THE EYE AND SOLAR ULTRAVIOLET RADIATION

New understandings of the hazards, costs, and prevention of morbidity

REPORT OF A ROUNDTABLE
June 18, 2011, Salt Lake City, UT, USA

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The idea that sunlight can be damaging to the eyes is not new—evidence of ultraviolet’s negative effects has been accumulating for over a century. Sunlight exposure has been implicated to varying degrees in a variety of ocular pathologies involving the eyelids, conjunctiva, cornea, lens, iris, vitreous, and possibly the retina. These ophthalmic conditions have been collectively described as “ophthalmohelioses,” the ophthalmic equivalent of dermatohelioses.¹,²

The evidence for a causative connection between ultraviolet (UV) light and ocular pathology ranges from strong to highly suggestive, depending on the disease state. In the case of pterygium, a common ocular disease with highest incidence in tropical, high-altitude, and highly reflective environments, sun exposure is the only scientifically proven risk factor, and the critical role of UV damage in pterygium pathogenesis is well established. On the other hand, while there is some evidence that UV exposure may play a role in the development of age-related macular degeneration (AMD), that role has not been definitively proven.

There is no question, however, that UV exposure—particularly the cumulative effect of long-term exposure to sunlight—is damaging to the eyes. While dermatologists have done a superb job alerting the public to the hazards of exposing skin to UV, the general population—and even many eyecare professionals—remain somewhat uninformed about the ocular hazards of UV. The result has been a low level of interest in and knowledge about sun protection for the eyes.

This may stem in part from a lack of effective communication of what we already know about the ocular hazards of UV exposure. More important in the longer term, perhaps, are gaps in our understanding of eye protection and the absence of consensus on standards for eye protection—we have, for example, nothing like the sun protection factor (SPF) that could tell sunglass consumers how effectively their new eyewear will protect them. Yes, we know that some clear and most sunwear lenses will block transmitted UV below 350 nanometers (nm) from reaching the retina, but what that does not tell us is how much UV still reaches the eyes without passing through the lenses. So while sunblock lotion buyers know the relative protection one preparation offers versus another, there is no similar scale for buyers of sunglasses.

Similarly, while the UV Index can tell consumers how much solar UV to expect on a given day; as this report documents, even that is flawed as a measure of ocular UV exposure. While excess exposure to UV is clearly hazardous, the situation is complex—moderate exposure to sunlight is important, perhaps even necessary, for good health. In dealing with UV risk, we must be thoughtful and sophisticated, balancing beneficial exposure with the need to protect both skin and eyes from overexposure.³

In an effort to raise awareness about the serious risks of ocular sun exposure and what can be done about them, Essilor brought together an expert panel in June 2011, comprising 11 optometrists, ophthalmologists, dermatologists, chemists, and physicists, for a comprehensive discussion of the dangers UV poses to the eye and ways to protect the eye from UV. Our goals were to:

⦁ Delineate what is known and not known about the damaging effects of UV on the eye,
⦁ Review the costs in terms of both dollars and morbidity of UV-induced eye disease, and
⦁ Identify the stumbling blocks to greater adoption of effective eye protection.

The high points of that wide-ranging discussion are reported here. One point came across with great clarity: we know that UV presents a serious hazard to the eye, but we have not found means to communicate that effectively enough to get the public or even the majority of eyecare practitioners to act on that knowledge. The goal of this work, then, is to inform and by that means to incite action to protect eyes from the very real dangers of long- and short-term solar injury.
UV Radiation: The Nature of the Hazard

UV radiation is electromagnetic radiation with wavelengths ranging from 100 nm to the edge of the visible light spectrum (Figure 1). The UV spectrum has itself been divided into bands based upon the biologic effects of the wavelengths: UVA comprises wavelengths from 380 to 315 nm, UVB from 315 to 280 nm, and UVC from 280 to 100 nm.* (The visible light spectrum runs from 380 to 760 nm.)

UVA, which can penetrate further into skin than UVB, is known to be responsible for sun tanning and skin aging and wrinkling. More biologically active than UVA, UVB causes tissue damage such as erythema and blistering, and is known to play a critical role in the development of skin cancer. UVC may also cause skin cancer; in addition, UVC can kill bacteria, hence the use of UVC as a germicidal agent.

Sources of UV

Natural sunlight is the primary source of terrestrial UV radiation. In normal circumstances, wavelengths below 290 nm are almost completely absorbed by the ozone layer of the stratosphere, so solar UVC is not a problem on the surface of the earth (although man-made UVC from industrial processes is sometimes a hazard). Because the ozone layer can more efficiently absorb short UV wavelengths than longer ones, the UV that reaches the earth’s surface is constituted by about 95% UVA and 5% UVB.⁴

UV can also come from artificial sources such as electric arc welding devices and some new, specialized, or unusual light sources. Lamps often used in tanning

* The precise cutoff points for various UV bands are somewhat arbitrary and differ slightly in work by different groups.
salons are a common and potentially dangerous source of UV radiation. The current trend in indoor lighting is to replace conventional incandescent lamps with more energy-efficient ones, such as compact fluorescent lamps; but light production by fluorescent lamps relies on the release of UV radiation. To help address this, one solution is a double glass envelope which can effectively filter out the emitted UV. However, compact fluorescent lamps with a single-envelope design may lead to an increased risk of UV exposure, particularly when they are used closer to the body (eg, table lamps) for long periods of time.

