ULTRAVIOLET RADIATION: THE EYE

Jean-Pierre Cesarini, M.D.[1] and David H. Sliney, Ph.D.[2]

[1]Laboratoire de Recherche sur les Tumeurs de la Peau Humaine, INSERM, Fondation Ophtalmologique Adolpe de Rothschild, 25, rue Manin, F-75940 Paris CEDEX 19, France
[2]Laser/Optical Radiation Program, US Army Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, MD 21010-5422 USA

INTRODUCTION

Under most conditions, the eye is well adapted to protect itself against ultraviolet radiation encountered in the outdoor environment as a result of the exposure geometry of the sun. Only when snow is on the ground does one experience acute effects of UV sunlight exposure (i.e., "snow blindness," or photokeratitis).[1-6]

With regard to artificial sources, there are many occasions where one views bright light sources such as tungsten-halogen lamps, arc lamps and welding arcs. Such viewing is normally only momentary because of the aversion response to bright light and due to discomfort glare. However, such an aversion does not take place for germicidal lamps and other UV lamps which do not contain a strong visible component in their spectrum. The adverse effects from viewing such sources has been studied for decades and during the last two decades guidelines for limiting exposure to protect the eye have been developed. The guidelines were fostered to a large extent by the growing use of lasers and the quickly recognized hazard posed by viewing laser sources.[3,7-12]

BIOLOGICAL EFFECTS

For each adverse effect upon the eye in the UV spectral region, the dominant injury mechanism is initially a photochemical reaction in the exposed tissue. The relative spectral risk for each type of injury is determined by a photobiological *action spectrum*. As with any photochemical reaction, the action spectrum describes the relative effectiveness of different wavelengths in causing a photobiological effect. The action spectra for UV photokeratitis (or "welder's flash") for humans as well as animals,[13-14] for photoretinitis[15-17] and acute cataractogenesis[18-19] have been published. Epidemiological studies of UV effects upon the eye support laboratory findings that acute effects are largely the result of UV-B radiation exposure.[3] The product of the dose-rate and the exposure duration always must result in the same exposure dose (in J/cm^2) to produce a threshold injury. This characteristic of photochemical injury mechanisms is termed the *Bunsen-Roscoe Law*, or the rule of reciprocity (of exposure duration and irradiance), and helps to distinguish these effects from thermal burns.

There are at least four separate types of hazards to the eye from UVR exposure, and protective measures must be chosen with an understanding of each:

1. *Photokeratoconjunctivitis* (acute inflammation of the cornea and conjunctiva as in "welders' flash") has been defined for wavelengths from 200 nm to 400 nm and cataractogenesis (lens cataract) has been demonstrated principally in the wavelength range from 290 to 320 nm, and perhaps occurs at greater wavelengths. The clear relationship between UV-B exposure and the onset of symptoms a few hours later conclusively prove that UVR causes this acute ocular effect. The signs and symptoms seldom last for more than a day or two.[13-14]

NOTE: The opinions or assertions herein are those of the author and should not be construed as reflecting official positions of the Department of the Army or Department of Defense.

2. *Pterygium and droplet keratopathies*, as age related pathologies of the cornea, which are common in island environments rich in UVR exposure have frequently been linked to chronic, life-long UVR exposure.[20] There are clearly other potential factors.

3. *Cataractogenesis*. There has long been a suggestion that UVR causes cataracts. The earlier age of onset of cataract in equatorial zones has led to a number of theories to explain this latitudinal dependence. Although some studies conclude (and it has long been argued) that exposure of the human eye to UVR plays an etiologic role in the development of some corneal pathologies,[17-20] this role in cataractogenesis has been questioned by others.[20-27] Even more under debate are theories that suggest that UVR and light may affect retinal disease.[22,26-27] However, epidemiologic studies which carefully consider individual exposure and ocular dosimetry show a clear relationship between life-long UV-B exposure and cataract.[23]

