UV and Blue-Violet Light
Ocular risks and prevention

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I. UV AND BLUE-VIOLET LIGHT: DEFINITION AND OCULAR RISKS
1. ULTRAVIOLET
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 AND HUMAN HEALTH

- Although a small amount of UV comes from artificial sources, the overwhelming bulk of the UV to which people are exposed comes from the sun.

- UV can cause health effects both through direct damage to DNA and through photosensitizing reactions that cause the production of free radicals and oxidative damage.

- The retina and other posterior ocular structures are protected from UV by the cornea and the crystalline lens, which together absorb almost all of the UV that enters the eye. This, however, puts the protective structures at risk.

- Although UV can be harmful, some UV exposure is necessary for good health.

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.

**Figure 1** The visible and invisible light spectrum.
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.
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...
available to large segments of their population; and it is
estimated that worldwide as many as 5 million people go
blind from cataract each year.²⁶

In industrialized nations, where crystalline lens re-
moval and replacement with an intraocular lens is a sim-
ple, effective, and near-universal procedure, the cost of
the surgery overall has a significant economic impact. In
the US alone, more than 3 million cataract surgeries are
performed each year, costing at least $6.8 billion annu-
ally for Americans over age 40.²⁷,²⁸

While further studies are needed to fully determine
the role of UV in the formation of nuclear and posterior
subcapsular cataract, UV has been established as an im-
portant risk factor for cortical cataract.²⁹-³³ Because the
cornea focuses and concentrates light on the nasal lim-
bus and nasal lens cortex, one would expect those sites
to be more prone to UV damage than other loci within
the eye.¹,³⁴ Epidemiologic studies of cortical cataract lo-
calization have consistently observed that early cortical
cataract most often occurs in the lower nasal quadrant of
the lens—exactly what one would predict if UV plays a
role in the development of cortical cataract.³⁵-³⁷

**AMD** Though extensively studied, the role of UV in
the development of AMD remains unclear. Epidemiologic
studies have some suggestive evidence but no clear asso-
ciation between sunlight exposure and AMD.³⁸-⁴⁴ This
is not altogether surprising: unlike the cornea, and to a
lesser degree, the crystalline lens, which are relatively
heavily irradiated with UV (in part due to Peripheral
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 pre-
disposition, 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.

**UV Exposure and Skin Cancer**

One major effect of excessive sun exposure is the de-
velopment 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-threat-
ening 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 di-
rect 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>EYELID</th>
<th>Wrinkles; sunburn, photosensitivity reactions, malignancy—basil cell carcinoma, squamous cell carcinoma</th>
</tr>
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<tbody>
<tr>
<td>OCULAR SURFACE</td>
<td>Pinguecula, pterygium, climatic keratopathy (Labrador keratopathy), keratitis (flash, snow blindness), dysplasia and malignancy of the cornea or conjunctiva</td>
</tr>
<tr>
<td>CRYSTALLINE LENS</td>
<td>Cortical cataract</td>
</tr>
<tr>
<td>UVEA</td>
<td>Melanoma, miosis, pigment dispersion, uveitis, blood–ocular barrier incompetence</td>
</tr>
<tr>
<td>VITREOUS</td>
<td>Liquification</td>
</tr>
<tr>
<td>RETINA</td>
<td>Age-related macular degeneration</td>
</tr>
</tbody>
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EXPOSURE FACTORS

PARTICULAR EXPOSURE FACTORS AND NEWLY UNDERSTOOD HAZARDS

- The intensity of ambient UV exposure is a function of solar angle, which varies with time of day, time of year, and latitude. Physical surroundings can increase ambient UV through reflection; and heavy cloud cover can decrease UV.

- UV is greater at higher altitudes, where there is less atmosphere to absorb or reflect incoming UV.

- UV exposure and associated eye diseases are expected to increase over the next few decades due to depletion of the ozone layer.

- Nearly half of the UV that reaches the eye comes from exposure to scattered or reflected light.

- Over 40% of the annual UV dose is received under conditions when people are less likely to wear sunglasses (Table 2).

- Peripheral light focusing increases the deleterious effect of reflected UV.

- At most times of the year (and in most locations) the greatest ocular sun exposure occurs in the early morning and late afternoon rather than at solar noon.

- Conventional sunglasses do not provide protection against side exposure.

- UV reflection from the back surface of anti-reflective ophthalmic lenses is a newly recognized hazard.

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.⁶⁰

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>
<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.

**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.*

*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.

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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.
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.⁶⁴)

**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.⁶⁵

**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.⁶⁶ Highly reflective substances, such as fresh snow, reflect as much as about 90% of incoming UV back into the atmosphere (Table 3A&B).⁶⁷,⁶⁸ Sand can reflect between 8% and 18% of incident UV, water from 3% to 13%, and lawn grass from 2% to 5%.⁶⁷

**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.⁶⁹ 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.⁷⁰-⁷²

### 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.⁶⁰ 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.⁷³

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 ridge shield the eye from the incident overhead light.⁷⁴

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**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°</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.

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**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>
<td>52</td>
<td>48</td>
</tr>
</tbody>
</table>
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). The maximum PLF effect at the limbus has been shown to occur when the angle of incidence is 104 degrees from the visual axis. While limbal stem cells are normally protected from direct UV exposure, PLF concentrates sunlight at the nasal limbus by a factor of 20 times.

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

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. 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.

NEW RESEARCH IDENTIFIES DISTINCT TIMES FOR PEAK UV EXPOSURE TO THE EYE

In their recent work, Sasaki and colleagues provide a clear demonstration of the relationship between solar angle and ocular UV exposure. Using a specially designed mannequin equipped with UV sensors, the group measured ocular UV exposure as a function of time of day in September and November in Kanazawa, Japan.

Surprisingly, they found that the level of UV entering the eye in the early morning (8:00 AM to 10:00 AM) and late afternoon (2:00 PM to 4:00 PM) is nearly double that of midday hours (10:00 AM to 2:00 PM) at most times of the year (Figure 5). When measured by a sensor on top of the skull, UV exposure rises and falls in parallel with the solar altitude. A sensor positioned at the eye, however, typically finds peak exposure times before and after solar noon. This suggests that, although it is widely believed to be the case, maximum ocular UV exposure may not occur at solar noon, and we very likely need to rethink our strategies about when is most important to protect the eyes from sunlight.

Figure 5 Change of UV intensity in the eye over time during the day.
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 transmission 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

**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

**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.
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**WHAT MOUNTAINEERS’ EYES TELL US**

A study of 96 alpine mountain guides was conducted in Chamonix, France. In the study, the high-mountain guides’ eyes were compared to those of people who, although living in the Alps, spent much less time at high altitudes. The goal was to compare ocular damage from sunlight exposure in the two groups, the assumption being that more time at significantly higher altitudes would equate with elevated UV exposure.

The study showed a significantly higher incidence of pterygium, pinguecula, and cortical cataract among the guides than in the age-matched group of locals who kept to lower altitudes, providing additional evidence for the critical role of UV exposure in these diseases. The study also found that the proportion of guides with retinal drusen deposits was nearly double that of the control group.


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.

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 side. 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.

**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.
PREVENTION AND RISK REDUCTION

CURRENT STATE OF EYE PROTECTION

- The level of public awareness of the ocular hazards of UV is dangerously low; eye protection is rarely included in the general consideration of UV protection
- High-risk populations such as children and aphakic patients are not properly protected
- Few practitioners incorporate UV protection into their daily patient routines
- There is no agreed-upon system for grading the comprehensive effectiveness of eyewear and specifically UV reflection, a newly recognized hazard

APPROACHES TO IMPROVING EYE PROTECTION

- Educate the public
- Educate healthcare professionals
- Develop a simplified eye protection factor similar to the SPF
- Fill knowledge gaps

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. 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.

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.

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.

Figure 9 Percent lifetime UV dose.
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 sunwear 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 eye and skin protection are clearly needed. One example of a campaign running for over two years is The Vision Council’s extensive UV awareness campaign toward the profession.

As part of educating the public about ocular UV hazards, it will be important to eliminate misconceptions about the solar conditions that create maximum risk. That the peak ocular UV hazard occurs in the early morning and late afternoon rather than the hours just before and after solar noon is little known within the eyecare community and virtually unknown outside it. Also, few members of either the public or the eyecare professions are aware of the dangers of albedo and other limitations of sunwear. The message that must get out is not only the need for eye protection, but also what constitutes effective protection and when to use it (see Table 2).

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.
GOALS FOR THE FUTURE

A number of short- and long-term needs were identified at the meeting. In addition to education, we need tests that will allow us to assess risk and standards that will allow clinicians to prescribe and wearers to buy appropriate protective solutions. A list of identified needs follows.

- 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.

  Corneal 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 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.
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Damage of the ultraviolet on the lens

Los daños que ocasionan los rayos ultravioleta en el cristalino

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The human lens

The lens is a key refractive element of the eye which, with the cornea, focuses images of the visual world onto the retina. This is achieved by its biconvex shape, high refractive index, almost perfect transparency[1]. Lens transparency is due to the three dimensional arrangement of the lens proteins and these proteins are prone to aggregation by heating, which increases the optical density[2].

The lens is clear for the first 3 years of life and then gradually develops yellow pigments (3-hydroxy kynurenine and its glucoside). This is a protective pigment, which absorbs UV radiation and safely dissipates its energy[3]. The crystalline lens filters UV and its total transmission of visible light decreases with age as the color becomes yellower[1]. An aged lens absorbs a great part of the short wavelength region of the visible light as it contains chromophores that help absorbing the radiation[3]. The crystalline lens readily absorbs UV –A and the remaining 2% of the UV-B not absorbed by the cornea and aqueous humour[4]. It is important to protect the crystalline lens against the potential hazards of UV exposure.

As the crystalline lens ages, a process known as brunescence occurs. The lens becomes denser and more opaque, allowing less light, especially at shorter wavelengths, to reach the retina[5].

Lens transparency

The transparency of the crystalline lens depends on its avascularity, paucity of organelles, narrow inter-fibre spaces and the regular organization of its cells and proteins. At the cellular level, there is limited light-scattering by cellular organelles, which are relatively sparse in the central epithelium and displaced to the equator in the fibres, away from the light path[1].

In the lens cortex, transparency is enhanced by the high spatial order of the fibre architecture and the narrow intercellular spaces. This compensates for light-scattering caused by fluctuations of the refractive index between membranes and cytoplasm[3].
Lens growth

Lens growth is achieved by the addition of new fibres to the surface of the fibre mass over the lifespan. At a certain depth, the superficial, active, nucleated fibres lose their organelles and become transcriptionally incompetent, relatively inactive metabolically and lacking in synthetic capability[1].

Aside from the skin, the eye is the organ most susceptible to sunlight and artificial lighting–induced damage. Solar radiation exposes the eye to ultraviolet-B (UV-B; 280–315 nm), UV-A (315–380 nm), and visible light (380–780 nm)[13].

Description of ultraviolet radiation

The eye dependent on the visible light energy and can be damaged by the contiguous ultraviolet and infrared wavelengths. The conditions in which sunlight is implicated in the pathogenesis is termed the “ophthalmoheliosis”, for example, pterygium and cataract formation[4]. Exposure to UV radiation from the sun is one of the widespread risk factors for the development if cataract and various skin diseases.

The spectrum of nonionizing radiation ranges from short wavelength UV RADIATION (wavelength 100 nm) through to far infrared radiation (1 mm or 1 000 000 nm). The visible spectrum lies between 380 nm to 780 nm. Above the visible spectrum is infrared radiation, and below the visible spectrum are the shorter wavelengths of nonionizing radiation called UV radiation. Wavelengths below 290 nm are totally absorbed by the ozone layer in the stratosphere, and longer wavelengths are absorbed to a lesser extent. Thus, in nature, one does not encounter UV radiation below 290 nm, although the physical spectrum of UV radiation ranges from 100 nm to 380 nm[13].

Although UV radiation is only 5% of the sun’s energy, it is the most hazardous portion encountered by man. UV radiation has been subdivided into three bands:

UV-A or near UV (315–380 nm): Produces sun tanning (the browning of the skin due to an increase in the skin content of melanin), as well as photosensitivity reactions.

UV-B (280–315 nm): It is the sunburn spectrum and causes sunburn and tissue damage (blistering) and also associated with skin cancer.

UV-C (100–280 nm): It is germicidal and may also cause skin cancer.

UV-C, or far UV, is not commonly encountered on the earth’s surface and comes entirely from artificial sources such as germicidal UV lamps or arc welding. Furthermore, UV-B is much more biologically active than UV-A[7, 8].

The temporal side of the eye is the most vulnerable to solar UV radiation, focusing the light on the nasal part of the cornea and lens[9]. The intensity of the light, the age of the recipient, the wavelength emitted and received by ocular tissues determines the damage to the eye due to UV radiation. However, the human lens is continuously exposed to small quantities of UV exposure every day, but, if this exposure exceeds a certain level, the lens may become irreversibly damaged[10].

Exposure to UVB and UVA radiation is associated with photochemical damage to cellular systems. UV radiation can generate free radicals including oxygen-derived species, which are known to cause lipid peroxidation of cellular membranes. It has also been shown that UV can damage DNA directly, decrease mitochondrial function, and induce apoptosis. Oblique rays entering the eye from the temporal side, can reach the equatorial (germinative) area of the lens.

En el córtex del cristalino, la transparencia es acentuada por el alto orden espacial de la arquitectura de las fibras, así como por los estrechos espacios intercelulares. Esto compensa la dispersión de la luz ocasionada por fluctuaciones del índice refractivo entre las membranas y el citoplasma[24].

Crecimiento del cristalino

El crecimiento del cristalino se lleva a cabo mediante la adición de nuevas fibras a la superficie de la masa fibrosa a lo largo de toda la vida. En una cierta profundidad, las fibras nucleadas superficiales, activas, pierden sus organelos y se convierten incompententes en el ámbito transcripcional, desde el punto de vista metabólico se vuelven relativamente inactivas y carecen de capacidad sintética[11].

Además de la piel, el ojo es el órgano más susceptible a los daños ocasionados por la luz solar y artificial. La radiación solar expone al ojo a los rayos ultravioleta B (UV-B; 280–315 nm), UV-A (315–380 nm), y la luz visible (380–780 nm)[13].

Descripción de la radiación ultravioleta

El ojo depende de la energía de la luz visible y puede ser dañado por las longitudes de onda infrarroja y ultravioleta contiguous a la misma. Las condiciones en las que la luz solar participa en la patogénesis se denomina “oftalmoheliosis”, por ejemplo, la formación de pterygium y cataratas[6]. La exposición a la radiación ultravioleta del sol es uno de los factores de riesgo mayormente difundidos del desarrollo de catarata y de varias enfermedades de la piel.

El espectro de la radiación no ionizante va de la radiación UV de longitud de onda corta (longitud de onda 100 nm) hasta la radiación infrarroja lejana (1 mm ≤ 1 000 000 nm). El espectro visible se encuentra entre 380 nm hasta los 780 nm. Por arriba del espectro visible se encuentra la radiación infrarroja y por debajo del espectro visible están las longitudes de onda más cortas de la radiación no ionizante denominadas radiación UV.

Las longitudes de onda inferiores a los 290nm quedan totalmente absorbidas por la capa de ozono en la estratosfera y las longitudes de onda más largas quedan absorbidas en menor medida. Por lo tanto, en la naturaleza, uno no encuentra radiación inferior a los 290 nm, aunque el espectro físico de la radiación UV va de los 100 nm a los 380 nm[13].

Aunque la radiación UV representa solamente el 5% de la energía solar, se trata de la porción más peligrosa para el ser humano. Se ha subdividido la radiación UV en 3 bandas:

Los UV-A o ultravioleta cercanos (315–380 nm). Producen el bronceado de la piel (el bronceado de la piel debido a un aumento del contenido de melanina en la piel), así como reacciones fotosensibles.

Los UV-B (280–315 nm). Es el espectro de quemaduras de sol causando así quemaduras de sol y daños tisulares (ampollas) también está asociado con cáncer de la piel.

Los UV-C (100-280 nm). Es germicida y también puede causar cáncer de la piel.

Los UV-C o UV distantes no se encuentran habitualmente en la superficie de la tierra y provienen completamente de fuentes artificiales como las lámparas germicidas con UV o soldadura de arco. Además, los UV-B son mucho más activos biológicamente que los UV-A[7, 8].

El lado temporal del ojo es el más vulnerable a la radiación UV, al focalizar la luz en la parte nasal de la córnea y el cristalino[29].
The intraocular filters effectively filter different parts of the UV spectrum and only allow 1% or less to reach the retina\(^{11}\).

The eye is largely shielded from this by the eyelids and brow ridges. Thus, for the eye, reflection (for example, off grass, sand, or snow) and scattering (for example, from patchy cloud cover) are important sources of UV exposure, with the dose and location of the incident UV radiation (Fig. 1).

**Penetration of UV radiation to various structures of the eye**

UV radiation incident on the eye is largely absorbed by the tear film, the cornea and the lens. The cornea is transparent to visible light but absorbs a significant portion of the UV-B radiation and a very small amount of UV-A radiation. The anterior layers of the cornea (epithelium and Bowman layer) are believed to be up to twice as effective at absorbing UV-B radiation as the more posterior layers.

Ultraviolet wavelengths from 295 to 317 nm are absorbed in the aqueous humor, due to the presence of ascorbic acid. It also provides antioxidant protection from UV-induced damage to the lens surface.

The UV radiation transmission also varies from the tear film to the retina. The figure below shows the percentage of light transmitted through each ocular tissue\(^{16}\) (Fig. 2).

![Fig. 2](image)

**The incidence of cataract is high in countries with excessive sunlight.**

Yellow to brown coloration of cataracts were noted in countries with higher solar intensities due to photooxidation of proteins such as tryptophan moiety, when compared to people living in higher latitudes.

High incidence of cataracts in countries with excessive light could be because of the photochemical generation of reactive oxygen species (ROS), including superoxide and its derivatization to other potent entities such as hydrogen peroxide, hydroxyl radicals, and singlet oxygen, in the aqueous and the lens resulting oxidative damage\(^{24}\).

La intensidad de la luz, la edad de la persona, la longitud de onda emitida y recibida por los tejidos oculares determinan el daño ocular ocasionado por la radiación UV. No obstante, el cristalino humano está continuamente expuesto a pequeñas cantidades de UV diariamente, pero, si esta exposición excede un cierto nivel, el cristalino puede tener daños irreversibles\(^{12}\).

La exposición a la radiación UVB y UVA está asociada con daño fotoquímico a los sistemas celulares. La radiación UV puede generar radicales libres incluyendo especies derivadas de oxígeno, conocidas por ocasionar la peroxidación lipídica de las membranas celulares. También se ha demostrado que los UV pueden causar daños directos al ADN, disminuir la función mitocondrial e inducir apoptosis. Los rayos oblicuos que penetran el ojo desde el lado temporal, pueden alcanzar el área ecuatorial (germinativa) del cristalino. Los filtros intraoculares filtran efectivamente las diferentes partes del espectro UV y sólo permiten el paso al 1% o menos hacia la retina\(^{11}\).

Los párpados y los arcos superciliares protegen al ojo. Por lo tanto, el reflejo proveniente del césped, arena o nieve; así como la dispersión de luz a través de una cubierta nubosa entrecortada, constituyen fuentes significativas de exposición a los UV, con la dosis y ubicación de la radiación UV incidente (Fig. 1).

**Penetración de la radiación UV en varias estructuras del ojo**

La radiación UV incidente en el ojo queda ampliamente absorbida por la película de lágrimas, la córnea y el cristalino. La córnea es transparente a la luz visible pero absorbe una gran parte de la radiación UV-B y una parte muy pequeña de la radiación UV-A. Se cree que las capas anteriores de la córnea (epitelio y capa de Bowman) son dos veces más efectivas en la absorción de la radiación de UV-B con respecto a las capas más posteriores.

El humor acuoso absorbe las longitudes de onda ultravioleta de 295 a 317 nm gracias a la presencia de ácido ascórbico. También brinda protección antioxidante de los daños ocasionados por los UV a la superficie del cristalino.

La transmisión de la radiación UV también varía de la película de lágrimas a la retina. La figura a continuación muestra el porcentaje de la luz transmitida a través de cada tejido ocular\(^{16}\) (Fig. 2).

La incidencia de cataratas es elevada en países con luz solar excesiva. Se ha observado una coloración de las cataratas que va del amarillo al marrón en países con intensidades solares más elevadas debido a la foto-oxidación de las proteínas como los triptófanos cuando se hace una comparación con poblaciones que viven en latitudes más elevadas. La alta incidencia de cataratas en países con exceso de luz podría explicarse mediante la generación fotoquímica de las especies reactivas al oxígeno (ROS en inglés "reactive oxygen species"), incluyendo el superóxido y su derivación a otras entidades potentes como el peróxido de hidrógeno, radicales hidroxilos y el oxígeno singlete, en el humor acuoso y en el cristalino resultando en daño oxidativo\(^{14}\).
The inferonasal localization of early cortical cataract has been confirmed in various epidemiological and animal model studies. The germinative zone of the crystalline lens is located equatorially, this region is more sensitive to UV radiation than other parts of the crystalline lens. It is for this reason, the resultant cataract is predominantly spoke shaped[6].

Damage to the ocular tissue by UV irradiation occurs by many mechanisms such as protein cross-linking, dysfunction of enzymes, ion pump inhibition, genetic mutations, and membrane damage. Short term complaints of UV exposure include excessive blinking, swelling, or difficulty looking at strong light. UV exposure can also cause acute photokeratopathy, such as snow blindness or welders’ flash burns.

It is estimated that in Australia, where UV levels are consistently high, almost half cases of pterygium treated annually are caused by sun exposure and 10% of cataracts are potentially caused by UV radiation exposure. By the year 2050, assuming 5% to 20% ozone depletion, there will be 167,000 to 830,000 more cases of cataracts[4].

UV exposure is based on environmental conditions (altitude, geography, cloud cover, ground reflection) and factors like extent of outdoor activities[4].

Ground reflectance (p) will determine if photokeratitis will result from spending time in outdoor daylight. The “global” (whole sky) reflection, and the typical, effective actinic UV reflectance is approximately 20%. Thus walking on a concrete pavement produces nearly 10-fold more UV-effective dose to the cornea than walking over green grass. Sunlight reflection from water gives the highest natural UV exposure. It has been found in various animal models that oral administration of vitamin E had a protective action against UV radiation-induced cataract[10].

Previous epidemiological studies have shown a significant frequency of cataracts in populations that have a high annual exposure to sunlight and UV radiation[16]. Higher odds ratios for cortical cataract were found in people who spend more than 4 hours outside in the daytime during their 20s to 30s and their 40s to 50s in comparison with people who spend hardly any time outside during the day. No similar relationship was found for nuclear cataract, although smoking was found to increase the risk of nuclear opacification[17-20].

The mechanism of light damage to the eye due to UV radiation is either due to inflammatory response or due to photooxidation.

In inflammatory response, acute exposure to intense radiation causes a burn in the eye similar to sunburn that can damage the cornea, lens, and retina. The eye is immune privileged, which means that under ordinary stress its immune response is suppressed. In the presence of very intense UV and visible light (for instance, emitted from lasers), this suppression is overwhelmed. There is a release of interleukin-1, a T-cell and macrophage invasion at the site of irritation and a subsequent release of superoxide and peroxides and other reactive oxygen species, which eventually damage the ocular tissues[31].

In photooxidation, chronic exposure to less intense radiation damages the eye through a phototoxidation reaction. In this, a pigment in the eye absorbs light, produces reactive oxygen species such as singlet oxygen and superoxide, and these damage ocular tissues[31].

La ubicación inferonasal de la catarata cortical precoz se ha confirmado en varios estudios epidemiológicos y con modelos animales. La zona germinativa del cristalino se ubica en el ecuador, esta región es más sensible a la radiación UV que otras partes del cristalino. Por este motivo, la catarata resultante tiene generalmente forma radiada[6].

Los daños al tejido ocular por irradiación UV ocurren mediante toda una serie de mecanismos como por ejemplo el envenenamiento de proteínas, la disfunción de enzimas, la inhibición del bombeo de iones, las mutaciones genéticas y los daños a la membrana. Algunas dolencias expresadas poco tiempo después de la exposición UV incluyen parpadeo excesivo, hinchazón o dificultades de mirar hacia la luz intensa. La exposición UV también puede ocasionar fotokeratopatía aguda, como ceguera del esquiador o quemaduras del soldador.

Se ha estimado que en Australia, donde los niveles de UV son regularmente elevados, casi la mitad de los casos de pterygium tratados anualmente con ocasionados por la exposición solar y el 10% de las cataratas son potencialmente ocasionadas por exposición a la radiación UV. En el año 2050, si se parte del supuesto que del 5% al 20% de la capa de ozono habrá desaparecido, se contarán de 167,000 a 830,000 casos adicionales de cataratas[4].

La exposición a los UV se determina basándose en condiciones medioambientales (altitud, geografía, cobertura nubosa, reflejo del suelo) y factores como el grado de actividades realizadas en exteriores[4].

La reflectancia del suelo (p) determinará si la fotokeratitis será el resultado de las actividades exteriores durante la luz del día. El reflejo “global” (todo el cielo) y la reflectancia UV actínica efectiva es de aproximadamente el 20%. Por lo tanto, caminar en la acera de hormigón produce casi diez veces más dosis efectivas de UV a la córnea que caminar sobre césped verde. El reflejo de la luz solar en el agua es la exposición natural más elevada a los UV. Se ha observado en varios modelos animales que la administración oral de vitamina E tenía una acción protectora contra la catarata inducida por radiación UV[18].

Estudios epidemiológicos previos han mostrado una frecuencia significativa de cataratas en poblaciones con una alta exposición anual a la luz solar y a la radiación solar elevada[16]. También se ha determinado un coeficiente de probabilidad superior de cataratas corticales en personas que pasaban más de 4 horas en el exterior durante el día de los 20 a los 30 años y de los 40 a los 50, en comparación con personas que casi no pasaban tiempo en el exterior durante el día. No se encontró ninguna relación similar para las cataratas nucleares, aunque se determinó que el tabaquismo aumenta el riesgo de opacificación nuclear[17-20].

El mecanismo de daño solar al ojo debido a la radiación UV se debe o bien a la respuesta inflamatoria o bien a la foto-oxidación.

En la respuesta inflamatoria, la exposición aguda a la radiación intensa causa una quemadura en el ojo similar a la quemadura de sol que puede dañar la córnea, el cristalino y la retina. El ojo es inmunológicamente privilegiado, lo cual significa que bajo estrés ordinario su respuesta inmunitaria queda suprimida. En presencia de UV y luz visible muy intensos (por ejemplo, emitidos con láser), esta supresión queda desbordada. Se libera la interleuquina-1, se inicia la invasión de células T y macrófagos en el lugar de la irriación con la subsiguiente liberación de superóxido y peróxidos así como otras especies de oxígeno reactivo, lo cual puede ocasionar daños a los tejidos oculares[32].
Antioxidants

As the normal production of antioxidants in the eye decreases with increasing age, increasing the intake of fruits and vegetables has been suggested to replace the missing protection and have been found to retard age-related cataracts and macular degeneration. In addition, supplementation with vitamins and antioxidants, including Vitamin E and lutein, quenches phototoxicative damage, whereas N-acetyl cysteine has been shown to be particularly effective in quenching UV phototoxic damage and inflammation. Other natural products such as green tea, which contains polyphenols (epigallocatechin gallate) and Ashwagandha (root of Withania somnifera) used in traditional Ayurvedic medicine has also been shown to retard light-induced damage to the lens[19].

Lens epithelial cells are a likely target for UVB damage because they are the first cells in the lens to be exposed to UV radiation. Epithelial cells, which serve key transport functions for the entire lens, are key sites of enzyme systems that protect the lens from oxidative stress. Exposure of cells to UVB radiation induces DNA damage and triggers alterations in the synthesis of specific proteins. Thus, the lens is particularly susceptible to the long-term effects of stressors such as environmental near-UV radiation. UV absorption by human lenses increases substantially with age[21, 22].

A concentration of cortical cataract in the lower nasal quadrant of the lens was found by many reviewers[19, 23]. The bony configuration of the orbit and the most probable gaze position during peak sunlight hours suggest that the lower nasal lens region receives the greatest dose of UVB. UVB is proved to be an established risk factor for cortical cataract, due to the fact that the differential exposure by region could account for spatial variation in cataract severity[19].

Age-related cataractous changes originating in the deep equatorial cortex of the lens are most likely exacerbated by UVB exposure through mechanisms such as increased oxidative radical burden and lipid peroxidation. UVB exposure had a variable effect on cataract severity, with little to no effect in the upper nasal regions of the lens and a maximum effect in the lower regions[24].

Prevention

Guidance from the World Health Organisation at its Intersun webpage advises people to wear “wrap – around” sunglasses under many conditions[6, 12].