**UV Damage Mechanisms**

UV can cause both direct and indirect cellular damage (Figure 2). Direct damage from UV penetrating a cell occurs when molecules absorb the radiation. DNA, which readily absorbs UVB, can be damaged this way. When UVB photons are absorbed by a DNA molecule, they add energy and raise the DNA molecule to an excited state; this, in turn, can initiate photodynamic reactions that result in structural changes to the DNA. One typical structural change is the formation of thymine dimers, the most abundant DNA lesions following direct UV exposure.⁵ Thymine dimerization has been shown to occur virtually instantly when UV is absorbed.⁶

UV-induced DNA damage can be repaired through multiple repair pathways inherent to organisms. These protective mechanisms, however, can be overwhelmed by sudden high levels of radiation or chronic lower-level UV exposure. Unrepaired lesions cause distortion of the DNA helix and transcription errors that can be passed on through replication, leading ultimately to mutagenesis or cell apoptosis.

UVA radiation causes no direct DNA damage because it is not absorbed by the DNA molecule. Its absorption by other cellular structures, however, can trigger photochemical reactions that generate free radicals known to be damaging to essentially all important cellular components including cell membranes, DNA, proteins, and important enzymes. Free radicals can also induce depolymerization of hyaluronic acid and degradation of collagen, changes found in photoaging of the skin and vitreous liquefaction of an aging eye.

**Beneficial vs Harmful Effects of UV**

It has long been known that the optimum wavelengths for vitamin D synthesis in human skin fall within a narrow band from 295 to 315 nm.⁷ Studies have found increasing rates of vitamin D deficiency worldwide, and some have suggested that this is attributable to reduced vitamin D production due to sun avoidance, as people take measures to prevent diseases such as skin cancer.⁸,⁹

The balance between beneficial and harmful effects of UV on human health appears to be the single area of disagreement among specialists in the physiologic effects of UV. For example, many dermatologists remain

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**REPTILE LIGHTS: THE GOOD, THE BAD, AND THE SURPRISING**

[The following story was related by Dr. Jan Bergmanson at the Roundtable*]

Reptiles, particularly lizards, gain part of the energy that they need for metabolism and reproduction from UV. In the desert, these creatures’ natural habitat, they can get adequate UV from bathing in the sun for half an hour. For captive (pet) lizards, however, a half hour of desert sunlight is hard to come by, so these reptiles require an artificial source of UV, typically a “reptile light,” that can be purchased at pet stores.

One day in the summer of 2010, Dr. Bergmanson was asked to buy one for his daughter’s pet lizard. Curious about them, he bought not just one but six different reptile lamps and brought them into his lab, where he tested them with his research partner.

What they found came as a surprise; many of the lights emit high levels of UVB—more UVB than one would get in the middle of a sunny summer day in Texas. Even at 30 cm from the bulbs, the recommended safe distance, UVB levels were very high. Some of the lamps also emit toxic shorter wavelengths (UVC) not found in ambient solar radiation.

Dr. Bergmanson and his colleagues also noticed that none of the lamps came with any warning about the potential danger of UV. They did find emission spectra on the packages, but the curves on the labels bore little relation to what they found in the lab. Interestingly, some of the lights did not emit any UV at all. So some UV lamps can harm people, while others, though safe for people, are no good for lizards!

The bottom line is that artificial sources of UV can be dangerous, and labeling is not necessarily an accurate guide to exposure. By asking patients their hobbies, practitioners may be able to identify potential UV exposure risks.

* This work on reptile lights by Dr. Bergmanson and his colleagues was presented at the 2011 meeting of the Association for Research in Vision and Ophthalmology in a poster titled “Commercially Available Reptile Lights—Good For Animal Bad For Handler?”
focused on skin cancer, and suggest that to raise vitamin D levels, sun exposure be replaced by vitamin D supplements; other groups question whether oral vitamin D is equivalent to vitamin D produced by the action of sunlight on skin.

**Absorption and Transmission of UV in the Eye**

The eye is rich in light-absorbing pigmented molecules (chromophores), making it particularly susceptible to photochemical reactions. The human retina should be at high risk for UV damage, but fortunately only 1% or less of the UV incident upon the eye reaches the retina.¹⁰ The overwhelming bulk of the UV is filtered out by anterior ocular structures, in particular the cornea and crystalline lens.

