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SENSITIVITY OF THE RETINA TO RADIATION DAMAGE AS A FUNCTION OF WAVELENGTH*

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Abstract—Exposure of the retina of the rhesus monkey to visible and infrared radiation from CW optical sources like the Sun, xenon lamps, etc. produces small lesions or scotomata which may be classified as thermal or photochemical, depending on the wavelength and duration of exposure. The action spectrum for the production of retinal lesions has been determined for eight monochromatic laser wavelengths extending from 1064 to 441 nm. The corneal power required to produce a lesion decreases by three orders of magnitude in going from 1064 to 441 nm. Exposure to 1064 nm radiation for 1000 s produces a typical thermal lesion at elevated retinal temperatures, whereas a 1000 s exposure to 441 nm light produces a photochemical lesion at power levels too low to raise the retinal temperature by an appreciable amount ($< 0.1°C$). The two types of lesion have entirely different characteristics as will be discussed in some detail. The photopathology of the photochemical lesion has been studied at postexposure times ranging from 1 h to 90 days and will be demonstrated in a number of histological slides. Moreover, this photopathology correlates well with monocular visual acuity tests in the rhesus monkey as defined by the Landolt ring technique.

To further elucidate the differential effects on the retina of short vs long wavelength CW radiation, we have divided a simulated solar spectrum at sea level into two spectral bands, 400–800 nm and 700–1400 nm, and determined the radiant exposures required to produce very mild lesions on the rhesus retina for exposure times of 1, 10, 100 and 1000 s. To correlate our data with solar retinitis and eclipse blindness the image diameter or spot size on the retina was 159 μm, corresponding to the image size of the Sun on the human retina. Exposure to the 400–800 nm spectrum for durations of 10 s or greater required approximately 400 J/cm^2 to produce a mild photochemical lesion. Reciprocity is maintained over the exposure range 10–1000 s. Radiant exposure to the 700–1400 nm spectrum, on the other hand, required roughly 69,100 J/cm^2 for a 1000 s exposure. This was a mild thermal lesion. We were unable to produce a lesion for exposure times less than 1000 s. We interpret these data to mean that solar retinitis and eclipse blindness are primarily photochemical events produced by the short wavelength component of the solar spectrum, and that the infrared component of the solar spectrum plays only a minor role in these retinal pathologies.

INTRODUCTION

Wavelengths between 400 and 1400 nm are transmitted through the ocular media of the mammalian eye to the retina (Ham *et al.*, 1973). Within this spectral range there are at least three types of retinal damage resulting from exposure to optical sources which produce a discrete or limited image on the retina. These are: mechanical damage from sonic transients induced by mode-locked ps pulses or Q-switched pulses from lasers (Ham *et al.*, 1974); thermal damage from pulses ranging from microseconds to seconds in duration and after prolonged exposure to CW radiation in the long wavelength visible and near infrared spectrum (Ham *et al.*, 1966); photochemical damage from short wavelength visible light at power levels too low to produce appreciable temperature rises in the retina (Ham *et al.*, 1976).

The type of retinal damage depends primarily upon wavelength, power level and exposure time. Pulse widths ranging from microseconds to seconds at relatively high energy levels produce elevated temperatures in the retinal pigment epithelium (RPE) and choroid, causing thermal denaturation in both the RPE and neural retina. If relatively minor variations with wavelength of melanin absorption and transmittance through the ocular media are accounted for, both mechanical and thermal damage are independent of wavelength, depending only upon sufficient energy absorption to produce sonic transient phenomena and/or temperatures which can damage the delicate neural tissue and RPE. Prolonging the exposure time while reducing retinal irradiance to a level where temperature rise is negligible produces a different type of damage which is photochemical in nature. The photochemical lesion is strongly dependent upon wavelength, such that retinal sensitivity increases sharply at the blue-violet end of the visible spectrum.

Although Friedman and Kuwabara (1968), Tso (1973) and Ham *et al.*, (1973, 1976) have described discrete retinal lesions in the rhesus monkey which could not be explained on a thermal basis, little atten-

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tion has been paid to the short wavelength photochemical lesion. In this paper we report on the action spectrum and photopathology of the photochemical lesion as investigated in the rhesus monkey and describe how it differs from a thermal lesion and how it affects visual function. We also show that photochemical injury is primarily responsible for solar retinitis and eclipse blindness. It is also possible that long term exposure to short wavelength light may be involved in certain retinal pathologies and aging processes in the lens and retina.