The use of UV-blocking contact lenses provides safe, effective, and inexpensive protection of the cornea, limbus, and crystalline lens, especially where sunglasses or hats are undesirable or impractical. Contact lenses can offer UV protection against all angles of incidences.

UV blocking contact lenses are labled as class 1 and class 2, with each of the different classes indicating the level of UV protection.

Class 1 contact lenses must block 90% of UVA (315 to 380 nm wavelengths) and 99% of UVB (280 to 315 nm wavelengths).

Class 2 contact lenses must block at least 70% of UVA and 95% of UVB radiation. Non – UV – blocking contact lenses have been documented to absorb on average, only 10% UV-A and 30% of UVB[24].

En la foto-oxidación, la exposición crónica a radiación menos intensa ocasiona daños oculares mediante una reacción de foto oxidación. En este proceso, un pigmento del ojo absorbe la luz, produce especies reactivas al oxígeno como oxígeno singlete y superóxido, los cuales dañan al tejido ocular[21].

Antioxidantes

Puesto que la producción normal de antioxidantes en el ojo disminuye con la edad, se ha sugerido que el aumento de la ingesta de frutas y verduras puede sustituir la protección que va escaseando y se ha demostrado que retrasan la aparición de la catarata asociada a la edad y la degeneración macular. Además, la ingesta de suplementos de vitaminas y antioxidantes, incluyendo la vitamina E y la luteína, contienen el daño foto-oxidativo y se ha demostrado que, por su parte, la N-acetil cisteína es particularmente efectiva para contener el daño y la inflamación foto-tóxicos de los UV.

Se ha demostrado que otros productos naturales como el té verde, que contiene polifenoles (epigallocatequín galato) y la Ashwagandha (raíz de Withania somnifera) utilizada en la medicina tradicional ayurveda, retrasan los daños que la luz ocasiona al cristalino[21].

Las células epiteliales del cristalino son una diana probable para los UVB porque son las primeras células del cristalino que se exponen a la radiación UV, con los daños consecuentes. Las células epiteliales, que realizan funciones de transporte clave para todo el cristalino son centros primordiales de los sistemas enzimáticos que protegen al cristalino del estrés oxidativo. La exposición de las células a la radiación UV induce daños al ADN y desencadena alteraciones en la síntesis de proteínas específicas. Por lo tanto, el cristalino es particularmente susceptible a los efectos a largo plazo de factores estresantes como la radiación cercana a los UV que se encuentra en el entorno. La absorción de los UV del cristalino humano aumenta significativamente con la edad[21, 22].

Se ha encontrado, en un gran número de estudios, una concentración de cataratas corticales en el cuadrante nasal inferior del cristalino[19]. La configuración ósea de la órbita y la posición más probable de la mirada durante las horas de luz solar más intensa sugieren que la región nasal inferior del cristalino recibe la mayor dosis de UVB. Se ha comprobado que los UVB son un factor de riesgo de la catarata cortical, debido al hecho de que la exposición diferencial por área puede implicar la variación espacial en la gravedad de la catarata[24].

Muy probablemente, los cambios en las cataratas asociadas con la edad y que se originan en el córtex equatorial profundo del cristalino se acentúan mediante la exposición a los UVB a través de mecanismos como la mayor carga de radicales oxidativos y la peroxidación lipídica. La exposición a los UVB ha tenido un efecto variable en la gravedad de las cataratas con poco o ningún efecto en las áreas nasales superiores del cristalino y con un efecto máximo en las áreas inferiores[24].

Previsión

Las directrices de la Organización Mundial de la Salud en su página web Intersun aconseja la utilización de gafas de sol “envolventes” en toda una serie de situaciones[6, 12].

La utilización de lentes de contacto con bloqueo de UV brindan una protección segura, efectiva y poco onerosa de la córnea, el limbo y el cristalino, particularmente en situaciones en las que el uso de gafas de sol o un sombrero o gorro no es deseable o poco práctico. Los lentes de contacto pueden brindar protección UV contra todos los ángulos de incidencia.
Diet

Sunlight-induced processes such as oxidative stress in the skin or in the eye would trigger inflammation. A protective effect for weekly consumption of fish, shellfish, drinking tea daily, and a high consumption of vegetables, in particular carrots, cruciferous and leafy vegetables and fruits, and of these in particular citrus fruits was found[6].

Above all, Public and practitioner awareness is of critical importance in advising a wrap-around sunglasses or contact lenses or a wide-brimmed hat in different situations. ❑

References- referencias

Transmission of solar radiation to and within the human eye

El espectro de la radiación solar en la superficie de la tierra se extiende desde 300 nm hasta 2500 nm, aproximadamente. Su punto máximo se sitúa en torno a los 550 nm. Fuera de esta franja, las absorciones que se llevan a cabo en la atmósfera bloquean toda la energía radiante. La concentración de ozono afecta la cantidad de absorción en las longitudes de onda más cortas de la franja de los ultravioletas (300 nm a 400 nm). La absorción por vapor de agua y dióxido de carbono se lleva a cabo en varios longitudes de onda de la franja de los infrarrojos cercanos (780 nm a 2500 nm). Debido al hecho de que el actinismo de esta longitud de onda más larga es muy pequeño, este artículo se focalizará en las radiaciones ultravioleta y visible (300 nm a 780 nm).

Toda una serie de mediciones de la composición espectral (poder radiante como función de longitud de onda) a nivel del suelo (varias altitudes) y por encima de la atmósfera han suministrado información excelente sobre los espectros solares. Toda una serie de cálculos computacionales complejos que incorporan varios de los parámetros físicos que afectan la transmisión de la radiación a través de la atmósfera suministran tablas fiables de irradiancia espectral que pueden utilizarse para calcular la irradiancia ocular correspondiente a una exposición determinada. Este artículo utiliza los espectros solares de la Publ. No CIE 851, 2.

Salvo por las ocasiones en las que el sol se encuentra muy bajo en el horizonte, la visión directa del disco solar y su aureola, extremadamente brillante, debería evitarse, y, de hecho, esto es así; e incluso, sólo debería observarse brevemente el sol bajo. Por lo tanto, se calcula el espectro solar sobre la base de una observación hacia el horizonte, en un día soleado, una masa de aire 1 y cielo despejado.

Calcular las exposiciones oculares a la radiación solar

La irradiancia difusa solar proveniente de todo tipo de cielo sobre una superficie horizontal a nivel del suelo es igual a la irradiancia global.
He also states that, although limited clouds in a particular configuration slightly increase the global irradiance, a long-term average of varied cloudiness shows that clouds should generally be assumed always to decrease global irradiance (hence, too, average sky radiance). Clear-sky conditions should be assumed when calculating retinal irradiance, thereby avoiding under-estimation.

The average radiance of the ground is $x^{-1} (0.3168)$ times the diffuse reflectance of the ground times the global irradiance.

The spectral radiance of the retina, $E_{\text{retina}}(\lambda)$, from a source with spectral radiance, $N(\lambda)$ is:\[ E_{\text{retina}}(\lambda) = N_{\text{source}}(\lambda) \times A_{\text{pupil}} \times \tau_{\text{eye}}(\lambda) / (f_{\text{eye}})^2 \]

where: $A_{\text{pupil}}$ is the area of the pupil

$f_{\text{eye}}$ is the focal length of the eye, nominally 17 mm, and

$\tau_{\text{eye}}(\lambda)$ is the transmittance of the elements of the eye anterior to the retina; it is mainly determined by absorption in the crystalline lens. Other absorptions are small enough to be ignored.

The area of the pupil is determined by calculating the luminance of the source using spectral radiances of the source from 380 nm to 780 nm.

To calculate the irradiance of the cornea, an average radiance for the scene viewed, part horizon sky, and part ground surface, is estimated. The solid angle subtense of the scene is estimated.

Transmittances of the elements of the eye

1. The cornea, aqueous, and vitreous

The cornea is about 78% water\[4\]; therefore it is a strong absorber of infrared radiation. Similar absorption in the aqueous ensures that almost no infrared radiation reaches the crystalline lens, but any that penetrates to the vitreous will be completely absorbed therein.

The reflectance of the tear film on the cornea is about 2%. It is too slowly varying with wavelength for the effect to be considered. Reflectances at interior interfaces are negligibly small.

The spectral transmittances of these three elements are high; this author does not have numerical values. The transmittance of the cornea (and probably the aqueous and vitreous, as well) rolls off below 380 nm to approach zero near 300 nm (Fig. 1).

1 - Lens of a newborn, one specimen.
2 - Average transmittances of 9 lenses, birth to 2 yrs.
3 - Average of 17 lenses, 2 to 9 yrs.
4 - Average of 27 lenses, 10 to 19 years.
5 - Average of 36 lenses, 20 to 29 years.
2. The crystalline lens

The crystalline lens is the strongest absorber of ultraviolet and visible radiation. Barker and Brainard\[5\] measured direct (visual axis) transmission of 1.4% (Table 1). D. Dehner\[6\] measured direct transmission of 1.0% (Table 1).

Transmittances of the elements of the eye

1. La córnea, el humor acuoso y el humor vítreo

La córnea está constituida de aproximadamente 78% de agua\[4\]; por tanto, es un gran absorbente de la radiación infrarroja. Una absorción similar en el humor acuoso asegura que prácticamente ninguna radiación de infrarrojos alcanza al cristalino, pero en el caso de que penetre alguna cantidad en el humor vítreo, ésta quedará absorbida por el mismo.

La reflectancia de la película lagrimal de la córnea es de aproximadamente del 2%. Esta varía con demasiada lentitud con la longitud del onda para que el efecto se tome en consideración. Las reflectancias en interfaces interiores son insignificantes.

Las transmittancias espectrales de estos tres elementos son elevadas y este autor no tiene valores numéricos. La transmittancia de la córnea (y probablemente el humor acuoso y vítreo también) se sitúa por debajo de los 380 nm para alcanzar cero cerca de los 300 nm (Fig. 1).

1 - Cristalino de un recién nacido, un espécimen.
2 - Media de transmittancias de 9 cristalinos, del nacimiento a los 2 años.
transmittances of excised eyes. Their report details spectral transmittances from 200 nm to 2500 nm and reports averaged spectral values by age groups: birth to 2 yrs; 2-9 yrs; 10-19 yrs; 20-29 yrs; and by decades to 90-99 yrs. Above 20 years of age, ultraviolet transmittances below 380 nm are less than 1%. There is a “window” around 320 nm in younger eyes. Figure 1 shows five spectra of the average transmittances, 300 nm to 400 nm. A peak transmittance of 21% at 320 nm, for one of the eyes, at birth, is listed.

Figure 2 shows average transmittances, 380 nm to 700 nm, for four decades of age: 2 – 9 yrs; 20 – 29 yrs; 40 – 49 yrs; and 70-79 yrs (Fig. 2).

1 – 2 to 9 yrs.
2 – 20 to 29 yrs.
3 – 40 to 49 yrs.
4 – 70 to 79 yrs.

Infrared transmittances are about 70%, 700 nm to 1350 nm; there is a very strong absorption band (water), 1350 nm to 1500 nm, after which transmittances range over 5% to 20%, and are essentially zero beyond 1900 nm. Average infrared transmittances do not vary appreciably with age.

Solar spectral irradiances and radiances

Global and direct solar spectral irradiances on a horizontal surface at sea level for an Am-1 sun and clear sky were used to calculate, in accordance with the procedures described in Clause 2, the diffuse irradiance from the whole sky, the average radiance of the sky, and the radiance of the horizon sky. Using the stated diffuse reflectance of the ground surface (20%), which affects the global irradiance, the spectral radiances of the ground were calculated. These results are displayed in Figure 3. From an analysis not shown in this report, a multiplier was determined for converting irradiances and radiances at sea level to their corresponding values at 3 km altitude. Curve 7 of figure 3 represents the radiance of the horizon sky at 3 km; it corresponds closely with curve 3 of figure 3.

1 – Direct irradiance on horizontal surface.
2 – Global irradiance.
3 – Irradiance from whole sky diffuse radiation.
4 – Average radiance of sky.
5 – Radiance of horizon sky.
6. – Radiance of ground
7 – Radiance of horizon sky at 3 km altitude.

Irradiance of the retina by radiation from the horizon sky at sea level

The spectral irradiances (μW cm⁻² nm⁻¹) of the retina over the wavelength range 380 nm to 700 nm are shown in figure 4. The diameter of the

El cristalino es el mayor absorbente de las radiancias ultravioleta y visible. Barker y Brainard(3) han podido medir transmisiones directas (eje visual) en ojos extirpados. En su informe se pormenorizan las transmisiones espectrales de 200 nm hasta los 2500 nm e incluye datos de los valores espectrales con las medias por grupo de edad: del nacimiento a los 2 años de edad; de 2 a 9 años; de 10 a 19 años; de 20 a 20 años y por décadas hasta los 90 a 99 años. Más allá de los 20 años de edad, las transmisiones ultravioleta por debajo de los 380 nm son inferiores al 1%. Existe una “ventana” alrededor de los 320 nm en los ojos más jóvenes. La figura 1 muestra cinco espectros de las transmisiones medias, 300 nm a 400 nm. Figura en la lista una transmision pico del 21% a los 320 nm en uno de los ojos, al nacimiento.

En la figura 2 se muestran las transmisiones medias, 380 nm a 700 nm, de cuatro décadas de edad: 2 - 9 años; 20-29 años; 40-49 años y 70-79 años (Fig. 2).

1 – 2 a 9 años.
2 – 20 a 29 años.
3 – 40 a 49 años.
4 – 70 a 79 años.

Las transmisiones de los rayos infrarrojos son aproximadamente del 70%, 700 nm a 1350 nm; existe una franja de muy fuerte absorción (agua), 1350 nm a 1500 nm, después de que las transmisiones sean superiores al 5% y hasta el 20% y son esencialmente de cero más allá de los 1900 nm. Las transmisiones medias de los infrarrojos no varían de manera apreciable con la edad.

Irradianzas y radianzas espectrales solares

Para calcular, de conformidad con los procedimientos descritos en la Cláusula 2, la radianza difusa de todo el cielo, la radianz media del cielo y la radianz del cielo del horizonte, se han utilizado las radianzas espectrales solares directas y globales sobre una superficie horizontal a nivel del mar a Am-1 con cielo claro y soleado. Se calcularon las radianzas espectrales del suelo utilizando la reflectancia difusa establecida de la superficie del suelo (20%), que afecta a la radianza global. Esos resultados se muestran en la figura 3. Del análisis n, que no figura en este informe, se determinó un multiplicador para convertir las radianzas e irradianzas a nivel del mar con sus valores correspondientes a 3 km de altitud. La curva 7 de la figura 3 representa la radianza del cielo en el horizonte a 3 km; se corresponde muy de cerca con la curva 3 de la figura 3.
pupil, 1.74 mm, was determined by calculating the luminance of the horizon sky at sea level. The spectral transmittances of the lens were the averages for the age-group, 10 – 19 years, from[5]. Because of the very small spectral transmittances of teen-age and adult lenses, ultraviolet irradiances of the retina are usually negligibly small for solar radiation when direct viewing of the solar disc is excluded.

Fig. 4 Spectral irradiances ($\mu W \text{ cm}^{-2}$) 300 to 700 nm, of the retina by radiation from the horizon sky, 1.74 mm pupillary diameter, using the average spectral transmittances of lenses in the age group 10 to 19.

La irradiancia de la retina con la radiación del horizonte a nivel del mar

Se muestran en la figura 4 las irradiancias espectrales de la retina ($\mu W \text{ cm}^{-2}$) por encima de la longitud de onda el rango de 380 nm a 700 nm. Se ha determinado el diámetro de la pupila, 1.74 mm, mediante el cálculo de la luminancia del cielo mirado hacia el horizonte a nivel del mar. Las transmittancias espectrales del cristalino eran las medias en el grupo de edad 10 - 19 años[5]. Debido al hecho de que las transmittancias espectrales de los cristalinos de adolescentes y adultos son muy pequeñas, las irradiancias ultravioleta de la retina son habitualmente insignificantes para la radiación solar cuando se excluye la visión directa de disco solar.

Fig. 4 Irradiancias espectrales ($\mu W \text{ cm}^{-2}$) 300 a 700 nm de la retina por radiación desde el horizonte, diámetero de la pupila 1.74mm, utilizando las medias de transmisiones espectrales de los cris-talinos en el grupo de edad de 10 a 19 años.

references- referencias

BLUE LIGHT HAZARD: New Knowledge, New Approaches to Maintaining Ocular Health

REPORT OF A ROUNDTABLE
March 16, 2013, New York City, NY, USA

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SUMMARY

Short wavelength visible light, the spectrum from 380 to 500 nm that includes violet, indigo, blue, and some blue-green light, plays a paradoxical role in health and vision. Not only is blue light essential for color vision, recent research has found that light in this band triggers critical physiological responses, including pupil constriction and circadian rhythm synchronization. However, blue light may also be damaging to the eye, and the term “blue light hazard” has been coined to describe the danger this light presents to critical structures within the eye.

Blue light can induce formation of toxic reactive oxygen species that cause photochemical damage, leading to the death by apoptosis first of critical retinal pigment epithelial (RPE) cells and then photoreceptors. This slow process, in which damage accumulates over a lifetime, has been implicated in the pathogenesis of retinal degenerative diseases such as age-related macular degeneration (AMD).

The fact that blue light is both beneficial and toxic raises a critical question: Can we protect the eye from harmful blue light without simultaneously denying it the physiologically necessary blue light? One way to accomplish this would be with a lens that selectively filters out the harmful wavelengths while transmitting the beneficial ones. Recent work has enabled this by more fully defining the range of harmful blue light.

To determine whether specific bands within the blue-violet spectrum are responsible for blue light’s phototoxic effects on the RPE, researchers from Essilor’s Paris research and development laboratories joined forces with scientists from the Paris Vision Institute to develop a unique illumination system that allowed cultured porcine retinal cells to be exposed to narrow (10-nm) bands of light at moderate irradiances normalized to typical retinal sunlight exposure. Using this test system, it was discovered that RPE phototoxicity was concentrated in a relatively narrow band, with little overlap of the wavelengths necessary for the beneficial physiological effects of blue light. This finding paved the way for selective photofiltration: the creation of lenses that reduce the level of exposure to the harmful portion of the blue-violet spectrum while permitting the rest of the visible spectrum to enter the eye at a normal level. Thus, the eye’s necessary visual and non-visual functions can be maintained while exposure to hazardous wavelengths is reduced.

With the creation of Crizal® Prevencia™ No-Glare lenses, Essilor has turned this concept into a reality. These lenses reduce exposure to ultraviolet (UV) light — coming from in front or reflecting off the back surface of lenses — and they attenuate the harmful wavelengths of blue light. Because they reduce (but don’t fully block) transmission of just a narrow band of blue-violet light, excellent color transmission, as well as transparency, are maintained, providing superior clarity of vision. Because the damaging effects of blue-violet light are cumulative, wearing Crizal® Prevencia™ No-Glare lenses may help protect the eye by reducing lifetime exposure to harmful UV and blue-violet light. With more and more clinicians prescribing spectacle lenses from the chair, Crizal® Prevencia™ No-Glare lenses provide a helpful tool for patients to protect themselves from UV and the harmful wavelengths in the blue-violet spectrum.
INTRODUCTION

The human eye is adapted to life in a world of light. Sunlight not only enables vision, it triggers essential physiologic functions, including circadian entrainment (synchronization of internal circadian rhythms) and the pupillary light reflex.1 But along with its many beneficial effects, sunlight exposure can also bring harm to both skin and eyes—the spectrum of optical radiation spans a wide range of wavelengths, not all of which are benign.

The eye is subject to injury from both acute and long-term exposure to solar and man-made optical radiation. The serious dangers that UV radiation presents to both eyes and skin are well established. Now, mounting evidence has alerted scientists and clinicians to the damage that long-term exposure to blue light may cause to retinal photoreceptors.

With this in mind, Essilor formed an expert panel that met in March 2013 to evaluate what is known about blue light hazard and the means of ocular protection available. This report, which summarizes the roundtable discussion, will:

- Provide an overview of the interaction between light and the eye;
- Describe the current understanding of the role blue light plays in health and vision;
- Review the present state of knowledge about blue light hazard and the mechanisms by which blue light may damage retinal cells;
- Discuss a recent research study identifying a specific, narrow band of blue light that is phototoxic to the retinal pigment epithelium cells; and
- Introduce a new spectacle lens solution that for the first time offers a way to reduce exposure to both UV and damaging blue light without affecting either color vision or blue light’s beneficial effects.

LIGHT AND THE EYE

Optical Radiation

The electromagnetic spectrum has three bands of what is termed optical radiation: UV encompasses wavelengths from 100 nm to 380 nm; visible light comprises radiation between 380 nm and 780 nm; and infrared (IR) consists of wavelengths from 780 nm to 10,000 nm (Figure 1). These can all be further divided into sub-bands. Within the UV spectrum there is UVA (315 nm to 380 nm), UVB (280 nm to 315 nm), and UVC (100 nm to 280 nm)2; the IR spectrum contains IRA (780 nm to 1,400 nm), IRB (1,400 nm to 3,000 nm), and IRC (3,000 nm to 10,000 nm); and the visible light spectrum can be generally classified as short- (blue), medium- (green), and long-wavelength (red) light.2

Visible light, like all electromagnetic radiation, has energy; the amount of photon energy is a function of wavelength, with shorter wavelengths being most energetic. Thus, blue-violet light is the highest-energy band of the visible spectrum.

Light Absorption in the Eye

Visual perception occurs when light strikes the retina, an intricate structure of highly specialized cells that form the innermost layer of the globe. Before reaching the retina, incoming light must penetrate the ocular media, the transparent tissues and fluids that lie between the front of the eye and the retina. The ocular media—consisting of the cornea, aqueous humor, lens, and vitreous humor—either absorb or transmit light, depending on its wavelength.

Almost all of the UV that reaches the eye is absorbed by the cornea or the crystalline lens, so that in adult eyes only 1% to 2% of incoming UV is transmitted to the retina.3 The cornea and crystalline lens also block IR above 980 nm; and the vitreous absorbs the IR above 1400 nm that is not absorbed by the lens. The net result of light filtering by the ocular media is that the retina is exposed almost exclusively to the visible portion of the solar spectrum (Figure 2).

Light Transduction: the Visual Cycle

Visual function depends on two types of photoreceptors within the retina: rods and cones. Required for scotopic vision, rod vision lacks color information and is characterized by high sensitivity but low resolution. Highly concentrated in the center of the macula, cones enable both sharp image resolution and color detection.

Rods and cones in the retina initiate the visual process when...
visual pigments absorb photon energy and convert it into neural signals. This biological conversion of light to electrical signals is supported by an enzyme-mediated process called the "visual cycle" that allows efficient reuse of key chemicals in the reaction.

The visual pigments that initiate the process are made up of an opsin combined with the chromophore 11-cis-retinal. The important photochemical reaction is the conversion of the 11-cis-retinal to all-trans-retinal, caused by photon energy striking the pigment. This changes the shape of the retinal molecule, breaking its connection with opsin and leaving the opsin free to initiate a series of reactions that leads to a neural signal and ultimately to vision.

In the meantime, the all-trans-retinal is converted to all-trans-retinol and transported to the retinal pigment epithelium (RPE) where it is either stored or reconverted to the 11-cis-retinal form to transport back to the photoreceptors. There it can recombine with opsin to complete the visual cycle (Figure 3).

The visual cycle takes place within the outer segment of the rods and cones and in the RPE cells. The RPE cells are not photoreceptive, but they are essential to the regeneration of visual pigments and also play a critical role in the survival and normal function of photoreceptors. With microvilli on their apical surfaces interdigitating with the outer segments of photoreceptors, the RPE cells supply the photoreceptors with nutrients and oxygen. They also help maintain the homeostasis of photoreceptors by phagocytosis and digestion of oxidized photoreceptor outer segments.

**Light Damage in the Eye**

Although light is essential to vision, light exposure can also cause pathological changes to ocular tissues through absorption of photon energy. When absorbed, photon energy can be dissipated and normal function of photoreceptors. With microvilli on their apical surfaces interdigitating with the outer segments of photoreceptors, the RPE cells supply the photoreceptors with nutrients and oxygen. They also help maintain the homeostasis of photoreceptors by phagocytosis and digestion of oxidized photoreceptor outer segments.

**COMMENTARY: An Insurance Policy for the Eyes**

Short wavelength visible light, particularly violet and indigo, reaches the retina in substantially greater doses than does ultraviolet (UV) radiation. Indeed, the conditions associated with UV exposure are generally confined to the anterior segment of the eye, due to nearly complete absorption of UV by the crystalline lens.1

When we think about how light interacts with the molecules that compose living cells and tissues, what concerns us is photon energy, which is inversely correlated with wavelength. At a 400-nm wavelength, for example, photons are much more energetic and have a greater potential to alter the molecules they strike than photons at 500 nm. Light at wavelengths in the neighborhood of 400 nm consists of the highest-energy photons to reach the retina, and there is reason for concern about this high-energy light’s effects there.

**The “Blue-light Hazard”**

The most certain impact on retinal health and vision from exposure to higher-energy visible (indigo and blue) light is acute phototoxicity, as seen in humans who stare directly at an arc lamp or the sun. It is established that this damage is photochemical, not thermal, and studies in primates have made it possible to define the action spectrum for this type of damage, which peaks around 440 nm.2

It is certainly reasonable to suppose that over the long term, and especially as aging changes erode cellular defense mechanisms, retinal exposure to high-energy light could have a damaging effect. Many in vitro studies, including those detailed in this report, have helped us to understand the photochemical and cellular mechanisms by which this damage occurs.

Visual pigment, retinoids, and bisretinoids (in particular A2E, a major photosensitive component of lipofuscin) have been implicated in photochemical damage to the outer retinal layers, and additional not-yet-identified chromophores may also act in this way. High energy-visible light exposure also induces oxidative damage, to which retinal cells are especially vulnerable.3

**Challenges to Research**

Corresponding epidemiological studies examining the link between light exposure and AMD have been less conclusive, in part because of the difficulties of conducting such studies. For example, the dosimetry necessary to conduct a conclusive epidemiological study of light exposure and AMD is extremely challenging. Two otherwise similar people, standing side by side at a beach and facing the same direction may easily have significantly different pupil sizes and lid-openings, and therefore different levels of retinal light exposure. But epidemiological studies tend to assume that two such people’s retinas would receive the same light dose.

In addition, much of the data on which these epidemiologic studies rely is retrospective, and thus subject to the vagaries of memory. I can’t say for certain...
as heat and/or trapped via a photochemical reaction. Acute exposure to intense light can cause thermal injury (e.g., skiers’ photokeratitis), while lower levels of exposure may, over a lifetime, cause the slow accumulation of harmful photochemical waste products that lead ultimately to cell death.

It is well established that solar UV is hazardous to ocular health. Chronic exposure to solar UV has been shown to increase the risk of developing pterygium, cataract, and a variety of other ophthalmic conditions. But because UV is almost fully absorbed by the ocular media before reaching the retina, the harmful effects of UV radiation are concentrated in the cornea and the crystalline lens. However, scientific findings on blue light suggest that fully protecting the eyes from light damage requires more than just blocking UV.

**Blue Light: Concept and Sources**

In the visible spectrum, wavelengths between 380 and 500 nm include violet-, blue-, and green-appearing wavelengths. This portion of the spectrum is also known as high-energy visible (HEV) light because of the high photon energy associated with these short wavelengths.

The sun is the primary natural source of blue light, but human beings are also increasingly exposed to blue light from artificial sources, which vary widely in spectral distribution. Solar radiation is 25% to 30% blue light, depending on the reference solar spectrum; and while conventional incandescent lamps emit very little blue light (about 3%), newer artificial light sources produce a considerably higher amount of blue light (Figure 4). Approximately 26% of the light from the energy-efficient and increasingly popular compact fluorescent lamps is in the blue portion of the spectrum; and the 35% of the optical radiation from cool white spectrum; and the 35% of the optical radiation from cool white light-emitting diodes (LEDs) is blue.4

### Figures

**FIGURE 4.** Spectral distribution of different light sources.

The sun is the primary natural source of blue light, but human beings are also increasingly exposed to blue light from artificial sources, which vary widely in spectral distribution. Solar radiation is 25% to 30% blue light, depending on the reference solar spectrum; and while conventional incandescent lamps emit very little blue light (about 3%), newer artificial light sources produce a considerably higher amount of blue light (Figure 4). Approximately 26% of the light from the energy-efficient and increasingly popular compact fluorescent lamps is in the blue portion of the spectrum; and the 35% of the optical radiation from cool white light-emitting diodes (LEDs) is blue.4

**Increased Exposure?**

While there is a global trend toward more energy-efficient lighting with LED and compact fluorescent lamps, consumer preference in the US has not favored those blue-rich light sources. Here, the bigger concern may be with modern, higher-luminance displays (computer monitors, smartphones, and tablets) which are blue-rich and virtually ubiquitous. It is unclear what long term effect this increased exposure to short-wavelength light will have on us; but it is certainly cause for further study and for taking some steps to reduce needlessly high exposures to short wavelength light. Therefore, lenses designed to reduce violet light exposure and accomplish this without interfering with vision and circadian function, seem like a very reasonable insurance policy.