The absorption of UV by ocular tissues is wavelength-dependent (Figure 3). The cornea absorbs light at wavelengths below 295 nm, including all UVC and some UVB.¹¹ Initially the majority of this absorption was thought to occur in the corneal epithelium, but the corneal stroma actually absorbs a significant amount of UV, and Bowman’s membrane is also an effective absorber.¹²,¹³

Unlike the cornea, whose UV absorbance characteristics are stable over time, the crystalline lens undergoes significant changes in UV absorbance as it ages. Specifically, the lens turns more yellow with age, resulting in greater absorption of UV wavelengths. So, while younger lenses can transmit wavelengths as short as 300 nm, the adult lens absorbs almost all wavelengths up to 400 nm.¹⁴,¹⁵ In children under age 10, the crystalline lens transmits 75% of UV; in adults over 25, UV transmission through the lens decreases to 10%.¹⁶,¹⁷ This makes it especially important for children to have UV protection for their eyes.

Thus, the cornea and lens function together as an efficient UV filtration system, removing essentially all UVC wavelengths and the overwhelming majority of UVA and UVB. The “flaw” in this natural design is that it puts the protective structures, the cornea and the lens, at great risk from cumulative UV exposure. Not surprisingly, the most common ocular pathologies associated with sun exposure (including climatic droplet keratopathy, pinguecula, pterygium, and cortical cataract) involve the anterior eye.

**Figure 2** Multiple processes activated by UV contribute to pathogenesis of pterygium.

**Figure 3** Absorption of UV by different ocular structures.
Chronic Diseases

Because of the difficulty involved in collecting quantitative data on UV exposure in large populations over periods long enough to allow estimation of lifetime dose, establishment of the relationship between specific eye diseases and sunlight exposure has had to rely heavily on epidemiological studies.¹⁸ These studies have implicated UV damage from chronic sun exposure in a number of ocular diseases, including climatic droplet keratopathy, pinguecula, pterygium, cataract, and possibly AMD (Table 1).

UV-associated ocular diseases have a tremendous impact on both individuals and society. Impaired vision often causes lost productivity and social limitations; treatment of the diseases increases healthcare costs, adding to the economic burden of lost productivity.

Pterygium

Pterygium is most prevalent in areas close to the equator and at higher altitudes, both of which are places with higher levels of UV exposure. An elevated incidence of pterygium is also found in places with high ground reflectivity.¹⁹,²⁰

In the southern US, for example, the incidence of pterygium is estimated to be more than 10%, and it affects about 15% of the elderly population in Australia and more than 20% in Pacific islanders and in high-altitude populations in central Mexico.²¹-²⁴

Without intervention, a pterygium may eventually invade the central cornea, causing blindness in severe cases. Although the abnormal tissue can be surgically removed and the affected bulbar conjunctiva/limbus reconstructed, surgery is time-consuming, costly, and may be associated with a relatively high recurrence rate.

Climatic droplet keratopathy

Climatic droplet keratopathy is a condition in which translucent material accumulates in the corneal stroma in the band between the lids. People who spend considerable time outdoors are at particular risk for this condition, which can cause significant visual disability. It is believed that the translucent material consists of plasma proteins denatured by exposure to UV.²⁵

Cataract

Cataract continues to be the leading cause of blindness worldwide. Although surgery can prevent vision loss in almost every case, many nonindustrialized countries lack the resources to make cataract surgery...
Light Focusing [PLF]), the amount of solar UV that reaches the retina is small, only 1% or less of the UV that strikes the cornea.

Also, AMD is a multifactorial disease; genetic predisposition, age, smoking, diet, and light toxicity are all likely risk factors. Future study of the link between UV and AMD is warranted to determine its place among the many other factors that have been implicated in AMD. One challenge in this process will be to get an accurate measure of retinal UV dose, which can vary with pupil size and increasing age as the absorption spectrum of the crystalline lens changes.

Ultraviolet Exposure and Skin Cancer

One major effect of excessive sun exposure is the development of skin cancer. Although UVA penetrates more deeply into the dermis and subcutaneous layers, it is not absorbed by DNA and thus previously deemed to be less harmful than UVB as a skin hazard. But we now know that, while UVA is less efficient in causing direct DNA damage, it can contribute to development of skin cancer through photosensitizing reactions that produce free radicals, which, in turn, cause DNA damage.⁴⁵

Over the past 31 years, there have been more cases of skin cancer than all other cancers combined.⁴⁶ Melanoma, while less common than other skin cancers, is life-threatening and accounts for the majority of skin cancer deaths. It is estimated that about 64% of melanoma and 90% of nonmelanoma skin cancers (basal and squamous cell carcinomas) stem from excessive UV exposure.⁴⁷,⁴⁸ The vast majority of the more than 33,000 gene mutations identified in the melanoma genome are caused by UV exposure, providing a strong link between UV exposure and the development of this skin malignancy.⁴⁸

In the US, nonmelanoma skin cancers increased at a rate of 4.2% per year between 1992 and 2006.⁴⁹ Equally alarming is that melanoma incidence also increased by 45%, or about 3% per year, between 1992 and 2004, a rate faster than any other common cancer.⁵⁰ Skin cancer places a significant economic burden on society—the direct costs for the treatment of nonmelanoma skin cancers in 2004 came to $1.5 billion.⁵¹ Treatment of melanoma in adults 65 or older costs about $249 million annually.⁵² These numbers are expected to rise in parallel with the rising incidence of skin cancer.