MATERIALS AND METHODS

The action spectrum for minimal photic damage was determined at eight monochromatic wavelengths, extending from 1064 nm to 441.6 nm, by means of CW lasers operating in the TEM_{00} mode. The criterion for minimal photic damage was the appearance of a funduscopically visible lesion at 24 h postexposure for thermal lesions and 48 h post-exposure for photochemical lesions. The entire laser beam, having a divergence of 29 mrad, entered the dilated pupil (>8 mm) of the anesthetized animal by means of a beam splitter so that a fundus camera could be aligned coaxially with the laser beam for viewing and photographing the retina. Maximum retinal irradiance, E_0 in W/cm^2, for a Gaussian distribution was calculated by the formula $E_0 = P_c T/2\pi\delta^2$, where P_c is power at the cornea in W, T is transmittance through the ocular media and δ is calculated from the laser beam profile, $E = E_0 e^{-r^2/2\delta^2}$ where r is the radius in cm. The beam size on the retina was 500 μm in diameter as calculated for the $1/e^2$ points. Power levels at the cornea were measured with a calibrated Scientech calorimeter.

For photopathology, macular and paramacular lesions 1 mm in diameter were produced by a 2500 W xenon lamp equipped with quartz optics and a 6 nm bandwidth interference filter at 441 nm. After enucleation and fixation of the eye, tissue plugs, 2 mm in diameter, containing both the lesion and a surrounding area of normal tissue for control purposes were cut out with a trephine. Following additional treatment and fixation, serial thick sections were cut normal to the retinal layers and transferred to slides, either for toluidine blue staining or for direct viewing by bright field, phase contrast, or Normarski microscopy. Thin sections were also prepared for transmittance electron microscopy on a Hitachi HU-12. Over 3000 serial sections from 20 eyes were evaluated at postexposure times ranging from 1 h to 90 days.

The effects on visual function after radiant exposure of the macula to 30, 60 and 90 J/cm^2 were assessed in terms of the Landolt ring technique (Farrer *et al.*, 1970; Moon *et al.*, 1978). Trained animals were exposed under anesthesia and with pupil dilated to 8 mm or more to the 2500 W xenon lamp with narrow bandpass filtration at 441 nm. The beam was 1 mm in diameter and centered on the fovea centralis retinae, covering most of the macula. Exposures were 1000 s in duration. Thereafter, visual performance was evaluated monocularly in each eye (exposed and control eye) over a period of several weeks and intermittently over a span of months.

The 2500 W xenon lamp with quartz optics was modified by means of an Aerospace Corp. reflectance filter to produce a simulated solar spectrum at sea level extending from 300 to 1400 nm. By means of a combination of 'hot' and 'cold' mirrors and filters this simulated solar spectrum was divided into two bandwidths, 400–800 nm, and 700–1400 nm, so that retinal sensitivity to visible and near infrared radiation could be investigated. Again animals with pupils dilated to greater than 8 mm were exposed under anesthesia to a beam having a divergence of 12 mrad. This produced a beam size on the monkey retina of 159 μm corresponding to the image size of the Sun on the human retina. Although the simulated solar optical system was capable of producing higher power levels at the cornea we limited our exposures to the maximum power level obtained under optimal conditions at sea level while gazing at the Sun with an 8 mm pupil. This was 39.6 mW over the solar bandwidth 300–1400 nm. Power levels greater than this were considered to be of no practical importance for a study of solar retinitis. Power levels were measured with a calibrated Scientech calorimeter.

RESULTS AND DISCUSSION

The sensitivity of the retina to light damage as determined for eight laser lines ranging from 441.6 to 1064 nm (Ham *et al.*, 1976) is summarized in Table 1. One striking feature in these data is the comparison of retinal sensitivity to blue light (441.6 nm) vs infrared (1064 nm). For extended exposures it requires three orders of magnitude greater corneal power, P_c, to produce a minimal lesion with 1064 nm radiation than with blue light (441.6 nm). Even more striking, 1064 nm radiation produces high temperatures and thermal damage in the retina, whereas 441.6 nm light produces negligible temperature rises in the retina at the power levels required to produce a lesion. We therefore ascribe this type of lesion to photochemical processes. Even a 1 s exposure to 441.6 nm light produces a photochemical lesion since the maximum temperature rise is only 2°C above ambient. The shorter the exposure time the lower the radiant exposure, H_0, required to produce the lesion. This may be due in part to thermal enhancement of the photochemical processes.

