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The College of Optometrists

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Radiation effects on the eye

Part 3b - Ocular effects of ultraviolet radiation

The electromagnetic spectrum is measured and expressed in radiometric units, since photometric units can only be used for visible radiation. In biological research, the power of a radiation source is called its irradiance, and is measured in watts per square centimetre (W/cm^2). The exposure is some duration of time, in seconds, and hence the biological system receives a radiant exposure measured in joules per square centimetre (J/cm^2).

Unfortunately a simple, reliable instrument is not available to measure the amount of UV radiation in the environment and indicate the extent and type of protection required. The measurement of UV requires expensive instrumentation, or estimates based on published data giving the power per unit area (W/cm^2) for various sources at the relevant wavelengths.

Protection standards indicate that between 315nm and 400nm, that is UVA, the irradiance incident on the eye should not exceed $1mW/cm^2$ for periods greater than 103 seconds (16 minutes). For exposure times less than 103 seconds, the total radiant exposure should not exceed $1J/cm^2$ in total. These values for exposure of the eye apply to UV radiation from arcs, gas and vapour discharges and incandescent sources, but not UV lasers or solar radiation.

The biological effects of UVA are far less serious than those of UVB and UVC. The potential ocular hazards from acute exposure to the 315nm to 400nm range are well known. Studies show that the thresholds for acute photokeratitis (from wavelengths below 400nm) and cataract (from wavelengths below 325nm) rise significantly with increasing wavelength, from approximately $10J/cm^2$ at 320nm to over $250J/cm^2$ at 390nm.

The typical UV dose experienced on a sunny day while sailing or skiing can be enough to cause keratitis, for which the threshold for 300nm UVB is 0.03-0.08J/cm2. It is possible to work out safe exposure times by a simple equation - $t = Q/E$ - where Q is the radiant exposure threshold for damage at the particular wavelength in J/cm^2 and E is the total UV radiance of the source in W/cm^2. This formula can also be used to find the appropriate optical filter to protect the eye when exposures exceed the safe time t.

One of the most important studies on the threshold value sufficient to produce damage to the eye was conducted by Pitts (1973). He used 40 human volunteers for whom extensive measures of visual function were made before and after exposures to UV radiation. Data were taken on visual acuity and corneal light scatter, together with extensive slit lamp examination. A reduction in visual acuity was seen within two hours of exposure for some subjects, while for others the reduction took seven hours. Visual acuity was typically reduced by two Snellen lines for most subjects. Frequent symptoms reported were foreign body sensation and tearing, although only in a few were the symptoms severe enough to interfere with normal activities. Photophobia and general visual discomfort were reported by most of the subjects.

CORNEAL DAMAGE FROM UV RADIATION

The cornea absorbs UVC and UVB with a peak effect at 270nm (**see Figures 1-4**). Studies over the last decade have indicated