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**REFERENCES**

spectrum; and the 35% of the optical radiation from cool white light-emitting diodes (LEDs) is blue.4

**BLUE LIGHT IN HEALTH AND VISION**

UV and visible light have long been observed to cause phototoxic damage to retinal photoreceptors and RPE cells.5,7 Since the anterior structures of a healthy eye naturally protect the retina from UV, retinal phototoxicity is primarily due to photochemical damage induced by the cumulative effects of long-term exposure to visible light, in particular blue light.

Being in the most energetic portion of the visible spectrum, blue light has the greatest potential to induce the photochemical damage that may ultimately be a factor in retinal disorders such as age-related macular degeneration (AMD).8,10 On the other hand, blue light is important to visual processes including color perception. More recent research has also demonstrated that blue light plays an essential role in non-visual functions, such as circadian entrainment and the pupillary light reflex.1,12,13

**Blue Light is Vital for Life**

These non-visual functions depend on a newly discovered third photoreceptor type that exists along with the rods and cones. Called intrinsically photosensitive retinal ganglion cells (ipRGCs), these cells contain melanopsin, a photopigment, and, unlike cone cells, they are not concentrated in the fovea. Instead ipRGCs form a photoreceptive network broadly across the inner retina.13 Because melanopsin is so important to the daily resetting of our biological clocks, the absorption spectrum of melanopsin is sometimes called the chronobiological spectral band. This band peaks at about 480 nm, within the blue range.15

The ipRGC response to light in the chronobiological band regulates many non-visual physiologic functions in the human body, including circadian entrainment, melatonin regulation, pupillary light reflex, cognitive performance, mood, locomotor activity, memory, and body temperature.13,14 Studies have shown that pupil constriction, the eye’s natural defense against exposure to strong light, is wavelength-dependent and peaks at 480 nm.14,15 The exact physiology by which ipRGCs control these functions have not been fully elucidated.

What is clear, however, is the essential role that blue light plays in daily life. Thus, simply filtering out the entire blue spectrum in order to reduce the “blue light hazard” may interfere with the physiological functions driven by the reaction between ipRGCs and light in the chronobiological band. Indeed, one recent study has shown that blocking light at 470 nm could disrupt the sustained phase of the pupil constriction reflex.17

**Blue Light Phototoxicity**

Blue light damage occurs when a photosensitizer absorbs photon energy of a specific wavelength, setting in motion a series of intracellular chemical reactions. Rods, cones, and RPE cells of the outer retina—the cells responsible for photon absorption and visual transduction—are rich in photopigments and therefore susceptible to photochemical damage.

Blue light can cause damage to both photoreceptor and RPE cells in primates.18 Cumulative exposure to light in the 380 nm to 500 nm range can activate all-trans-retinal accumulated in the photoreceptor outer segments (Figure 5).19 This blue light photostimulation of all-trans-retinal can induce production of reactive oxygen species (ROS), such as singlet oxygen, hydrogen peroxide, and other free radicals, in the photoreceptor outer segments.

The ROS attack many molecules, including polyunsaturated fatty acids, a major component of cell membranes. The large concentration of cell membranes in the retina makes it highly sensitive to oxidative stress. In particular, this stress may disrupt the membranous structures of the photoreceptor outer segments, causing incomplete phagocytosis and digestion of oxidized outer segments in the RPE. The consequence is an accumulation of the waste product lipofuscin in RPE cell granules.

In the eye, lipofuscin, also known as “the age pigment,”

![Figure 5. Phototoxicity mechanisms in outer retina.](image)

![Figure 6. Light transmittance of clear ocular media in aging human phakic eye.](image)
accumulates over the years and builds up at a faster rate in some retinal diseases.20 Composed of lipids, proteins, and a number of chromophores, lipofuscin is highly susceptible to photochemical changes that can produce permanent cellular damage.21 Lipofuscin accumulation has been implicated in the pathogenesis of AMD, and intense lipofuscin autofluorescence is frequently observed in regions surrounding the leading edges of geographic atrophy lesions in the retina.22

A2E (N-retinylidene-N-retinylethanolamine) is a key photosensitive fluorophore that mediates lipofuscin phototoxicity.23,24 (A fluorophore is a chromophore that can re-emit light after excitation.) With maximum absorption at around 440 nm, A2E is excited by blue light.19 The photosensitization of A2E leads to the formation of ROS and to an inhibition of lysozyme’s ability to break down cellular structures for recycling.25,26

Excessive oxidative stress can cause dysfunction in the RPE cells and, eventually, cell death by apoptosis. Without the supportive functions of the RPE, photoreceptors cannot function properly and will degenerate as well. Lipofuscin accumulation and A2E photosensitization are involved in this cascade of phototoxic effects, which has been implicated in the pathogenesis of AMD.20

**Aging and Susceptibility to Phototoxicity**

Retinal changes associated with age have significant influence over the potential for photodamage. As the eye ages, light transmission and absorption change, primarily owing to the gradual yellowing of the crystalline lens. As a result, the aging lens transmits less visible light overall, with a disproportionate drop in transmission of blue light due to yellow discoloration of the lens (Figure 6).27-28 But even though it decreases with age, the level of blue light transmitted to the retina remains significant throughout life. Early in life, blue represents about 20% of the visible light reaching the eye; by 70 years, the level of exposure drops.29

Some blue light transmission and absorption change, primarily owing to the gradual yellowing of the crystalline lens. As a result, while less blue light reaches the retina in elderly eyes, the natural defenses and repair mechanisms simultaneously become less effective. The aging retina therefore remains susceptible to photochemical damage from blue light, even as its level of exposure drops.

**Link with AMD**

AMD, a degenerative retinal disease that affects the photoreceptors, the RPE, Bruch’s membrane, and the choroid, is a leading cause of legal blindness among people over age 65.36,37 AMD is responsible for about half of severe visual loss (defined as visual acuity of 20/200 or worse) in Caucasian Americans over age 40.37

With the elderly population growing, AMD is rapidly becoming a major public health concern. By 2020, the number of Americans with early-stage AMD is expected to double from 9.1 million to 17.8 million.38 Extrapolations from current trends indicate that the AMD population worldwide will grow to between 100 and 200 million people over the next 30 years.

Multiple factors increase a person’s risk of developing AMD,
including age, tobacco use, genetic factors, and an antioxidant-deficient diet. Blue light exposure, owing to its impact on lipofuscin accumulation and A2E-mediated phototoxic effects, has come to be considered another potential risk factor.

Several epidemiological studies have found evidence of a relationship between chronic sunlight exposure and AMD. The Beaver Dam eye study found that levels of sun exposure in the teen and early adult years were strongly associated with a higher risk of developing retinal pigment abnormalities and early AMD. In the Chesapeake Bay Waterman Study, a group of subjects with advanced AMD had high levels of blue light exposure over the preceding 20 years. Recently, the European Eye (EUREYE) Study reported a significant association between lifetime blue light exposure and AMD in individuals with low dietary levels of antioxidants (including vitamins C and E, zeaxanthin, and dietary zinc).

### Breakthrough Science

The potential connection between blue-light phototoxicity and retinal diseases such as AMD suggests that reducing blue-light exposure would be beneficial to long-term ocular health. Although research in animal models and in-vitro experimental settings has generated substantial evidence that blue light can cause cellular damage to photoreceptors and RPE cells, the wavelengths within the blue-violet spectrum responsible for this damage have not been as precisely identified until now.

Eyes could be protected by simply blocking all blue light (as yellow “blue blocking” glasses aim to do), but this solution distorts color, has unwanted cosmetic effects, and eliminates the physiologically critical light in the chronobiological band. Selective blocking of the hazardous wavelengths (and just those wavelengths) required investigation to determine just what those wavelengths are.

To delineate the damaging bands within the blue-light spectrum, research scientists from Essilor partnered with the Paris Vision Institute (Paris, France) to create an in vitro model for the study of retinal phototoxicity.

**COMMENTARY: Preventive Eyecare — Lens Technology Gets Specific**

**DIANA L. SHECHTMAN, OD, FAAO**

The role of ultraviolet (UV) radiation in the pathogenesis of ocular conditions like cataract, pterygium, and UV keratopathy is well known. Most of the UV incident upon the eye is absorbed by the cornea and crystalline lens, and is thus associated primarily with conditions of the anterior segment. On the other hand, high energy blue-violet visible light, lying just outside the UV band, typically passes through the cornea and lens. Thus, this light is the highest energy visible light to reach and affect the posterior segment.

While it has been challenging to accurately measure and prove a causal link between age-related macular degeneration (AMD) and long term retinal light exposure, there is evidence that long term sunlight exposure is one of the risk factors contributing to AMD.

AMD can have a devastating effect on a patient’s vision and quality of life. Anti-VEGF therapy and AREDS-type supplements have been used to manage patients with AMD, but these options do not provide a cure or restore vision to its pre-morbid state. It would be far better to find effective ways to reduce the risk of developing AMD in the first place.

The need for good preventive measures is given urgency by the rapid growth of the elderly population and the prevalence of AMD within that population. In addition, exposure to high energy blue light is likely to increase significantly as people convert from incandescent and halogen lighting to compact fluorescent lights and LEDs, which produce a far higher proportion of blue light. In addition, the proliferation of digital screens in use today has caused an increase in our exposure to blue wavelengths. The impact of this increase is potentially concerning, though further studies are warranted.

Recently, research by Essilor in collaboration with the Paris Vision Institute has contributed to the growing body of evidence surrounding the mechanism of blue-light mediated retinal damage. Their study isolated the specific narrow band of blue-violet light (435 nm ± 20 nm) that contributes to retinal pigment epithelium (RPE) cell apoptosis in an in vitro AMD model. Given the fact that blue light is still a necessity for color perception and physiological functions like the regulation of circadian rhythms, selectively blocking only the dangerous band(s) of blue light is critical. This discovery, and the lens technology that enables it, may prove to be a public health breakthrough.

We already counsel patients about UV exposure and offer specific lenses and filters to help protect their eyes. Further research is necessary; but lenses designed to provide optimum vision, protect against UV, and selectively block the narrow band of blue-violet light implicated in RPE apoptosis could become an important element of preventive eyecare going forward.

**REFERENCES**

New Methods

A large body of prior research had demonstrated that blue light causes phototoxic damage to RPE cells and is far more damaging to those cells than green or yellow-red light.46-49 In addition, it had been determined that blue-light-induced RPE cell death is mediated by apoptotic, rather than necrotic, processes.46,47,50,51

These studies, however, had a number of methodological limitations. For example, the cells typically used for in vitro experiments were from immortalized RPE cell lines (rather than freshly harvested RPE cells), and the culture media were not always entirely free of visible light chromophores. Nor were the experimental light levels normalized to approximate actual physiological conditions. Most importantly, all studies prior to the joint study between Essilor and the Paris Vision Institute work used broadband blue light illumination and so were not able to define the specific toxic sub-band(s) within the blue-violet spectrum.

Knowing this, scientists from Paris Vision Institute and Essilor used their respective areas of expertise to develop improved experimental techniques and overcome the limitations of prior studies. Instead of immortalized cell lines, they employed primary cultures of swine RPE cells grown in a cell medium free of visible light-absorbing chromophores. In addition, they devised a unique illumination system that allowed them to normalize light irradiances to sunlight retinal exposure. They were able to expose the RPE cells to extremely narrow (10-nm) spectral bands (across the range from 390 to 520 nm in 10-nm increments) with tight photometric control.

Before light exposure, the RPE cells were treated with A2E at different concentrations. Because, again, A2E is a key photosensitive fluorophore in lipofuscin, A2E-loaded RPE cells are frequently used to model aging RPE cells.18,47,49,52,53 Very recently, however, some authors have challenged the A2E model, proposing instead to measure lipofuscin directly. [Ablonczy Z, Higbee D, Anderson DM, Dahrouj M, Grey AC, et al. Lack of correlation between the spatial distribution of A2E and lipofuscin fluorescence in the human retinal pigment epithelium. Invest Ophthalmol Vis Sci. 2013 Jul 11.]

The A2E-containing cells were exposed to controlled doses of light in 10-nm bands at irradiance levels mimicking sunlight retinal exposure, and RPE cell damage was assessed by measuring cell viability, necrosis, and apoptosis (Figure 9).

Results

The greatest damage followed exposure to the four 10-nm sub-bands within the blue-violet spectrum between 415 nm and 455 nm. In those test cells, morphological changes to RPE cells (cell rounding, loss of confluency, and decrease of density) were observed 6 hours after exposure (Figure 10). In addition to wavelength dependence, the toxic effect was A2E-dose dependent, with the greatest apoptosis rates occurring with 20 μM and 40 μM concentrations of A2E. In cells exposed to the narrow band...
of blue-violet light centered on 440 nm, though, there was a significant increase in apoptosis, even with 12.5 μM A2E, indicating the phototoxicity of those wavelengths.

The damage observed in the study was clearly apoptotic rather than necrotic. Irradiated RPE cells had necrosis rates no higher than those maintained in dark, irrespective of the A2E concentration, which is consistent with the experiments conducted in physiological light conditions.

**Significance of these Findings**

The A2E concentration dependence seen here demonstrates that the photodamage to RPE cells in this test system was not due simply to the high photon energy of short-wavelength blue-violet light. Rather, this apoptotic cell death represents blue-light phototoxicity specifically mediated by the photosensitizer A2E. This is significant because it provides evidence that the test system can be used as an in vitro model of the suspected mechanism of cell death in AMD.

The key learning from this series of experiments is that blue-light phototoxicity to RPE cells appears to be concentrated in a narrow band of wavelengths centered on 435 nm ± 20 nm. For the first time, the toxic wavelength range within the blue-violet spectrum has been identified in physiological sunlight conditions using an aging RPE model.

The data further suggests that selectively attenuating the hazardous portion of the blue spectrum (wavelengths from 415 nm to 455 nm) may provide protection for the retina without significantly affecting the igRGCs, whose primary action spectrum lies between 465 nm and 495 nm. This is in contrast to broad filtration of blue light (“blue blocking”), which has the potential to affect the regulation of the pupillary light reflex and other critical physiological functions. The establishment of a narrow phototoxicity spectrum paves the way for developing new ophthalmic filters that deliver selective photoprotection.

**Preventive Measures**

Given the probable role of blue-light phototoxicity in degenerative retinal diseases, selective photoprotection offers one potential means of helping eyes stay healthy longer. There may be added benefit to this in the world of blue-rich artificial light that is building around us due to the growing popularity of energy-efficient compact fluorescent lamps and LEDs.

Because these new lighting sources are more cost-efficient, energy-efficient, long-lasting, and environmentally friendly than incandescent and halogen bulbs, they are quickly becoming the next-generation light sources. By 2016, traditional incandescent light sources will, by law, no longer be available for domestic lighting in Europe.1 LEDs are also becoming progressively more popular in backlight mobile phone, tablet, television, and computer displays.

As LEDs and other blue-rich solid state light sources become more important in domestic and workplace lighting, and as people spend more and more time staring at TV, computer, and mobile phone screens, blue light exposure will gradually increase, and its ocular hazards may become more problematic.

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**Crizal® Prevencia™ No-Glare Lenses: Truly Selective Eye Protection**

**Crizal® Prevencia™ No-Glare lenses with Light Scan™ represent the first application of new patent-pending technology that enables selective attenuation of harmful light—both UV and blue-violet—while allowing beneficial light to pass through and maintaining exceptional transparency at all other visible-light wavelengths. The goal is to enable patients to enjoy the best vision with significant protection against UV and high-energy blue-violet wavelengths.**

**Crizal® Prevencia™ No-Glare lenses reduce the quantity of harmful blue-violet light (415 nm to 455 nm) reaching the eye by 20%*. Unlike common yellow-tinted “blue blocking lenses,” Crizal® Prevencia™ No-Glare lenses cause minimal color distortion—indeed these lenses are almost perfectly clear.**

The efficacy of Crizal® Prevencia™ No-Glare lenses has been demonstrated using the same A2E-loaded RPE tissue culture model used to discover the sub-band of blue-violet light that causes RPE apoptosis. When A2E-containing-RPE cells were exposed to white light that mimicked the solar spectrum, placing the new lens between the light source and the cells reduced cell apoptosis by 25% compared to no light filtering at all.46 Designed to selectively block harmful light and maintain transmittance of visible light essential to color vision as well as critical chronobiological processes, Crizal® Prevencia™ No-Glare lenses offer the most selective eye protection on the market today.

**Crizal® Prevencia™ No-Glare lenses also feature an Eye-Sun Protection Factor (E-SPF®) of 25, which means they provide 25 times more UV protection for the eye than wearing no lens at all. Integrating Essilor’s superior No-Glare technology, Crizal® lenses are easy to clean, resistant to smudges, scratches, dust, and water, and protect against distracting glare and reflections. Maintaining excellent transparency, Crizal® Prevencia™ No-Glare lenses offer optimal color vision at all times.**

*Slight differences in attenuation may occur with different lens materials.
From Science to Solution

Efforts have been made to develop prophylactic and therapeutic methods to protect retinal cells from phototoxic damage. In cataract surgery, yellow intraocular lenses that block both UV and blue light (< 500 nm) have been introduced to reduce retinal phototoxicity in pseudophakic eyes; however, the clinical value of these lenses is debatable, as they block both hazardous wavelengths and those that most effectively activate the ipRGCs.54,55

The use of small-molecule compounds is also being investigated as a treatment method to modulate the visual cycle and reduce lipofuscin accumulation in RPE cells.56,57 The most viable preventive approach, however, may simply be wearing spectacle lenses that are able to stop hazardous blue light from entering the eye.58,59 Blue-light blocking glasses have existed for years and are recommended for patients with retinal diseases; but current lenses absorb a very large portion of the blue-light spectrum, distorting colors, reducing scotopic vision and possibly interfering with nonvisual ipRGC-controlled functions. Also, the absorptive technology makes the lenses appear yellowish (absorbing blue).

Based on the discovery of the precise spectrum of RPE-toxic blue light, Essilor has developed a new No-Glare lens, Crizal® Prevencia®, a unique narrow-range blue light filter that selectively attenuates the hazardous portion of blue-violet light (415 nm to 455 nm) while remaining transparent to other wavelengths of visible light. Designed to reduce exposure to potentially harmful blue light, Crizal® Prevencia™ No-Glare lenses also protect eyes from UV light coming through the front or reflecting off the back surfaces of the lenses. This new lens can benefit everyone by reducing exposure to the phototoxic wavelengths of blue-violet light.

Optometrists and Eye Protection

There is scientific evidence to support the finding that high-energy blue light is harmful to the retina and that reducing exposure to the most toxic wavelengths of this light is likely to be beneficial.

Today, optical dispensing is becoming more doctor-driven, with optometrists no longer hesitant to discuss eyewear and make specific spectacle lens recommendations to patients in the chair. This is fortunate because the exam room is the ideal place to educate patients about the nature of blue light hazard and to explain how spectacle lenses can better protect themselves from it. In recommending selective filtering of phototoxic wavelengths, optometrists have an ideal opportunity to perform a truly beneficial function—protecting vision for a lifetime—even if the patient has simply come in for a refraction and new glasses.

This role will become ever more important as LED and compact fluorescent lighting find their way into more homes and workplaces—and as blue rich digital screens come to occupy even more of our days and evenings.

Crizal® Prevencia™ No-Glare lenses, which cut the hazardous blue light in the 415 nm to 455 nm band by 20% and provide protection from back-side UV reflection, can be beneficial for patients at all ages. It is important for clinicians prescribing Crizal® Prevencia™ No-Glare lenses to gain the support and commitment of their staff members, who can contribute tremendously to communication with patients. Once staff members understand the nature of blue light hazard and its association with ocular health, they can bolster the doctor’s recommendation and help patients understand the importance of blue-light protection for the eye.

CONCLUSIONS AND FUTURE DIRECTIONS

Certain wavelengths in the blue-violet range are now known to be detrimental to the retina, and cumulative blue-light damage is implicated in retinal disorders such as AMD. The most hazardous blue wavelengths for retinal pigment epithelium, as determined by the joint work of Essilor and the Paris Vision Institute, fall in the narrow band between 415 nm and 455 nm. This is relatively distinct from the spectral band that is responsible for critical physiological functions such as the pupillary light reflex and circadian entrainment.

For spectacle lenses to protect the retina, this means that in addition to protecting against UV wavelengths, attenuation of high-energy blue-violet light in the 435 ± 20 nm band is of value. But for normal physiologic functioning, lenses must block this light without reducing transmission in the chronobiological spectral band.

Furthermore, patient acceptance may be limited when lenses are visibly colored and distort color perception, as is the case with most blue absorber lenses. To enhance vision and support color perception, lenses should offer high transmittance of all visible light wavelengths outside the UV and phototoxic blue bands.

Crizal® Prevencia™ No-Glare lenses offer selective photofiltering and superior clarity of vision, taking blue blocking lenses and eye protection to the next level.

References

BLUE LIGHT HAZARD


THE COLOUR BLUE INSPIRES THE ARTS, blue vibrates through literature, but we really should be referring to blues: from Aragon’s *Blue sun of dreams*, and Balzac’s *Life as blue as a pure sky*, there is only a breath, a ray to tip us towards Gorki’s *Blue fires of anger* or Bobin’s *The blue of disasters seen through the window*. “Bad Blue v. Good Blue”, there’s the challenge and the focus of this latest issue of *Points de Vue*, which seeks to answer the new questions that have arisen from recent scientific discoveries and clinical observations linking the blue-violet fraction of the visible spectrum – 380 to 500nm – to the eye and vision:

- Is high energy blue harmful to ocular tissue?
- What more do we know today about the physiological roles of blue light?
- What would be the benefits for human health of suppressing some of the blue and what would be the risks of suppressing too much of it?
- Are we exposed more today to harmful blue, and if so, why?

Significant progress has been made since the mid-nineties in terms of physiopathological knowledge about the consequences of exposing the eye to various types of blue light.

Previously, and since the advent of lasers in the seventies, the scientific community and public authorities controlling radio- and photo-protection performed experiments on animals in order to establish the thermal and photochemical danger thresholds of light, mainly involving UV rays and the anterior segment of the eye. This research also involved “high energy visible light”, the blue-violet light renamed “blue light” for simplification, which is the light that potentially presents a danger of photochemical lesions in the retina. We know in fact that, except during childhood, ocular tissue filters out almost all UV rays and that it is indeed this “blue light” which is today incrimented in certain ocular pathologies.

In the nineties, progress made in cellular and molecular photobiology enabled exploration into which bands of visible light were the most harmful for the retina, which toxicity mechanisms were activated, distinguishing acute toxicity from chronic toxicity. This work was stimulated by the increased use of new intra-ocular implants that filter out blue, and also by the need to assess the risks to the retina of exploratory or eye surgery instruments.

Acute toxicity is the consequence of exposure to high intensity light over a short period, and results in thermal destruction of the retina’s cells and cell death by necrosis.

Chronic toxicity is more insidious because phototoxic mechanisms of oxidant stress lead to the accumulation of photo-sensitising components and oxidising reactive species (singulet oxygen, hydrogen peroxide, etc.) which, year after year, increase the danger to exposed cells from blue light and contribute to certain chronic ocular pathologies, such as AMD – Age-Related Macular Degeneration – or pigmentary retinopathies.
The dangers of blue light to photoreceptors have been demonstrated in animals. C. Remé and C. Grimm showed in 2000 (2) in rats that blue light, unlike green, causes photoreversal of the whitening of photoreceptors; this rapid regeneration of the rhodopsin caused by high energy blue light leads to degeneration of the photoreceptors by apoptosis. Molecular mechanisms were explored further by M. Rozanowska (3) who showed a combined role played by rhodopsin and the 11-cis-retinal and 11-trans-retinoids (“ATR” all-trans-retinal) the accumulation of which contributes to the phototoxicity mechanism on photoreceptors.

The action spectrum of light phototoxicity on RPE cells was studied by J. Sparrow and M. Boulton (4) who demonstrated the central role of lipofuscin accumulation in the amplification of photo-oxidation mechanisms, resulting in cell death by apoptosis. Death of the RPE leads, in turn, to the loss of photoreceptors, because they are inter-dependent. The granules of lipofuscin form in large numbers when the phagocytosis of the oxidised segments of photoreceptors is incomplete, which leads to cascades of inflammation and oxidant stress. Made of lipids and proteins, these granules contain a particularly photosensitising molecule, bisretinoid “A2E”, made from two ATR, which has an absorption peak in blue at around 440 nm, which explains the particular toxicity of blue light for the RPE, with a spectrum of action that does not follow the light energy level exactly. The collections of lipofuscin in the RPE increase with age, during childhood and then again after the age of 45 (fig.1), as well as in pathological conditions such as AMD or pigmentary retinopathy. Moreover, with age, ocular diseases and bad diet, the natural mechanisms of retinal defence against oxidant stress are reduced: reduced “detoxifying” enzymatic activity (catalase, SOD, etc.), reduced fixing of the macular pigment in the centre of the retina, notably of lutein and zeaxanthin, which are absorbed from food, the maximum levels of absorption and protection of which are astonishingly close to the maximum toxic absorption of A2E.

Recently, a team of photobiologists from the Vision Institute in Paris (UPMC, Inserm, CNRS), Dr Serge Picaud and Dr Emilie Arnault, under the direction of Professor José-Alain Sahel, and in collaboration with Essilor, sought to narrow the spectrum of action of blue light phototoxicity on RPE cells, by putting the cells, for the first time, in chronic toxicity illumination physiological conditions, in stages of 10nm, taking account of the spectral ratios of the solar spectrum and of filtering by ocular media. They present their work here, for Points de Vue.

### References

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Thus, all the in vitro work done confirms the dangers of cumulative exposure to a certain blue light, Bad Blue.

But, in 2002, chronobiologists discovered a 3rd photoreceptor in the retina, which furthered the clinical knowledge of the eighties in terms of the extent and mechanisms of the eye’s non-visual functions, modulated by a blue-turquoise band, Good Blue, centred at 480nm (ca. 465-495nm). This photoreceptor projects onto several non-visual areas of the brain, enabling resynchronisation of the so-called circadian physiological functions over the 24 hours of the Earth’s rotation: sleep, vigilance, mood and body temperature are just a few examples of these functions, demonstrating the importance of not disturbing this Good Blue, if ever we were to seek to cut out all or some of the Bad Blue. Doctor Claude Gronfier (Inserm, Lyon) develops, in this issue of Points de Vue, the current level of knowledge of blue light and circadian rhythms.

Bad Blue, Good Blue, between “chagrin of Azure” (Louis Aragon, Elsa’s Eyes) and “the magnificent radiation of a heavenly eye” (Victor Hugo, The Rhine, Letters to a friend), our eyes, our exposure to the new artificial lighting (see C. Martinsons in this issue), our vision of colours (see F. Vienot in this issue), our predisposition to eye diseases, or quite simply to glare (see B. Girard in this issue), our body, our rhythms, in short our whole physical and psychic life is influenced by light acting on our retinal and cortical sensors and, more specifically, by its proportions of Good Blue and Bad Blue.


--- INTRODUCTION ---

Over the past ten years there has been a wealth of discoveries in the field of chronobiology. Since the discovery of a new retinal photoreceptor in 2002 (melanopsin ganglion cells), shown to be involved in the synchronisation of the circadian clock, it is now clear that the eye is not for seeing only, it is also involved in a range of non-visual functions, directly stimulated by light. The mechanisms involved are mainly yet to be explored but all biological responses to photic stimulus show the way to clinical applications of light in a range of disorders and pathologies, from sleep to alertness, from cognition to memory and mood.

--- LIGHT AND THE CIRCADIAN BIOLOGICAL CLOCK ---

The link between light and the internal biological clock was discovered in humans in 1980. The circadian clock (from the Latin circa “close to” and dies “day”) is a physiological component that is essential to life since it has been observed in almost all the living organisms that have been studied, from prokaryotes through to humans[4]. Two fundamental properties characterise the circadian clock[4]:

1. Its rhythmic activity is endogenous. Located in the suprachiasmatic nuclei (SCN) of the hypothalamus in mammals[5], its circadian electric activity is supported by around ten clock genes whose cyclic activity is responsible for the near 24-hour rhythm of each of its neurons[6].
2. Its activity must be synchronised to 24 hours. Its endogenous period is actually close to but slightly different from a 24-hour period.

Therefore, the clock has to be synchronised (reset in time) in order to enable its activity to be in phase with the solar day. In mammals, light is the most powerful clock synchroniser, and its effect takes place solely through the eye.

--- FUNCTIONS CONTROLLED BY THE CIRCADIAN CLOCK ---

Lots of physiological functions work according to circadian rhythm. Figure 1 shows circadian control over several functions in humans. The clock acts like an orchestra conductor, enabling the expression of physiological activities at the right time. Alertness, cognitive performance, memory, body temperature and blood pressure are at their highest during daytime (awake). On the contrary, secretion of the hormone melatonin, muscle relaxation and sleep pressure are at their highest during the night (sleep).