The two types of lesion not only arise from different basic mechanisms, but also produce entirely different effects in the retina. Minimal thermal lesions are always smaller than the beam diameter on the retina because the center of the image is the hottest place. Very minimal thermal lesions are characteristically 50–100 μm in diameter and are always funduscopically visible 24 h postexposure. Minimal photochemical lesions, on the other hand, are characteristically uniform across the entire beam diameter and require at least 48 h to become funduscopically visible.

The action spectrum for minimal lesions as observed funduscopically at 24–48 h postexposure (see Fig. 1, Ham *et al.*, 1976) does not correspond to the absorption spectrum of rhodopsin or of the other visual pigments, since it continues to rise steeply toward the short wavelengths in the blue-violet end of the visible spectrum. It does seem to correspond roughly with the absorption spectrum of melanin and this correlates with our histological findings (Ham *et al.*, 1978) in that the primary site of the lesion appears to involve the melanosomes in the retinal pigment epithelium (RPE).

In Fig. 1 we have plotted log–log, radiant exposure in J/cm^2 vs exposure time in s for each of the eight wavelengths, as shown in Table 1. The four longer

Table 1. Sensitivity of the retina to photic damage as a function of wavelength λ. T is the transmittance through the monkey ocular media as measured on eight eyes in this laboratory. P_c is power incident on cornea in mW, H_0 the maximum radiant exposure on retina in J/cm^2, E_0 the maximum irradiance on retina in W/cm^2, and K is the maximum temperature above ambient on the retina during irradiation in °C as determined from the mathematical model of Clarke *et al.*, 1969. Exposures were for 1, 16, 100 and 1000 s and the beam size on retina was 500 μm in diameter to the $1/e^2$ points of the Gaussian distribution. Criterion for a minimal lesion was the appearance of a funduscopically visible lesion 48 h postexposure.

Wavelength λ nm	Transmittance T	1 s Exposure				16 s Exposure				100 s Exposure				1000 s Exposure			
		P_c mW	H_0 J/cm^2	E_0 W/cm^2	K °C	P_c mW	H_0 J/cm^2	E_0 W/cm^2	K °C	P_c mW	H_0 J/cm^2	E_0 E/cm^2	K °C	P_c mW	H_0 J/cm^2	E_0 W/cm^2	K °C
441.6	0.45	2.0	0.91	0.91	2°	0.9	6.6	0.41	1°	0.67	20	0.2	0.4°	0.062	30	0.03	0.05°
457.9	0.69	7.3	5.1	5.1	10°	4.6	51.2	3.2	6°	0.74	52	0.52	1°	0.082	60	0.06	0.1°
488	0.83	11.0	9.4	9.4	17°	7.1	97.6	6.1	11°	0.90	77	0.77	1°	0.17	150	0.15	0.1°
514.5	0.87	16.3	14.5	14.5	25°	11.6	165	10.3	18°	2.5	220	2.2	4°	0.36	320	0.32	1°
580	0.91	28.2	26.1	26.1	43°	12.4	184	11.5	19°	8.2	760	7.6	12.5°	4.3	4,000	4.0	6.6°
610	0.92	23.5	22	22	36°	13.3	200	12.5	20°	8.6	810	8.1	13°	6.2	5,800	5.8	9.5°
632.8	0.93	31.5	29.9	29.9	49°	16	243	15.2	25°	8.9	840	8.4	14°	29	5,400	5.4	9.0°
1064	0.76	145	56.1	56.1	55°	97	600	37.5	37°	84	3,250	325	32°	62	24,000	24	23°