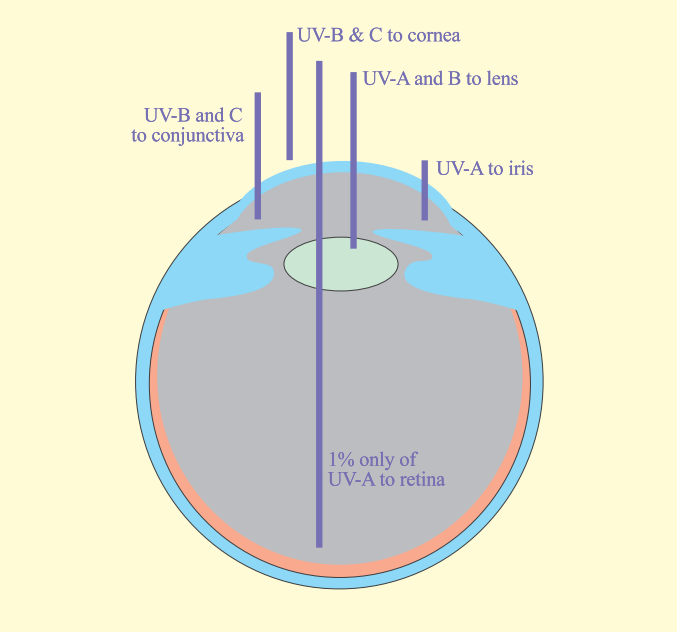

Figure 1 Ultraviolet absorbed by the normal phakic eye

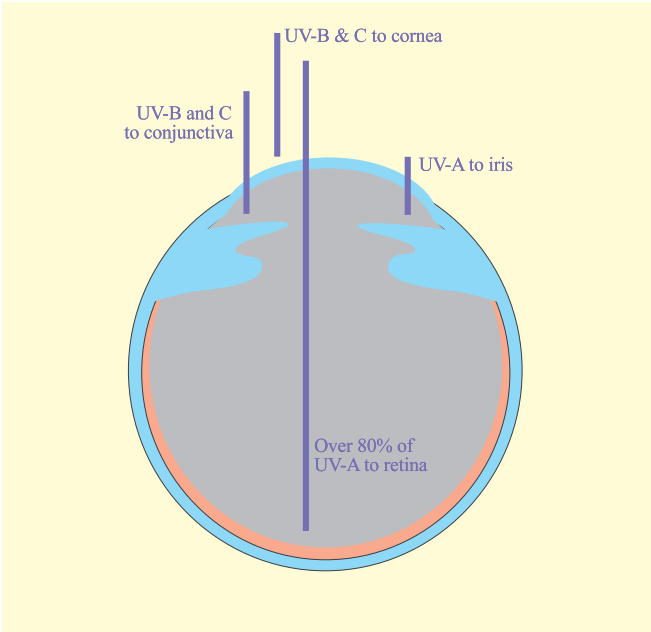

Figure 2 Ultraviolet radiation absorbed by the unprotected eye

that exposure to UV radiation of the cornea is considerably more damaging than was previously thought. It had been known for many years that the outer epithelial layer was the region most damaged by UV radiation. More recently, optometrists working in the USA have shown that the endothelial layer in the primate cornea is also damaged (specifically by UVB of wavelength 300nm) and this, unlike the epithelial damage, is permanent (Pitts et al, 1987). High exposure to UV radiation at the cornea causes a change in the normal fluid balance. It is believed that this arises at the endothelial layer, from possible increased permeability of this layer. Studies involving specular microscopy of the corneas of welders during the acute stage of photokeratitis have shown an immediate reaction by the endothelial layer and a reduction in the number of hexagonal cells. A higher dosage of UV is needed to induce endothelial damage than epithelial damage.

Corneal damage from UV radiation exposure is difficult to separate from the normal ageing processes, because both produce a thinning of the cornea and changes in the endothelium. UV damage causes a decrease in corneal sensitivity, but rapid recovery is usual within a few hours. Recent work suggests that the nerve fibre action is inhibited, but it is not thought that there are structural changes (Bergmanson et al, 1995). There is a close relationship between the epithelial cells and the nerves that supply it. The cornea has two nerve plexuses, one epithelial and one just below this outer layer in conjunction with the corneal stroma, which acts as a back-up to protect the delicate corneal tissue from mechanical and radiation threats. It is this nerve complex that causes so much pain to the cornea damaged by UV radiation.

After exposure, corneal oxygen uptake decreases, glucose levels increase as do those of glycogen. UV radiation is therefore capable of severe disruption of corneal metabolism. As the cornea has a low threshold for UVB, it has been suggested that corneal damage precedes the development of cataract (Ham et al, 1982).

The main response of the cornea to UV exposure is the condition called photo-ophthalmia, or photokeratitis, thought to be due to primarily to wavelengths between 210nm and 315nm. This is the 'welder's flash', known for over 100 years, that is experienced by arc welders who strike the arc before lowering a protective helmet. After exposure to UV, there is a latency period of approximately six to 12 hours, depending on the intensity of the exposure.