Many circadian biological activities have been discovered over the past 30 years, both in the periphery and at central level. Depending on the tissue, between 8 and 20% of the genome is expressed rhythmically via the endogenous clock.

The circadian system is involved in the control of cell division, apoptosis in cancer[10] and in the repair of DNA[11]. Because of this, these results can be used to understand how desynchronisation of the circadian system could be responsible for the increased prevalence of certain cancers in shift work[12].

The importance of the circadian system and its synchronisation therefore appears to be crucial to human health.

--- FIG. 1 ---

Diagram of the biological functions controlled by the circadian biological clock (non-exhaustive list).

The structures indicated in colour are respectively in red: the suprachiasmatic nucleus, in orange: the pineal gland, in blue: the hypothalamus (containing the VLPO (ventrolateral preoptic area), known as the sleep switch), in beige: the brain stem (containing the ascending activator cortical pathway and the slow wave / paradoxical sleep sleep switch), in green: the thalamus (responsible for cortical activation and synchronisation of the EEG).


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_ THE CONSEQUENCES OF CIRCADIAN DESYNCHRONISATION _

In humans the importance of synchronisation is clear in symptoms of "jet lag" or in night work (20% of the population in industrialised countries). A lack of synchronisation of the clock is generally translated by a change in numerous physiological functions (sleep, alertness, cognitive performance, cardio-vascular system, immune systems [4,13,14]), the deterioration of neurocognitive processes (cognitive performance, memory) and a disturbance of sleep and alertness[13]. These changes are also found, chronically, in night workers, elderly patients, blind people, in certain psychiatric pathologies and in certain degenerative diseases of the central nervous system (Alzheimer’s and Parkinson’s disease[15]). Chronobiological disorders associated with these normal or pathological conditions have major socio-economic consequences since they can lead to a fall in the general state of health and to an increase in associated pathological risks. The French Society of Occupational Medicine has just published a report under the aegis of the High Health Authority (Haute Autorité de Santé) on the consequences of shift work, including recommendations for detecting them and ways in which to minimise them[13].

_ ENDONIC PROPERTY OF THE CIRCADIAN CLOCK _

In light conditions that are unsuitable for the synchronisation of the circadian system, the endogenous clock functions according to a rhythm that is no longer that of a 24 hour day, In this case it expresses its own endogenic rhythmicity (period). Just like a mechanical clock that has not been adjusted to time regularly, the circadian clock loses time or runs fast, depending on the individual (according to the length of the period of their own clock) in the absence of any synchronisation by the environment. This phenomenon, known as “free run”, is observed in blind people in whom the absence of any light means that the biological clock cannot synchronise to the 24-hour period[15]. This explains why about 75% of blind people complain that their sleep is not of good quality and consult their doctors for recurrent sleep disorders[15]. It should be noted that the length of the clock’s period is a highly precise individual characteristic. It does not vary with age in adults[20], but is relatively flexible during childhood and adolescence (lengthening of the period in adolescence could explain in part the late-to-bed factor, or even disorder of the delayed phase type observed in the 15-25 age range[21]). Thanks to the use of strictly controlled experimental protocols[20], it has been possible to demonstrate that the length of the clock period in humans is very close to 24 hours (24.2 hours on average[20]). One of the direct impacts of the endogenous period in everyday life is the chronotype. Individuals with a short period (a fast clock) are generally those who go to bed early (morning chronotypes) whereas people who go to bed late (evening chronotypes) have a longer period (a slower clock)[22].

_ SYNCHRONISATION OF THE CLOCK _

Because the endogenic period is close to, but not exactly, 24 hours, the circadian clock must be constantly synchronised to 24 hours. In mammals it is light that is the most powerful synchroniser of the internal clock.

The term synchronisation of the biological clock corresponds, just as with a wrist watch, to setting the time, whether the watch is running fast or slow, in order to get it back into phase with the environment. For an “evening” individual, whose endogenic period is 24 hrs and 30 mins, the clock has to be put forward by 30 minutes every day in order to be synchronised to 24 hours, if not it will be another 30 minutes late every day. On the other hand, in a “morning” person, whose period is 23 hrs and 30 mins, the circadian clock has to be delayed by an average of 30 minutes every day. Animals have different synchronisers, which are less efficient in humans. They are known as “non-photic” synchronisers because they do not involve light. Eating and physical exercise have a synchronising effect on the human clock but this is not very strong. Studies carried out in the fifties had led researchers to believe that social synchronisers were more powerful than light in Humans[23]. We now know that this is not the case.

The best proof that non-photic synchronisers have, if anything, an extremely limited effect, has been obtained from the observation that the vast majority of blind people – with no perception of light – are in a state of non synchronised “free run”, despite a social life and activities set out according to the 24-hour period (work, going to bed / rising, eating meals, physical and intellectual activities, etc.). The hormone melatonin is the only non-photic synchroniser for which the effect on the human circadian clock is without a doubt[24]. It should be considered as a priority approach in the treatment of “free run” in blind people.

_ CIRCADIAN PHOTORECEPTION _

Until recently it was accepted that the cones and rods of the external retina were the only photoreceptors responsible for the transduction of light information to the endogenic clock. Studies carried out since the year 2000 in both humans and animals show that two retinal systems are involved in circadian photoreception (fig. 2):
These rhabdomeric type cells also show the property of bistability, a peak of sensitivity at around 480nm (in all the mammals studied). Unlike cones and rods, (fig. 2 and 4) blue light 100 times more intense (comprising 100 times more photons) can be as efficient on the circadian system as a fluorescent white light 100 times more intense (comprising 100 times more photons). This property is based on the sensitivity of melanopsin ganglion cells.

Finally, the effect of light depends on its spectrum. As shown in figure 3, the circadian system is at maximum sensitivity to a coloured light of between 460-480nm. A monochromatic blue light (wavelength 480nm) can be as efficient on the circadian system as a fluorescent white light 100 times more intense (comprising 100 times more photons). A stimulus given at the same time for the same length of exposure, with a light intensity of 100 lux, i.e. 10% of the maximum intensity tested, produces a delay of about 1 hour, i.e. 50% of the maximum observed. Recent studies show that the circadian clock is actually particularly sensitive to low light intensities, and that exposure to a LED computer screen (between 40 and 100 lux) for 2 hours partially inhibits melatonin secretion, activates alertness, and delays the biological clock and sleep onset.

The circadian system’s response to light depends on photic characteristics. The effect of light on the clock depends on the intensity of light and how long it lasts. The more intense the light stimulus, and/or the longer it lasts, the greater the effect. For example, nocturnal exposure to light lasting for 6.5 hours leads to a delay of more than 2 hours in the melatonin rhythm when intense white light is used (10000 lux). A stimulus given at the same time for the same length of exposure, with a light intensity of 100 lux, i.e. 10% of the maximum intensity tested, produces a delay of about 1 hour, i.e. 50% of the maximum observed. Recent studies show that the circadian clock is actually particularly sensitive to low light intensities, and that exposure to a LED computer screen (between 40 and 100 lux) for 2 hours partially inhibits melatonin secretion, activates alertness, and delays the biological clock and sleep onset.

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Finally, the effect of light depends on the time at which it is perceived. The phase response graph shows that the light to which we are exposed in the evening and at the beginning of the night (on average between 5pm and 5am) has the effect of delaying the clock, whereas light received at the end of the night and in the morning (on average between 5am and 5pm) has the reverse effect of advancing the clock. It is this specific temporal sensitivity that explains the clock’s daily synchronisation under normal circumstances and its non-synchronisation in the presence of jet-lag and night work.

LIGHT AND NON-VISUAL FUNCTIONS

Since the discovery of melanopsin ganglion cells in the retina 10 years ago, a range of non-visual, light-sensitive functions have been described. These functions involve anatomical pathways and cerebral structures.

1. The photoreceptors involved in conscious vision (cones and rods).
2. The intrinsically photosensitive retinal ganglion cells: (ipRGC) involved in a large number of non-visual functions. In the absence of these 2 systems, the circadian system is “blind” in rodents and functions in “free run”, expressing its endogenous rhythmicity. It is currently thought that the light information responsible for synchronisation of the biological clock passes through the melanopsin ganglion cells, either by stimulating these cells directly or by stimulating them indirectly through cones and rods. Because of this fact, it is now considered that the eye is not used for vision only, but that it possesses both visual and non-visual functions (fig. 2 and 4).

The two types of photoreceptors in the external and internal retina are phylogenetically and functionally different. Unlike cones and rods, melanopsin ganglion cells require high illuminances and show a peak of sensitivity at around 480nm (in all the mammals studied). These rhabdomeric type cells also show the property of bistability, which makes them virtually insensitive to bleaching. These photoreceptors are currently the subject of a great deal of research, aimed at developing methods for treating certain chronobiological disorders (including disorders of the circadian rhythms of sleep and seasonal affective disorders), which could be faster and more efficient than the current methods which use fluorescent white lights.

The circadian system’s response to light depends on photic characteristics. The effect of light on the clock depends on the intensity of light and how long it lasts. The more intense the light stimulus, and/or the longer it lasts, the greater the effect. For example, nocturnal exposure to light lasting for 6.5 hours leads to a delay of more than 2 hours in the melatonin rhythm when intense white light is used (10000 lux). A stimulus given at the same time for the same length of exposure, with a light intensity of 100 lux, i.e. 10% of the maximum intensity tested, produces a delay of about 1 hour, i.e. 50% of the maximum observed. Recent studies show that the circadian clock is actually particularly sensitive to low light intensities, and that exposure to a LED computer screen (between 40 and 100 lux) for 2 hours partially inhibits melatonin secretion, activates alertness, and delays the biological clock and sleep onset.

The effect of the light depends on its spectrum. As shown in figure 3, the circadian system is at maximum sensitivity to a coloured light of between 460-480nm. A monochromatic blue light (wavelength 480nm) can be as efficient on the circadian system as a fluorescent white light 100 times more intense (comprising 100 times more photons). This property is based on the sensitivity of melanopsin ganglion cells.

Finally, the effect of light depends on the time at which it is perceived. The phase response graph shows that the light to which we are exposed in the evening and at the beginning of the night (on average between 5pm and 5am) has the effect of delaying the clock, whereas light received at the end of the night and in the morning (on average between 5am and 5pm) has the reverse effect of advancing the clock. It is this specific temporal sensitivity that explains the clock’s daily synchronisation under normal circumstances and its non-synchronisation in the presence of jet-lag and night work.

LIGHT AND NON-VISUAL FUNCTIONS

Since the discovery of melanopsin ganglion cells in the retina 10 years ago, a range of non-visual, light-sensitive functions have been described. These functions involve anatomical pathways and cerebral structures.
that are different to those involved in vision, and do not lead to the formation of images (fig. 4). Studies in animals\(^1\)\(^2\) show projections of melanopsin ganglion cells towards structures involved in the regulation of biological rhythms, the regulation of alertness and sleep states, the regulation of locomotor activity, the pupil reflex, etc. In humans, studies show that melanopsin ganglion cells, via non-visual pathways, are involved in the effect of light on the resetting of melatonin phase\(^3\)\(^4\), the increase in alertness, body temperature and heart rate\(^5\)\(^6\), expression of the PER2 gene\(^7\)\(^8\), resetting of the rhythm of the PER3 gene\(^9\)\(^10\), the increase in psychomotor performances and EGG activity\(^11\)\(^12\), sleep structure\(^13\)\(^14\), and activation of cerebral structures involved in memory and mood regulation\(^15\)\(^16\). Light, via non-visual retinal projections, will therefore directly stimulate the cerebral structures involved in the control of alertness, sleep, mood and cognitive and psychomotor performances.

Before the identification of two anatomical pathways (visual and non-visual), it has been known since 1995 that some blind people who do not have any conscious visual perception can have a light-sensitive circadian system\(^1\)\(^2\). The visual system of these patients is blind, but their non-visual functions (including their circadian clock) are not blind and receive photic information. These cases are probably rare (very few individuals have been studied) and the majority of patients with ocular pathologies leading to partial or total privation of photic information have an increased prevalence of sleep and biological rhythm disorders (their circadian rhythms are most often expressed through “free run” and this clinical condition is associated with sleep disorders in over 75% of cases\(^1\)\(^2\)).

**CONCLUSIONS**

In view of the importance of the circadian system synchronisation and the nature of the non-visual functions, light appears to be a biological requirement essential to health. It is predictable that light will be used in the future in the treatment of numerous normal or pathological conditions, in which a physiological malfunction will be corrected through activation of the eye’s non-visual functions. •
P E R C E P T I O N  O F  B L U E  A N D  S P E C T R A L  F I L T E R I N G

INTRODUCTION

The sky is blue. Physicians give us an explanation for this: it is due to the preponderance of short wavelengths in the light diffused by the atmosphere. But why do we see it blue? Seeing the world in colour and identifying its characteristics requires processing of the image formed by the distribution of photons on the retina.

1. HOW IS THE COLOUR SENSE CREATED?

First we need to remember the various stages involved in how colour vision works. The photons reaching the retina are absorbed by photoreceptors: cones for daytime vision and rods for vision when the light is dim, and very often both cones and rods if light is slightly reduced. The photoreceptors generate a signal when they capture a photon, whatever the wavelength involved. Due to very extensive spectral sensitivity in the field of wavelengths, almost all the photoreceptors are able to absorb short wavelength photons. It is only the rate of absorption that differentiates them. So, “S” cones (improperly named “blue”) are preferentially sensitive to short wavelengths of around 450nm, “M” cones (“green”), to medium wavelengths of around 540nm, “L” cones (“red”), to around 570nm, and rods to around 507nm. However, the probability exists that, for example, a 450nm photon hitting the retina is absorbed by a photoreceptor other than an “S” cone. Immediately on exit, the photoreceptors signals are recombined, and it is mainly contrast signals, of luminous or spectral origin, that enter the numerous visual paths in the retina. As for the retinal signals that head for the cortex, they are subject again to several recombinations, of variable importance, before resulting in the colour sense. In general, in these recombinations, signals from all the cone groups come into play, with variable importance. Colour is therefore an appearance attribute, constructed by our visual system. It is the tone that essentially characterises the colour of materials, and its definition is exceptionally stable within our natural environment. This phenomenon of relative stability is known as colour constancy.

With regard to the effect of spectral filtering, we note that: in practice, every group of photoreceptors can be stimulated at short wavelengths. An imbalance in the signals generated in cones can lead to a change in the contrasts perceived and a disturbance in colour perception which is not radical, however, as long as the three cone groups remain intact.

2. SPECIFIC CHARACTERISTICS OF BLUE VISION

In colour vision, blue, or more exactly the retinal pathway of signals issuing from the “S” cones, has a particular status. These signals contribute only slightly to luminous contrast at high spatial or temporal

FIG. 1 | Spectral sensitivity of the three groups of retinal cones.
frequencies. Because of this fact, neither acuity nor sensitivity to flicker is based on these signals. We even speak of foveal tritanopia or small field tritanopia to indicate the reduction of colour vision due to the inability of “S” cones to process certain colour contrasts. On the other hand, “S” cone signals contribute massively to the distinction of colours and play an essential role in identifying shades of colours. For example the difference between yellow or white, or the distinction between warm white or cold white lights, is based on the response of “S” cones.

In summary, in terms of spectral filtering: A strong reduction in signals from “S” cones should not affect acuity, but could lead to deterioration in the distinction of shades of colour and change colour sense. But as long as a few “S” cone signals, even weak signals, pass through into the networks of retinal neurons, modifications to colour often go unnoticed.

### 3. WHAT WOULD BE THE IMPACT OF A BREAK IN VISIBLE SHORT WAVELENGTHS?

As long as the three groups of cones can maintain activity, colour vision, which is based on contrasts, is possible. So, everything depends on the position of the break in the visible spectrum. A break at around 450nm, which leaves a gap at the entrance in “S” cones of almost 50% of the available photons, will have only a low impact on colour vision. Moreover, this is what happens naturally with ageing and cataract. The sky remains blue through until advanced old age. The effect of perceptive constancy, and in this case of “colour constancy”, stabilises the colours of materials in the environment, each in relation to the others, whatever the light variations. If the break happens at around 500nm, a marked deterioration in the distinction of shades of colour is foreseeable in blue-green and purples, as well as for certain colour pairs such as yellow and white or dark blue and black. Acuity should be preserved.

On the other hand, in night vision, the subject may suffer from a notable lack of light.

### CONCLUSION

Any kind of spectral filtering leads to perception deficiency. Although colour distinction is always weakened, higher functions, that is to say the appearance and recognition of colours, are actually well preserved. In terms of colour, the visual response adjusts to the environment. As long as the light is polychromatic, the physiological adaptation capacities of humans compensate for a deficiency of light at source.

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LIGHT EMITTING DIODES (LEDs) AND THE BLUE LIGHT RISK

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__This article presents an overview of the knowledge regarding the potential toxicity of light emitted by LEDs on the retina. Due to their high brightness and their emission spectrum containing a significant fraction of blue light, the so-called “blue light hazard” has been considered and studied for several years. Several independent studies carried out by health agencies have shown that the risk posed by LEDs used as general lighting sources is low, but cannot be neglected in the case of some sensitive populations, considering the increasing optical performances of LEDs and their fast mass market distribution.

Traditional lighting sources such as the well-known incandescent lamp and the compact fluorescent lamp are rapidly being replaced by products based on light emitting diodes (LED) (fig. 1). The so-called “solid-state lighting” (SSL) presents many advantages such as longer lifetime, reduced energy consumption and lower environmental impact. Many governments have therefore started to progressively ban older lighting technologies, paving the way for the massive usage of LEDs in the general lighting market. As a matter of fact, leaders of the lighting industry believe that over 90% of all lighting sources in the world will be based on SSL products and LEDs by 2020.

As any new and emerging technologies, SSL products should be proven to be at least as safe as the products they intend to replace. Furthermore, some unique properties of LEDs such as their compactness have generated many new lighting applications for which older technologies could not be employed. For instance, some kinds of toys and clothes now incorporate LEDs. The safety of products using LEDs should be assessed considering the interactions with the human body in existing and new ways of using them.

The potential adverse effects of optical radiation on the skin and on the eyes are known as photobiological hazards. LEDs currently used in lighting applications have the advantage of emitting a negligible amount of ultraviolet (UV) and infrared (IR) radiation.

The only photobiological hazards to consider when assessing the safety of LEDs are linked to visible light, and more particularly the blue part of the spectrum.

Several health agencies such as ANSES and SCENIHR have investigated and reviewed the scientific literature on photobiological hazards related to the use of LEDs. Two key features of LEDs have drawn the attention of experts:

- LEDs are very bright small sources of visible light, which can be glaring. Due to their high brightness, LEDs also have very high radiance.

FIG. 1 | Photographs of several types of solid-state lighting products.
- a. Directional luminaire (spot light) using an LED.
- b. SSL lamp based on three LEDs and used to replace an incandescent lamp.
- c. Outdoor high power SSL luminaire using 121 LED modules.
- d. Typical single LED component, used in many SSL products. This type of LED consumes about 1 W of electricity and generates a luminous flux of about 100 lm. Its luminance can be as high as 10^7 cd/m^2.

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1 As they emit negligible amounts of UV and IR, LEDs should not be expected to contribute to the onset of photokeratitis and cataract.
2 Agence nationale de sécurité sanitaire de l'alimentation, de l'environnement et du travail (French National Agency for Food, Environmental and Work Safety).
3 Scientific Committee on Emerging and Novelly Identified Health Risks.
As they emit negligible amounts of UV and IR, LEDs should not be expected to contribute to the onset of photokeratitis and cataract.

Many governments have therefore started to progressively ban older lighting technologies, paving the way for the massive usage of LEDs. "Solid-state lighting" (SSL) presents many advantages such as longer lifetime, reduced energy consumption and lower environmental impact. "Solid-state lighting" (SSL) presents many advantages such as longer lifetime, reduced energy consumption and lower environmental impact.

The only photobiological hazards to consider when assessing the safety of products using LEDs should be assessed considering the interactions of optical radiation on the skin and the retina, as a result of cellular oxidative stress. Blue light is also suspected to be a risk factor in age-related macular degeneration (ARMD).

Retinal blue light exposure can be estimated using the ICNIRP guidelines. A quantity called the blue-light weighted radianc L_b can be estimated as a function of the viewing distance and the exposure time. Maximum permissible exposure values (MPEs) were set by ICNIRP to provide limits for L_b as a function of exposure time.

For the past three years, blue light exposure data about LEDs have been provided by LED manufacturers and professional lighting associations but also by independent laboratories and governmental agencies. It was found that the retinal blue light exposure levels L_b produced at a distance of 200mm from the user by blue and cold-white LEDs (bare LEDs and LEDs equipped with a focusing lens) exceed the MPE limits set by ICNIRP after an exposure time comprised between a few seconds for high power blue LEDs to a few tens of seconds for high power cold-white LEDs. As a consequence, the potential toxicity of some LED components viewed at short distances cannot be neglected. However, when the viewing distance is increased to one metre, the maximum permissible exposure time rapidly increases to a few thousands of seconds, up to a few tens of thousands of seconds. These very long exposure times provide a reasonable safety margin to assert that there is virtually no possible blue light retinal damage caused by LEDs at longer viewing distances (statement valid for state of the art LEDs at the time of writing).

Several classes of products and applications based on bare LEDs or LEDs covered by a focusing lens (collimator) are directly related to a potentially high level of retinal blue light exposure when short viewing distances are possible. Examples are (but are not limited to):

- Tests and adjustments of high power blue and cold white LEDs by operators in lighting manufacturing facilities or by lighting installers
- Toys using LEDs, given that the higher degree of transparency of the crystalline lens of children makes them more susceptible to higher blue light retinal exposures
- Automotive LED daytime running lights when activated near children and other sensitive subjects
- Some types of directional LED lamps sold for home applications. These lamps can be viewed from distances as short as 200mm

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**FIG. 2**

The blue curve represents the typical emission spectrum of a white LED. The blue peak reaches its maximum value at about 435nm. It corresponds to the primary light generated by the LED semiconducting structure itself (the LED die). The secondary peak reaches a maximum value at 550nm (yellow colour) and is the secondary light emitted by luminophores excited by the blue light (fluorescence). The combination of the direct blue light and the yellowed secondary light produces white color.

The red curve is a plot of the blue light retinal phototoxicity function. It reaches a maximum value at wavelengths corresponding to the blue light peak emitted by LEDs.

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The conclusions drawn for single LED components or LED modules cannot be extended to all SSL applications because the photobiological safety of a final SSL product must be assessed independently of its LED components. As a matter of fact, the Lₚ value of an SSL product is generally very different from the Lₚ value of the LED components that it uses. For instance, a higher Lₚ can be obtained with a lamp using an assembly of low Lₚ LEDs. Reversely, a lower Lₚ can be obtained with a lamp using a diffuser in front of a high Lₚ LED. For all LEDs and products using LEDs, a photobiological blue light risk assessment must be carried out to determine whether or not the MPEs can be exceeded in the conditions of usage. Such risk assessments can be performed by test laboratories specialised in light sources photometry such as CSTB⁴ and LNE⁵ in France.

The main tool used to perform photobiological risk assessment is the CIE¹ S009 publication whose content was included in an international standard (IEC 62471) and other national standards (IESNA RP27, JIS C8159, etc.).

### THE PHOTOBIOLOGICAL SAFETY STANDARD IEC 62471

This standard deals with the photobiological safety of lamps and devices using lamps and includes a classification of the light source in several risk groups. The standard considers all of the photobiological hazards that may affect the skin and the eye (thermal and photochemical hazards) from ultraviolet to infrared wavelengths. Four risk groups are defined: Risk Group 0 (RG0, no risk), Risk Group 1 (RG1, low risk), Risk Group 2 (RG2, moderate risk), Risk Group 3 (RG3, high risk). The risk group depends on the maximum permissible exposure time (MPE time) assessed at a given viewing distance.

### RISK ASSESSMENTS METHODOLOGY

IEC 62471 defines two different criteria to determine the viewing distance. Light sources used in general lighting should be assessed at a distance corresponding to an illuminance of 500 lx. Other types of light sources should be assessed at a fixed distance of 200mm. For LED components, there is no ambiguity in the distance since LED components are not used per se in general lighting. In this case, IEC 62471 requires using the distance of 200mm. The application of the IEC 62471 measurement technique at 200mm leads to RG2 classification (moderate risk) for some high power blue and cold white LEDs.

However, the choice of the viewing distance in IEC 62471 is sometimes ambiguous and not realistic in the context of the real usage conditions. For instance, in the case of stage lighting (theatres, concert halls) where artists are exposed to an illuminance level higher than 500 lx. Applying the 500 lx criterion would underestimate the exposure while the 200mm criterion would largely overestimate it. In a more usual situation, directional household lamps fall under the 500 lx criterion, which corresponds to a typical viewing distance of a few metres. It is however quite common to have shorter viewing distances, as short as 200 or 500mm at home. Another example is street lighting where the illuminance level is much lower than 500 lx, typically a few tens of lx. Assessing the exposure to blue light emitted by a street lighting luminaire at the distance giving an illuminance of 500 lx is clearly not appropriate. A future revision of IEC 62471 should bring a more accurate definition of the distance at which the risk group is determined.

It is interesting to note that the strict application of CIE S009 and IEC 62471 to indoor LED lamps and luminaires lead to RG0 and RG1 classifications, similar to traditional indoor light sources (fluorescent lamps, incandescent and halogen lamps). Nevertheless, when the 200mm viewing distance is chosen, several measurement campaigns reveal that a small number of indoor LED lamps and luminaires belonged to RG2 while traditional indoor light sources (fluorescent and incandescent) were still in RG0 or RG1. This result shows that LED technology potentially raises the blue light risk in home applications where the viewing distance is not limited and light sources are accessible to children and other sensitive people. At the time of publication, the general public remains unaware of potential risks to the eye since no mandatory labeling system is currently in place for consumer SSL products.

The notion of a safety distance would actually be more appropriate to communicate to installers and to users, especially the general public. The safety distance of an SSL product would be the minimum distance for which the blue light hazard risk group does not exceed RG1. Measurement campaigns carried out by several laboratories showed that the vast majority of indoor LED lamps and luminaires have a safety distance of 200mm which is compatible with most lighting applications.

It is important to note that other widely used lighting sources, particularly high intensity discharge lamps used for outdoor lighting are in RG2 (moderate risk). However, these lamps are intended for clearly identified uses and can only be installed by professionals who should be aware of the safety distance required to limit the exposure.

### OTHER LIMITATIONS OF IEC 62471 AND CIE S009 AND SENSITIVE POPULATIONS

The maximum exposure limits defined by the ICNIRP and used to define the Risk Groups in both IEC 62471 and CIE S009 are not appropriate for repeated exposures to blue light as they were calculated for a maximum exposure in one 8-hour day. They do not take into account the possibility of exposure over an entire lifetime.

Neither CIE S009 nor IEC 62471 takes into account the sensitivity of certain specific population groups, which can be characterised by an accrued sensitivity to visible light:

- People having pre-existing eye or skin conditions for which artificial lighting can trigger or aggravate pathological symptoms
- Aphakic (people with no crystalline lens) and pseudophakic people (with artificial crystalline lenses) who consequently either cannot or can only insufficiently filter short wavelengths (particularly blue light)
- Children
- Elderly people as their eyes are more sensitive to optical radiation

The photobiological standards for lighting systems should be extended to cover children and aphakic or pseudophakic individuals, taking into account the corresponding phototoxicity curve published by the ICNIRP in its guidelines.

In addition to proven photochemical damage of the retina resulting from acute exposure to blue light, uncertainty still remains surrounding the effects of chronic exposure at low doses. These effects are still being investigated by ophthalmologists, biologists and optical scientists.

In France, the RETINALED project⁶ is investigating the effects of chronic low exposure of rodents to light emitted by LEDs.

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¹ Centre Scientifique et Technique du Bâtiement (French Technical and Scientific Research Center on Construction and Building).
² Laboratoire National de Métrologie et d’Essais (National Testing and Metrology Laboratory).
³ Commission Internationale de l'Éclairage (International Commission on Illumination).
⁴ The RETINALED project is carried out by INSERM, CSTB and ENCA. It is supported by ADEME (French Environmental and Energy Management Agency).
⁵ Identité et collective de l’Agence Nationale de Sécurité des Éclairages utilisant des diodes électroluminescentes (LED)»,
⁶ Progress in Retinal and Eye Research, Volume 30, for domestic lighting: Any risks for the eye?, Commission, March 2012, ISSN 1831-4783, biochemistry/index_en.htm
Certain categories of workers are exposed to high doses of artificial light (long exposure times and/or high retinal illuminances) during their daily activities (examples: lighting professionals, stage artists, etc.). Since the damage mechanisms are not fully understood yet, exposed workers should use appropriate individual means of protection as a precautionary measure (glasses filtering out blue light for instance).

