Position Paper on Ocular Ultraviolet Radiation

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Introduction

The human eye is exposed to toxic Ultraviolet Radiation (UVR) from natural sunlight and man made artificial sources. UVR induced damage and related diseases can occur in a number of tissues within the eye, ranging from the corneal surface to the retina (Bergmanson and Söderberg 1995). At the same time, the cornea and the crystalline lens provide inherent UVR protection (Boettner et al. 1962, Sliney 2002, Walsh et al 2008). Shading headwear and certain designs of UVR-blocking sunglasses can reduce UVR exposure but do not provide the high degree of ocular protection afforded by UVR-blocking contact lenses (Walsh et al 2003), particularly when the latter is combined with shading and sunglasses. The design of UVR-blocking sunglasses and how they are worn are important in achieving optimal protection from these devices. Sunglasses providing a tight fitting wrap-around design, as opposed to small flat lenses mounted off the eye, offer the best protection to the ocular media, providing that they adhere to the highest standard of inherent UVR-blocker in their lens material (Rosenthal et al 1988, Leow and Tham 1995). Well designed sunglasses also offer some protection for the eyelids and the bulbar conjunctiva.

Ultraviolet Radiation

UVR extends from 1-400 nm and is divided into very high photon energy far UVR (1-100 nm), high photon energy UVR-C (100-280 nm), medium photon energy UVR-B (280-315 nm), and low photon energy UVR-A (315-400 nm) (CIE 1987).

UVR below 300 nm is not present in sunlight at the Earth's surface due to protective absorption by the ozone layer (Floyd et al 2002). However, a man-made depletion of atmospheric attenuating factors of solar UVR, such as ozone, would change the spectrum and increase the intensity of UVR-B below 300 nm incident on the surface of the Earth. This would lead to an increase in sun induced ocular pathology as the wavelengths around 300 nm are critical in the action spectra of many human UVR related diseases (Farman et al 1985, Scotto et al 1988).

Due to atmospheric attenuation of UVR and the cosine-law, the incident intensity of solar radiation is highest at the equator and decreases towards the poles. In addition, the incident intensity is inversely related to the altitude at each latitude. Shading will efficiently block directly incident solar radiation but diffuse solar UVR, due to atmospheric scattering and surface reflection, will still reach the eye in significant amounts (Sliney 2002). Therefore, shade, sunglasses and prescription glasses only provide relative protection for the eye and in the absence of direct solar UVR may actually decrease the normal defence reactions such as squint and pupillary constriction (Nemeth et al 1996, Segre et al 1981).

Ultraviolet radiation, a risk factor for human disease

It is now well established and widely accepted that UVR exposure is implicated in the development of skin cancer. There is also increasing evidence that UVR exposure plays a causative role in the development of a range of ocular diseases. For example, an overdose of solar UVR causes an acute photo-keratoconjunctivitis or snow-blindness. There is a very strong association between UVR and the development of pterygium (McCarty et al. 2000, Hirst et al. 2000, Mukesh et al. 2006), climatic droplet keratopathy (Gillan 1970), cortical cataract (Hollows et al. 1981, Taylor et al. 1988, Klein et al. 1992, West et al. 1998, McCarty et al. 2000) and probably pingueculum (Bergmanson and Söderberg 1995). UVR exposure has been epidemiologically associated with intraocular tumours, although considering that almost no UVR is transmitted to the retinal surface this seems highly unlikely (Boettner et al. 1962).

It has been speculated that the limited window of UVR transmittance around 320 nm may contribute to age related macular degeneration (AMD) (Boettner et al. 1962) but there is an ongoing debate as to which light wavelengths are the most important in this vision threatening pathology (Lim 2007). Some forms of AMD are associated with neovascularisation through sprouting from the capillaries in the choroid and the retina. UVR has been shown to induce a number of angiogenic factors, such as vascular endothelial growth factor (VEGF). Strategies designed to block VEGF have demonstrated considerable promise in the treatment of AMD (Mainster 2006, Yanagi et al 2006, Kernt et al 2009).