Both melanoma and nonmelanoma skin cancers occur in the eyelids, which is the site of approximately 5–10% of nonmelanoma skin cancers.⁵³ It has been noted clinically that eyelid cancers are four times more likely to occur in the lower than the upper lids, perhaps because the upper orbital rim shades the upper lid more than the lower.⁵⁴ In addition to eyelid malignancy, UV exposure has also been associated with an increased risk of uveal melanoma.⁵⁵,⁵⁶

Table 1: Ophthalmic Conditions in which UV has been Implicated in Pathogenesis

<table>
<thead>
<tr>
<th>Table 1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eyelid</strong></td>
</tr>
<tr>
<td>Wrinkles; sunburn, photosensitivity reactions, malignancy—basal cell carcinoma, squamous cell carcinoma</td>
</tr>
<tr>
<td><strong>Ocular Surface</strong></td>
</tr>
<tr>
<td>Pinguecula, pterygium, climatic keratopathy (Labrador keratopathy), keratitis (flash, snow blindness), dysplasia and malignancy of the cornea or conjunctiva</td>
</tr>
<tr>
<td><strong>Crystalline Lens</strong></td>
</tr>
<tr>
<td>Cortical cataract</td>
</tr>
<tr>
<td><strong>Uvea</strong></td>
</tr>
<tr>
<td>Melanoma, miosis, pigment dispersion, uveitis, blood–ocular barrier incompetence</td>
</tr>
<tr>
<td><strong>Vitreous</strong></td>
</tr>
<tr>
<td>Liquification</td>
</tr>
<tr>
<td><strong>Retina</strong></td>
</tr>
<tr>
<td>Age-related macular degeneration</td>
</tr>
</tbody>
</table>
Sources of Exposure

Multiple factors determine the intensity of ambient UV, which can vary dramatically with location and time of day or year. Direct sunlight contributes to only a portion of the ambient UV, more than 50% of which actually comes from localized light scattering and cloud reflection and scattering.⁵⁷

In general, adults and children get exposed to about 2 to 4% of the total available annual UV while adults working outdoor get about 10%.⁵⁸ The average annual UV dose is estimated to be about 20000 to 30000 J/m² for Americans, 10000 to 20000 J/m² for Europeans, and 20000 to 50000 J/m² for Australians, excluding vacation, which can add 30% or more to the UV dose.⁵⁸

UV that reaches the ocular surface can be measured by contact lens dosimetry as the ratio of ocular-to-ambient UV exposure, which was reported to range from 4 to 23% at solar noon.⁵⁹ Unlike the skin or ambient exposure, UV exposure of the eye is further determined by natural protective mechanisms, including squinting, pupil constriction, and geometric factors related to the orbital anatomy. These unique factors mean that peak ocular UV exposure may not coincide with peak skin exposure.

There are many popular misconceptions with respect to ocular UV exposure.⁶⁰ Understanding the factors that determine ocular exposure is challenging but critical for accurate assessment of ocular UV risks and determination of specific defense strategies against them.

### Table 2

<table>
<thead>
<tr>
<th>Condition</th>
<th>Sunlight exposure (Lx)</th>
<th>Percent of UV exposure per year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indoor</td>
<td>500</td>
<td>8%</td>
</tr>
<tr>
<td>Clouded sky</td>
<td>5000</td>
<td>5%</td>
</tr>
<tr>
<td>Clear sky</td>
<td>25000</td>
<td>30%</td>
</tr>
<tr>
<td>Summer sky</td>
<td>100000</td>
<td>58%</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>100%</td>
</tr>
</tbody>
</table>

*Calculation based on urban workers in Northern hemisphere.
Critical Factors in Determining Atmospheric UV Intensity

The ozone layer The ozone layer absorbs virtually all solar UVC and up to 90% of UVB, providing a natural shield from UV light. In the past three decades, however, human activity has reduced the concentration of atmospheric ozone. Between 2002 and 2005, the ozone at mid-latitudes was depleted by about 3% from 1980 levels in the northern hemisphere and by about 6% in the southern hemisphere.

This ozone reduction can be expected to increase human exposure to UV. It has been estimated that for every 1% reduction in the ozone layer there will be penetration of between 0.2% and 2% more UV. A greater proportion of the increased radiation will be shorter wavelengths, which are absorbed by the ozone layer.

Solar angle Solar angle is the most significant determinant of ambient UV intensity. Sunlight intensity peaks when the sun reaches its zenith, because perpendicular light projects to a smaller surface area than oblique light projection, so the light energy per unit area is more concentrated when the spot size is smaller. Also, when the sun is high in the sky, sunlight travels less distance through the atmosphere to reach the surface, so it is less diffused and attenuated.

UV INDEX

The UV Index, which ranges from 0 to the mid-teens, is a linear scale developed to describe the UV intensity at the earth’s surface. The Index is calculated by an international standard method that takes into account the date, a location’s latitude and altitude, and forecast conditions for ozone, clouds, aerosols, and ground reflection. The higher the value, the more intense the ambient UV and the greater the likelihood of UV damage to exposed skin.