The anterior part of the eye, the eyelids and the skin surrounding the eyes become reddened. There is some sensation of a foreign body (grittyness) and the person becomes photophobic, produces excess tears, and tightly closes both upper and lower lids (blepharospasm) to avoid pain. These acute symptoms last up to 24 hours but all discomfort disappears within about 48 hours. Fortunately, only extreme over-exposure causes permanent damage, but the person is visually incapacitated during the acute attack. The eye, unlike the skin, does not develop any tolerance to UV radiation; rather, it becomes more sensitive to repeated exposure. If the eye is exposed to repeated doses at times less than about eight hours apart, there is a likelihood of greater damage. To some degree, the corneal tissue can recover between doses, provided the time is sufficient.

The radiant exposures necessary to produce photokeratitis are quite low. Only 4mJ/cm^2 at 270nm wavelength is the threshold for causing damage, and radiant exposures around twice the threshold value can result in permanent damage to the cornea.

Inflammation occurs when the dosage is high because the iris and related tissues are also involved. White blood cells which migrate to the edge of the corneal tissue to help in its recovery from the radiation threat may also complicate the recovery process. It is thought that the high level of pain suffered when photokeratitis occurs from UV exposure is a result of the combined effects of epithelial cell loss and damage to the sub-epithelial axons of the neurones.

PTERYGIUM AND OTHER CORNEAL CONDITIONS

Evidence is clear of an association between chronic UVB exposure and pterygium, a fleshy growth on the normally clear cornea.

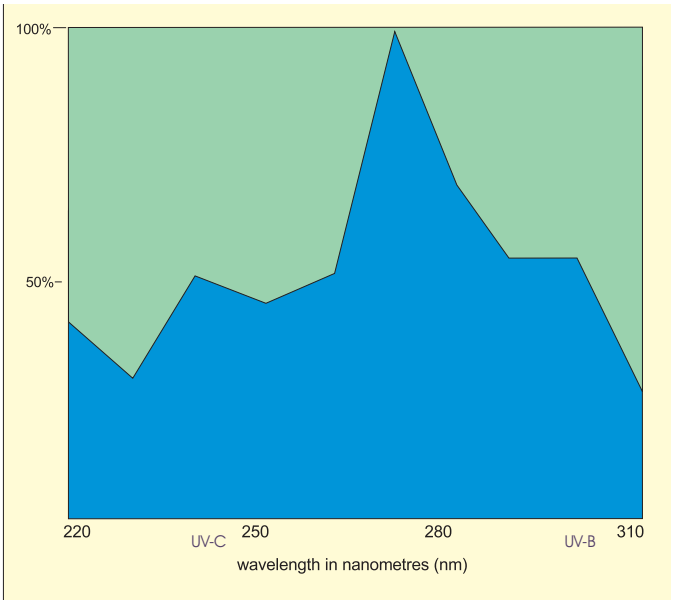

Figure 3 Relative sensitivity of the human cornea to UVC and UVB, (reciprocal of threshold energy for damage) after Pitts (1974)

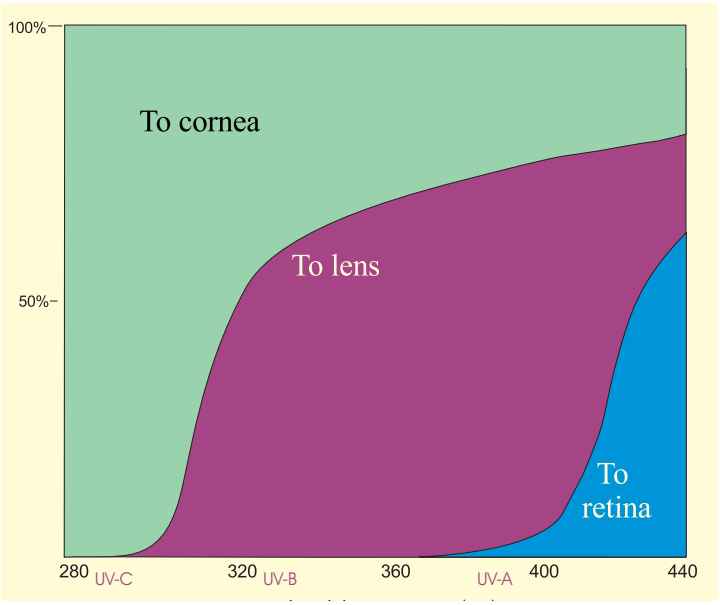

Figure 4 Transmittance of near UV to the lens and retina after Charman (1990)

Various types of keratopathy involving deposition of altered proteins in the superficial cornea between the epithelium and Bowman's membrane, leading to opacification, have been reported by several authors (Moran and Hollows, 1984; Taylor, 1989).