Repetitive exposure to high intensity short wavelength blue light has been shown to damage the retina (Harwerth and Sperling 1975) with a maximum spectral sensitivity around 505 nm – Type I damage.

Further, blue light exposure has been demonstrated to cause photochemical damage in the retina with a maximum spectral sensitivity in the phakic eye around 435 nm – Type II damage (Ham et. al. 1976). However, the evidence that naturally occurring short wavelength blue or violet light is a risk factor in macular disease is inconclusive and devices filtering out this waveband can distort colour perception (Wirtitsch et al 2009).

The ozone layer has been predicted to continue to decline in thickness, by possibly 20 %. Such a decline would increase UVR exposure at the Earth surface causing an increase in associated diseases and an elevation of health care costs. Population UVR-exposure is therefore a public health issue (West et al 2005).

The implications of corneal diseases associated with thinning of the corneal stroma, such as keratoconus and pellucid marginal degeneration and some refractive surgeries, should also be considered by lens prescribers.

Since a great deal of UVR-B attenuation takes place in the corneal stroma, a thinning of the stroma increases the intraocular exposure to UVR behind the cornea, threatening the crystalline lens (Walsh et al. 2008).

As the crystalline lens efficiently attenuates UVR (Boettner et al. 1962), UVR blocking intraocular lenses (IOLs) should be used for lens implants in cataract surgery to avoid UVR damage to the retina. When IOLs are contraindicated in cataract surgery the aphakic eye must be prescribed UVR absorbing refractive correction (Bergmanson et al 2007, Bergmanson 2007).

Ocular protection against ultraviolet radiation exposure

In the bare eye, the anterior corneal surface is exposed to the full force of solar UVR. If the ocular media are intact and undamaged, the cornea filters out most of the UVR-B and the crystalline lens the UVR-A (Boettner et al. 1962). However, UVR-induced mutations in the ocular surface stem cells are associated with pterygium formation, one of the most common ocular pathologies worldwide. Further, the anterior surface of the lens is exposed to significant levels of toxic solar UVR-B and this exposure has been epidemiologically associated with cataract formation. The ideal preventive measure against toxic solar UVR is to fully block UVR in front of the cornea and the adjacent limbal and conjunctival stem cells. This would simultaneously protect the interior of the eye as well as the vital surface cells.

UVR-blocking spectacles provide adequate protection against normally incident UVR striking the anterior ocular surface. However, since many frame designs are small and leave a gap between the face and the frame, they do not efficiently block ocular exposure from diffuse atmospheric and surface reflected UVR. In addition, there is a significant body of research suggesting that laterally incident UVR can be focused across the cornea onto the nasal limbus where pterygium occurs (Coroneo 1994, Walsh et al 2001). UVR-blocking contact lenses that extend over the limbus and palisades of Vogt, provide more complete protection allowing the user to remain safely outdoors for significantly longer periods because their UVR filtering capacity, although not total, brings UVR levels down to what is considered safe levels (Walsh et al 2003).

Summary

1. There is a need to educate the public and healthcare providers regarding the toxic effects of solar radiation in the eye and how this can be prevented with UVR blocking spectacles and contact lenses. A scientifically based measure of a true relative protection factor for eyewear that can easily be understood by the public and ocular healthcare providers is currently lacking. The development of such a labelling is recommended.
2. UVR ocular trauma may result from an acute overdose or from the accumulated lifetime dose. With increased life expectancy, the lifetime UVR dose becomes an increasingly important consideration when offering advice and prescribing eyewear for patients.
3. The ideal UVR-blocker should transmit only visible radiation and block solar UVR incident on the eye from all directions. This is particularly important for those who work outdoors in the high solar intensities encountered in Southern European and near equatorial latitudes (Walsh et al 2003).

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