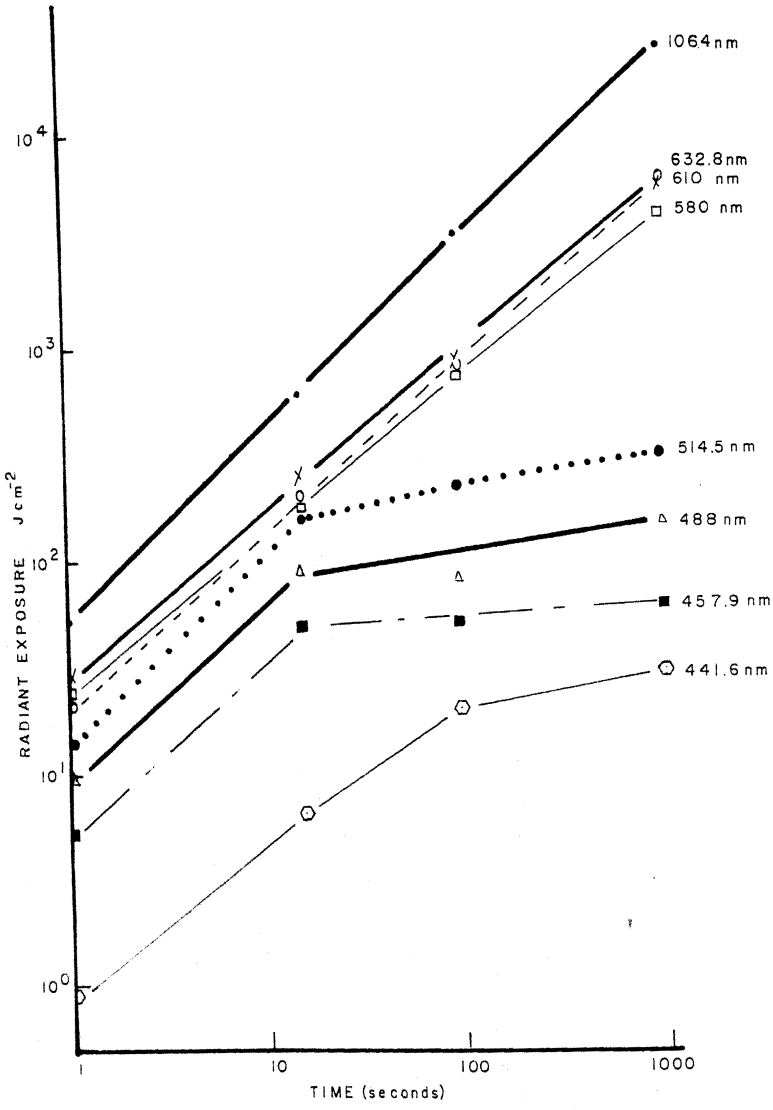

Figure 1. Radiant exposure required to produce a minimal lesion at various exposure times for eight wavelengths. H_0 in J/cm^2 is plotted on the ordinate vs exposure time in s along the abscissa in a log–log plot.

wavelengths, 1064–580 nm, seem to indicate a straight line relationship with approximately the same slope for each wavelength, although there is considerable scatter due to experimental errors and the very real variations in fundus pigmentation from animal to animal. We interpret this to mean that a single mechanism, thermal injury, predominates for wavelengths greater than approximately 580 nm. In the case of the four shorter wavelengths, 514.5–441.6 nm, a single straight line will not fit the data which seem to call for a curve flattening out to zero slope at the longer exposure times. We interpret this as indicating a totally different mechanism of injury.

The tendency for the short wavelengths to flatten out into a plateau at the long exposure times indicates cumulative photochemical damage at low irradiancies and extended exposures. We have collected some data at 10,000 s with 441.6 nm light which support this interpretation. A radiant exposure of 50 J/cm^2 in 10,000 s produced a moderately severe photochemical lesion. We estimate that only 30 J/cm^2 are required to produce a minimal photochemical lesion. Thus, for extended exposures (100–10,000 s) to 441.6 nm light the radiant exposure remains constant at approximately 30 J/cm^2 for minimal photochemical damage. Two repetitive 1000 s radiant exposures of 15 J/cm^2 each, spaced 48 h apart, produced a minimal lesion. However, four repetitive 1000 s exposures to 7.5 J/cm^2 each, spaced 48 h apart, did not produce a lesion. In fact, 10 repetitive exposures of 7.5 J/cm^2, spaced 48 h apart, did not produce a lesion. Apparently repair mechanisms in the retina are able to cope with the damaging effects of short wavelength light for intermittent exposures at low levels of irradiance.