Intended to guide people who need to make ordinary decisions such as how long they can stay outside on a given day and whether or not they need to wear sun protection, the Index has been widely incorporated into weather forecasts to predict the peak UV level at solar noon.

A vital shortcoming of the UV Index is that what it projects is only the predicted degree of UV danger to the skin. The Index does not correlate well with the risk of ocular UV damage, due in large part to the exposure geometry of the eye.

FIGURE 4 The Antarctic ozone hole on the day of its maximum depletion (the thinnest ozone layer, as measured in Dobson Units [DU]) in four different years.*

Top left: on September 17, 1979, the first year in which ozone was measured by satellite, the ozone level was at 194 DU.

Top right: ozone dropped to 108 DU on October 7, 1989. This was the year that the Montreal Protocol went into force.

Bottom left: ozone measured 82 DU on October 9, 2006.

Bottom right: the measurement was back up to 118 DU by October 1, 2010.

*The ozone measurements were made by National Aeronautics and Space Administration (NASA)’s Total Ozone Mapping Spectrometer (TOMS) instruments from 1979 to 2003 and by the Royal Netherlands Meteorological Institute (KNMI) Ozone Monitoring Instrument (OMI) from 2004 to present. Purple and dark blue areas are part of the ozone hole.
For this reason, surface level of UV varies with time of day and time of year, as well as with latitude: all factors that affect the solar angle. All other things being equal, UV intensity is greatest when the solar angle is closest to perpendicular. (This is thought to explain the observation that pterygium is most common in equatorial regions and highly reflective environments.)

Cloud cover Clouds are complex and ever changing, facts that have a significant bearing on the variability of ambient UV. While a thick cloud cover substantially reduces the amount of UVA and UVB that reaches the earth’s surface, thin and broken clouds have much less effect. Also, cumulus clouds can actually increase UVB radiation by 25% to 30% due to reflection from their edges.

Surface reflection (albedo) Reflection from the ground and surrounding surfaces, known as albedo, can add significantly to ambient UV levels—especially the level measured at the eye, which, as noted, is protected from overhead UV. Due to reflection, one can be exposed to UV in completely shaded areas. Highly reflective substances, such as fresh snow, reflect as much as about 90% of incoming UV back into the atmosphere (Table 3A&B). Sand can reflect between 8% and 18% of incident UV, water from 3% to 13%, and lawn grass from 2% to 5%.

Altitude Since UV passes through less atmosphere to reach higher grounds, it has less chance to be absorbed by atmospheric aerosols, which, like the ozone, can absorb and attenuate UV. As a result, populations at higher altitudes are generally exposed to higher levels of UV. In the United States, there is 3.5% to 4% percent decrease in UV for each 300 m of descent in elevation.

Ocular UV Exposure

Exposure geometry Since our eyes are set deep in the orbital bone structure, sunlight entering the eye parallel to the visual axis has the clearest path. When the sun is directly overhead near its zenith, little direct UV strikes the corneal surface due to the natural shield of the brow and upper eyelids. Thus, despite the fact that the ambient UV usually reaches its maximum strength at solar noon (at which point skin exposure is at its peak), the level of UV that enters the eye may be lower than it is at earlier and later times of the day.

Contribution of scattered and reflected light Short-wavelength radiation (UVB) is effectively scattered by air particles and highly reflected by certain surfaces (Table 3A). This indirect radiation from light scattering and reflection actually contributes to nearly half of the UV we receive, warranting its significance in any consideration of UV protection.

When the solar altitude reaches about 40 degrees, direct UV exposure in the eye decreases rapidly, presumably because the upper eyelids and possibly the eyebrow shield the eye from the incident overhead light.