4. *Photoretinitis*. The principal retinal hazard resulting from viewing bright visible light sources is photoretinitis, e.g., *solar retinitis* with an accompanying scotoma ("blind spot"), which can result when one stares at the sun for several minutes. Solar retinitis was once referred to as "eclipse blindness" with an associated "retinal burn." Only in recent years has it become clear that photoretinitis results from a *photochemical* injury mechanism following exposure of the retina to shorter wavelengths in the visible spectrum, i.e., from violet and blue light. Prior to conclusive animal experiments at that time (1976),[3] photoretinitis was thought to result from a thermal injury mechanism. However, it has been shown conclusively that an intense exposure to short-wavelength light (hereafter referred to as "blue light") can cause retinal injury. The action spectrum has been defined principally from 400 nm to 550 nm in the visible; but, wavelengths from 310 nm to 400 nm in the UV can also produce effects in aphakic eyes (i.e., eyes with lens surgically removed), and possibly even somewhat in normal eyes. Normally, UV-A radiant energy is <u>not</u> implicated in this type of retinal injury unless the normal (UV-absorbing) crystallin lens of the eye has been surgically removed, as during cataract surgery. Accelerated ageing of the macula (age-related macular [central retinal] degeneration) may also be related to chronic retinal exposure to UV-A and short-wavelength visible light.[15]

Blue-light retinal injury (photoretinitis) can result from viewing either an extremely bright light source (e.g., the sun) for a short time, or a bright light of lesser brightness (e.g, a tungsten-halogen filament) for longer exposure periods.[3] Figure 1 summarizes the aforementioned biological effects.

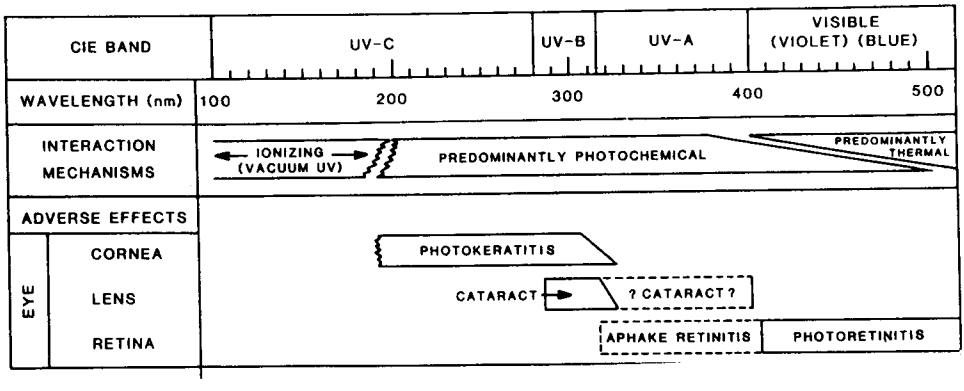

Figure 1. Adverse Biological Effects Attributed to Ultraviolet Radiation and Short-Wavelength Light.

GEOMETRY OF EXPOSURE

Exposures of the anterior structures of the human eye to UVR depend upon the relative position of the light source and the degree of lid closure. In bright sunlight, the orbital ridge above the eye and the upper lid considerably shade the cornea from excessive UVR exposure, thus limiting the chance for UVR photokeratitis in bright sunlight, except when viewing highly reflective snow.[3-6] Geometrical factors dominate the determination of ultraviolet radiation (UVR) exposure of the eye. The degree of lid opening limits ocular exposure to rays entering at angles near the horizon. Clouds redistribute overhead UVR to the horizon sky. Mountains, trees and building shield the eye from direct sky exposure. Most ground surfaces reflect little UVR. The result is that highest UVR exposure occurs during light overcast where the horizon is visible and ground surface reflection is high. By contrast, exposure in a high mountain valley with green foliage results in a much lower ocular dose. Other findings of these studies show that retinal exposure to light and UVR in daylight occurs largely in the superior retina.[6]