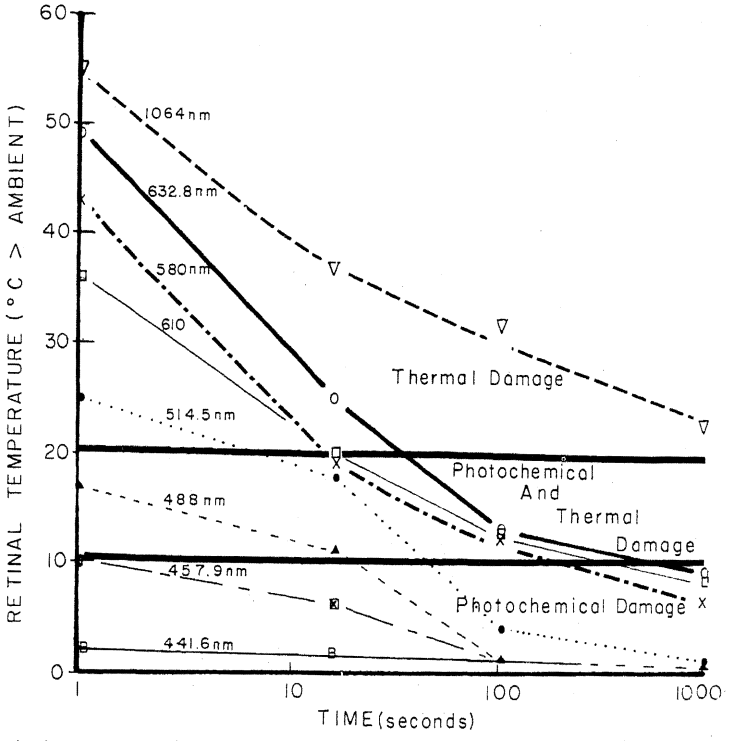

Figure 2. Retinal temperature in °C above ambient, as calculated from model of Clarke *et al* (1969), is plotted against exposure time in s for each of eight laser lines. Lines parallel to the abscissa at 10° and 20°C divide the graph into the three zones, thermal, photochemical and thermal, and photochemical.

Another interpretation of the data in Table 1 is shown in Fig. 2 where we have plotted temperature rise in the retina in °C above ambient against exposure time in seconds for each of the eight wavelengths. It is generally accepted that temperatures below 10°C do not produce permanent denaturation of biological macromolecules, while temperatures above 20°C produce irreparable thermal damage. Accordingly, we have assumed that retinal lesions produced at temperatures below 10°C are predominantly photochemical and lesions produced at temperatures above 20°C are primarily retinal burns. The intermediate region, 10–20°C, represents a combination of thermal and photochemical damage which is difficult to define. For 1 s exposures, wavelengths 514.5–1064 nm produce preponderantly retinal burns, whereas the wavelengths 488 nm and 458 nm produce temperatures in the intermediate region where both thermal and photochemical effects exist. However, the 441.6 nm wavelength remains well inside the photochemical region, even for 1 s exposures, while the infrared line at 1064 nm remains well within the thermal zone. It may reasonably be asked why the temperature required to produce a lesion decreases (55–25°C) with decrease in wavelength (1064–514.5 nm) for 1 s exposures. The logical conclusion is that even for 1 s exposures at the longer wavelengths there must be photochemical components in the damaging mechanisms which become more and more prominent as the wavelength approaches the shorter end of the visible spectrum. The longer the exposure time the more prominent the photochemical component becomes in determining the final nature of the lesion. Superimposed on this photochemical component is the time–temperature history of thermal damage. For example, taking the curve for the 1064 nm wavelength, 55°C for 1 s may be equivalent to 23°C for 1000 s as far as thermal damage is concerned.

The histopathology of the photochemical lesion produced by blue light (441.6 nm) has been submitted for publication elsewhere (Ham *et al.*, 1978). Here, we shall limit ourselves to a brief discussion of the salient features of the photopathology of the paramacula area as observed by light microscopy at intervals ranging from 1 h to 90 days. There are no detectable changes in the neural retina, RPE and choroid at 1 h postexposure except for a few rod nuclei with clumped chromatin and a few dense ellipsoids in the cone cells. The same holds true at one day postexposure but at two days postexposure the RPE displays an inflammatory reaction throughout the exposed area. In some lesions the choroid also appears inflamed. Macrophages, presumably from the reticulo-endothelial system, invade the RPE via the choriocapillaris and phagocytize the melanosomes, thereby producing hypopigmentation of the RPE. It is this hypopigmentation that becomes funduscopically visible as a lesion at 48 h postexposure. The outer segments (OS) of the photoreceptor cells appear undamaged at this stage of the photopathology, so

that the initial lesion appears to be located predominantly in the RPE and sometimes also in the choroid.

The OS of the rods and cones first show damage and disorientation at 5 and 6 days postexposure when macrophages are found in the subretinal space along with disruption, proliferation and hypopigmentation of the RPE.

Ten and 11 days postexposure, the RPE shows remarkable reparation. Cell proliferation has ceased and the cells are well aligned in a single layer. Hypopigmentation remains with macrophages in the subretinal space. The OS of rods and cones are less disoriented and appear almost normal except for some shortening in length. At 30 days postexposure both neural retina and RPE appear normal except for hypopigmentation and the continued presence of macrophages in the subretinal space. These macrophages have disappeared from the subretinal space at 60 days postexposure. The lesion is less hypopigmented and is still faintly visible funduscopically. By 90 days, hypopigmentation is morphologically subtle and the lesion is very faintly visible in the funduscope.