### Table 3A

<table>
<thead>
<tr>
<th>Material</th>
<th>Percent Reflectance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lawn grass, summer, MD, CA, and UT</td>
<td>2.0-3.7</td>
</tr>
<tr>
<td>Lawn grass, winter, MD</td>
<td>3.0-5.0</td>
</tr>
<tr>
<td>Wild grasslands, Vail Mountain, CO</td>
<td>0.8-1.6</td>
</tr>
<tr>
<td>Lawn grass, Vail, CO</td>
<td>1.0-1.6</td>
</tr>
<tr>
<td>Flower garden, pansies</td>
<td>1.6</td>
</tr>
<tr>
<td>Soil, clay/humus</td>
<td>4.0-6.0</td>
</tr>
<tr>
<td>Sidewalk, light concrete</td>
<td>10-12</td>
</tr>
<tr>
<td>Sidewalk, aged concrete</td>
<td>7.0-8.2</td>
</tr>
<tr>
<td>Asphalt roadway, freshly laid (black)</td>
<td>4.1-5.0</td>
</tr>
<tr>
<td>Asphalt roadway, two years old (grey)</td>
<td>5.0-8.9</td>
</tr>
<tr>
<td>Housepaint, white, metal oxide</td>
<td>22</td>
</tr>
<tr>
<td>Boat dock, weathered wood</td>
<td>6.4</td>
</tr>
<tr>
<td>Aluminum, dull, weathered</td>
<td>13</td>
</tr>
<tr>
<td>Boat deck, wood, urethane coating</td>
<td>6.6</td>
</tr>
<tr>
<td>Boat deck, white fiberglass</td>
<td>9.1</td>
</tr>
<tr>
<td>Boat canvas, weathered, plasticised</td>
<td>6.1</td>
</tr>
<tr>
<td>Chesapeake Bay, open water</td>
<td>3.3</td>
</tr>
<tr>
<td>Chesapeake Bay, specular component of reflection at $Z = 45^\circ$</td>
<td>13</td>
</tr>
<tr>
<td>Atlantic Ocean, NJ coastline</td>
<td>8.0</td>
</tr>
<tr>
<td>Sea surf, white foam</td>
<td>25-30</td>
</tr>
<tr>
<td>Atlantic beach sand, wet, barely submerged</td>
<td>7.1</td>
</tr>
<tr>
<td>Atlantic beach sand, dry, light</td>
<td>15-18</td>
</tr>
<tr>
<td>Snow, fresh (2 days old)</td>
<td>88</td>
</tr>
</tbody>
</table>

All measurements performed with cosine-corrected hemispherical UVB detector head of IL 730 radiometer. Reflectance is ratio of “down”/zenith measurement.

### Table 3B

<table>
<thead>
<tr>
<th>Surface</th>
<th>UVA albedo,%</th>
<th>UVB albedo,%</th>
<th>Percent of UVA albedo,%</th>
<th>Percent of UVB albedo,%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sand</td>
<td>13</td>
<td>9</td>
<td>59</td>
<td>41</td>
</tr>
<tr>
<td>Grass</td>
<td>2</td>
<td>2</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>Water</td>
<td>7</td>
<td>5</td>
<td>58</td>
<td>42</td>
</tr>
<tr>
<td>Snow</td>
<td>94</td>
<td>88</td>
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With the higher sunlight angles, the eye is primarily exposed to scattered and reflected radiation—contrary to the popular belief that direct sunlight around noon puts us at risk for maximal UV exposure.

**Peripheral light focusing (Coroneo effect)** The configuration of the human eye and face permits a large temporal field of vision and thus allows a significant amount of the incident light that reaches the cornea to come from the side. The groundbreaking work of Coroneo and colleagues established that this radiation from the side represents a particularly significant hazard due to the way it is focused on the nasal limbus by the PLF mechanism.

In PLF, oblique light (including UV) is refracted by the peripheral cornea, causing it to travel across the anterior chamber and focus at the nasal limbus, where the corneal stem cells reside (Figure 6A).\(^1\)\(^,\)\(^3\)\(^4\)\(^,\)\(^7\)\(^5\) The maximum PLF effect at the limbus has been shown to occur when the angle of incidence is 104 degrees from the visual axis.\(^7\)\(^6\) While limbal stem cells are normally protected from direct UV exposure, PLF concentrates sunlight at the nasal limbus by a factor of 20 times.\(^1\)

Compelling epidemiologic evidence and laboratory results have demonstrated that this peripherally focused light plays a critical role in the development of pterygium.\(^7\)\(^7\) The prevalence of pterygium is thought to rise by 2.5% to 14% with every 1% increase in UV exposure.\(^2\)\(^2\) Almost 20 years ago, Coroneo suggested that pterygium could be an indicator of UV exposure.\(^3\)\(^4\)

We know today that, in addition to the nasal limbus, PLF also affects the nasal crystalline lens equator and the eyelid margin (Figure 6B), which, like the limbus, are sites of stem cell populations. Stem cell damage resulting from focused peripheral light at these loci is believed to be accountable for onset of early cortical cataract and skin malignancy in the eyelid margin.\(^7\)\(^8\)\(^,\)\(^7\)\(^9\)

**Spectacle lenses and back surface reflection** The back surface of clear spectacle lenses has been found to reflect light coming from behind onto the eye, increasing ocular UV exposure.\(^8\)\(^0\)\(^-\)\(^8\)\(^2\) Anti-reflective coatings, intended to enhance the optical performance of spectacle lenses by increasing light transmission and eliminating reflection and glare, turns out (surprisingly) to significantly increase UV reflectance of the back lens surface (Figure 7).\(^8\)\(^2\)

Reflectance measurements have demonstrated that, while clear lenses without anti-reflective treatment reflect about 4% to 6% of UVA and UVB (and less than 8% of UVC), anti-reflective lenses reflect an unexpectedly high level of UV light—an average of 25% for most UV wavelengths and close to 90% for certain wavelengths.\(^8\)\(^2\) This reflected UV can potentially reach the temporal limbus or the central cornea; however, it can be prevented with a high-wrap frame design that protects against back surface exposure, or with an optimized anti-reflective coating with low UV reflection.\(^8\)\(^2\)
Even when the Z80.3 standard is closely adhered to, the transmittance value of sunglasses can be misleading, since it is at best a partial measure of eyewear’s ability to protect the eye from UV exposure. In particular, the transmittance value does not address the radiation coming from around the lenses, the quantity of which is determined by the shape of the frame and its fit to the face. Unless the glasses have a goggle frame, a significant amount of UV can reach the eye via routes around the lenses (Figure 8). Measurements in mannequins have found that just