Despite a substantial literature on the adverse effects of ultraviolet radiation (UVR) and intense visible light upon ocular structures--particularly upon the retina--controversy still surrounds the question of whether natural and man-made light sources are really hazardous under normal viewing conditions. Acute studies of UVR and light injury of the cornea, lens and retina of experimental animals have generally employed excessive light levels. Although scientific evidence accumulates to indicate that chronic exposure conditions may accelerate ageing processes in the retina, the quantitative question of: What is safe?" remains to be conclusively answered. In recent years the potentially adverse effect of UVR exposure of the eye have been the subject of numerous scientific reviews and meetings. Present scientific knowledge in this area impacts upon the determination of optical product safety, ophthalmic instrument safety, and the design of sunglasses, and ophthalmic lenses. The one apparent controversy with respect to thresholds of UV photokeratitis and the ACGIH EL for UV-B can be seen at 310 nm in Figure 2. The reported threshold may be higher, since the bandwidth of the xenon-arc monochromator exposure system was at least 5 nm, and the effective wavelength would be less than 310 nm along such a steep slope.

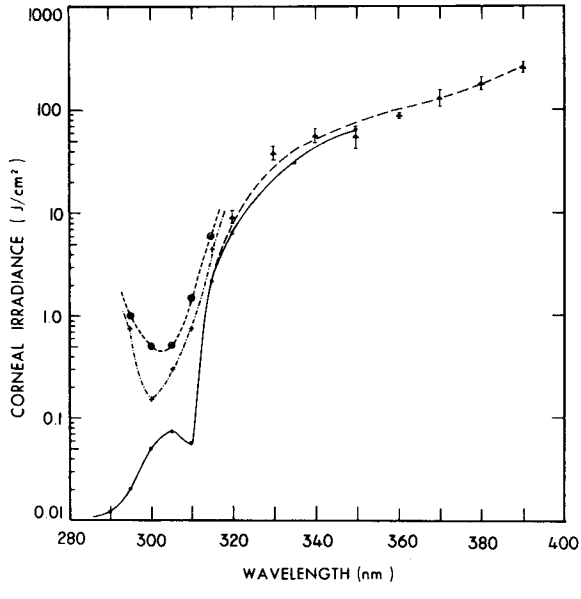

Figure 2. The thresholds (inverted action spectra) for photokeratitis[13-14] and cataractogenesis[18] are shown as a function of wavelength. Note the logarithmic ordinate.

Ultraviolet radiant energy at wavelengths shorter than 280 nm (referred to as UV-C) is totally absorbed in the cornea and is totally imperceptible. Wavelengths in the UV-B (280 - 315 nm) are absorbed largely in the cornea, but also reach the crystallin lens, and at wavelengths greater than 300 nm, there is a very small fraction of energy (of the order of 1 % or less) that may reach the retina.[3,15-17] Higher UV-A transmittance of the lens is most notable during youth.[3,20] Wavelengths in the UV-A (315 - 400 nm) spectral band are strongly absorbed in the lens; much of this energy is re-emitted as fluorescence in the visible spectrum.[28] In the absence of visible light, UV-A radiant energy may be perceived by fluorescence of the lens and some visual sensitivity to wavelengths below 400 nm which reach the retina. This general perception of a UV-A source can be described as a fuzzy, somewhat discomforting-to-view, blue-grayish light.[28] UV-A emitting lamps (frequently termed "black lights") are used extensively in industry for non-destructive testing by fluorescence techniques, and in entertainment and novelty applications to illuminate fluorescent signs, posters, in discos for special effects, in mineralogy to classify certain minerals, etc. In these applications, the eye is normally exposed to UV-A irradiances less than 1 mW/cm^2.[3] Although the UV-A at ground level from sunlight is 1 - 5 mW/cm^2, the actual UV ocular exposure to ground reflections and skylight is normally about 1 mW/cm^2 or less.[21]