Macular lesions are similar to those in the paramacula except that it requires nearly twice the radiant exposure to produce a lesion because of the yellow macular pigment which strongly attenuates blue light. Typically, 30–35 J/cm^2 produces a mild lesion in the paramacular but none in the macula. It requires 50–60 J/cm^2 to produce a mild macular lesion.

We have investigated the relationship between the developing histopathology of the photochemical lesion and visual performance in trained monkeys who were exposed for 1000 s to 441.6 nm light at radiant exposure levels which ranged from 30 to 90 J/cm^2. The reader is referred to Moon *et al.*, 1978, for details regarding the visual acuity tests employing the Landolt ring technique. Animals exposed in the macular area to 30 J/cm^2 showed no loss in 20/20 or 20/30 monocular visual acuity. However, animals exposed to 60 J/cm^2 did show a sharp loss in 20/20 performance on the fifth day postexposure, followed a day or two later by a similar drop in 20/30 performance. These sharp declines in visual performance coincide with our first observations of damage to the OS of the photoreceptor cells at 5–6 days postexposure. Also, the finding that 30 J/cm^2 failed to impair visual performance accords with our observations that 30 J/cm^2 does not produce a funduscopically visible lesion in the macula, presumably because the yellow macular pigment attenuates blue light. The animals exposed to 60 J/cm^2 recovered 20/30 visual performance at about 20 days postexposure and 20/20 vision in 30 days after exposure. This return to normal visual performance in 20–30 days supports our observations of recovery of both photoreceptor cells and RPE at 30 days postexposure. Thus, both visual acuity tests and histopathology support the hypothesis that the primary lesion from blue light is in the RPE and not in the photoreceptor cells. It seems logical to assume that primary damage to the RPE impairs its supportive role in maintaining the photoreceptors, leading after a few days to deterioration of the OS. If the damage is not too severe, both photoreceptors and RPE recover their normal functions except that the RPE is slightly hypopigmented. However, an animal exposed to 90 J/cm^2 has not recovered 20/20 vision even a year after exposure. We assume therefore that macular exposures somewhere between 60 and 90 J/cm^2 produce irreparable damage to the visual processes in the retina.

In 1973 we pointed out (Ham *et al.*, 1973) that eclipse blindness and solar retinitis could not be explained wholly in terms of thermal damage because the maximum temperature on the retina during solar or eclipse gazing did not exceed 3°C above ambient. It now seems abundantly clear that photochemical processes induced in the retina by short wavelength light must play a major role in solar retinitis and eclipse blindness. To further elucidate the role of thermal and photochemical processes in the retina as a function of wavelength, we have divided the simulated solar spectrum (SSS) at sea level, 300–1400 nm, into two broadband components, 400–800 nm and 700–1400 nm; we have exposed a group of eight monkeys to each of these three spectra.

The results of exposure to the SSS at sea level (300–1400 nm) for time intervals of 1, 10, 100 and 1000 s are shown in Table 2. Calculations demonstrate that in humans a pupillary diameter of 7 mm is required to obtain a retinal exposure of 109 J/cm^2 in 1 s, and 5.6 mm for 633 J/cm^2 in 10 s. Pupillary dilations of these magnitudes occur only under abnormal conditions, i.e. drugs or prolonged dark adaptation. However, a pupillary diameter between 1.5

Table 2. Exposure of the rhesus monkey retina to a SSS (300–1400 nm) for time intervals of 1, 10, 100, 1000 s. Criterion was a funduscopically minimally visible lesion at 48 h postexposure. Beam diameter on retina, 159 μm. Power, P_c, incident on cornea in mW, retinal radiant exposure, H_0, in J/cm^2. Retinal temperature in °C above ambient estimated from model of White *et al.*, 1971. Transmittance through monkey ocular media, 0.702.

Exposure time (s)	P_c (mW)	H_0 (J/cm^2)	Max. retinal tem. (°C)
1	30.5 ± 2.6	109	16.4
10	17.7 ± 1.8	633	9.5
100	2.0 ± 0.17	710	1.1
1000	0.23 +0.04	800	0.1

Table 3. Exposure of the rhesus monkey retina to the visible component of the SSS at sea level, 400–800 nm, for time intervals of 1, 10, 100, 500 s. Biological criterion was a minimal retinal lesion as funduscopically detectable at 48 h postexposure. Beam diameter on retina, 159 μm. Power, P_c, incident on cornea in mW, retinal radiant exposure, H_0, in J/cm^2. Retinal temperature in °C above ambient estimated from model of White *et al.*, 1971. Transmittance through monkey ocular media, 0.794.