**Figure 6** Focused peripheral light reaches (A) the nasal limbus and (B) the equatorial crystalline lens.

**Figure 7** UV reflection from the back surface of spectacle lenses.

**Sunglasses**

Most sunglasses can efficiently block UV coming from directly in front of the lens. The American National Standards Institute (ANSI) Z80.3 standard is based on measurement of UV transmission and classifies sunglasses into one of two categories: Class 1 lenses absorb at least 90% of UVA and 99% of UVB; and Class 2 lenses block at least 70% of UVA and 95% of UVB. As voluntary consensus standards, however, these criteria may or may not be followed by all sunglass manufacturers.⁸³,⁸⁴
uV-blocking Contact lenses

For patients who already wear contact lenses, UV-blocking contact lenses can offer significant UV protection. Typically, contact lenses are inserted in the morning and worn all day, providing full-time protection. Soft contact lenses that extend to or past the limbus can block UV from all angles, protecting the stem cells in the limbal region by blocking peripheral radiation and negating the PLF effect. The geometrical factors of the eye are complex, and only a goggle frame or a full coverage contact lens can provide complete protection for the eye.

The ANSI Z80.20 standard recognizes two levels of contact lens protection: Class I lenses must absorb more than 90% of UVA (316 to 380 nm) and 99% of UVB (280 to 315 nm), and are recommended for high exposure environments such as mountains or beaches. These criteria were adopted by American Optometric Association (AOA), which has offered a seal of acceptance for qualified lenses. Class II lenses, recommended for general purposes by the FDA, block more than 70% of UVA and 95% of UVB. However, contact lenses do not offer protection for the eyelids.

A goggle frame that wraps around the eye can effectively reduce the side exposure, but the majority of sunglasses do not offer protection from radiation incident from the sides. Under certain conditions, sunglasses without side protection can expose wearers to dangerous doses of UV. Skiers, for example, are at high risk for UV exposure due to the high level of UV reflectance from snow. Unaware of the side exposure issue, however, skiers in standard sunglasses may spend an extended period of time on the slopes, assuming their eyes are adequately protected with ordinary sunglasses. If the sunlight is sufficiently intense, these skiers may suffer painful photokeratitis—literally the ocular equivalent of sunburn. (Welders who fail to wear proper protection and tanning bed users who are not careful in using the right eyewear can also cause themselves to suffer from photokeratitis.)

Sunglasses that allow light to enter from the sides may actually increase a wearer’s level of UV exposure. The darkness of the lenses may reduce the eye’s natural squinting reflex and increase pupil size, increasing the UV entering the eye.

14% of ambient UV reaches the eye when the sunglasses are worn close to the forehead, but up to 45% reaches the eye when the distance between the glasses and forehead is as little as 6 mm.

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Importance of Protection from Cumulative UV Exposure

Although new ozone layer data is encouraging, indicating that atmospheric ozone levels may be beginning to stabilize, ozone layer thickness will not rebound to pre-1980s levels for several decades, at least.⁹³,⁹⁴ Ongoing reduced ozone levels mean that accumulated sunlight exposure will have a growing impact on eye health, and prevention of eye diseases associated with UV exposure will become correspondingly more important.⁹⁵

Also, the population is growing older worldwide, and with longer life comes greater risk for cumulative UV damage. As shown in Figure 9, the accumulative UV dose received by an individual increases linearly with age. Based on an 80-year lifespan, people will, on average, receive about a quarter of their lifetime dose every 20 years.⁵⁸

Higher incidence of ocular diseases associated with chronic UV exposure implies both higher morbidity and increased healthcare costs. In contrast to the high cost of treating UV-related disease, reducing exposure to UV is relatively simple and inexpensive. UV exposure can be readily reduced by sun avoidance and wearing proper prescription or sunwear lenses. If the majority of the population were to become aware of the ocular hazards of UV and were to wear eye protection, significant morbidity and costs could be prevented.
Current State of Eye Protection

Despite what professionals know about the ocular hazards of UV, what the public knows about eye protection is low, compared to the message about skin protection. A 2002 survey found that 79% of the population knew about the skin hazards of UV exposure, but only 6% was aware of the association between UV and eye disease. A survey done by Glavas et al has shown that 23% of people are not wearing any sunwear protection among a population of 1,000 participants in the US. Another more recent survey by the AOA found that although two-thirds of Americans were aware of the need for eye protection when spending extended time in the sun, only 29% of parents made sure their children wore sunglasses while outdoors.

More concerning, perhaps, than public ignorance of ocular UV hazards, is the lack of discussion on UV hazards between eyecare professionals and their patients. As we have seen, there is very little discussion of UV hazards between practitioners in different specialties. Dermatologists educate their patients every day about UV hazards to the skin without ever making reference to the need for eye protection.