CONCLUSIONS

Due to their unique light emission properties, LEDs are currently on the verge of becoming the dominant lighting source of this century. However, the risks posed by these new sources of light are also rooted in their intrinsic characteristics: high optical output in a small package (producing a high radiance level) associated with a significant blue light emission. The combination of these two factors can potentially increase the risk of photochemical damage of the retina, in comparison with the incandescent lamp and the fluorescent lamp.

Lighting industry leaders are well aware of the photobiological safety of their products. Many lighting products using LEDs now emit warmer shades of white light (reduction of the blue light content in the spectrum) or use diffusers to reduce glare (reduction of the radiance). Most lighting products are found to present low risks or no risk at all for the general population when the viewing distance is equal to or greater than 200mm.

However, measurement campaigns carried out by independent agencies pointed out a few lighting products with significantly higher risk levels below a distance of one metre or more. At the present time, no mention is made by lighting manufacturers of a “safety distance”. It is therefore impossible for the public to identify lamps or luminaires with a higher risk level.

The blue light risk assessment related to LEDs can be performed by test laboratories using the IEC 62471 standard which is not perfectly clear about the viewing distance to consider. In addition, this standard does not consider sensitive populations such as children, aphakic, pseudophakic and elderly people, despite the fact that these populations are exposed to a higher level of blue light on the retina. The current knowledge of the mechanisms of blue light phototoxicity is far from being complete. The effects of chronic exposure and accumulated low exposure over very long periods of time are still an active subject of research. As far as LEDs are concerned, the better comprehension of the possible long term effects of the blue light on the retina is fundamental to guaranteeing that the “LED revolution” will not compromise our vision of the future.

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BIography

Christophe Martinsons received a Ph.D in Physics from the University of Reims Champagne-Ardenne in 1998. Up to 2000, he held a research scientist position at the National Physical Laboratory (NPL).

From 2000 to 2007, he worked in the field of home automation for the HAGER Group. In 2007, he joined CSTB to head the Lighting, Electricity and Electromagnetism division.

He currently conducts research and consultancy work in the field of combined daylighting and artificial lighting in order to promote energy-efficiency in buildings while providing the best visual comfort conditions for users. His approach to lighting is put forward in the new French building energy code (RT 2012).

For the past four years, Christophe Martinsons has been leading laboratory measurement campaigns for French governmental agencies while working on independent studies concerning health and environmental aspects of solid-state lighting and LEDs.
Hazards of Solar Blue Light

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INTRODUCTION

A retinal condition known as photoretinopathy occurs in people who have stared fixedly at the sun without adequate protection, usually for more than a few minutes (see references [1]). Photoretinopathy is photochemical damage caused by visible light, especially in the wavelength region of approximately 400–500 nm (Fig. 1). Light in this wavelength region appears blue to the eye and therefore is called blue light.

According to the guidelines of the International Commission on Non-Ionizing Radiation Protection (ICNIRP) [2] and the American Conference of Governmental Industrial Hygienists (ACGIH) [3], the hazard of blue light is generally measured by blue-light radiance. Blue-light radiance is obtained by weighting the spectral radiance of a light source against the blue-light hazard function (fig.1) and integrating this in the wavelength range of 305–700 nm. The maximum permissible exposure duration per day is calculated by dividing 106 Jm-2sr-1 by the blue-light radiance. Thus, solar blue-light radiance should be known as a first step toward preventing sun-induced photoretinopathy.
Hazards of Solar Blue Light | Points de Vue

Fig. 1: Blue-light hazard function [2, 3]. The blue-light hazard function shows the relative effectiveness of optical radiation to produce photochemical retinal damage as a function of wavelength.

The intensity of sunlight observed on the earth's surface generally increases with solar elevation. As solar elevation increases, sunlight travels a shorter distance through the atmosphere to reach the earth's surface and therefore is less attenuated by atmospheric scattering and absorption. The intensity of sunlight is also expected to be influenced by temporary local atmospheric conditions such as clouds and dust. Thus, these factors should also influence solar blue-light radiance. In this study, solar blue-light radiance was determined for solar elevations up to almost 90° in summer in Ishigaki, Japan (latitude 24°20’N). The effect of solar elevation was studied using a mathematical model of atmospheric extinction.

METHODS

Measurements were made on 10 consecutive days from 21 June (the summer solstice) to 30 June 2006 in Ishigaki from sunrise to sunset at 15-min intervals, except when the sun was completely invisible because of clouds. Since Ishigaki is a small remote rural island, urban atmospheric pollution is expected to be very low.

Spectral radiance in the wavelength range of 380–780 nm at 2-nm intervals was measured at the center of the solar disk with a measuring field of 0.125° (0.0022 rad) diameter by a spectroradiometer (PR-705, Photo Research Inc., 9731 Topanga Canyon Place Chatsworth, CA 91311-4135, USA). Two neutral density filters of about 1 % transmittance (ND-100, Photo Research Inc.) were attached to the aperture of the instrument, because solar radiance was too high to measure directly. Corrections for the spectral transmittance of the filters were made automatically by the instrument. The spectroradiometer was calibrated by the manufacturer prior to the
measurements. With the use of PC software (MyPlanet, Japan, Mitsunori Asami), the solar elevation was calculated from the geographic coordinates (longitude and latitude) of the measurement site and the date and time of each measurement.

The blue-light radiance at the center of the solar disk was obtained by weighting the measured spectral radiance against the blue-light hazard function and integrating it with respect to wavelength. In this case, the integration was started at 380 nm instead of 305 nm. This modification is acceptable, because the blue-light hazard function is very small in the wavelength range of 305-380 nm (Fig. 1) and therefore radiant energy in this range is expected to contribute little to blue-light radiance for white-light sources. For example, a simple calculation shows that the contribution of this wavelength range is only 1% for light sources with a flat spectral distribution.

Data were corrected for the limb darkening of the sun. The central blue-light radiance obtained was multiplied by the ratio of the mean to central radiance at 450 nm of 0.755 [4] to obtain the blue-light radiance of the sun (i.e., the mean of the solar disk).

The blue-light radiance was then multiplied by \((0.0093/0.011)^2\), because the sun subtends an angle of 0.0093 rad, which is less than 0.011 rad [2,3].

According to the ICNIRP [2] and ACGIH [3] guidelines, the maximum permissible exposure duration per day in seconds is obtained by dividing 106 Jm-2sr-1 by the measured blue-light radiance in Wm-2sr-1.

The combined data on blue-light radiance versus solar elevation for all 10 days were compared with the prediction of a model of atmospheric extinction. Assuming that the optical density of the atmosphere that sunlight traverses to reach the earth’s surface is proportional to the amount of that atmosphere (air mass), the solar blue-light radiance observed on the earth’s surface depends on the solar elevation, as follows:

\[
L(\gamma) = L_0 \exp(-k M(\gamma)), \quad (1)
\]

where:
- \(\gamma\) = solar elevation;
- \(L(\gamma)\) = solar blue-light radiance observed on the earth’s surface;
- \(L_0\) = solar blue-light radiance observed outside the atmosphere;
- \(M(\gamma)\) = air mass, which is normalized to 1 at 90°;
- and \(k\) = extinction coefficient per unit air mass.

The air mass is approximated as follows [5]:

\[
M(\gamma) = \frac{1.002432 \cos^2(90° - \gamma) + 0.148386 \cos(90° - \gamma) + 0.0096467}{\cos^3(90° - \gamma) + 0.149864 \cos(90° - \gamma) + 0.0102963 \cos(90° - \gamma) + 0.000303978}. \quad (2)
\]

The data were least-squares fitted to eqn (1) with \(L_0\) and \(k\) as parameters under the constraint that the solar blue-light radiance measured is lower than that predicted by the model. This constraint was imposed because the solar blue-light radiance may actually be reduced by temporary local atmospheric conditions such as clouds and dust. Fitting was performed using the solver add-in in spreadsheets software (Microsoft Excel).

**RESULTS AND DISCUSSION**

A total of 461 measurements were made of the solar spectral radiance on 10 consecutive days.
Although the overall intensity of sunlight varies greatly from measurement to measurement, the spectral features remain basically unchanged (fig.2).

Fig. 2: Solar spectral radiance measured on 23 June 2006. The time and solar elevation at which the measurement was taken are indicated for each line.

The solar blue-light radiance and the maximum permissible exposure duration per day were calculated for each measurement of the solar spectral radiance, according to the ICNIRP [2] and ACGIH [3] guidelines. The solar blue-light radiance generally increases from sunrise to about noon and then decreases toward sunset, but it varies when the sun goes behind a cloud, as shown by the sharp valleys in fig.3. The solar blue-light radiance also fluctuates to some extent, even when no clouds are seen in front of the sun, probably due to invisible moisture or dust in the atmosphere.
Fig. 3: Solar blue-light radiance measured on 22 June 2006, plotted against time of day. The maximum permissible exposure duration per day can be read from the right-hand scale.

The blue-light-radiance data for all 10 days are shown in fig. 4 as a function of solar elevation. Higher blue-light radiances are associated with higher solar elevations. The solar blue-light radiance ranges from 8.39×10 to 1.71×106 Wm-2sr-1 with the median 1.31×106 Wm-2sr-1. The maximum exposure durations per day corresponding to the maximum and median blue-light radiance are only 0.82 s and 1.07 s, respectively, meaning that viewing the sun can be very hazardous. In fact, it is not unusual to view the sun for more than these maximum exposure durations in everyday situations such as scanning the sky for a scenic view. Thus, it is necessary to avoid viewing the sun directly except at very low solar elevations.

Data on blue-light radiance versus solar elevation were well fitted by eqn (1) (fig.4), indicating the validity of this model. The best-fit parameters are L0 = 2.26×106 and k = 0.272. Thus, the maximum solar blue-light radiance at each solar elevation and the corresponding maximum permissible exposure duration per day can be calculated as,

\[
L_m(\gamma) = 2.26 \times 10^6 \exp(-0.272 M(\gamma)) \tag{3} 
\]
\[
t_{max}(\gamma) = 0.619 \exp(0.272 M(\gamma)) \tag{4}
\]

where \(L_m(\gamma)\) = maximum solar blue-light radiance at solar elevation \(\gamma\); and \(t_{max}(\gamma)\) = maximum permissible exposure duration per day at solar elevation \(\gamma\).

Eqns (3) and (4) are of practical importance, because the maximum hazard at any time and place can be evaluated by calculating the solar elevation from the geographic coordinates, the date and the time and substituting it into these equations. This knowledge can be used when discussing
measures or strategies to prevent sun-induced photoretinopathy.

Fig. 4: Solar blue-light radiance plotted against solar elevation. The letters A–J represent data for 10 days, respectively, and the line represents the prediction of the best-fit model. The maximum permissible exposure duration per day can be read from the right-hand scale.

CONCLUSIONS

This study demonstrates that the sun is generally very hazardous to view. It is necessary to avoid viewing the sun directly except at very low solar elevations. This study also presents a mathematical model to predict the maximum hazard at each solar elevation and the corresponding maximum permissible exposure duration per day. This knowledge is important when discussing measures or strategies to prevent sun-induced photoretinopathy.

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PHOTOSENSITIVITY AND BLUE LIGHT

PHOTOPHOBIA IS THE PAINFUL SENSATION felt by a patient on exposure to light. It is responsible for the reflex closing of the eyelids, which protects the retina from too much exposure to light rays, and particularly the sun’s rays, due to the phototoxicity of light on the chorioretinal layers.

Photosensitivity occurs only within the spectrum of visible light. This sensorial information can be exacerbated and in this case we then refer to it as photophobia. Some diseases cause photophobia and it is seen as one of the symptoms. The most common diseases of this type affect the integrity of the eye or vision paths, such as corneal lesions, traumatic corneal ulcers, corneal abscesses or superficial punctate keratitis, which are common in all dry eye syndromes. Uveitis may also be mentioned here, along with retrobulbar neuropathy or extra-ocular conditions such as migraine or meningitis.

SPECIALISED GANGLION CELLS

Photophobia originates in specialised ganglion cells known as “ipRGCs” (intrinsically photosensitive retinal ganglion cells). At the current stage of research we do not yet know whether these cells sub-divide according to the wavelength presented. These ipRGCs are located in the retina’s layer of ganglion cells. At the outset their axons take the same path as all the retinal nerve fibres and head towards the optic nerve. Their specific path has only recently been discovered, and is called the non-visual path of the optic nerve, which arrives at the posterior section of the thalamus or pulvinar[8]. These non-visual paths, individualised using the techniques of Diffusion MR tractography provide an anatomo-physiological basis for the pain engendered by light. There are also nerve connections between the pulvinar and the nucleus of the trigeminal nerve which can explain photosensitivity in all ocular lesions that stimulate the ophthalmic branch of the trigeminal nerve.

After direct connection by the optic nerve to the pulvinar, the route of the non-visual path connects the cortex, both visual (Brodmann occipital areas 18, 19, 20), parietal (association area, Brodmann area 7), frontal and pre-frontal. The connections of this non-visual path interact with motor and sensorial paths (olfactive). This non-visual path, activated by photic stimulation, acts on the excitation limit of the trigeminal neurones in the lateral posterior and posterior nucleus of the thalamus (rat) increasing the feeling of pain to light exposure in migraine. A functional IRM study[8] has also shown an increase in pulvinar activity during central cerebral sensitisation (migraine), thus explaining photophobia. The pulvinar is divided into four areas, three of which (medial, superior and inferior) concentrate visual information[3]. The pulvinar is therefore a major centre for the integration and modulation of sensorial inputs, particularly those conveyed by the ipRGCs and the non-visual path which itself has connections with the suprachiasmatic nucleus (SCN), the habenula, the pineal gland, the intergeniculate leaflet (IGL) and the olivary pretectal nucleus (OPN). The latter is connected to the ciliary ganglion and to the Edinger-Westfal nucleus which is involved in photo-dependent pupil reflexes.

THE TOXICITY OF BLUE LIGHT

To protect itself from the harmful effects of high energy light radiation, nature has established numerous filters. A, B and some C ultraviolet rays, which have even higher energy than blue light, do not reach the retina because they are halted by the ozone layer, then the cornea and the crystalline lens. On the other hand, the various radiations of the visible spectrum of light do reach photoreceptors. The blue light wavelength has the most high-energy. It is located at between 400 and 510nm. It includes violets, indigo-blue and cyan (fig. 1). Blue light is absorbed by the yellow pigments of the crystalline lens (fig. 2), which gradually appear as age progresses (fig. 3) and in the retina by pigments, rhodopsin, lipofuscin and the macular pigments (lutein, zeaxanthin, meso-zeaxanthin).

The photochemical reaction is responsible not only for phototransduction but also for the formation of free radicals during...
The pulvinar is therefore a major centre for the integration of the non-visual path. At the outset their axons take the same path as all the retinal nerve fibers. These ipRGCs are located in the retina’s layer of ganglion cells. At the current stage of research we do not yet know whether they are responsible for the photic sensitivity of the retina, the hampering of our ability to see in the dark, or for the suppression of our circadian rhythm.

Photophobia originates in specialised ganglion cells known to interact with motor and sensorial paths (olfactive). This sensorial information can be exacerbated and in this case consider it as photophobia. Some diseases cause photophobia due to a loss of control hormonal rhythms, with regulation of the internal biological clock. Lesions with apoptosis of the photoreceptors during photochemical reactions or abnormalities of oxygen metabolism, non-renewal mechanisms that release toxic oxidative residues.

Photophobia is a natural phenomenon that gives humans their diurnal behaviour, with regulation of the internal biological clock. The ipRGCs mediated by the non-visual path control hormonal circadian cycles, sleep and mood. Photophobia triggers retinal protection against light energy and more particularly blue light, which has the most high-energy and is responsible for irreversible cellular lesions with apoptosis of the photoreceptors during photochemical mechanisms that release toxic oxidative residues.

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INTRODUCTION

Age-related Macular Degeneration, AMD, is one of the major causes of visual impairment in industrialised countries, along with diabetic retinopathy and glaucoma. In the United States, AMD is considered to be the cause of 54.4% of visual impairments and 22.9% of cases of blindness[1]. It is estimated that in 2010, 9.1 million Americans aged over 50 presented early-stage AMD[2] and that this number is set to double by 2050, to reach 17.8 million. At least 12% of the American and European populations aged over 80 is affected by advanced AMD[3-5]. Amongst risk factors for AMD identified in literature, sunlight is indicated as being a factor that can cause cumulative damage to the retina. The only recurrent information is that the highest toxicity levels are in front of the retina, particularly the cornea and the crystalline lens[17, 18]. In the retina, light is mainly absorbed by the visual pigments of vertebrates, rhodopsin. The visual pigments of vertebrates are made up of a transmembrane protein, opsin, combined with a vitamin A derivative 11-cis-retinal. The most energetic light that reaches the retina is therefore mainly blue light, at between 400nm and 500nm. Because of its photosensitising characteristics, is thought to be involved in the development of AMD[7, 8]. In the rod photoreceptors, this visual pigment is rhodopsin. The highest energy portion of the visible spectrum, at between 400nm and 500nm, also known as blue light, is incriminated here. Ophthalmic appliances already claim to offer protection against blue light. Spectacle lenses or intraocular implants mostly contain high-pass filters that absorb a wide band of blue light. However, such unselective filtering can lead to maladjustment of the eye’s visual and non-visual functions. Colour perception is disturbed, scotopic vision is limited and the body clock of wake/sleep cycles, which is controlled by certain wavelengths of blue light, is potentially thrown out of kilter.

LIGHT: A RISK FACTOR FOR AMD

Since the causes of AMD are currently poorly identified, there are no efficient preventive and curative solutions. Numerous epidemiological studies demonstrate a large variety of potential risk factors. Although the first proven factors are age[5], tobacco consumption[9-11] and lack of carotenoids[9], light is also being blamed for a lack of information concerning the relative toxicity to the retina of each wave length within the visible spectrum. This is the reason why Essilor International and the Vision Institute went into partnership in 2008 in order to define the harmfulness of blue light to the retina more clearly and develop more selective, protective filtering lenses.

BLUE LIGHT: HOW DANGEROUS IS IT FOR THE RETINA?

In the retina, light is mainly absorbed by the visual pigments contained in the external segments of the photoreceptors. The visual pigments of vertebrates are made up of a transmembrane protein, opsin, combined with a vitamin A derivative 11-cis-retinal. In the rod photoreceptors, this visual pigment is rhodopsin. Most ultraviolet radiation is naturally filtered by ocular tissues located in front of the retina, particularly the cornea and the crystalline lens[17, 18]. The energy of the toxic spectrum of light on the cells of the retina. The only recurrent information is that the highest toxicity levels are in front of the retina, particularly the cornea and the crystalline lens[17, 18]. Under moderate light exposure conditions, the all-trans-retinal is recycled continuously into 11-cis-retinal by the cells of the retinal pigment epithelium and does not cause any danger to the cell. When exposure to light happens over a longer or more intense period, the all-trans-retinal accumulates and its activation by blue light may be the cause of oxidative stress which damages the cellular components of the photoreceptors. This oxidative stress is normally compensated for by the presence of the numerous antioxidants in the retina. However, with age and certain genetic and environmental factors, such as tobacco consumption or a diet that is low in antioxidants, anti-oxidative defences are reduced[19, 20] and can no longer compensate for the stress caused by prolonged or intensive exposure to blue light.

The function of the cells in the retinal pigment epithelium is to ensure renewal of the external segment of photoreceptors. They eliminate the distal part of them by ingestion, or “phagocytosis”, whilst the growth of these external segments occurs continuously[21]. When the external segments are too damaged by oxidative stress, their membrane components are difficult for the retinal pigment epithelium to break down. Intracellular digestion is then incomplete and generates an accumulation of residual granular bodies, in the form of lipofuscin. The granules of lipofuscin contain a large amount of polyunsaturated fat, a target for oxidation. The lipophilic extract of lipofuscin contains a potential photosensitiser, which forms a triplet excited state with a maximum of absorption in blue at 440nm[24, 25]. One of the components of lipofuscin, A2E, has been identified as being involved in the photosensitising nature of the lipid residue. The energy of the triplet state is sufficient to be transferred and react with oxygen in the blood.
Photoactivation of the lipofuscin granules by blue light then generates reactive oxygen species (superoxide, hydrogen peroxide, lipid hydroperoxides and malondialdehyde)\[^{[26, 27]}\]. When the number of these species exceeds the cellular defence capacity, the retinal pigment epithelium cells die by apoptosis. Deprived of these support cells that provide their energy supply, the photoreceptors deteriorate in turn, contributing to the loss of vision diagnosed in patients suffering from AMD.

In conclusion, the suggested mechanism by which light is involved in the appearance and progression of AMD may happen at two levels: on the one hand in photoreceptors via absorption of blue light by rhodopsin and then in the near ultraviolet blue by the all-trans-retinal, and, on the other, in the retinal pigment epithelium via absorption of blue by lipofuscin.

\section*{THE LIMITATIONS OF EXISTING STUDIES}

The toxic effects of visible light and blue light in particular on the retina have already been demonstrated experimentally on cellular\[^{[28-30]}\] and animal\[^{[31]}\] models of degenerative retinal pathologies. However, the studies performed to date have not enabled characterisation of the respective toxicity of each wavelength. Also, they suffer from certain limitations. In fact comparisons of results are difficult from one study to another because units fluctuate between energetic and visual units. Also, the illumination systems used are not calibrated on the illumination of the light sources existing in our environment, whether natural (the sun) or artificial (neon, LED, halogen, etc.) and therefore do not reflect true conditions of exposure to light. Finally, none of the illumination systems used to date enables step by step definition of the toxic spectrum of light on the cells of the retina. The only recurrent information is that the highest toxicity levels are contained within the spectral interval [400nm; 500nm].

\section*{THE CONTRIBUTION MADE BY THE VISION INSTITUTE AND ESSILOR INTERNATIONAL}

The objective of this contribution was, in partnership with Essilor International, to establish a photobiology laboratory at the Vision Institute, to enable us to define precisely the specific toxicity on the retina of each wavelength in the blue section of the visible spectrum. The first action taken involved the development of a cellular illumination system. This enabled the production of visible wavelengths of very narrow bandwidths and at given illumination in order to model the desired luminous spectrum. The light source to which we are the most exposed and which is the most intense is the sun and the work was therefore carried out using, for each wavelength, radiation values relative to the sun’s spectrum.

The second direction for work involved development of a model of cultured cells, reproducing \textit{in vitro} the degeneration of retinal cells, as observed in AMD, with the presence of a lipofuscin component: A2E.

\section*{EQUIPMENT AND METHOD}

The system of illumination that has been developed is a multi-wavelength generator used to illuminate the cells being cultured inside an incubator. The light source comprises a set of light-emitting diodes (LEDs), each connected to the incubator and the cells by means of optical fibres. The range of wavelengths covered extends from 390nm to 520nm in bandwidths of 10nm (\textit{fig. 1}). The whole unit can thus, with each optical fibre, restrict illumination to 10nm of the spectrum arriving in the retina.

In order to model the accumulation of lipofuscin in the retina, cells cultured in pig’s pigmentary epithelium were treated with various concentration of A2E, one of the components of lipofuscin (\textit{fig. 2}). These cells were then exposed to a light bandwidth of 10nm.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image1.png}
\caption{View from above of a cell culture plate lit by various wavelengths, from 390 to 520nm.}
\end{figure}
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was essential in setting up this innovative ophthalmological project. •

The association of the respective skills of the Vision Institute in terms of the photoreceptors, such as pigmentary retinitis and Stargardt’s disease. This type of therapeutic solution could be extended to other retinal sources of artificial light (neon, LED, xenon, halogen, etc.) and wavelengths are also present in variable proportions in the various experimental conditions. Our results also show that the greater the concentration of A2E, the greater the toxic effect of light. These results demonstrate that an A2E dose-dependent effect exists, and therefore probably one of lipofuscin too, in induction of phototoxicity.

This can be related to the influence of age in AMD, because it has been observed that drusen and lipofuscin accumulate with age and are present in greater quantities in elderly patients suffering from AMD [13, 16, 32, 33].

CONCLUSION AND PROSPECTS

The joint work carried out by the Vision Institute and Essilor International has resulted in the establishment of an experimental process using a cellular model of AMD to define the precise spectrum of sunlight toxicity on the retina. These results provide information of capital importance in terms of the need to be protected from highly specific blue light wavelengths. It is important to note that these wavelengths are also present in variable proportions in the various sources of artificial light (neon, LED, xenon, halogen, etc.) and that the potential effects of lengthy exposure should not be neglected. This project supplies elements of understanding of the physiopathological processes taking place in AMD, with the possibility of therapeutic or preventative solutions for this major pathology.

This type of therapeutic solution could be extended to other retinal pathologies involving oxidative stress processes leading to degeneration of the photoreceptors, as in pigmentary retinopathy and Stargardt’s disease.

The association of the respective skills of the Vision Institute in terms of the cellular biology of the retina, and of Essilor International in optics was essential in setting up this innovative ophthalmological project. •

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UN D E R S TA N D I N G R I S K S O F P H OT O T O X I C I T Y O N T H E E Y E

Certain portions of the light spectrum can be detrimental to ocular health and lead to accelerated eye ageing and diseases. With an influx of modern short wavelength light sources on the market, the human eye is susceptible to greater exposure to these lights. Prof. John Marshall, Professor of Ophthalmology at University College London, recipient of the Junius-Kuhnt Award and Medal for his work on AMD, sheds some light on phototoxicity risks and the need for prevention for Points de Vue.

PROFESSOR JOHN MARSHALL
University College London

Points de Vue: Professor Marshall, could you describe some of the research areas you have been involved with over the years that are linked to vision and light?

Prof. John Marshall: I started in vision back in 1965, when I was given a PhD grant with the Royal Air Force to investigate the potential damaging effects of lasers on the retina. At that time we needed to have a much better understanding of how light interacted with the retina and what mechanisms could potentially damage it. Collectively our work together with some German and American teams developed a data base that formed the basis for the international codes of practice to protect individuals against the potential damaging effects of lasers. It also extended into the potential damaging effects of incoherent light. These data were also incorporated into the codes of practice used by large international organizations such as the World Health Organization (WHO), the United Nations environmental programme and the International Red Cross.

After looking at the effects of the acute intense light I become very interested in the effects of chronic irradiation with incoherent light such as sunlight and commercial and domestic light sources in the UK. Our subsequent research showed that the retina was most sensitive to short wavelength visible radiation in the blue region of the spectrum and strangely the cones were more vulnerable than the rods in diurnal animals. Previous data which has confused a lot of the literature was derived from experiments on rats and mice that have predominantly rod retina and as a consequence showed damage to rods.

Subsequently, was your transition into studying the effects of incoherent light, away from lasers, more of a personal interest?

Originally it was personal interest because light is light, whether generated within a laser or an incandescent bulb. Light sources emit photons. I was interested in the interaction between photons and biological tissue, and how photons gave rise to the sensation of vision. Eventually I got interested in how excessive exposure, whether high level, high power or prolonged periods of exposure, had

KEYWORDS
UV, blue light, phototoxicity, laser, cataract, AMD, Retinitis Pigmentosa, RP, IOL, Crizal® Prevencia®, prevention
the potential to damage the visual system. From the evolution standpoint our eyes were designed to have roughly 12 hours of light and roughly 12 hours of dark, something that modern lifestyles have changed considerably.

From your personal point of view, do you think changes in illumination have had an impact in this regard?

Yes, because for thousands of years the only light source under man’s control was fire found in systems such as burning braid, oil lamps or candles. The next progression in the series was gas lighting, which was also essentially fire. However, all of these sources created heat and a lot of light meant a lot of heat. It wasn’t until the advent of the incandescent bulb in the mid-1800s that we had daylight levels of illumination at any time of night or day. Further, with the advent of fluorescent lighting in the 1940s, we could have high light levels without significant actual heat. Unfortunately unlike incandescent bulbs, which produced light mainly towards the red end of the spectrum, fluorescent lighting had emissions in the blue and ultra violet regions. At present, due to environmental concerns of conservation of energy, we are seeing compact fluorescent and LED lights in the market in the name of energy saving, but again these produce ultraviolet and blue light. There should have been much more consultation with the biological vision community before these biologically unfriendly sources were introduced. It is only now that a committee has been formed to consider the unexpected health hazards of such devices. The dermatologic and ophthalmic community could have told the manufacturers that such potential health hazards were certainly not unexpected.

What do you expect the impact of this new form of low energy lighting to be now and in the future?

Researchers on skin have already expressed some concern over ultraviolet and high-intensity blue, increasing the chances of skin problems from commercial and domestic lighting. My concern would be that any short wavelength radiation involves high-energy photons and can exacerbate the ageing process in our eyes in a manner similar to how excessive sunlight exposure during your lifetime can lead to ageing effects such as wrinkly skin. Certain wavelengths may well implicate an accelerated ageing process leading

“Yes, the longer wavelengths of blues are the blues we need portion of the spectrum?”

“Any short wavelength radiation involves high-energy photons and can exacerbate the ageing process in our eyes.”
to an earlier onset of cataract and could also exacerbate other age-related conditions such as age-related macular degeneration. They represent environmental risks factors to which we really do not need to expose ourselves, as incandescent bulbs had illuminated our homes satisfactorily for a hundred years.