A study of fishermen in Maryland, USA, found a substantially enhanced risk of both pterygium and keratopathy in individuals receiving higher exposures of UV (Taylor, 1989) but pingueculae (conjunctival lesions) were very weakly associated with UVB exposure (Taylor et al, 1989). We do not know which type of UV radiation may contribute most to these conditions, but since any damage must depend on the absorption of photons, the energetic UVB photons are likely to have a greater effect than UVA or visible photons, for which the cornea is relatively transparent. On the other hand, solar radiation levels are much greater at the longer wavelengths, and higher levels of solar UVB also imply higher levels of UVA and visible light. Analysis of the same data on Maryland fishermen showed that the correlation of pterygium and keratopathy with blue light exposure was just as good as the correlation with UVB exposure (Taylor et al, 1992).

Pterygium affects the bulbar conjunctiva near the nasal limbus. The characteristic position is thought to be a direct consequence of radiation entering from the temporal side at a specific angle, owing to the shape of the outer eye. A growth of conjunctival epithelium into the corneal region is typical, since UV radiation disrupts the cell division and reproduction. The explanation of the wedge shape of a typical pterygium is not clear, although there is a definite migration of cells with a leading edge towards the cornea.

A pinguecula is a benign degenerative tumour which is normally seen on the bulbar conjunctiva within the palpebral aperture. It is weakly associated with UV exposure (Taylor et al, 1989; Charman, 1994) since the link is based only on a small number of epidemiological studies (Bergmanson et al, 1995). Corneal and conjunctival tumours may well be a consequence of excessive UV exposure, usually of the epithelial cells. There have been reports of such carcinomas (Guex-Crosier et al, 1993) in patients in their 30s, who were contact lens wearers exposed to substantial levels of solar and artificial UV radiation.

A permanent condition called climatic droplet keratopathy is produced by long-term exposure to UV radiation, usually arising from a life in the open air. It is thought likely that plasma protein in the limbal region of the cornea is degraded photochemically by the UV radiation breaking this down to material that accumulates in the corneal stroma and Bowman's layers. The deposits look like small amber-coloured granules. This process is also known to be similar to the normal ageing seen in corneal tissue.

The human cornea typically reacts to UV radiation in the 220-310nm range and with a threshold of 4J/cm^2. For the cornea, the lowest threshold is at 270nm and for the lens at a slightly higher wavelength of 300nm.

DAMAGE TO THE UVEA

UV wavelengths between 295nm and 310nm cause anterior uveitis - an inflammation of both Descemet's layer and the endothelium layer of the cornea, the iris and even the lens. The effect is redness of the eye close to the cornea and fibrous material deposited on the endothelial back surface of the cornea. Usually this is only a temporary problem.

CRYSTALLINE LENS

UVB radiation is absorbed by the crystalline lens of the human eye and even some UVA (longer wavelengths) are absorbed in the older eye, into the violet end of the visible range. The absorption of small amounts of UV daily over many years may be responsible for inducing or accelerating the formation of cataracts. There is increasing evidence implicating UV radiation as the causative factor in the production of cataracts, particularly the age-related brown or brunescent cataract occurring in the nucleus of the lens. Epidemiology indicates that the incidence of cataract is related both to the exposure to sunlight and to UV exposure for persons living at different latitudes. For instance, the incidence of cataract is increased for all age groups in Israel compared to Oxford, and in Tampa, Florida, as compared to Rochester, New York. Areas of the USA with greater sunlight duration have a higher incidence of cataract than areas with shorter duration. Nevertheless, the relationship between solar UV exposure and cataract is still open, as the epidemiological data cannot establish a cause-and-effect relationship, and the biochemical theories are experimentally unproven.