In the US, standards for protective eyewear are voluntary, whereas in Europe and Australia, mandatory standards are used as ways of implementing public policy. This puts the US at a disadvantage when it comes to eyewear regulation and UV protection.

Improving Eye Protection

Preventing UV damage to the eye requires that we translate existing knowledge of UV hazards and eye protection into effective multi-component interventions. These must be implemented among all parties involved: the public, healthcare providers, and industry. The most fundamental and important strategy involves education of the public and eyecare providers.

Public education Public education is the cornerstone of any serious effort to reduce the effects of UV on ocular health, because implementation of eye protection is ultimately a matter of what individuals do each day—the habit of UV-protective eyewear in real-life situations.

There have been large public education programs on UV protection, but, unfortunately, almost all have focused on the skin rather than the eyes. The upside, though, is that at least the public is aware that UV in sunlight is a potential danger. More campaigns aimed at increasing eye protection or both the public is aware that UV in sunlight is a potential danger. A 2002 survey found that 79% of the population knew about the skin hazards of UV exposure, but only 6% was aware of the association between UV and eye disease. A survey done by Glavas et al has shown that 23% of people are not wearing any sunwear protection among a population of 1,000 participants in the US. Another more recent survey by the AOA found that although two-thirds of Americans were aware of the need for eye protection when spending extended time in the sun, only 29% of parents made sure their children wore sunglasses while outdoors.

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The task is daunting—human behavior is not easily changed. In Australia, despite decades of strong messages about the need for sun protection, public compliance is still relatively low. There is much to be learned about how to educate the public. Going forward, cooperation between dermatologists and eyecare professionals will be an important part of successful education with respect to UV hazards and protection.

Education of eyecare professionals The challenge in educating eyecare professionals is not in disseminating information but in making sure that that information is used to counsel patients appropriately. The importance of sun protection is a message frequently taught in schools and at professional meetings, but often that message gets lost between the classroom and the clinic. It should, therefore, be a goal of every practitioner education effort to ensure that practitioners use the knowledge they gain to educate patients about UV protection of the eye and prescribe proper UV-protective solutions.

High-risk populations Everyone who is at risk for UV exposure (which is to say anybody who spends time in the sun) should consider adopting protective measures for their eyes. People with darker skin may not have to worry about sunburn and skin cancer to the degree that fair skinned people do, but this may actually increase their risk of ocular exposure because they may feel it less important to wear a hat to protect facial skin.

Certain populations are particularly vulnerable to UV damage. Adults spending extended time or working outdoors is one such group. Children are at elevated risk for two reasons: they typically spend more time outdoors than adults, and their crystalline lenses transmit much more short-wavelength radiation than do the crystalline lenses of older eyes. Young children should start wearing sunglasses with a proper frame design as soon as practicable when they go outdoors.

Aphakic patients, who lack a crystalline lens to absorb UV, may also be at elevated risk. Similarly, patients whose corneas are thin—including those whose corneas have been thinned by laser vision correction and those with naturally occurring corneal ectasias, such as keratoconus and pellucid marginal degeneration —may be at elevated risk, because the corneal stroma absorbs a very significant amount of UV. Also, patients who are taking photosensitizing medications may be more susceptible to potential adverse effects of UV. For all patients with elevated risk, sun protection is extremely important.
UV damage is cumulative, and some people will be well ahead of their contemporaries in the amount of UV they have absorbed due to heavy exposure in their early years. These people are at higher risk for UV-associated diseases later in life. Today, we have no practical means of discovering who these people are so they may be counseled to protect themselves from additional exposure. Thus, a biomarker for UV exposure would be extremely useful for preventing future disease.

Coroneo has developed an ocular UV fluorescence photographic technique that appears able to demonstrate preclinical ocular surface evidence of solar damage. Conceivably this technology could be developed as an “early warning system” to detect excess UV exposure.

An index for eyewear similar to the SPF system for sunblocking lotions would enable rational purchase decisions by people seeking UV protection. Such a system would take into account frame design as well as the transmission spectrum of the lenses.

The current UV Index is far more relevant to skin exposure than ocular exposure. A system that adjusts the current UV Index for the effects of solar angle is needed.

Cooperation with dermatology is necessary to harmonize messages. A method must be found to recognize the importance of skin protection without slighting the special needs related to eye protection.

Research is needed in many areas, including:

a) The importance, in quantitative terms of UV reflection, for the backside of ophthalmic lenses
b) Mechanisms by which UV causes ocular damage
c) Mechanisms of light damage to the retina, including photochemical, photothermal, and photomechanical mechanisms
d) Effective treatment for pterygium
e) Pathogenic role of other environmental factors, such as the ambient temperature in ocular diseases like as nuclear cataract

There is much work to be done. It is vital for eyecare professionals to do more to understand UV hazards and protect our patients. Simply talking to patients on a routine basis about the importance of owning and wearing a pair of glasses that provides good UV protection is a valuable and simple first step.