Are there any calls to government agencies on the dangers of this new push for low energy light bulbs?

In my opinion there should have been a committee of experts assessing the health hazards of low-energy lighting before they became available in the marketplace and certainly before incandescent bulbs were banned! Unfortunately this is closing the door after the horse has bolted. It should have been more important to consult the relevant experts before making important policy decisions in order to avoid a potential downstream problem.

How does this phototoxicity act on ocular tissue?

High-energy photons in the presence of oxygen give rise to reactive oxygen species that are potentially dangerous for cells. Light damage to the skin is minimised by the surface cells of the skin being constantly replaced by cells from deeper layers, thus simplistically the system is renewed approximately every five days. By contrast the cells that line the inside of the eye, the retina, are in essence an outgrowth of the brain and therefore like all neurons incapable of dividing. The rods and cones have to absorb light and are in the presence of high levels of oxygen. They have developed a mechanism whereby the light-sensitive portion of the cell is constantly renewed on a daily basis. Every hour of every day approximately three to five new light-sensitive membranes are manufactured and every morning on awakening rods lose approximately 30 old membranes to a layer of cells called the retinal pigment epithelium (RPE). Cones lose their old membranes about every four hours during our sleep period. Over a human lifetime, the RPE cells that also don’t normally divide have to contend with huge amounts of degraded biological material. From one’s mid-thirties onwards, the RPE cells get progressively clogged with toxic products. At a later stage these waste products lead to further changes between the RPE cells and their underlying blood supply. This sequence of buildup of age-related waste products generated by an attempt to protect the light-sensitive cells against the damaging effects of light throughout a lifetime is the biggest risk factor in age-related macular degeneration (AMD). More light stress produces more debris, and has the potential to accelerate the ageing process. We certainly need some exposure to blue lighting in order to balance our biological well-being and stop us becoming affected by seasonally adjusted disorder (SAD). However this is a requirement for longer wavelength blue light and there is no advantage associated with short wavelength blue light or ultraviolet.

So to expand on this point, do you see a difference in phototoxicity between the bands within the blue portion of the spectrum?

Yes, the longer wavelengths of blues are the blues we need to keep happy and prevent ourselves from getting SAD. It’s the blue light near the ultraviolet and the blue indigo violet that are the most harmful and the wavelengths that we ought to get rid of. Not all wavelengths cause concern. Only short wavelength photons are individually capable of producing photochemical events, and these tend to be from the short wavelength blue end of the visible spectrum down through the ultraviolet. From the red end of the visible spectrum up through the infrared, photons do not have enough energy by themselves to produce photochemical damage and here damage results by large concentrations of them arriving in tissue, causing vibrational modes which are heat.
Could you elaborate on the particular ocular conditions that you have some concern about?

Many patient groups that suffer from conditions where the photoreceptor cells or light-sensitive cells are most vulnerable have been advised in the past to wear protective eyewear which typically looks “reddish” or “brownish” and such devices filter out harmful wavelengths whilst letting in the useful wavelengths required for vision. Large patient groups such as those with Retinitis Pigmentosa (RP) would be an example of a disease group that benefits from such protection.

Would you contend that from your personal belief that protective eyewear would be useful for people who are in early stages of any other ocular condition?

Several clinicians would advise patients in the early stage of AMD to wear peaked hats and to wear protective eyewear as well. The big problem is that patients do not get good advice currently as to which protective eyewear is going to be helpful; they are merely instructed that the device blocks 100% of ultraviolet, but usually they are given no information on how much blue is transmitted.

What role do you think clinical practice could play in prevention of the ocular problems you’ve described linked to blue-violet light?

I think the scientific base is pretty incontrovertible: short wavelength visible radiation is more harmful than long wavelength visible radiation. It should be remembered that we do not have any short wavelength photoreceptor cells, blue cones, in our foveas and that the macular region of the retina is protected by the presence of a yellow pigment thus blue plays no role in high acuity vision. We all suffer with foveal tritanopia and as a consequence we lose nothing by filtering out short wavelength blue in terms of our visual life. There is some resistance to wearing highly pigmented protective eyewear because many individuals don’t like walking around in bright yellow or brown lenses. This is why I think the current innovation from Essilor is quite interesting, because these lenses (Crizal® Prevencia®) are apparently transparent, and also reflect blue from the surface while absorbing the ultraviolet. This innovation is pretty interesting, because they now offer protection without being stigmatised for aesthetics.

Would you suggest that this innovation would be a useful correction that an eye care professional could deliver to a younger patient?

I think it’s extremely useful because wearing protective eyewear is similar to wearing sun cream. It won’t do any harm and probably it will do a lot of good over the course of one’s lifetime.

Earlier, you mentioned the shifts in internal lighting historically over the last hundred years. Do you see the more recent changes as a cause of concern?

Yes, both in terms of domestic and commercial lighting. Although lighting companies are working very hard to try and get rid of potentially harmful wavelengths, they’ve not been successful so far. The light sources they have produced with filters to filter out the harmful radiation are significantly more expensive compared to the light bulbs in our homes. In terms of fluorescent tubes, there is one sodium line which is almost 40% of the blue light hazard and accounts for less than 8% of the light, but they can’t get rid of it, because it facilitates lower costs and ease of manufacture.

What do we need to do to bring a level of public awareness around blue light and its potential harmfulness?

It would be very helpful to bring optometrists and eye care professionals up to date and to make sure they are in full possession of the basic knowledge. They would then be in a position to help their potential clients. Specifically in the field of the cataract surgery, we remove the natural yellow lens and implant a plastic intra-ocular lens; now virtually all intraocular lenses have UV block, and in recent years many IOL companies have introduced lenses with blue blocking or blue attenuating filtration. This is because when you remove the crystalline lens, the retina gets exposed to even more light damaging blue light and ultraviolet.
The benefits of the yellow sort of blue filter IOLs have been raised with the ophthalmologist community. What are your thoughts on this?

In Europe, the proportion of IOLs having blue filters varies from country to country; the highest ratio of blue blocking lenses is in France, where I believe 70% of the lenses have yellow filtration. It is less in many other countries. In the UK, ophthalmologists sometimes prefer clear lenses over blue blocking ones. They would like to see more established evidence of the benefits of blue blocking. There is mixed opinion on the subject, although experimental evidence does point in that direction. It comes down to education at the end. The mindset of ophthalmologists is progressively moving, but these things take time. When it comes for me to have my cataracts removed, I will certainly have a blue-filtering IOL implanted.

Interviewed by Andy Hepworth

B I O

Professor John Marshall
University College London

Professor John Marshall is the Frost Professor of Ophthalmology at the Institute of Ophthalmology in association with Moorfield’s Eye Hospital, University College London.

He is Emeritus Professor of Ophthalmology at King’s College London, Honorary Distinguished Professor University of Cardiff, Honorary Professor the City University and Honorary Professor Glasgow Caledonian University.

Primarily, he has concentrated his research on the inter-relationships between light and ageing, the environmental mechanisms underlying age-related, diabetic and inherited retinal disease, and the development of lasers for use in ophthalmic diagnosis and surgery.

He invented and patented the revolutionary Excimer laser for the correction of refractive disorders.

He also created the world’s first Diode laser for treating eye problems of diabetes, glaucoma and ageing.

Professor Marshall has been the recipient of several awards: the Nettleship Medal of the Ophthalmological Society of the United Kingdom, the Mackenzie Medal, the Raynor Medal, the Ridley Medal, the Ashton Medal, the Ida Mann Medal, the Lord Crook Gold Medal, the Doyle Medal of the Oxford Congress, the Barraquer Medal of the International Society of Cataract and Refractive Surgery and the Kelman Innovator Award of the American Society for Refractive and Cataract surgery. More recently in 2012 he received the Junius-Kuhnt Award and Medal for his work on AMD.

Professor Marshall has authored over four hundred research papers, 41 book chapters and 7 books.

“Wearing protective eyewear is similar to wearing sun cream. It won’t do any harm and probably it will do a lot of good over the course of one’s lifetime.”

KEY TAKEAWAYS

• Photons interact with biological tissue and may potentially lead to ocular health hazards.

• The red end of the visible spectrum up to the infrared can generate heat, while short wavelength photons can produce photochemical damage and accelerate ocular ageing process.

• Short wavelength blue-violet may exacerbate age-related macular degeneration (AMD) and UV radiation can potentially lead to earlier onset of cataract.

• Not all wavelengths cause concern. Long wavelength blue light is needed to balance biological well-being and Seasonally Adjusted Disorder (SAD).

• Selective photo-protection (filtering UV and short blue-violet light) is a necessity for eye health in the long term.

• Crizal® Prevencia® lenses selectively filter UV and the bad part of the spectrum while allowing good blue light to pass through. They maintain perfect transparency.
THE ROLE OF BLUE LIGHT IN THE PATHOGENESIS OF AGE-RELATED MACULAR DEGENERATION

Blue light exposure is one of the modifiable risk factors involved in the pathogenesis of Age-Related Macular Degeneration (AMD). Several studies have evaluated the relationship between light exposure and AMD, as well as clinical trials evaluated the visual function effect of blue filtering IOLs versus conventional IOLs. However, the authors encourage further clinical trials to assess the preventive filtering effect of ophthalmic lenses, particularly those with narrow bandwidth filters, in the development and/or progression of AMD.

KEYWORDS
AMD, neovascularization, blue-violet light, IOL, lipofuscin, rhodopsin, chromophore, RPE cells, photoreceptors, photopigment, photoreactivity, Crizal® Prevencia®
Age-related Macular Degeneration (AMD) is the most common cause of blindness in the elderly population in developed countries and accounts for 8.7% of all the blindness worldwide.\(^{1,2,3}\) In the future, the prevalence of AMD is likely to increase as a consequence of exponential population aging. The early stages of AMD are characterized by yellowish deposits (drusen) and/or pigmented changes of retinal pigment epithelium (RPE) but without overt functional loss of vision. In advanced stages of AMD, there is dysfunction and death of photoreceptors secondary to an atrophic (geographic atrophy, GA) and/or a neovascular (choroidal neovascularization, CNV) event leading to irreversible loss of central vision.

The early stages of AMD, compared to its later stages, affect a significantly larger proportion of the population and increase the risk for visually significant advanced AMD by 12- to 20-fold over 10 years.\(^4\) There have been significant advances in the management of neovascular AMD and the introduction of anti-angiogenesis therapy can now prevent blindness and in many cases restore vision.\(^5,6\) However, the treatment modalities are expensive and not available to patients in many countries.\(^7,8\) Therefore, identification of modifiable risk factors that may inform disease prevention programme is of priority. This review evaluates the long held belief that blue light exposure has a role in the pathogenesis of AMD.

Light is necessary for vision but it can damage the sight organ itself – a property that has long been recognized. The human retina is exposed to the “visible component” of the electromagnetic spectrum from 400 to 700 nm and some short wavelength infrared because ultraviolet radiations are naturally filtered by ocular tissues located in front of the retina, particularly the cornea (295 nm) and the crystalline lens (less than 400 nm). Therefore, high-energy visible light, the blue-violet light renamed “blue light” for simplification, between 400 and 500 nm wavelength reaches the retina.

Blue light may damage the retina in a number of ways involving different chromophores and cellular events; however, retinal damage by photochemical mechanism is most likely to correspond to the aerobic photoreactivity of the lipofuscin.\(^9\) The key component likely to contribute to lipofuscin’s photoreactivity is A2E (N-retinylidene-N-retinylethanolamine), a photosensitizer that has been demonstrated to produce ROS, trigger RPE cell apoptosis and lead to RPE cell death.\(^10,11\)

Long term exposures (typically 12-48 hrs) to less intense exposures produce damage at the level of the photoreceptors. The photopigments absorb the blue light and acts as photosensitizer resulting in photoreceptor damage. It is believed that deep blue light is 50-80 times more efficient at causing photoreceptor damage than green light due to rhodopsin photo reversal.\(^12\) Blue light promotes the photoisomerization of all-trans-retinal...
that leads to the regeneration of rhodopsin and an increase phototransduction signaling in turn leads to photoreceptor apoptosis. Photoreceptor damage may also take place from liberation of ROS by all-trans-retinal, which is a well-known photosensitizer.

**Blue light damage increases substantially with aging and may play a role in the pathogenesis of AMD.**

Phototoxicity contributed by lipofuscin increases substantially with age because of substantial increase in the concentration of photoreactive elements. Past studies have shown that aging significantly increased the potential for blue light hazard by nine-fold over a life span. Lipofuscin is of particular importance because of several reasons: first, the chronology of lipofuscin accumulation within RPE cells is coincident with the development of AMD; second, *in-vivo* autofluorescence studies have shown that degenerative changes in the retina corresponds with the areas of highest autofluorescence; thirdly, RPE cells are retained throughout life and their repair system operates at a molecular level and this type of closed-system is more prone to ROS induced damage.

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**TABLE 1**

List of studies that have evaluated the relationship between light exposure and Age-Related Macular Degeneration (AMD)

<table>
<thead>
<tr>
<th>INVESTIGATOR (YEAR OF PUBLICATION)</th>
<th>TYPE OF STUDY</th>
<th>SAMPLE SIZE</th>
<th>TYPE OF AMD</th>
<th>ASSESSMENT OF LIGHT EXPOSURE</th>
<th>CONCLUSION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Taylor H.R. et al. (1992)*</td>
<td>Cross-sectional</td>
<td>838</td>
<td>Late AMD (GA+CNV)</td>
<td>Blue light exposure at leisure and working time for the previous 20 years</td>
<td>High levels of exposure to blue and visible light in late life may play a role in the pathogenesis of late AMD (OR: 1.35, 95%CI:1.0-1.81)</td>
</tr>
<tr>
<td>Cruickshanks K. J. et al. (1993)* Beaver Dam Eye Study</td>
<td>Population-based</td>
<td>4926</td>
<td>Early AMD</td>
<td>Time spent outdoors in summer</td>
<td>The amount of time spent outdoors in summer was associated with an increased risk of early AMD (OR: 1.44, 95%CI:1.01-2.04)</td>
</tr>
<tr>
<td>Fletcher A.E. et al. (2008)*</td>
<td>Population-based</td>
<td>4753</td>
<td>Late AMD (CNV)</td>
<td>Leisure time spent outdoors in summer</td>
<td>The amount of leisure time spent outdoors in summer was significantly associated with neovascular AMD (OR, 2.26; 95% CI, 1.06 to 4.81) and GA (OR: 2.19; 95% CI 1.12 to 4.26)</td>
</tr>
<tr>
<td>Darzins P. et al. (1997)</td>
<td>Case-control</td>
<td>409/286**</td>
<td>Any type of AMD (early+GA+CNV)</td>
<td>Annual sun exposure</td>
<td>Sun exposure was relatively greater in control subjects than in cases with AMD (p &lt; 0.01)</td>
</tr>
<tr>
<td>Delcourt C. et al. (1997) POLA study</td>
<td>Population-based</td>
<td>2584</td>
<td>Early AMD</td>
<td>Annual ambient solar radiation</td>
<td>A decreased risk of early AMD was observed in subjects exposed to high ambient solar radiation (OR:0.73, 95%CI:0.54-0.98)</td>
</tr>
<tr>
<td>Taylor H.R. et al. (2007)</td>
<td>Cross-sectional</td>
<td>623</td>
<td>Late AMD (GA+CNV)</td>
<td>Leisure time spent outdoors in summer</td>
<td>The amount of leisure time spent outdoors in summer was significantly associated with neovascular AMD (OR, 2.26; 95% CI, 1.06 to 4.81) and GA (OR: 2.19; 95% CI 1.12 to 4.26)</td>
</tr>
<tr>
<td>Tomany S.C. et al. (2004)* Beaver Dam Eye Study</td>
<td>Population-based</td>
<td>3684</td>
<td>Early AMD</td>
<td>Leisure time spent outdoors aged 13-19 years and aged 30-39 years</td>
<td>Significant associations were observed between extended exposure to the summer sun and the 10-year incidence of early AMD (RR:2.09; 95%CI:1.19-3.65)</td>
</tr>
<tr>
<td>Khan J.C. et al. (2006)</td>
<td>Case-control</td>
<td>446/283**</td>
<td>Late AMD (GA)</td>
<td>Sun exposure index (per unit increment)</td>
<td>No associations between late AMD (GA) and sun exposure or related factors were observed (p = 0.44)</td>
</tr>
<tr>
<td>Khan J.C. et al. (2006)</td>
<td>Case-control</td>
<td>446/283**</td>
<td>Late AMD (CNV)</td>
<td>Sun exposure index (per unit increment)</td>
<td>No associations between late AMD (CNV) and sun exposure or related factors were observed (p = 0.29)</td>
</tr>
<tr>
<td>Hirakawa M. et al. (2007)</td>
<td>Case-control</td>
<td>148/67**</td>
<td>Late AMD (GA+CNV)</td>
<td>Facial wrinkle length (direct correlation with sunlight exposure)</td>
<td>Significantly more facial wrinkling was found in patients with late AMD (p = 0.047, OR: 3.8; 95% CI: 1.01 - 13.97)</td>
</tr>
<tr>
<td>Vojnikovic B. et al. (2007)</td>
<td>Population-based</td>
<td>1300</td>
<td>Any type of AMD (early+GA+CNV)</td>
<td>Exposure of sunlight</td>
<td>Significant correlation was observed between chronic exposure to sunlight and prevalence of any type of AMD</td>
</tr>
<tr>
<td>Plesitana-Borjan I. et al. (2007)</td>
<td>Cross-sectional</td>
<td>623</td>
<td>Any type of AMD (early+GA+CNV)</td>
<td>Mean daily exposure (in hours) to solar radiation</td>
<td>A positive relationship was observed between long-term sunlight exposure and increased risk of any type of AMD</td>
</tr>
<tr>
<td>Fletcher A.E. et al. (2008)*</td>
<td>Population-based</td>
<td>4753</td>
<td>Late AMD (CNV)</td>
<td>Blue light exposure</td>
<td>Significant associations were found between blue light exposure and neovascular AMD in patients with lowest antioxidant levels (OR:1.09,95% CI:0.84-1.41)</td>
</tr>
</tbody>
</table>

* significant and positive association; ** no. of controls; GA: Geographic atrophy; CNV: Choroidal neovascularization; OR: Odds ratio; RR: Relative risk; CI: Confidence interval
Several studies in the past have evaluated the role of blue light on the development of AMD (Table 1). A study by Taylor et al. on 838 watermen of the Chesapeake Bay demonstrated that patients with advanced AMD had significantly higher exposure to blue or visible light over the preceding twenty years. Similarly, the Beaver Dam Eye Study observed that visible light rather than UV light might be associated with AMD. Furthermore, the EUREYE study found a significant association between blue light exposure and late neovascular AMD in individuals having the lowest antioxidant levels.

Recently, a systematic review and meta-analysis included fourteen studies that evaluated the association between sunlight exposure and AMD. In this review article, twelve out of fourteen studies identified an increased risk of AMD with greater sunlight exposure, six of which reported significant risks. The pooled odds ratio was 1.379 (95% confidence interval 1.091 to 1.745). The subgroup of non-population-based studies revealed a significant risk (odds ratio 2.018, confidence interval 1.248 to 3.265, p=0.004). The authors concluded that individuals with more sunlight exposure are at significantly increased risk of AMD.

It is important to note that epidemiological studies evaluating light exposure and risk of AMD have several limitations. The pathogenesis of AMD is very complex and lifetime light exposure cannot be measured accurately. Also, there are notable dif-
Difficulties in such studies that depend on the patients’ own recall about cumulative exposure to blue light. Moreover, other factors including variability in genetic susceptibility or diet may obfuscate the true relationship between light exposure and AMD.

The nature of the blue light induced damage is dependent not just on the photoreactivity of a variety of chromophores but also on the capacity of the defense and repair systems. One of the defense systems that deserve special mention is macular pigment (MP). MP is composed of two dietary carotenoids, lutein (L) and zeaxanthin (Z), and has peak concentration within the central 1-2 degrees of the fovea. MP carotenoids are natural protective filters attenuating short-wavelength light prior to photoreceptor light capture with absorbance spectra ranging from 400 to 500 nm (lutein = 452 nm; zeaxanthin = 463 nm). It is therefore particularly effective at reducing the potentially damaging effect of lipofuscin whose photo reactivity peaks at 450 nm in elderly population. MP acts, uniquely as an antioxidant, both passively and actively, the former mechanism being dependent on its ability to limit photo-oxidative damage by filtering short wavelength light at a pre-receptorial level and the latter mechanism attributable to its capacity to quench ROS.

Implantation of blue-light filtering intraocular lens (IOLs) following cataract surgery may have the potential to protect the retina from oxidative damage secondary to blue light and slow the progression of AMD. In experimental studies, these IOLs have been demonstrated to significantly reduce the death of RPE cells from light induced damage mediated by lipofuscin fluorophore A2E. Furthermore, blue light filtering IOLs may provide additional visual benefit for AMD patients because blue light is selectively scattered by the ocular media and its attenuation has been associated with improvements in contrast sensitivity and a reduction in glare sensitivity.

There have been theoretical speculations about the potential negative ramifications of filtering blue light. Blue light provides 35% of scotopic vision, 53% of melanopsin, 55% of circadian and 32% of s-cone photoreception. Blue light filtering IOLs eliminate 27-40% of incident blue light depending on their dioptric power. The decrease in blue light photoreception therefore may result in impairment of color vision, scotopic vision, and circadian rhythm. Several randomized clinical trials have been conducted to compare visual performance using blue filtering IOLs and conventional IOLs in healthy volunteers and in patients with AMD (Table 2). The results from these trials suggest that there are no clinically significant effects on various measures of visual performance, including color vision, photopic and scotopic sensitivities and contrast sensitivity with blue filtering IOLs.

Also, given the great improvement in light transmission achieved simply by removing the cataract, it seems unlikely that blue filtering IOLs cause any significant disruptions to the circadian rhythm. However, there is a current lack of evidence that demonstrates that blue filtering IOLs have any effect on AMD. No randomized prospective studies have been conducted to prove claims of macular protection against progressive disease.

Furthermore, a recent study in animal model suggested that the 415-455 nm spectral range might be the most damaging light for patients at risk of AMD. The authors suggest that filters in this narrow bandwidth would not occlude light in the 460-500 nm range, not only essential for color vision but also for circadian rhythm regulation mediated by melanopsin-sensitive retinal ganglion cells. However, it remains to be evaluated if new selective ophthalmic filters in the defined bandwidth could provide macular protection in patients at risk of AMD.

Similarly, another proposed option is to use eyeglasses that attenuate short-wavelength light in bright environments for effective photo-protection. Crizal® Prevencia® No-Glare clear lenses represent the first application of new patent-pending technology.

“In the future, well-designed clinical trials should be undertaken to evaluate the effect of blue light filtration in the development and/or progression of AMD.”

KEY TAKEAWAYS
that enables selective attenuation of harmful light, both UV and blue-violet, while allowing beneficial light to pass through and maintaining exceptional transparency at all other visible-light wavelengths. The goal is to enable patients to enjoy the best vision with significant protection against UV and high-energy blue-violet wavelengths. The advantage of eye-glasses (c.f. IOLs) lies in the fact that there is freedom to remove sunglasses for optimal scotopic and circadian photoreception, if necessary.

In summary, there is persuasive theoretical and experimental evidence suggesting that blue light exposure may damage the retina and possibly play a role in the pathogenesis of AMD; however, there is a paucity of clinical evidence to support this notion. In the future, well-designed clinical trials should be undertaken to evaluate the effect of blue light filtration, particularly those with narrow bandwidth, in the development and/or progression of AMD. •

**KEY TAKEAWAYS**

- Blue light provides 35% of scotopic vision, 53% of melanopsin, 55% of circadian and 32% of s-cone photoreception. Yet blue-violet light may damage the retina.
- The nature of the blue-violet light induced damage is dependent on the photoreactivity of a variety of chromophores and on the capacity of the defense-repair systems.
- A systematic review and meta-analysis indicates that people with more sunlight exposure are at significantly increased risk of AMD.
- However, individual patients’ cumulative exposure to blue-violet light is complex to measure. Several other individual factors involved in AMD pathogenesis can vary, including genetics, diet, etc.
- Implantation of blue-light filtering intraocular lens (IOLs) following cataract surgery may have the potential to protect the retina from oxidative damage secondary to blue light and slow the progression of AMD.
- Blue light filtering IOLs eliminate 27-40% of incident blue light depending on their dioptric power.
- It remains to be evaluated if new selective ophtalmic filters in the defined bandwidth could provide macular protection in patients at risk of AMD and/or patients operated on from cataracts.
The Benefits and Dangers of BLUE LIGHT

By Christian Sotty

The blue light region in the visible light spectrum has captured the interest of scientists due to its role in non-visual biological mechanisms such as regulation of the circadian cycle. This part of the light spectrum can have a positive impact on health, and it ranges from 465 to 495 nanometers (nm) (Blue-Turquoise light). However, in the range of 415 to 455 nm (Blue-Violet light), it has been established that light induces high levels of mortality in the retinal pigment epithelium (RPE) cells.

Blue light (also known as high energy visible light) ranges from 380 nm to 500 nm. It is emitted by both natural (sun) and artificial light sources, such as LED lighting.

Synchronizing our biological clock

Light, and in particular “good” blue light, also known as “chronobiological light,” regulates our individual circadian rhythm. We need to reset our biological clocks daily in order to synchronize our biological rhythm. Our clock transmits to a number of parts of the body, such as the liver, muscles, heart, kidneys and other organs. All biological functions need to work at the right moment, and because our biological clock drives this particular rhythm, it ensures particular functions are active at the right time.

“Light acts on the retina through the action of specific cells—melanopsin-containing ganglion cells—which are different from the cones and rods that are the photoreceptors used in vision,” said Claude Gronfier, INSERM (French Institute of Health and Medical Research) chronobiology researcher. “When these ganglion cells are activated by blue light, they transmit a nerve signal that runs along the optic nerve and, rather than activating the visual structures in the brain, activates non-visual structures such as our internal circadian clock. So it’s exposure to light that resets the time on the biological clock.”

Blue light and AMD

Recently, it has been shown that exposure to light contributes to the early occurrence of age-related macular degeneration (AMD). In-vitro experiments on porcine cell cultures point specifically to blue light, which is more energy intensive. Macular pigments are natural filters for these wavelengths. Unfortunately, pigments don’t accumulate well in the retina as we age or when disease starts.

“It’s essential to combine several approaches to help explain the pathophysiological impact of light on the retina and the part played by these effects on retinal conditions,” said Serge Picau, INSERM director of research at the Paris Vision Institute. “This multidisciplinary aspect was one of the challenges of a recent project in which we tried to define toxic wavelengths in the visible spectrum. Our main aim was to calculate the relative quantity of light reaching the retina in each wavelength. We measured the toxicity of these relative irradiances using an AMD porcine cell model.

“The work enabled us to define the most phototoxic spectral bands against this cellular model,” he said. “Optics specialists from Essilor took part in the project to help us design optical devices to calculate retinal light irradiances and to manipulate concepts involving light.”

References
Exploring the latest findings and research in Blue Light

Report of expert round table discussion, 14th January 2016, London, UK
Introduction

A round table discussion held in January 2016 – chaired by Professor John Marshall with a panel of experts representing research, ophthalmology, academia and retail optometry – set out to determine the extent to which blue light is a hazard to the human eye and to establish whether it is implicated in disease such as age-related macular degeneration (AMD).

Discussions included the availability of existing research and the likelihood of future studies being conducted, which will help support the increasing body of evidence that blue light is a concern for eye health. They concluded by suggesting how this potential risk should be discussed in the practice environment.
The Panel

John Marshall MBE
Professor at University College London’s Institute of Optometry in association with Moorfield’s Eye Hospital. John has sat on many of the world’s safety committees concerned with protecting individuals against lasers and other sources of optical radiation. He has generated substantial data, now used in codes of practice and is interested in utilising light as a form of intervention in terms of surgery and diagnosis. He had the first patents for UV lasers to carry out refractive surgery and has had a lifelong interest in the interaction between light and cellular ageing, especially ageing in the retina. He has published many papers on the interactions between light, ageing and the membrane between the neural retina and the underlying choroidal blood supply membrane which is one of the first elements to undergo change during the process we know as AMD.

Tom Margrain
Based at Cardiff University Tom has had a long running interest in age-related macular disease and in particular the effects of light on the condition. Amongst other things he has worked as an optometrist and electrophysiologist.

Mike Killpartrick
An optometrist and independent practitioner based in Bath and Cheltenham, Mike is interested in light as a contributing factor in macular degeneration and in ensuring his customers are well informed on the latest evidence and thinking.

Bill Harvey
An optometrist with a specialism in low vision, Bill has lectured in low vision at City, Plymouth and Surrey Universities for many years and he is also involved in professional training for Boots Opticians. He is interested in prevention rather than heavy back-end management of macular degeneration – and ensuring he has appropriate, accurate and evidence-based information to share with practitioners.