The lens begins absorbing UV at about 290-295nm and absorbs much of the UV spectrum up to about 375nm. It appears that this absorption band, centred near 365nm, could be the cause of UV-induced cataracts. Some studies suggest that the most effective wavelength in producing cataracts is shorter, 300nm, but in fact less than 5% of the 300nm waveband striking the eye is transmitted by the cornea through the aqueous to the lens. Radiant exposure as high as 130J/cm^2 at 365nm will produce severe corneal damage but no cataracts, while exposure of only 0.15J/cm^2 at 325nm can produce cataracts.

BIOCHEMICAL CHANGES

The photochemical and biochemical mechanisms underlying the response of the eye to UV radiation above 300nm involve some significant alterations to the proteins of the lens. UVB caused oxidation of the tissues and disrupted the biochemistry in the study of UV-induced cataracts in rhesus monkeys by Ham et al (1989), but the exact role that UVB plays in cataract development in man is still debated since no single modified molecule has been found (Charman, 1994).

Some researchers suggest that UV photons interact with molecules in the epithelial membranes of the lens and cause an imbalance in the protein environment. A change to the calcium/ATPase balance, which regulates the exchange of ions across membranes, may well be responsible.

The cornea, in effect, acts as a filter for the lens and retina, absorbing harmful UV. This buffer breaks down in two situations. The first is for long-term, low level repeated exposures that are in the dangerous wavelength range, but with radiant power levels below the corneal damage threshold - for instance sunlight exposure. The cumulative effects may then produce lenticular and retinal damage. The second situation occurs when exposure to a very high radiant level in the dangerous wavelength range is delivered over a very short duration - for instance laser bursts. Such exposures may result in minimal effect on the cornea but extensive damage in the form of cataracts and retinal lesions.

RETINAL DAMAGE

Since the mid-1960s, we have known that even moderate intensities of light can damage the retinas of rats. Photopic damage to the retina after long-term exposure has been reported in pigeons, rats, mice, rabbits, piglets, monkeys and even humans. Body

temperature is found to play a role in determining the magnitude of the damage.

Damage to the light-sensitive human retina can be seen even years after a single exposure to the macula of relatively low intensity short-wavelength light. In 1989, Kremers and van Norren found the threshold damage in monkeys for white light to be near 230J/cm^2 occurring from either low radiation levels applied for a long time or higher levels for a short time. The time and strength of the radiation together have a compounding effect on biological tissue.

In the human eye, the initial effects involve the outer segments of the photoreceptor cells in which the tip of the photoreceptor shows vacuole formation. The damage continues until the outer segment loses its normal lamellar structure and breaks off from the inner segment of the visual cell. As the outer segments are phagocytosed by the pigment epithelium, the inner segments change and then also disappear. The final result is a retina in which most of the photoreceptor cells have disappeared but in which the remaining layers appear to be intact.

There is some controversy regarding the effect of light on the pigment epithelium. Some researchers believe that damage to this layer occurs before the destruction of photoreceptors; others believe that it occurs at the same time as photoreceptor cell destruction. It has also been suggested that damage to the pigment epithelium is subsequent to the destruction of receptor cells. We do know from direct evidence that photopic damage to the photoreceptor neurones of the retina occurs very soon after the eye has been exposed to light.

The eye has very fine repair mechanisms if exposure is no greater than 600J/cm^2. Usually, special cells involved in repair called phagocytes migrate into the retina and transport cell debris away if photochemical damage occurs.

Receiving less than 1% of radiation below 340nm and 2% between 340nm and 360nm, the retina is shielded by the powerful UV absorption of the cornea and crystalline lens. Although most of the UV radiation entering the eye is thus absorbed (in the phakic eye) by the cornea and lens, the retina is still considerably at risk from photochemical damage. Retinal exposures are difficult to quantify because of problems in determining the size of the retinal image and the ocular transmittance for UV radiation. The retinal image size is critical in calculating the exposure. The aberrations of the eye and focal distances must be guessed since they are not known for UV wavelengths. Typically, UV radiation at 300nm does reach the retina of the phakic eye. The melanin present in the pigment epithelial layer absorbs UV close to the visible spectral wavelengths, even into the UVA band.

