at a wavelength of 1.54 μm, and its output beam has a Gaussian irradiance profile.

Thresholds were determined for 1/*e* beam diameters of 0.05 to 0.70 cm for exposure durations ranging from ≈1 to 100 s and for a fixed beam diameter of 0.1 cm for exposure durations from 0.036 to 0.260 s.[26,27] Figure 10 compares the threshold radiant exposures for the Er fiber laser to those for CO_2[2,10] and Tm:YAG[11,12] lasers. Although the absorption coefficients for these lasers span nearly 2 orders of magnitude (cf. Fig. 1), the slopes of the threshold plots are nearly identical. The plots demonstrate clearly that injury thresholds for wavelengths having greater absorption coefficients, where the incident energy is more confined, are lower than those for more penetrating radiation, where the energy is absorbed in a larger volume.[27]

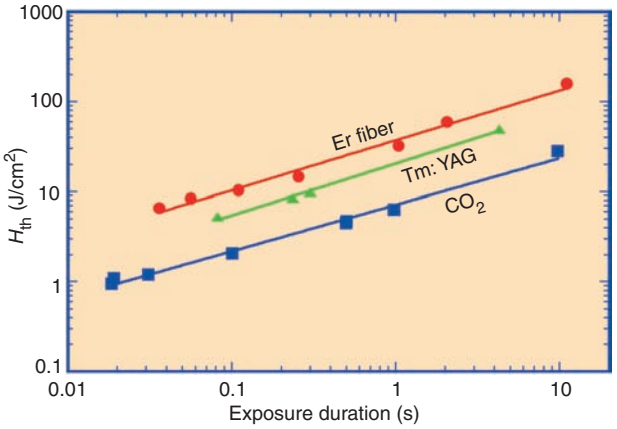

Figure 10. Comparison of threshold radiant exposures for CO_2,[2,10] Tm:YAG,[11,12] and Er fiber laser radiation,[26,27] with beam diameters of 0.2 cm for CO_2 exposures and 0.1 cm for the Tm:YAG and Er fiber exposures. Although the absorption coefficients vary over nearly 2 orders of magnitude (from 12.0 cm^{-1} for Er fiber to 950 cm^{-1} for CO_2), the slopes of the plots are nearly identical.

The predicted temperature increases for 2.05-, 11-, and 100-s exposures average 39.4 ± 4.1, 36.8 ± 4.9, and 32.8°C, respectively, and are therefore consistent with a critical temperature or modified critical temperature damage model. The standard deviation is not included for the 100-s exposures because only two thresholds were determined for that duration. The predicted temperature increases for the 1.04-s exposures with nominal beam diameters of 0.05 and 0.10 cm average 38.4°C, which is similar to those for the longer exposure durations; however, the temperature increases for exposures with 0.2- and 0.5-cm-dia. beams are anomalously low (≈25°C). The appearance of near threshold lesions for these two exposures also was different in that they looked uniform edge to edge and their edges were sharply defined.

Theory predicts that the irradiance required to produce a given temperature increase for a given exposure depends on the diameter of the laser beam.[2,10] Therefore if critical (or modified) temperature damage models are valid, threshold irradiances for epithelial damage should have the same dependence on beam diameter (recall that the temperature increase is directly proportional to the irradiance). This hypothesis was validated for damage from CO_2 laser radiation in a previous study.[2,10] Figure 11 shows the measured threshold irradiances for exposures having durations of 1.04, 2.05, and 11 s plotted as a function of beam diameter. These data show that the irradiance required to produce a threshold lesion increases as the beam diameter is made smaller and becomes relatively independent of beam diameter for 1/*e* diameters greater than ≈0.3 cm. This dependence results from radial heat conduction along the temperature gradient caused by the irradiance profile of the Gaussian beam, which becomes greater for small diameter beams. The curves in the figure are the calculated irradiances that would be required to produce the average temperature rises noted above. The relatively close agreement between the measured thresholds and these calculated irradiances provides strong support for the hypothesis that a "modified" critical temperature damage model can describe these damage thresholds.

On the other hand, the calculated temperature increases for exposure durations <1 s, which range between 17° and 28°C, are not consistent with either a critical or modified critical temperature damage model. First, they are significantly lower and second, unlike the modified critical temperature damage model, they decrease rather than increase as the exposure duration becomes shorter. The reason for this behavior for