THE EYE'S EXPOSURE OUTDOORS

Our exposure to UVR and light outdoors constantly changes during the day. We are largely unaware of the degree of these changes--especially the change in exposure rate of UV-B (the shortest wavelengths in terrestrial sunlight). This spectral change is not apparent in the visible part of the sun's spectrum until nearly sunset; and an attempt to take a color photograph either very early or very late in the day will result in a picture that is yellowish or orange in hue. We are fortunate that the Rayleigh scattering of sunlight by air molecules favors UVR and blue light (hence the blue sky). For longer pathlengths through the atmosphere when the sun is low in the sky, much more UVR and sunlight is scattered and the sun which is white at noonday becomes yellow and then orange as less UVR and blue light are present in the direct rays. When the sun is overhead and "white," it would take only 90 seconds to stare at the sun and receive a blue-light retinal burn. A few hours later, it would take several minutes, and it is virtually impossible at sunset. Thus, the geometry of exposure as well as the spectrum (hue) plays a major role in determining the hazards of viewing the sun. Fortunately, we seldom look directly overhead when the sun is very hazardous to view, and the sun is not very hazardous to view when the sun is sufficiently low in the sky to fall within our normal field-of-view. Furthermore, when the sun is greater than about 10° above the horizon, we squint, thus shielding the retina from direct exposure. Figure 3 illustrates the limited angular exposure.

Although the cornea is more sensitive to UVR injury than the skin, we seldom experience a corneal burn when out in sunlight. Again, the geometry of exposure helps. When the sun is overhead and UVR exposure is most severe, the brow ridge and upper lid shield the cornea, and if the eye is turned away from the sun, the more intense scattered UVR from overhead strikes the cornea at a grazing incidence where most is reflected and little is absorbed. Only when the incident UV rays are parallel to the pupillary axis are most rays absorbed (in fact, 98 % are absorbed). When one looks down at the snow the UVR is reflected directly into the eye, hence the eskimos' traditional eye protector, the slit in whalebone, provided geometrical rather than spectral protection against the UVR exposure. The strong dependence of reflections with angle of incidence is termed Fresnel's Law of Reflection. This Law not only explains the survivability of the cornea in an overhead bath of UVR, but also the glare experienced over water. When the sun is overhead, a body of water reflects the UVR upward, but only approximately 2% is reflected. When the sun is low in the sky, much of the incident light is reflected, but now the UVR and blue light have been filtered out of the direct rays by the atmosphere and are therefore harmless. Nevertheless, discomfort glare originates from the strong water reflections which because of polarization by reflection and scattering can be selectively filtered by polarizing lenses. Such lenses may not necessarily filter out the UVR.