Thermal damage, on the other hand, acts through denaturation of structural proteins. This damage produces scars (for instance, after laser treatment in ophthalmology) and destroys cells irreversibly because the melanin of the pigment epithelium absorbs approximately 60% of the light around 500nm and dissipates the heat into the near surroundings. Thermal damage is seen clearly, but it is particularly difficult to establish a relationship between radiation levels and photochemical damage because of the latency period between exposure and the detection of pathological alterations to the retina.

Exposure to the sun, particularly deliberate staring, can cause solar retinopathy, with clearly defined macular lesions. A macular hole may occur with oedema to a wide area. Yanuzzi et al (1987) found an increased incidence of solar retinitis in areas of ozone depletion, but the major consequence to the eye of decreased levels of ozone in the atmosphere is still corneal and conjunctival (Charman, 1994).

The potentially damaging effect of UVB to the retina is especially important for children whose corneas and crystalline lenses are less well developed and absorb less of the damaging wavelengths than for adults, as shown by a WHO study (1993).

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MULTIPLE CHOICE QUESTIONS

Radiation effects on the eye
Ocular effects of ultraviolet radiation Parts 3a & 3b

1. UV radiation of wavelengths 200-280nm poses little threat to eyes as:
a. the wavelength is too short to cause damage
b. it does not reach the earth's surface
c. it is reflected by the cornea
d. it is re-radiated by the lens

2. Blue visible light contributes significantly to the development of:
a. photokeratitis
b. pterygium
c. age-related cataract
d. age-related macular degeneration

3. Depletion of the ozone layer has which of the following implications for eyes?
a. Little, overall
b. Significantly increased risk of cataract in the tropics
c. Significantly increased risk of cataract in temperate latitudes
d. Significantly increased risk of cataract in polar regions only

4. Blue light is more damaging than red visible light by a factor of:
a. 5
b. 30
c. 500
d. 3000

5. UV damage to body tissue principally takes the form of:
a. thermal burns
b. photochemical damage
c. stimulation of immune response
d. carcinogenic action

6. The protection against UV offered by contact lenses is:
a.. limited and variable for all types, especially soft lenses
b. limited and variable for hard lenses, but excellent for soft lenses
c. limited and variable for soft lenses, but excellent for hard lenses
d. excellent for all types of lenses

7. PMMA intraocular lenses for cataract patients:
a. always provide better UV protection than the natural lens
b. provide inadequate UV protection, so patients must use sunglasses
c. are degraded by UV radiation transmitted through the cornea
d. need to be manufactured with UV absorber to protect the retina

8. Patients taking photosensitising drugs have:
a. an abnormal retinal reaction to ultraviolet
b. an increased risk to their eyes from normal UV exposure
c. no change in their risk from UV exposure
a. a decreased risk to their eyes from normal UV exposure

9. Corneal damage from UV exposure has:
a. a delayed reaction after 24 hours, with full recovery within a week
b. an acute phase lasting up to 24 hours, often leading to permanent damage
c. an acute phase lasting up to 24 hours, unlikely to produce permanent damage
d. an acute phase lasting one hour, gradually reducing over two days

10. Pterygium is believed to be caused by:
a. blue light
b. UVA
c. UVB
d. UV or blue light, the exact waveband being unknown

11. Deliberate staring at the sun is likely to cause:
a. solar cataract
b. photokeratitis
c. solar retinitis
d. anterior uveitis

12. The UVA waveband is:
a. the least dangerous type of UV radiation, though widespread in the environment
b. the most dangerous and most widespread type of UV radiation
c. the most dangerous type of UV radiation, but fortunately not widespread in the environment
d. the least dangerous and least widespread type of UV radiation

Please note there is only ONE correct answer. An answer return form is included in this issue.
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