Exposure time (s)	P_c (mW)	H_0 (J/cm^2)	Max. retinal temp. (°C)
1	17.7 ± 0.96	72	10.6
10	11.2 ± 0.89	453	6.7
100	0.92 ± 0.04	370	0.55
500	0.18 ± 0.03	365	0.11

and 2.0 mm would allow an exposure of 710 J/cm^2 for a 100 s continuous exposure. Thus, Sun gazing for 100 s would produce a photochemical lesion in the monkey retina. Obviously, exposure for 1000 s would produce a more severe lesion in spite of minimal pupillary diameter. The retinal temperatures involved in these longer exposures are too low to produce thermal damage, so that we are forced to postulate some type or types of photochemical damage, aided perhaps by thermal enhancement.

The phenomena underlying solar retinitis become even more apparent when we examine the results of exposing the monkey retina to only the visible wavelength components in the solar spectrum, 400–800 nm, as shown in Table 3. Here, exposure times were not extended beyond 500 s because lesions well above the minimal level resulted even for pupillary constrictions of 1 mm or less. A comparison of the corneal power, P_c, required to produce a minimal lesion for the two spectra, 300–1400 nm and 400–800 nm, deemonstrates that there is a drastic reduction in P_c (almost 50%) for the 400–800 nm spectrum. We attribute this to the fact that 49% of the power in the 300–1400 nm spectrum is composed of wavelengths beyond 700 nm and that these wavelengths in the infrared play a very minor role in the production of retinal lesions. Again, we are forced to conclude that the visible spectrum from 400–800 nm plays a predominate role in the mechanisms responsible for retinal damage. Since the retinal temperatures for exposures of 100 s or greater are negligible from the standpoint of thermal damage, the lesion must be produced primarily by photochemical processes.

Finally, exposure of the retina to wavelengths in the SSS beyond 700 nm conclusively demonstrates that the infrared component in the solar spectrum plays a very minor role in solar retinitis. Calculations show that the power entering the eye through an 8 mm pupil at sea level for that part of the solar spectrum between 700–1400 nm is 19.4 mW. Although the optical system used to simulate the solar spectrum at sea level was capable of delivering much greater powers to the eye than 19.4 mW, we limited our exposures to this figure, since greater powers would have no significance for solar retinitis. Exposure durations of 1, 100 and 500 s were completely negative, i.e. did not produce a retinal lesion. Exposures of 1000 s at a power level of 19.4 mW produced a minimal lesion in three out of eight animals. The average value required to produce a minimal lesion in eight animals was 19.8 ± 0.4 mW. This corresponds to a retinal irradiance of 69.1 W/cm^2 or a radiant exposure of 69,100 J/cm^2. The temperature in the retina during exposure is estimated to be 10.3°C above ambient. Thus, it seems quite clear that gazing at the Sun under worst case conditions through a filter which eliminated the visible spectrum (400–700 nm) would not produce a lesion, even for long exposure times unless the pupil was dilated to 8 mm or more.

The results of these experiments are summarized in Fig. 3 which is a log-log plot of retinal irradiance in W/cm^2 along the ordinate vs exposure time in s along the abscissa for the three broadband solar spectra, 300–1400 nm, 400–800 nm and 700–1400 nm. Each point on the graph represents the retinal irradiance required to produce a minimal lesion for a given exposure time, the criterion being the appearance of a funduscopically visible lesion at 48 h postexposure. For the 700–1400 nm spectrum we have included some data published in Ham *et al.*, 1973. These are the points at 10, 30, 60 and 180 s. The final point at 1000 s represents our most recent data on eight animals. The straight line relationship for all the data points on the 700–1400 nm line and those for 1 and 10 s exposures on the other two spectra suggest that a single mechanism, thermal injury, is predominant for short exposure times and for long wavelengths. The slope of the 700–1400 nm line is roughly equivalent to that of the other two spectra at short exposure times. The longer exposures for 300–1400 nm and 400–800 nm have a different slope which we interpret as indicating a different mechanism, namely photochemical processes. The higher irradiancies for the 300–1400 nm spectrum as compared to the 400–800 nm spectrum are due almost entirely to the infrared component in the former. Approximately 49% of the energy in the 300–1400 nm spectrum is at wavelengths beyond 700 nm. This infrared component makes an almost negligible contribution to the production of lesions as compared to the visible component. Even at short exposure times