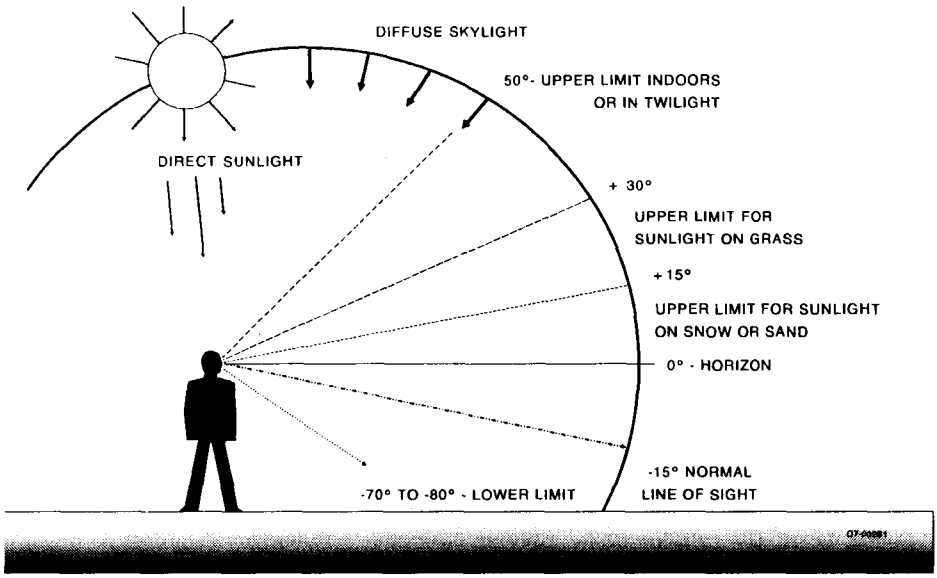

Figure 3. The limited solid angular exposure to UVR in the outdoor environment is limited by the geometry of the brow-ridge, but also strongly affected by lid position (squinting), which depends on the brightness of the visible light environment.[4-6]

UVR EXPOSURE FROM ARTIFICIAL SOURCES

The envelopes of most commercial lamps used for illumination filter out UV-B or are installed in a filtering lighting fixture. However, in some industrial processes, workers may actually be exposed to levels greater than one would experience in the outdoor environment. In this figure, the relative risk of fixating upon different types of bare lamps after the spectral distribution has been weighted by biological action spectra for uv photokeratitis and the retinal "blue-light" hazard. Since the ambient optical radiation levels in our natural environment are barely tolerated, it is hard to imagine that an exposure to an artificial light source would be tolerated at an exposure level exceeding the outdoor levels. Aside from pulsed lasers, where the individual may have no natural protective mechanisms such as the blink reflex of the eye, I have not encountered an instance where an individual repeatedly exposed himself or herself to levels where acute injury took place.

PROTECTIVE MEASURES

Where feasible, it would appear to be desirable to provide eye protection against an hypothesized chronic-exposure hazard simply to be on the safe side. If eye protection is worn, peripheral protection is paramount because of the *Coroneo Effect.*[6] However, when protective measures are extremely costly, or they possibly introduce another hazard, one has a serious dilemma. Such a dilemma has apparently arisen with regard to ultraviolet absorbing intraocular lenses.[17]

CONCLUSIONS

The present ICNIRP EL's and ACGIH threshold limit values (TLV's) for UVR are based upon an understanding of both acute and chronic effects upon the eye. Although exposures to optical radiation in the physiological range have generally been accepted as safe, greater knowledge today suggests that the absence of acute effects does not mean that some damage has not occurred at the

molecular level. Delayed effects appear to be possible from UVR exposure to the eye as well as the skin. Furthermore, growing evidence suggests that retinal degradation may also occur from chronic exposure to short wavelength light, and possibly from the trace amount of UV-A that reaches the retina as well. The present exposure limits probably reduce the risk from all of these delayed effects quite significantly, but one cannot argue that the risk is zero. As with almost all physical and chemical agents, one cannot speak of "zero risk" -- it is scientifically specious.