Serge Picaud
As a scientist and physio-pathologist at the Vision Institute in Paris, Serge is interested in understanding how retinal cells degenerate and how this can be prevented. The aim of this research is to examine the mechanisms used by the retina to process visual information and to use this to develop new neuro-protective or rehabilitation strategies. He is also concerned with the effect of light on retinal cell degeneration and restoration of vision in blind patients.

John Nolan
Principal investigator of the Macular Pigment Research Group based in Waterford in Ireland, John’s primary interest involves the study of nutrition for the eye and how this can be optimised in macular pigment, which plays a key role in filtering blue light. He believes that filtering blue light optically has a key role in visual functions and that enhancing visual function today while protecting our vision into later years is something that needs to be understood particularly by the optometry community.
What does the science and current thinking tell us?

Is there adequate data to say we ought to treat short wavelength blue-violet with suspicion and perhaps take protective measures to limit the amount we are allowing to pass into the retina?

“We do not have a great deal of chronic data so we have to balance that against recent studies – from behavioural psychologists rather than vision scientists – which show that there is a requirement for the longer wavelength blue – at around 480 nanometres (nm) – in order to harmonise our lives and prevent us from getting acute depression.

“At one end there’s the blue we don’t want which is the short wavelength blue – blue-violet as some people refer to – and at the other end we have the longer wavelength blue – blue-turquoise - that we absolutely do need.

“It is quite clear that UV light and short wavelength visible light impacts on skin ageing so even in a system like the skin, which is renewing itself, accumulated damage will result from chronic exposure to light. The retinal system is not turning over so does that give rise to special problems? We don’t need to live beyond the age of 30 but we are living much longer. We also need to consider changed environment. Because of various government misconceptions, we’ve moved to low energy sources and now LEDs with very bright blue components and some UV components are creeping into our homes. At the flick of a switch we can have daylight illumination anytime we want.”


“I think there is a pretty large body of evidence which does implicate light in the development of AMD and the paper that did it for me is by Sui et al1 in the British Journal of Ophthalmology in 2013 - a meta-analysis of all of the epidemiological data. Although the emphasis is on sunlight exposure, we might deduce that blue light is the major damaging component in sunlight.”

Tom Margrain

“The Sliney2 paper showed how important the geometric analysis of exposure is in all of these studies - just monitoring how long a person stays outdoors gives you no idea of their ocular exposure. Sui et al3 is a good clarifying paper and it would be even better if someone could look at the geometry of exposure and better analyse the reality of exposure.” John Marshall

“There are so many factors associated with the progression of AMD – some which you can’t do much about such as your family history, many of which you can such as smoking and diet – but it’s difficult, unless you’ve got a really good meta-analysis of a load of papers, to establish any definites because most of these things are difficult to control.

Stephen Dane does repeat the fact that there is surface damage in the short wavelength; it is accepted now that it will damage the replication of cells and there will be surface problems with basal cell carcinomas (BCCs), corneal changes and so on. But the further back into the eye there are question marks about cumulative damage at different ages. Do these things accumulate over time or does it more likely depend on how your recovery processes are ingrained in you genetically? There is agreed significant output, for example, from LED sources and significant potential danger in the ophthalmic equipment we use day to day so I don’t think it would be controversial to suggest that short wavelength visible light has safety concerns especially in younger people where significant amounts can access the retina and the macula.

“Do we act now before the evidence bank is enough to confirm that younger patient exposure is damaging and should we be intervening now with younger patients in filtering out the potentially phototoxic wavelengths? And I think this is where the debate has to focus.

“I think the blue light impact on surface tissues is difficult to argue against, but it seems to me we’re still having to make a reasoned decision on what is, as yet, not conclusive evidence.” Bill Harvey

“We’ve got this confusion between UV and blue. Even in the very young not a lot of UV is going to get through except through the little window. Blue is certainly going to get through and it is certainly going to fall on the retina. Hazardous blue, i.e. the high photon blue, is around 440 nm whilst the melanopsin blue is around 480 nm so it would be easy to differentially block those two.

“Ask an audience of ophthalmologists today “how many of you would put in an intraocular lens without a UV blocker?” and not one person would put their hands up. That’s on the basis of no clinical evidence. Ask the same audience how many will use these new intraocular lenses with a degree of blue blocking and, depending on which country you’re in it’s about 50/50. The argument there is that there is no clinical evidence, but there was no clinical evidence previously. Should we act now and prevent something or wait until we get the data by which time we’ll have lots more people with problems?”

John Marshall

“Concerning the evidence of blue light toxicity and light toxicity related to AMD, I was quite convinced by all the clinical evidence which has shown that blue and violet light can be toxic. It does seem that blue light in general can enter the eye and reach the retina and these wavelengths can be toxic to the cells.

“In animals, when you deprive the antioxidant defence you do see some damage to the cells at low light levels. So with patients with low antioxidant defence you may see this kind of damage as well.
Why would you see this in animals and not in humans? This is not acute light damage, but chronic effects. It’s difficult to reach a clear conclusion for patients but we have shown that the blue-violet light is much more toxic to the retinal pigment epithelium cells (RPE) at the back of the eye when you load them with chromophore like A2E, which is a natural pigment that you find edging a retinal pigment cell.

“We do believe that in ageing patients where you have an accumulation of this kind of chromophore you could have damage from blue-violet light. Although we normalise the light used in our experiments to the light of the sun reaching the retina, it’s clear that we always use higher intensities than those which reach the retina. So it’s possible this type of light would damage other cells such as the photoreceptors. But we are quite convinced blue light - and maybe more blue-violet light - can be really toxic not only to retinal pigment cells, but also other neurons such as ganglion cells and the photoreceptors.” Serge Picaud

“We have to address the difference between cumulative damage and ageing and cumulative damage and the flip between ageing physiology and overt disease like AMD. In my mind it’s clear that light exposure certainly is a rate limiting driver for ageing processes. The consensus is that we all feel we have an issue here with short-wavelength radiation and it’s not biologically friendly, but how far are we prepared to stick our necks out?” John Marshall

“Where do we sit in terms of the evidence for short wavelength blue light? From a human perspective it’s difficult to quantify light exposure. The answer to the human question is that we can’t attribute retinal disease to any one factor such as blue light. We’re talking about a disease that’s the result of cumulative (chronic) impacts over a person’s lifetime with many contributing factors - some of which are set in stone - such as genetics. We can begin by looking at animal studies – where you can accelerate a process such as blue light exposure and create irreversible retinal changes, but it’s a multi factorial disease and we have to understand many other factors, such as the antioxidant potential, the shape of the eye and the quantity of light. In summary, I would have no issue with making a comment that is scientifically backed that we need to be aware of the impact of shortwave light on the human population.” John Nolan

**Why it is difficult to prove a link between blue-violet light and ocular disease**

Although there was general agreement that blue light could well be a factor in ocular disease, an emerging theme throughout the discussion was the lack of appropriate human research to demonstrate the link between blue light and macular degeneration and support the compelling animal studies that exist. The panel agreed on the usefulness of such research but there was a lack of consensus as to whether such research could succeed or indeed be funded given the scale, complexity and duration required.
"There is one research design that’s tried and tested and used when introducing a new intervention and that’s the randomised control clinical trial. There is a lot of great underlying cell biology and some good epidemiological data but the clinical trials evidence is missing.

“One challenge we have with AMD is that if you look at studies such as AREDS (Age Related Eye Disease Study) for example they typify the difficulties, i.e. a massive sample size and people followed up over a long period of time, so there are challenges but it is achievable.”

Tom Margrain

“Do a clinical trial in a human population is impossible.” John Nolan

“You can’t even do that with intraocular lenses. It would be very difficult to do a randomised control trial.” John Marshall

“I’m personally dubious about how much a randomised controlled study trial would be available and how trustworthy it would be.” Bill Harvey

“It’s very difficult for non-scientists to understand the difficulties involved here and even for scientists to discriminate between ageing and AMD because if you look at ageing you see many of the clinical symptoms of AMD.” John Marshall

Risk factors for AMD, risk groups and comparisons with sunlight and skin

Comparisons were made with sunblock – especially in terms of compliance and consumer understanding of risk. There was also broad agreement about the risk factors for AMD.

“Everyone in this room will happily put on sunblock when you go out in the sun and we all believe that’s a good thing. Does it significantly reduce the incidence of skin cancers? We haven’t got that evidence for the same reason it will be difficult to do. We’re still happy to say it, it does work!”

Mike Killpartrick

“Sunblock is probably not a bad analogy as a lot of the problems are to do with compliance and understanding on the patient’s part. There is a potential danger someone will slap sun cream on once at the beginning of the day. It has a minimal impact as the day progresses. It might even give them an inherent belief that they are invincible and stay in the sun longer. Increasingly, the primary care sector has an astoundingly important role to reduce the burden on secondary care by giving good solid advice. Ten years ago you never asked a patient if they smoked in their history of symptoms. Hopefully now that’s taught at all the universities.”

Bill Harvey

“We’re very aggressive. I now say to my patients, if you want to increase the risk of macular degeneration start smoking.”

Mike Killpartrick

“What macular pigment is doing and the pigments at the back of the eye that are likely to be sensitive... it’s an interplay of all these things that are likely to be taken into account. The feeling is that older adults stand to benefit more than younger people.”

Tom Margrain

“I think we would all agree that the ability of the eye to function begins to degrade with age. We can all agree that age is the biggest risk factor in AMD and smoking is accepted as a significant risk factor in AMD. Then we’ve got genetics and we all agree genes are playing a role. Then we get into dietary issues and light exposure. There is agreement that light exposure may have a role but we’re not defining it and all the work we’ve done has shown it does increase ageing in experimental models.”

John Marshall

“The AMD story is not conclusive but based on the evidence that’s available from the basic science all the way up to the gold standard clinical trials, I think the evidence for nutrition is absolutely favourable that we should be active in that space and the patients we’ve worked with will confirm that.”

John Nolan

“Regarding risk factors from blue light and whether risk is higher at certain stages in the disease, we don’t have the data, but my research would seem to indicate it is a cumulative effect and from the twenties onwards you’re beginning to build up debris in the system. The evidence is not there but, the earlier the intervention the better.”

John Marshall

The potential for negative effects when filtering out blue light

Although some blue light is needed to regulate sleep, memory and brain performance, the use of spectacles to filter out unwanted blue light was not seen as a concern. Experts support the idea of precisely filtering harmful wavelengths, while allowing transmission of beneficial blue.

“I cannot personally see anything that’s negative about this.”

John Marshall

“From a spectacle point of view I agree. From the macular pigment point of view the evidence is all supportive that vision gets better. So you can infer from what the
pigment does and what the lenses do, I think it’s complimentary.” John Nolan

“It goes back to ‘will’ versus ‘is likely,’ Now I think we have to say to people are you aware smoking causes damage? Are you aware that UV causes significant surface damage and some internal damage depending on the exposure? I think we are now in the realms of saying there is some evidence that a blue light filter on the spectacle lens has some protective benefit.” Bill Harvey

“When we talk about intraocular lenses with blue filters – I’m much more comfortable as a vision scientist to say to a patient that if the evidence is in support of blue filtering to do so with spectacles rather than intraocular filters.” John Nolan

“If I was wearing glasses, especially outdoors, I would like to keep bright light for the activation of the chronobiology which is around 480 nm. We know it’s quite useful to have some kind of bright exposure and maybe it also has a role in progressive myopia control. So I wouldn’t want to completely block all wavelengths with sunglasses.

“When we apply photosensitisers like A2E on the retinal pigment epithelium (RPE) cells the toxicity is from 415 to 455 nm so I would take no risk in blocking these wavelengths and keeping those at 480 nm to excite my chronobiology because I want to be awake and also because it has a lot to do with your well-being.” Serge Picaud

“If we could block this end and not block that end, in terms of the spectra, we really want to have this significant reduction around 450 down and we want good transmission up around 465.” John Marshall

What does this mean for those in practice when talking to patients?

“I think patients should be aware of light damage. They should be aware that there’s some evidence of short wavelength not necessarily being good long term, in terms of external particularly and then less so with internal exposure. I think the UV message has to be got across absolutely to all patients and I don’t have an issue discussing what we currently know regarding blue light with patients the way I think we should be discussing what we know regarding nutrition. I think those two things need to be out there in the primary care sector.” Bill Harvey

Key Takeaways

We need longer wavelength blue (blue-turquoise) exposure to synchronise our biological clock and preserve health functions.

Sunlight is strongly incriminated not only in acute ocular damage, but also in the development of chronic changes such as cataracts and even severe diseases such as AMD. There is growing evidence that, in particular, blue light could be implicated in the development of AMD.

AMD has a multifactorial pathogenesis: age, genetics, smoking, diet low in vitamins, retinal phototoxicity, obesity and hypertension are all likely to play a role.

Prevention matters. Blocking sunglasses, specialist lenses to filter out UV and possibly blue-violet light and nutraceuticals can all play a part. Clear everyday lenses that filter harmful wavelengths (blue-violet), whilst allowing the transmission of beneficial blue light (blue-turquoise) could also help protect against long term damage of the eye.

Informing patients of UV danger and growing evidence on blue-violet light is important and particularly with patients who have a strong family history of macular degeneration, already have signs of it or have a high exposure to sunlight.

Those most vulnerable to the chronic effects of light exposure are children as well as the elderly; people with a family history of AMD; those who have had cataract surgery; outdoor workers or people who are exposed to sources of radiation and heat, or in prolonged contact with LEDs – and people with fair complexions.

References

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The purpose of this paper is to explore evidence-based findings on the effects of harmful light. Based on literature and articles published in peer-reviewed journals, the authors offer a critical analysis of the current therapeutic implications of the selective manipulation of short-wavelength high energy light. With the appearance of new medical devices, in particular clear ophthalmic lenses capable of filtering both UV radiations and blue-violet light, this is an area of growing interest for clinical practice and potential preventive measures.

**EXPLORING CLINICAL EVIDENCE AND THE BENEFITS OF FILTERING OUT HARMFUL LIGHT**

Visible light is composed of wavelengths ranging between approximately 380 and 780 nm. In humans, the components of this spectrum do not only interact with photoreceptors of the eye, but also have multiple local and systemic effects, which are yet to be fully documented. Blue light has recently been the subject of much interest. “Blue light” is generally considered as covering the part of the visible spectrum with wavelengths between approximately 400 and 500 nm. However, given the significant differences, in terms of phototoxicity and biological effects, between the components at either end of this range, it is more appropriate to distinguish between blue-violet light (400-455 nm) and blue-turquoise light (465-500 nm).

Because of its high energy, short-wavelength light in the narrow range (415-455 nm) – blue-violet light – has been associated with possible harmful effects, particularly on the retina [1]. The macula is especially vulnerable to damage from high-energy radiation. Most UV radiation is absorbed by the cornea and crystalline lens and it is in these structures that excessive exposure to these frequencies primarily manifests, typically in the form of photokeratitis, conjunctival alterations (acute damage) and cataracts (chronic damage). However, almost all visible radiation passes through the anterior segment of the eye with little attenuation (85-90% transmittance) [2, 3] before being absorbed by the retina and the retinal pigment epithelium (RPE). Although less harmful to biological tissue than UV radiation, the short-wavelength end of the

**KEYWORDS**

UV, blue-violet light hazard, harmful light, tear film dysfunction, dry eye, visual fatigue, visual discomfort, glare, AMD, cataract, prevention with ophthalmic lenses filtering.
visible spectrum can also cause photochemical damage, especially from prolonged and cumulative exposure. The damage caused to the RPE and the neuroretina by blue light has been documented in a number of scientific publications. Recent studies have drawn attention to the toxicity of blue-violet light to retinal ganglion cells, whose axons make up the optical nerve. These cells are less protected by the macular carotenoids due to their location in the retina, they are also rich in mitochondria which produce the energy necessary for the continuous generation of action potentials. The chromophores contained in these organelles are most stimulated by blue-violet light, impairing their function and increasing the production of oxygen free radicals. These effects constitute an important area of research with the potential for new strategies of neuroprotection, a topic of central importance in retinal vasculopathies such as diabetic retinopathy. The use of selective filters in clear ophthalmic lenses is also an increasingly reliable source of retinal protection. Due to their high energy, short wavelengths are also capable of causing damage to cellular DNA, either directly or by increasing the formation of reactive oxygen species (ROS), and are among the suspected risk factors in uveal melanoma.

Blue-violet light and age-related macular degeneration
While age-related macular degeneration (AMD) is a multifactorial condition, various studies have suggested a connection with blue light. As the leading cause of vision impairment in over-50s, this pathology has significant social impact. Prolonged and continuous exposure to short wavelengths is believed to contribute to the development and progression of AMD. The use of lenses that can selectively filter the light, especially at the blue-violet end of the spectrum, reaching the retina is thus likely beneficial. The blue-violet band in fact has greater phototoxic potential than the blue-turquoise band. It has recently been reported that the implantation of pigmented intraocular lenses (IOL) which block blue-spectrum light reduces, over time, the development of autofluorescence anomalies in the fundus of the eye compared to patients with transparent IOL implants with anti-UV blocking only. Fundus autofluorescence is a standard test for early diagnosis of RPE alterations associated with AMD. The findings of this study have evoked strong interest, although they need to be confirmed by randomized trials. These alterations are triggered by various mechanisms, including the production of oxygen free radicals. The type of IOL in the anterior segment has been demonstrated to have a significant impact on the levels of oxidative stress measured in the vitreous gel. In nuclear cataracts there is a yellowing of the lens, increasing its ability to filter blue light up to 60% and the oxidative stress to which the retina is exposed is considerably lower when nuclear cataracts are present than in patients with transparent IOL implants with UV blocking only.

Blue-violet light and refraction
The potential benefits of filtering out blue-violet light are not limited to broad protection of the retina and RPE, but also extend to vision quality. The eye is a complex dioptric system, and the light rays which pass through it undergo scattering before arriving at the retina. The components of a beam of white light are deflected differently in accordance with their wavelengths, producing chromatic aberrations. This occurs because the refraction index of a light-carrying medium differs according to the wavelengths transmitting through it. The shorter the wavelength, the higher the refraction. This principle accounts for the formation of rainbows, in which water droplets in the air act as microscopic prisms.
Absorption of blue light by specific ophthalmic lenses reduces chromatic aberrations, thereby improving the sharpness of images. In patients with an unstable tear film, this effect on visual quality is even more pronounced. Patients with dry eye and an unstable tear film are known to have decreased visual acuity. [22] This is because light rays undergo greater dispersion when they pass through an irregular tear film, and this dispersion is greater with short-wavelength light. These aberrations create blurred images, increasing visual fatigue and photo-stress, ultimately causing headaches and epiphora. Since blue-violet light is the most critical part of the visible spectrum causing such aberrations, blocking this light should reduce discomfort. Supporting this, a recent study showed that patients with an unstable tear film achieved better results in visual acuity tests when using a blue-violet filter. [23] Another study showed that the use of blue light-blocking ophthalmic lenses can reduce the glare and photo-stress associated with prolonged exposure to intense light. [24] Clear ophthalmic lenses with these blocking properties would avoid the need for tinted lenses in situations where photo-stress is particularly strong, such as after cataract surgery.

« Prolonged and continuous exposure to short wavelengths is believed to contribute to the development and progression of AMD. »

FIG.1: Prof. Loperfido with a patient

Prolonged and continuous exposure to short wavelengths is believed to contribute to the development and progression of AMD. "

Absorption of blue light by specific ophthalmic lenses reduces chromatic aberrations, thereby improving the sharpness of images. In patients with an unstable tear film, this effect on visual quality is even more pronounced. Patients with dry eye and an unstable tear film are known to have decreased visual acuity. [22] This is because light rays undergo greater dispersion when they pass through an irregular tear film, and this dispersion is greater with short-wavelength light. These aberrations create blurred images, increasing visual fatigue and photo-stress, ultimately causing headaches and epiphora. Since blue-violet light is the most critical part of the visible spectrum causing such aberrations, blocking this light should reduce discomfort. Supporting this, a recent study showed that patients with an unstable tear film achieved better results in visual acuity tests when using a blue-violet filter. [23] Another study showed that the use of blue light-blocking ophthalmic lenses can reduce the glare and photo-stress associated with prolonged exposure to intense light. [24] Clear ophthalmic lenses with these blocking properties would avoid the need for tinted lenses in situations where photo-stress is particularly strong, such as after cataract surgery.
These studies also demonstrated how the use of filters which block short-wavelength light can produce significant and measurable clinical effects, including when incorporated into glasses lenses. In optical terms, therefore, the ability to selectively filter out blue-violet light is likely to be of value, as it helps to improve the quality of vision without significantly affecting cone and rod function. In this way, scotopic and photopic sensitivity remain almost unchanged. Preserving night vision is an essential requirement for permanent lenses. The number of rod cells in the retina diminishes with age, while the number of cones remains fairly constant throughout an individual’s lifetime. This phenomenon explains the reduced ability to adapt to darkness and the problems with night vision reported by so many adults.

The duality of blue light
Although using lenses with short-wavelength cut-off filters may provide the retina with better protection against the harmful effects of photoexposure and reduce chromatic aberrations, the non-selective attenuation of blue light spectrum will also eliminate any associated beneficial effects. Many studies have shown that blue-turquoise light is one of the principal regulators of the circadian rhythms, contributing to chronobiological functions, with melanopin stimulation peaking at 482 nm. This photopigment does not contribute to the generation of a visual signal, but sends impulses to the neurosecretory nuclei regulating circadian rhythms by releasing mediators such as melatonin. During the day, exposure to blue-turquoise light promotes alertness, improves reaction times and regulates mood.

As blue light exhibits pathological as well as physiological characteristics, the ideal non-tinted ophthalmic lens would therefore block the harmful effects of blue-violet light while preserving chronobiological functions associated with blue-turquoise light. A study on tinted (yellow) IOLs analysed the biological effects of these lenses, locating the equilibrium point between photoprotection and photoexposure at 445 nm. However, it is very important to bear in mind that not all blue-filtering IOL implants are equivalent, with very large variations in the amount and type of light filtered by these implants.

Conclusion
In recent years, significant clinical evidence has emerged for the benefits of reduced exposure to blue-violet light. Those who particularly stand to benefit from these include sufferers of tear film dysfunctioning and dry eye (especially when associated with prolonged use of digital devices and...
intense exposure to natural light), patients suffering from, or with a family history of, macular impairment, and patients requiring additional neuroprotection.

In recent years, the risk of repeated and prolonged exposure to blue light sources has increased. Several reasons contribute to this, including the widespread adoption of low-consumption light sources such as LEDs, and intensive use of tablets and computers. In theory, filtering blue light could reduce the cumulative damage associated with chronic exposure, such as by incorporating filters in the light sources themselves, or individual photoprotection. The latter option could take the form of tinted IOls for patients undergoing cataract surgery or glasses for all other patients.

There are numerous aesthetic and functional advantages to incorporating selective blue-violet filters in the transparent lenses of conventional eyewear. Currently, most available non-tinted lenses have surface coatings for filtering blue-violet light, but lenses with a built-in capacity for absorbing UV and blue-violet light are now coming to the market with high transparency and improved aesthetics.

**KEY TAKEAWAYS**

- The harmful effects of short-wavelength high energy light, specifically UV and blue-violet light, have been clearly demonstrated by a large body of peer-reviewed literature.
- The potential benefits of filtering out UV and blue-violet light offer not only greater protection of the anterior segment of the eye and the retina, but also extend to the quality of vision.
- The use of blue-violet light filtering opthalmic lenses can reduce the glare and photo-stress associated with prolonged exposure to intense light.
- Those who particularly stand to benefit from clear blue-violet light filtering lenses are:
  - Patients who suffer from tear film dysfunctioning and dry eye, especially when associated with prolonged use of digital devices and intense light exposure,
  - Patients suffering from, or with a family history of, macular impairment,
  - Patients requiring additional neuroprotection.
- The new opthalmic lenses coming to the market reflect a growing interest for clinical implementation of light filtering from the perspective of visual comfort and/or potential preventive measures.

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EYE COMPLICATIONS OF EXPOSURE TO ULTRAVIOLET AND BLUE-VIOLET LIGHT

Harmful effects of ultraviolet (UV) and blue-violet light manifest differently in the human eye. Cumulative exposure to blue-violet light has been linked with the development of age-related macular degeneration (AMD). This article highlights the potential hazards of exposure to UV and blue-violet light, and summarizes some of the commonly prescribed protective/preventative measures.

KEYWORDS
UV, blue-violet light hazard, eye damage, age-related macular degeneration, cataract, harmful light, electronic devices, climatic droplet keratopathy, population at risk, prevention, protection, risk factors, IOL.

Light is part of the electromagnetic spectrum, which ranges from radio waves to gamma rays. Visible light is not inherently different from the other parts of the electromagnetic spectrum with the exception that the human eye can detect visible waves. Light can be classified into different categories depending upon its wavelength, not all of which are visible to the human eye. The human eye can see light of wavelengths ranging from 380-780 nm (also known as the visible spectrum). Shorter wavelengths than 380 nm are classified as ultraviolet (UV) light while wavelengths longer than 780 nm are classified as infrared light.

1. UV radiations: definition and transmission
Ultraviolet light is categorized into UV-C (100-280nm, short wavelength), UV-B (280-315nm, medium wavelength) and UV-A (315-380nm, long wavelength). The sun is the single largest source of UV light. UV-C is effectively filtered out by the earth’s ozone layer and atmosphere. UV-A and UV-B penetrate the earth’s atmosphere to reach its surface. Some of the common factors affecting the transmission of UV radiation from the sun to the earth’s surface include:

1. Sun elevation: The more vertical the sun is in the sky, the more intense the UV radiation. UV radiation levels are therefore highest around noon and in summer. However, it is postulated that as our eyes are set deep in the orbital bone structure, the level of UV entering the eye at noon may be lower than at other times of the day.

2. Latitude: Regions that are closer to the equator receive higher UV radiation levels.

3. Cloud cover: UV-A and UV-B radiation can penetrate through light cloud cover. Different thicknesses of cloud
cover have variable effects on levels of transmission of UV radiation. 7

4. Altitude: At higher altitudes the atmosphere is thinner and absorbs less UV radiation, allowing more UV light to reach the earth. 8

5. Ozone: Ozone absorbs UV-C radiation. 9 The filtering mechanism is becoming less efficient as the layer is increasingly depleted.

6. Ground reflection: While grass, soil and water reflect less than 10% of UV radiation reaching the earth, fresh snow reflects as much as 80% while dry beach sand reflects around 15% and sea foam about 25%. 5

2. The beneficial and harmful components of blue light

The wavelength adjacent to UV-A is called blue light. Blue light (also known as high energy visible, HEV) has wavelengths of approximately 380–500 nm. Most blue light, together with other visible light, is transmitted through the cornea and the crystalline lens to reach the retina. Blue light is vital for performing a number of visual functions including perceiving objects of different color and contrast in our surroundings. Blue-turquoise light, whose wavelength lies close to the green light, helps regulate our circadian cycle, which in turn maintains and regulates non-visual functions such as memory, mood and hormonal balance. 10 Blue-violet wavelengths lie close to UV light and therefore have higher energy (being inversely proportional to the wavelength). An excess of blue-violet is considered to be hazardous to the human retina if exposed for a long period of time as it radiates more energy than blue-turquoise and other visible light.

3. Anterior segment lesions due to UV exposure

Ultraviolet light (mainly UV-B) is absorbed by the cornea and ocular adnexa (lids, conjunctiva). The chronic absorption of UV-B light by the cornea can lead to photo-keratitis.11 If the cornea is also exposed to sand and dust then climatic droplet keratopathy can occur, which is a degenerative process characterized by golden-brown translucent material in the anterior corneal stroma, Bowman’s layer, and sub-epithelium. Initially, deposits are found near the limbus, which then progress to large nodules in the central cornea, thereby blurring vision. Deposits may also infiltrate the epithelium and the conjunctiva causing painful eyes. 12

Absorption of UV light by the conjunctiva can cause conditions such as pterygium and pinguecula. 13 (Fig. 1 & 2)

In the eyelids, prolonged exposure to UV light is also a risk factor for cancers such as basal cell carcinomas, squamous cell carcinomas and melanomas. These usually occur in the lower eyelids which are exposed to most sunlight. 14 The longer wavelength UV light (UV-A) which lies adjacent to the blue end of the visible spectrum is transmitted through the cornea to reach the crystalline lens. The lens absorbs most of the UV-A. Hence, chronic exposure of the eye to UV-A can promote cataract formation. 15

Cataracts are typically treated by removing the crystalline natural lens and replacing it with an artificial intra-ocular lens (IOL). However, the artificially implanted lens is typically less efficient at filtering UV light compared to the natural lens, thereby making the retina vulnerable to the hazardous effects of UV light. This can lead to photo retinitis and also increase the rate of progression of macular degeneration. The damage is even more acute and severe
In 2008, researchers at the Paris Vision Institute split the visible light spectrum into multiple bands of 10 nm and examined the effect of several hours exposure of each band on porcine retinal pigment epithelial cells. Their data showed that specific bands of blue-violet light of wavelengths ranging from 415-455 nm were most harmful to these retinal cells.\textsuperscript{1,21}

It is also known that digital screens (TVs, computers, laptops, smart phones and tablets), fluorescent lights, welders’ flashes, and LED backlit screens emit blue light. It is possible therefore, that excessive exposure to these devices over a period of time may increase the risk of damage to the eyes. However it has also been suggested that, while LED backlit screens are rich in blue-violet light, they have low levels of irradiance so their main effect may be short-term (e.g., eyestrain) rather than permanent retinal damage.