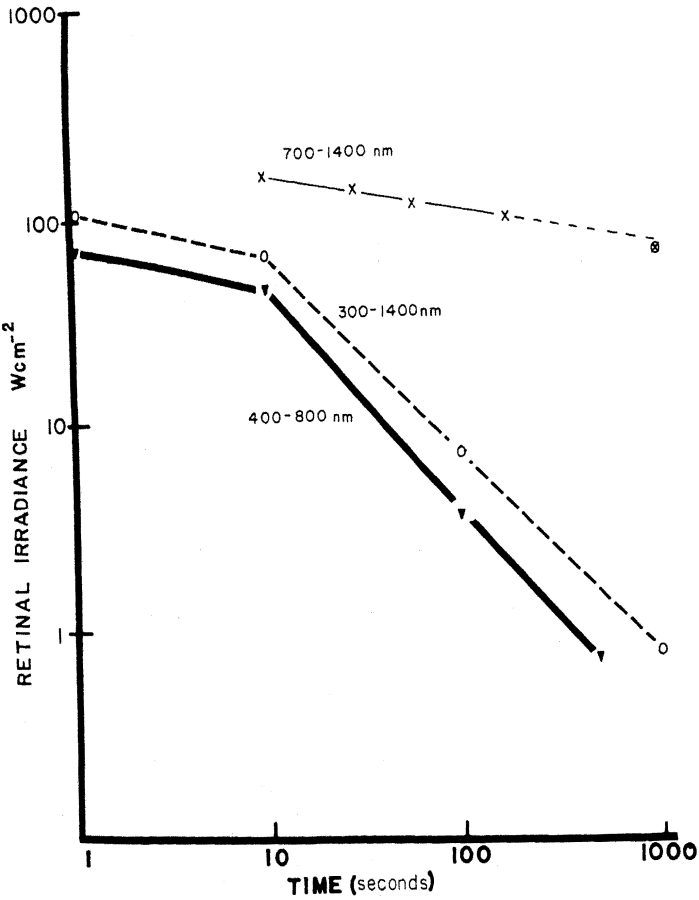

Figure 3. Retinal irradiance in W/cm^2 is plotted along the ordinate vs exposure time in s along the abscissa in a log-log plot. Points on the graphs represent the retinal irradiance required to produce a minimal lesion with each broadband spectrum for a given exposure time.

where the lesion is predominately thermal in character the white light component far outweighs the infrared in the ability to produce a lesion. This is partly because melanin absorbs more energy in the visible region than in the infrared region of the spectrum and partly because photochemical processes play a role in producing the lesion.

Occasional mitotic figures have been found in both the RPE and the choroid of the rhesus monkey after radiant exposures of 30–35 J/cm^2 of 441 nm light. This finding, in addition to proliferation of cells in the RPE and the residual hypopigmentation after the lesion has healed suggest that long term exposure to blue light may be a causative agent in certain retinal pathologies and degenerative diseases. Until more data are available it would seem wise to shield the eyes of aphakics and possibly retinitis pigmentosa patients from the blue end of the spectrum. This is easily accomplished by adding a yellow filter as a protective device to eye wear. Such a filter also protects the retinae of aphakics from near UV light.

SUMMARY

From these data we conclude the following:

1. That the long wavelengths in the visible spectrum (>580 nm) and near infrared radiation (700–1400 nm) produce predominantly thermal damage to the retina while short wavelengths in the visible spectrum (400–580 nm) produce predominantly photochemical damage. Transition from one type of injury to the other is gradual, not abrupt.

2. That power level and exposure time are important parameters in determining which type of damage prevails.

3. That the two types of retinal lesions differ not only in the basic mechanisms producing them but also lead to entirely different biological effects which are distinguishable both histologically and funduscopically.

4. That moderate photochemical lesions are reparable with time, whereas moderate burn lesions produce permanent damage.

5. That both histopathology and tests of visual function suggest that the melanosomes of the retinal pigment epithelium (RPE) are the initial site of photochemical damage and that only after the supportive functions of the RPE have been impaired do the photoreceptor cells begin to show damage.

6. That solar retinitis and eclipse blindness are primarily photochemical events produced by the short wavelength visible component of the solar spectrum and that the infrared component of the solar spectrum plays only a minor role in these photoretinopathies.

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