REFERENCES

1. WHO, Environmental Health Criteria No. 160, Ultraviolet Radiation, joint publication of the United Nations Environmental Program, the International Radiation Protection Association and the World Health Organization, Geneva (1994)
2. Rosenthal, F.S., Phoon C., Balkalian A., and Taylor, HR., The ocular dose of ultraviolet radiation to outdoor workers, Invest Ophthalmol Vis Sci, 29:649-656, 1988.
3. Sliney DH, Wolbarsht ML *Safety with Lasers and Other Optical Sources*, Plenum Publishing Corp., New York (1980).
4. Sliney DH (1983) Eye protective techniques for bright light, Ophthalmology, 90:937-944.
5. Sliney, D.H., Epidemiological studies of sunlight and cataract: the critical factor of ultraviolet exposure geometry, Ophthalmic Epidemiology, **1**(2):107-119, 1994.
6. Sliney, D. H. UV radiation ocular exposure dosimetry, J Photochem Photobiol B: Biology, 31(11):69-77 (1995).
7. ACGIH, *TLV's, Threshold Limit Values and Biological Exposure Indices for 1995-1996*, American Conference of Governmental Industrial Hygienists, Cincinnati, (1995).
8. ACGIH, Documentation for the Threshold Limit Values, 5th Edn., American Conference of Governmental Industrial Hygienists, Cincinnati (1992).
9. International Non-Ionizing Radiation Committee of the International Radiation Protection Association, "Guidelines on the limits of exposure to ultraviolet radiation of wavelengths between 180 nm and 400 nm (incoherent optical radiation)," Health Physics, 49, 331-40 (1985) [with amendment, Health Physics, 56, 971-2 (1989)].
10. A. S. Duchene, J. R. A. Lakey, and M. H. Repacholi, (Eds.) (1991), IRPA Guidelines on Protection Against Non-Ionizing Radiation, New York, MacMillan.
11. Sliney DH (1972) The merits of an envelope action spectrum for ultraviolet radiation exposure criteria, *Amer Industr Hyg Assn J* 33(9):644-653 (1972).
12. Health Council of the Netherlands, *Acceptable Levels for Micrometer Radiation*, Gezondheidsraad, Rijswijk (1979).
13. D. G. Pitts, "The ocular ultraviolet action spectrum and protection criteria," Health Physics, 25, 559-566, (1973).
14. J. A. Zuclich, "Ultraviolet-induced photochemical damage in ocular tissues," Health Phys, 56(5):671-682, (1989).
15. Ham WT, Jr, Mueller HA, Ruffolo JJ, Jr, Guerry D, III Guerry RK (1982) Action spectrum for retinal injury from near-ultraviolet radiation in the aphakic monkey, Am J Ophthalmol 93(3): 299-306.
16. Marshall J (1986) Light damage and the practice of ophthalmology. In: Rosen E Arnott E and Haining W (eds.). Intraocular Lens Implantation, Moseby-Yearbook, Ltd., London.
17. Mainster MA (1978) Spectral transmission of intraocular lenses and retinal damage from intense light sources, Am J Ophthalmol 85:167-170.
18. Pitts DG, Cullen AP, Hacker PD (1977) Ocular effects of ultraviolet radiation from 295 to 365 nm, Invest Ophthal Vis Sci 16(10):932-939.
19. Jose JG & Pitts DG (1985) Wavelength dependency of cataracts in albino mice following chronic exposure. Exp Eye Res, **41**:545-563.
20. World Health Organization, "The Effects of Solar UV Radiation on the Eye, Report of an Informal Consulltation, Geneva 30 August - September 1993," Publication WHO/PBL/EHG/94.1, Program for the Prevention of Blindness Program, World Health Organization, Geneva, 1995.

21. Sliney DH (1986) Physical factors in cataractogenesis: Ambient ultraviolet radiation and temperature, Invest Ophthalmol Vis Sci 27(5): 781-790.
22. Young, R. W., Age Related Cataract, Oxford, Oxford University Press, 1991.
23. H. R. Taylor, West, S. K., F. S. Rosenthal, B. Munoz, H. S. Newland, H. Abbey, and S. K. Emmett, "Effect of ultraviolet radiation on cataract formation," New Engl. J Med., 319, 1429-1433 (1989).
24. Weale, R. "Senile cataract, the case against light." Ophthalmology **90**:420- 423, 1983; see also: Brit J Ophthalmol **66**(1):31-34, 1982.
25. Harding, J.J. "The untenability of the sunlight hypothesis of cataractogenesis," Doc. Ophthalmologica, **88**:345-349, 1995.
26. Varma SD, Lerman S (eds.) (1984) Proceedings of the First International Symposium on Light & Oxygen Effects on the Eye, IRL Press, Oxford [also published as Current Eye Res 3(1)].
27. Waxler M, Hitchens V (eds) (1986) Optical Radiation and Visual Health, CRC Press, Boca Raton.
28. E. Wolf, "Effects on visual thresholds of exposure to radiation below 4000 angstroms," Trans. Am Acad. Ophthalmol, 53, 400-414 (1949).