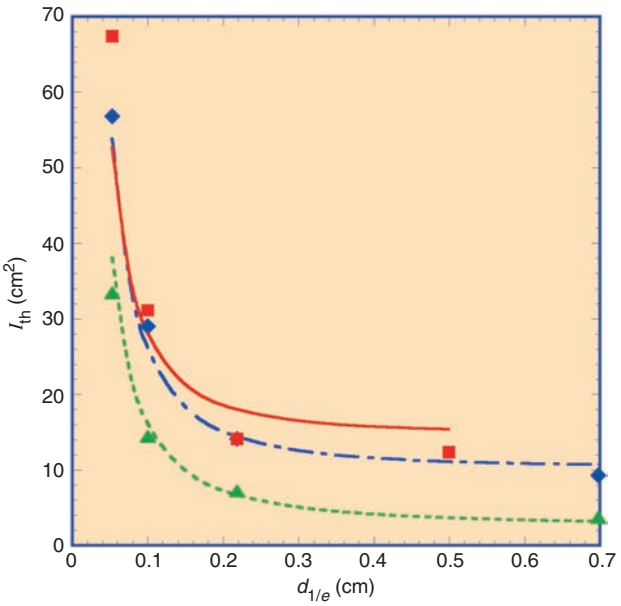

Figure 11. Dependence of Er fiber laser injury threshold exposures on beam diameter. The red squares, blue diamonds, and green triangles are the measured peak threshold irradiances for exposure durations of 1.04, 2.05, and 11 s, respectively. The curves with corresponding colors are the calculated irradiances required to produce a fixed temperature increase ΔT_c on the beam axis 10 μm below the anterior tear surface. For the red curve, ΔT_c = 32°C; for the blue curve, ΔT_c = 39.5°C; and for the green curve, ΔT_c = 36.8°C. These values of ΔT_c are the averages of the calculated temperature increases for the three exposure durations.[27]

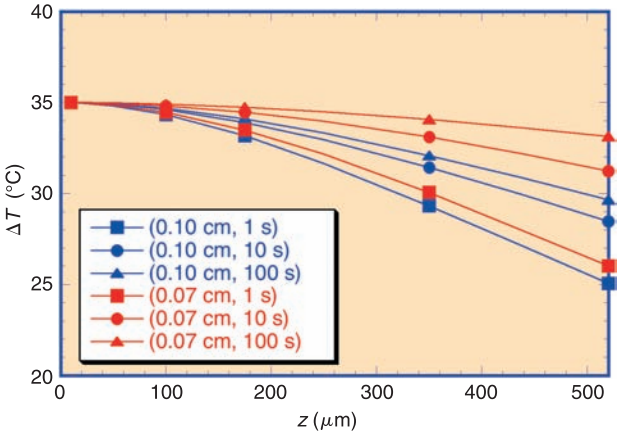

Figure 12. Depth dependence of the temperature increase in the cornea caused by an exposure that produces a rise of 35°C at a depth of 10 μm. Curves are shown for beams with 1/*e* diameters of 0.1 cm (blue) and 0.7 cm (red) for exposure durations of 1, 10, and 100 s.

exposures <1 s is not yet understood.[27] This result is particularly perplexing in view of the Er fiber data shown in Fig. 10. These data indicate that the threshold radiant exposures (and therefore the threshold irradiances) measured for the shorter Er fiber exposures are consistent with those of the longer Er fiber exposures as well as with the threshold exposures for CO_2 and Tm:YAG laser radiation, which are correlated by critical temperature damage models.

Figure 2 shows that 1.54-μm radiation is absorbed throughout the depth of the cornea. Because we have demonstrated that all corneal cells have essentially the same critical temperature for threshold damage,[2,14,28,29] it is important to examine how temperature depends on depth into the cornea for this penetrating radiation, particularly when considering its potential for damage to the corneal endothelial cell layer. Figure 12 shows the calculated maximum temperature increase as a function of depth into the cornea at several exposure durations for incident beams having diameters of 0.1 and 0.7 cm. The incident irradiance for each exposure was that required to produce a calculated temperature increase of 35°C at a depth of 10 μm. At short exposures there is very little difference in the depth dependence for the 0.1- and 0.7-cm-dia. beams; however, as the exposure duration increases, the temperature distribution becomes almost uniform for the 0.7-cm beam, differing by only about 2°C between 10 and 520 μm for a 100-s exposure and 4°C for a 10-s exposure. This is again a manifestation of the effect of radial heat conduction. The clear implication for these conditions is that the threshold for damage to the endothelium is only about 4% higher than the threshold for epithelial damage. In general this small percentage difference is within the experimental error of damage threshold determinations. These results imply that if a person were to receive an exposure slightly above the epithelial damage threshold from a beam having a diameter of 0.7 cm (which is the diameter of the exit pupil of 7 × 50 binoculars), he or she could be at risk for sustaining endothelial damage.

CONCLUSIONS

We have provided a review of APL research on IR laser damage to the cornea. The research encompasses the effects of single- and multiple-pulse exposures to radiation wavelengths ranging from those that are strongly absorbed by the cornea to those that are only weakly absorbed. We showed that

- Threshold damage from exposure to sequences of very short (80-ns) pulses of strongly absorbed radiation from a CO_2 laser is correlated by an empirical power law relating the threshold radiant exposure to the number of pulses.
- Threshold damage is consistent with a critical temperature damage model, but that susceptibility to thermal damage from such exposures may be potentiated by thermoelastic stress waves generated by the short pulses.
- Injury thresholds for multiple-pulse exposures to moderately penetrating radiation from a Tm:YAG laser are correlated by an empirical power law of the same form as for the CO_2 laser exposures;

however, for some exposure conditions, the relationship breaks down for small numbers of pulses.

- An empirical critical temperature model in which the critical damage temperature has a weak dependence on the duration of the single pulses or the entire train of pulses correlates injury thresholds for Tm:YAG radiation.

Finally, we discussed injury thresholds for highly penetrating radiation from an Er fiber laser and showed that the dependence of the threshold radiant exposures on laser beam diameter for exposures greater than 1 s provides strong evidence supporting a critical temperature damage model; thresholds for shorter exposures, however, are not in accord with a critical temperature damage model. We are currently investigating threshold damage for sequences of pulses from the Er fiber laser.

In the near future, we will investigate damage to deeper layers of the cornea, including the endothelial cell layer, and the healing response of lesions resulting from exposures above the injury threshold.

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