Okuno and co-workers examined the blue light hazard of various light sources.\textsuperscript{22} They found that arc welding was among the most hazardous sources, with an exposure time of 0.6-40s being acutely hazardous to the retina. Viewing very bright sunlight and its reflection on the ocean or desert with unprotected eyes can acutely damage the macula due to blue light.

5. Populations at risk and preventative requirements

The human eye has inbuilt mechanisms to filter UV and harmful blue-violet light; the cornea, the crystalline lens, and macular pigments. However, experience shows that these mechanisms do not always provide adequate protection from the harmful effects of UV and blue-violet light. It has been reported that by the age of 65, the ability of the human eye to protect itself against UV and blue light is reduced by half.\textsuperscript{20} Various populations are at increased risk of being prone to ocular damage.

Outdoor workers who are exposed to sunlight for prolonged
periods of time are more prone to the harmful effects of UV and blue-violet light. These include various occupations such as construction workers, farmers and truck drivers. Engaging in activities such as arc welding, curing of paints/inks, working in environments that are brightly lit with fluorescent lights/cool indoor white light and also where blue light is used to disinfect equipment in hospitals and laboratories may lead to retinal damage due to the cumulative effect of blue-violet light. Furthermore, with the increased use of compact fluorescent bulbs in the home, exposure to blue light has risen even more significantly in recent years.

In addition, adult eyes are at increased risk simply because of the aging process and the reduced efficiency in filtering out blue light as melanin levels are reduced. Melanin is the natural substance that determines hair and skin color and is responsible for protecting skin and eyes from the damaging rays of sunlight. Given the combined effects of ever increasing amounts of blue light in our surroundings and daily activities, along with increasing life expectancy interest has grown amongst researchers to study the harmful effects of blue-violet light on the human eye. However more research is needed to establish precisely what kinds of preventive measures are required for people living in different geographical regions and for those involved with different types of jobs/activities.

Below, we list some of the commonly prescribed preventive measures:

Indoor activities:
1. Reduce blue light exposure by keeping digital devices out of the bedroom to protect the circadian rhythm.
2. Limit the use of electronic devices: take frequent breaks from their use to reduce visual fatigue and to minimize the cumulative effect of blue light reaching the retina. Studies suggest that 60% of people spend more than 6 hours a day in front of a digital device. 3
3. Use filters in spectacles that filter out harmful blue-violet and UV light.
4. Use halogen lights to reduce cool indoor white lighting.

Outdoor activities:
1. Limit extended sun exposure whenever possible.
2. Wear wide-brimmed hats while in the sun.
3. Consider a melanin pigmented polarized lens. Although it may cause changes in color perception, it cuts down outdoor blue-violet light exposure. To have unchanged color perception, however, one may use Transitions® lenses that can block up to 88% of blue-violet light.
4. For welders adequate protection is vital as the pupillary constriction in response to striking the arc is too slow to block the initial surge of radiation.

Conclusion
Blue light has both beneficial and harmful effects. Whereas blue-turquoise light regulates the circadian rhythm, blue-violet light cumulatively affects the retina and thus may have a link with macular degeneration. It is vital that the eye is adequately protected against harmful effects of UV and blue-violet light. Proper eyewear when working in the sun and avoiding excessive use of devices that are rich sources of blue-violet can help reduce hazardous effects of blue-violet light in the eye.

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It is vital that the eye is adequately protected against harmful effects of UV and blue-violet light.

UV and blue-violet light are potentially hazardous to the eye. Prolonged exposure to UV increases the risk of various forms of eye damage such as cataract, pterygium, pinguecula, climatic droplet keratopathy, and eyelid cancers. Several factors affect UV radiation: Sun elevation, altitude, cloud cover, latitude, ozone layer and ground reflection. The blue-violet light wavelengths from 415-455 nm are the most harmful to retinal pigment epithelial cells. Adult eyes and specific populations are at increased risk from blue-violet light hazards and of developing macular degeneration.

REFERENCES


The potential dangers of blue light on the human eye are a subject of increasing debate, notably since the widespread use of LED lamps. This review addresses what blue light is, demonstrates where it is found, and explains why it can be harmful and under what conditions, and concludes with some recommendations for reducing associated risks.

**BLUE LIGHT: WHAT ARE THE RISKS TO OUR EYES?**

Dr. Jean Leid

Jean Leid is an independent ophthalmologist, a specialist in color vision and author of numerous publications on the subject. He has been the director of the International Color Vision Society for many years and is currently a lecturer at the Paris 7 Faculty of Medicine. He was the Report Coordinator of the France Ophthalmologic Societies for dyschromatopsias in 2001.

Blue light is everywhere, originally mainly in sunlight. This is nothing new. What has changed is our way of life. In short, we have gone from darkness to light within a few decades. Consider the changes to our habitat, where living spaces are now facing south and have large windows, whereas our elders tended to protect themselves from the sun; then there is extensive exposure of our bodies to sunlight in Western countries where garments are lighter and leisure is geared toward the sun (sea, mountains, ski, etc.). But that’s not all. Two major technologies have emerged in recent years that have contributed to blue light over-exposure: LED lamps and the last generations of screens. At the same time, the elderly are now suffering from age-related macular degeneration (AMD) on a large scale, and the use of screens by all of us, especially the younger generations, is literally exploding. These changes are now giving rise to fears of potentially associated health dangers, and an increasing number of questions.

**What is blue light?**

Most of the time, blue light cannot be “seen” as such. It is just one of the different spectral components of any given light. This is true for both sunlight and for artificial lights.

The human eye is a highly selective receptor of electromagnetic waves, being sensitive to only a very small...
number of them. Its “spectrum” covers wavelengths from approximately 400 to 700 nanometers, allowing us to successively see the colors of the rainbow from the so-called “cold” hues of violet, blue, and blue-green from 400 to 500 nm, to “hot” colors: orange and red from 590 to 700 nm, through to intermediate colors: green and yellow from 500 to 590 nm. This spectrum corresponds to what we call visible light. Other animal species have receptors that are capable of perceiving other regions of the electromagnetic spectrum.

These lighting characteristics can sometimes be found coded on commercial artificial light sources, especially neon lights. Code 840, which is often found on basic fluorescent tubes, means that the light’s CRI will be greater than 80 and its color temperature will be 4000 K. However, manufacturers prefer to give names that are more evocative than numbers. This light will bear a name such as “neutral white”, “warm white”, “cool white” or “daylight”.

Where is blue light?
So from this it is obvious that a given light source can produce lights of different qualities, and that the colder the light, the more blue there will be in the spectrum.

In the past, when we were using good old incandescent bulbs (the famous “lightbulbs” known since Edison in 1879), we would talk about the amount of light as the power supplied by the bulb. The quality of light was not an issue, because this type of lamp only gave an orangey to yellowish light, barely clearer than 100W lamps. The CRI was really poor, and the color temperature was low. On the other hand the spectrum of these lights contained very little blue light (fig. 1). They are now something of the past, as various lobbies managed to have them phased out in Europe by 2012; this is a shame for the comfort and health of our eyes. What’s more, it is astonishing that these drastic decisions were based on the poor energy.

These terms of hot or cold light are related to a feature reflecting the general color sensation produced by a given light source: the color temperature. This is expressed in kelvin (K) (although kelvin degrees have not been used since 1967!), ranging from 2000 to 3000 K for a reddish light (an incandescent lamp on low power, for example) to values of 6000 to 7000 K giving the appearance of a bluish light like that of a summer sky at noon, via intermediate values in the order of 4000 to 5000 K, producing a yellowish appearance, such as that emitted by a halogen lamp (fig. 1).

In the field of lighting, another characteristic of light is also important, the color rendering index (CRI). This characteristic is of a different nature because it measures the ability of a light source to provide the eye with a rendering of colors as close to reality as possible, up to an upper limit value of 100.

**FIG. 1** The different color temperatures in K representing the color of the radiation of a black body (theoretical body heated at these temperatures) for an observer in daylight standard D65. (http://www.cie.co.at/index.php)
efficiency of incandescent lamps and the overall energy savings expected from the use of more modern lamps, without taking into account the carbon footprint of manufacturing, and especially recycling, of the latter. This is another subject, on which there is much to say.

As far as incandescent lamps go, the halogen variety are still available. They appeared much later (the last quarter of the twentieth century, they were invented in 1959 by Zubler and Mosby) and retain the advantage of a spectrum that is poor in blue light (fig. 3), but with a higher color temperature. Their CRI remains low because we are still far from the solar spectrum. European authorities have also decided to ban them and they should be phased out by 2018.

Fluorescent lamps have been around for some time (Germer 1926) in their long tube form known as “neon”. In fact, there has not been any neon in these lamps for a long time; their light production system results in a very different spectrum from the aforementioned since it is a line spectrum (fig. 4).

As we have seen, this spectrum can vary according to the desired light characteristics (fig. 5).

Modern development of this type of lamp (in the early 80s) is epitomized by “compact fluorescent” lamps said to be “low energy”. Their spectrum is similar, they are of the same type and their small size makes them practical. Fluorescent lamps containing little blue in their spectrum are now easy to find.

Finally, LED lamps have ruthlessly invaded the market in only the last few years (1990s), but with lightning speed. The leaders of the lighting industry estimate that over 90% of all global light sources will be based on solid-state and LED lighting products by 2020.
Manufacturers attribute these lamps with all kinds of qualities: long-life, consistency of light emitted, relative insensitivity to the number of ignitions and to shock, cold light and especially the significant energy savings due to their exceptional energy efficiency. Unfortunately, this is far from proven, starting with lifespan that is only theoretical and which depends heavily on the manufacturing quality and the lamp’s ability to cool; the light might be cold, but the lamp is not! However, most disturbing to ophthalmologists is these lamps’ double disadvantage of their significant emission of blue light (most LED lights today) and their tremendous luminance of about 1,000 times that of a conventional lamp, due to the extremely concentrated beam.

LED lamps are discharge lamps in solid phase using semiconductors so that they can only issue one peak light (i.e. only one “color”). So, white LEDs do not exist. For white light, one must either: combine multiple colored LEDs (three primary colors), but this is very expensive; add a phosphor to the outer surface of the diode (making a daylight white LED) (fig. 6), or deceive the user’s eye through blue LEDs with a very high luminance that produces a feeling of “white” light. This is currently the case for the majority of commercially available LEDs, especially for cheap lamps and flashlights. Their spectrum is devastating for the eye with a single peak that is more toxic the higher it is (fig. 7). Fortunately, warm white LED lamps are now available where the proportion of blue is much lower thanks to a technology that uses two phosphors. But this means costs are high and energy efficiency is much lower (fig. 8).

And the sun in all this? It remains by far the first producer of blue light. The solar spectrum depends heavily on the time of day, the observation latitude, the altitude, the season and the atmosphere (presence of clouds). The reference spectrum of daylight said to be natural is that of a sky observed to the North, in the Northern Hemisphere, at noon. This spectrum is fairly balanced which allows us to consider that this light is “white” in appearance (fig. 9).

It contains a significant portion of blue light that we need to be wary of.

According to standards (ASTM G173-03 and D65), blue light represents 24%-30% of daylight. When we know that the luminance of a sunny sky is at least 5,000 cd/m² and that of a computer screen 250 to 300 cd/m², it makes you think.

Blue light and screens
Apart from LED lamps, the increasingly protracted use of screens is also a major cause for concern. Sixty percent of the population spends more than six hours a day in front of a digital device (Study “Blue in light”). [1]

We have seen that the luminance of screens is small compared to that of sunlight. Nevertheless, not only do we use screens for hours a day, but we do not think of protecting ourselves like we do from sunlight. Televisions may be viewed at a safe distance, but that is not the case for computer screens, and even less so for tablets and mobile phones used especially by young people, sometimes for hours and hours a day.

The proportion of blue light emitted by screens basically depends on the technology used.

In conventional LCD screens, the panel is backlit by fluorescent tubes. The perception of blue light by the user is very small, and there is very little risk. In LCD-LED screens, LEDs either backlight the panel or are located on the sides to reduce the thickness of the screen. The fact that the light passes through the LCD panel greatly diminishes the risk.

However, OLED or AMOLED (Active Matrix Organic Light Emitting Diode) screens produce their own light, directly visible to the user. These are said to be emissive in contrast with LCD screens that are said to be transmissive. Very thin, they are increasingly used in smartphones and tablets. The blue light emitted is directly collected by the retina at a very short distance. The danger comes not from the total amount of light emitted (luminous flux) but from the twofold risk of closeness and duration of exposure. Watching movies on a smartphone with an AMOLED screen can therefore be very dangerous. Recently, there
have been reports of transient unilateral blindness in young women who had watched their smartphone in the dark for extended periods of time lying on their side. These are only the immediate risks. Our troubles are only just beginning. The simple, straightforward comparative view of an AMOLED smartphone screen and an LCD-LED screen shows anyone who wants to see that it is not the same category of brightness. The skill of the salesman is to make it appear an advantage where in fact there is only discomfort and danger. The same goes for desktop monitors. Even the CEO of the French Agency for Lighting has harsh words about such marketing trends: “the market has been corrupted by opportunistic manufacturers selling products of very poor quality”. [3]

What are the ocular risks of blue light?

It is clear that we all receiving more and more blue light. So is this famous “blue light” really harmful and should we be afraid?

First of all, why would blue be more dangerous than red or green?

The answer is a simple formula from 1900 by Max Planck: 

$ W = h \nu \lambda $  

showing that the energy of an oscillator (the concept of photon emerged in 1926 after the work of Einstein and Compton) is directly proportional to its frequency, the inverse of its length wave (h is Planck’s constant). The shorter the light’s wavelength, the more energy it carries. Blue is therefore on the front line for this.

It seems that the first study of the phototoxicity of blue light (on rat optical rods illuminated by fluorescent lights) dates from half a century ago (Noell 1966). [4]

It is however the important work of John Marshall that clarified the issue. In 1972, he started to show the toxicity of short wavelength light on pigeon cones. [5] It was followed by very important studies showing the mechanisms of destruction of photoreceptors by blue lights in vitro in AMD.

It has been shown that the photo-activation of the retinal all-trans by blue-violet light can cause oxidative stress in the outer segments of the photoreceptors. More specifically, it is A2E, the lipofuscin photosensitive component which can be triggered by the radiation of blue light of 440 nm, eventually resulting in the degeneration of the photoreceptor and of the retinal pigment epithelium cells.
It would nevertheless be unwise to completely banish blue light; even if its wavelengths are barely above 480 nm, there is a real benefit to receiving this light because it corresponds to the peak sensitivity of melanopsin ganglion cells that are directly involved in the synchronization of the circadian clock (study by Provencio in 1998, [6] and Brainard in 2001 [7]).

The line is very thin between the “good blue” that is beneficial for our circadian cycle whose effects on the general economy of human physiology are considerable, and the “bad blue” capable of destroying our precious visual receptors.

So we should limit the retinal risks of blue light, but preserve the essential function of the circadian clock. Blue light should therefore be avoided up to about 455 nm, but not filtered beyond 465 nm. This means that the room to maneuver is very small. (Fig. 10).

In 2010, ANSES, the French national agency for the safety of food, the environment and labor, published a key report under the direction of Professor Béhar-Cohen on the “health effects of lighting systems using LEDs”. [9] The report was followed by two French publications in 2011 [10] and 2013 [11] and cautions about the risks of LED lamps in direct relation with the spectral imbalance of these lamps, in favor of low wavelengths whose in vitro toxicity has been thoroughly demonstrated and with the very high brightness of these lamps, finding in particular that the photochemical risk was related to the cumulative dose of blue light to which the person was exposed.

The report made recommendations, including the need to restrict the release of LED lighting systems on “mainstream” markets, as well as to adapt standards and to enforce them. It also considered it necessary to inform the consumer (informative labeling of lighting systems).

To date, these recommendations are far from being implemented and it is not clear that they will be. There is no awareness yet of the risk of LEDs on public health and one can only wonder why and be concerned that the precautionary principle is not yet applied, while a few unfortunate events on the subject in recent decades should encourage reflection and prudence.

It is clear that there are not enough studies with conclusive in vitro findings on the macular toxicity of short wavelength light to transpose them in vivo. Few long-term studies are available to allow us to clearly establish a relationship between the prolonged exposure to blue light and ARMD (Beaver Dam Eye Study [12], Eureye Study [13]). It is, after all, only a question of means and methodology. Research teams need to agree to undertake long and difficult studies to take into account new lifestyle parameters which will change in years to come (screens, LED lamps). Otherwise the voice of caution will be silenced because economic stakes are too high.

Conclusion

We cannot seriously deny the potential ocular risks from overexposure to blue light.

In order to remain level-headed but lucid before this much debated issue, we must remember the main producers of this high-energy light capable of destroying macular cells in vitro. First there is the sun, then there are artificial lights with cold white LEDs and AMOLED screens.

**FIG. 10** The dilemma of blue light

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<td><img src="image-url" alt="Blue Light Diagram" /></td>
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**BLUE LIGHT**

<table>
<thead>
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<th>HELIOLOGY</th>
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<td><img src="image-url" alt="Blue Light Diagram" /></td>
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**HARMFUL**

Blue-violet (415 - 455 nm)

**BENEFICIAL**

Blue-turquoise (465 - 495 nm)
Permanent eye protection against solar radiation using good quality tinted glasses is above all necessary to guard against all harmful effects. Precautions against screens that have a high emission of blue light: avoiding close, long-term exposure and using protection against toxic blue light as far as possible. These processes already exist and will only improve and become increasingly widespread. It is more difficult to protect oneself against bad light LED lamps that are becoming the norm, except at least by using them sparingly at home. The awareness of public authorities themselves could be effective in this regard.

It is important not to forget that it is the cumulative effect over time that is dangerous and must be fought. Precautions should be taken early and should be long-term. Particularly close attention must be paid to children whose ocular media allow these high-energy lights pass in large volumes, and to young people, prone to long exposure to the sun and screens.

Finally, emphasis should be placed on the need for increased protection for older groups at risk: people who have had cataract surgery, especially if they have not received yellow implants and those affected by age-related maculopathies.

Let us not deprive ourselves of light that is so basic but let us, as we do for so many natural elements, better understand it in order to better reap its benefits without its risks.

REFERENCES
9. ANSES. Effets sanitaires des systèmes d’éclairage utilisant des diodes électroluminescentes (LED) ; 2010 (Disponible sur le site www.anses.fr).

Blue light is not visible as such. It is a spectral component of visible light corresponding to wavelengths of approximately 400 to 500 nm.

The main producers of high-energy blue light capable of destroying macular cells in vitro are, primarily, the sun, and secondly, artificial lights with cold white LEDs, and AMOLED screens.

The recent emergence of LED lamps and the latest generations of screens contribute to over-exposure to blue light.

Blue light of a wavelength of 440 nm can induce oxidative stress in the outer segments of photoreceptors, which can ultimately lead to their degeneration as well as of the cells of the pigment epithelium.

Increased caution should be taken especially for children, people undergoing cataract surgery, and individuals with age-related maculopathies.

Not all blue light should be avoided - wavelengths of just over 480 nm are beneficial as they are directly involved in the synchronization of the circadian clock.
Phototoxicity is a current vision health concern and there is evidence that UV and blue-violet light may cause adverse effects to the eye. Blue-violet light sources include the sun, but also the widespread light-emitting diode (LED) technologies, resulting in around-the-clock exposure. Chronic exposure to blue-violet light, among other factors, may contribute to retinal diseases such as age-related macular degeneration (AMD), or speed-up AMD progression after cataract surgery, because of increased transmission of short-wavelength light. This link has not been definitively proven, due to a lack of clinical trials. Nevertheless, it has been shown that photoprotective measures such as blue-blocking IOLs or spectacles with blue-violet filtering lenses have no detrimental effects, making them a sensible choice in high-risk patients or patients with a longer pseudophakic life.

The risks of ocular light exposure are a current concern and health providers are frequently faced with questions on this topic. With recent advances in lighting sources and technology, modern man’s environment includes not only exposure to light in the form of solar radiation but also in the form of domestic lighting.1,2 Sunlight is the main source of ultraviolet (UV) radiation which is composed of electromagnetic radiation with wavelengths from 100 nm to 380 nm, while visible light ranges from 380 to 780 nm.3 Long-term exposure to UV radiation is known to cause anterior segment diseases such as keratitis, pterygium, cataracts and melanoma.4,5 Also, within the visible light spectrum, blue light exposure (380-500 nm) has been linked to photoreceptor and retinal pigment epithelium (RPE) toxicity1,4-8 and can induce a photochemical reaction culminating in cell apoptosis.8 This phenomenon is referred to as the “blue light hazard” and has a peak at 440 nm.9 Recent in vitro studies have demonstrated the phototoxic action spectrum in a RPE model of age-related macular degeneration as ranging from 415-445 nm with a peak at 435 nm.10 As far as artificial light is concerned, LEDs have evolved significantly in the last two decades and not only have replaced traditional lamps in the domestic setting, but have also become the primary light source in tablets, TVs, computers and smartphones. The LEDs emit in the blue light spectrum (380–500 nm) and therefore increase the exposure of the human eye to harmful radiation.1,2,7,11

**KEYWORDS**
Light exposure, blue-violet light, light-emitting diodes (LED), cataract surgery, retina, photoprotection, blue-blocking intraocular lenses (IOL), age-related macular degeneration (AMD)
Nevertheless, the eye has defences against phototoxicity. The cornea and crystalline lens naturally protect the retina and the posterior ocular structures against different sources of radiation, with less than 1% of UV light reaching the retina\cite{4,8,12}, while the macular luteal pigment attenuates the blue light that reaches the retina, mainly at 440 nm\cite{1} (Figure 1 & 2). Also with age, the yellowing of the crystalline lens leads to increased short-wavelength light blockage, partially protecting the aged retina and RPE. This protective effect disappears with cataract surgery, which is now one of the most common ocular procedures worldwide\cite{8,9,12}.

This article discusses the potential risks of light exposure on the retina after cataract surgery and photoprotective solutions.

1. Cataract surgery

Cataract surgery technology has been continuously evolving in order to allow safer and more predictable outcomes. Advances are not only at the level of preoperative and intra-operative testing, but also include femtosecond laser-assisted cataract surgery (FLACS) and improvements in intraocular lenses (IOLs)\cite{13}.

- IOLs

In cataract surgery, the yellowed aged lens (Figure 3) is removed and replaced by an IOL (Figure 4). IOLs have been modified over time to try to compensate for the properties of the removed crystalline lens. The first IOLs did not block UV light, however this obstacle was overcome by the 80s following concerns over retinal toxicity. Since then, transparent UV-blocking IOLs have been widely...
accepted.\textsuperscript{12} Later, further knowledge that blue light exposure could also contribute to retinal damage, led to the introduction of blue-blocking IOLs, which are yellow-tinted, and block both UV light and blue-violet wavelengths (380-500 nm), mimicking healthy crystalline lenses.\textsuperscript{12,14} The protective effect of these lenses on the retina has been well demonstrated by both animal and experimental studies.\textsuperscript{14-19}

However, contrary to simple UV-blocking IOLs, these lenses have been the subject of debate because of their photoprotective benefit in humans and their impact on the circadian rhythm, scotopic and color vision.\textsuperscript{12-14} Enthusiastic users claim there is decreased risk of age-related macular degeneration (AMD), reduced glare disability and improved photostress, without compromising contrast perception, color vision, scotopic vision, contrast sensitivity and circadian rhythm.\textsuperscript{12-14,20,21} Opponents report there is no evidence of increased photoprotection against AMD and negative impact on sensory and physiologic factors.\textsuperscript{12-14,20,21} While it has not yet been definitively proven that blue-blocking IOLs are photoprotective in humans,\textsuperscript{12,21,22} the majority of studies involving them have not shown changes in scotopic vision, color and contrast vision or the circadian rhythm, making it safe to opt for the blue-blocking IOLs.\textsuperscript{12,14,20-25}

2. Evidence of retinal phototoxicity and post-cataract risks for the retina

Replacement of the natural lens with an IOL implant increases retinal exposure to visible light and UV.

Many experimental and animal models have demonstrated retinal susceptibility to light exposure and blue light hazard.\textsuperscript{8,12,15,18} Light has a detrimental effect on photoreceptors and RPE, inducing cell damage.\textsuperscript{8} Recent studies have also shown retinal lesions following exposure to ubiquitous LEDs by inducing oxidative stress\textsuperscript{26} and various countries, such as the UK, have created dedicated groups to determine the potential health risks of these new light sources.\textsuperscript{1}

Regarding the association between light exposure and retinal disease in humans, the main attention has been on AMD, but some studies have also focused on pre-existing retinopathy and inherited retinal diseases.\textsuperscript{27}

- **Age-related macular degeneration (AMD)**

AMD is the leading cause of irreversible blindness in the elderly in developed countries.\textsuperscript{28,30} It has been established that it is a complex multifactorial disease associated with demographic, genetic, and environmental risk factors.\textsuperscript{28,30} AMD has two forms: dry (nonvascular or atrophic) (Figure 5) and wet (neovascular or exudative) (Figure 6).\textsuperscript{29,31}

Recent studies estimated that AMD will affect about 196 million people in 2020 and 288 million in 2040,\textsuperscript{32} emphasizing the need for improved treatment and preventive measures.\textsuperscript{12}

Light exposure has been implicated in macular toxicity and as an environmental risk factor for the development of AMD, in both experimental and animal studies. Phototoxicity induces oxidative stress and photoreceptor apoptosis which damages the ageing retina.\textsuperscript{8,10,12,26,28,29,33}
This injury is cumulative and appears to be mediated by the lipofuscin chromophore A2E, therefore increasing with the amount of lipofuscin present.\textsuperscript{10,15}  

Nevertheless, the evidence in humans supporting the hypothesis that environmental light exposure is associated with AMD progression is controversial.\textsuperscript{12,22} While some studies support the relationship,\textsuperscript{5,9,34-36} the Beaver Dam Eye Study found few significant relationships\textsuperscript{37-39} and other important epidemiology studies found no association.\textsuperscript{40-44}  

Regarding the post-cataract surgery risk of AMD progression, again the literature describes conflicting results.\textsuperscript{45-47} The major epidemiology studies report prevalence and have different results when considering early or advanced disease or dry versus wet AMD.\textsuperscript{50} A combined analysis from the Beaver Dam and the Blue Mountains Study, two large epidemiology studies, showed that the prevalence of advanced AMD was higher in pseudophakic versus phakic eyes (6.7\% versus 0.7\%, respectively) and also supported the hypothesis that cataract surgery may be associated with late AMD.\textsuperscript{51} Similarly at 10 years follow-up in The Blue Mountains Study, patients who underwent cataract surgery showed an increased long-term risk of developing late AMD.\textsuperscript{52} In contrast, the Rotterdam Eye Study found an association with early AMD\textsuperscript{53} and some studies,\textsuperscript{12,54-57} including the Age-Related Eye Disease Study (AREDS)\textsuperscript{58,59} and the Visual Impairment Project,\textsuperscript{60} found no relationship. A Cochrane review\textsuperscript{61} and a meta-analysis\textsuperscript{62} also found no conclusive evidence of association.\textsuperscript{50}  

It should also be taken into consideration that some cases of AMD might not have been recognized pre-operatively because of significant lens opacity or that the primarily cause for visual loss was AMD and that these pathologies frequently coexist.\textsuperscript{54,62}  

- **Inherited retinal diseases**  

Light deprivation has in the past been considered a possible treatment for some patients with inherited retinal diseases, although no therapeutic benefit has been demonstrated. As current knowledge in understanding the genes involved in these diseases increases, there have been suggestions of the value of light deprivation in selected cases.\textsuperscript{27}  

**Autosomal dominant retinitis pigmentosa (ADRP)**  

Rhodopsin mutations are a frequent cause of ADRP with several mutations, including P23H, causing a distinctive phenotype with regional variation of retinal damage - classified by Cideciyan et al as class B1.\textsuperscript{63} In line with this animal models of class B1 ADRP have demonstrated modification of degeneration by light, which was also supported by reports of familial cases with the P23H mutation.\textsuperscript{64} The hypothesis is that light increases retinal degeneration by photoreceptor toxicity or interaction with the mutated rhodopsin.\textsuperscript{27,65}  

**Oguchi Disease**  

Oguchi disease is a rare autosomal recessive disorder caused by mutation in either rhodopsin kinase (RK) or arrestin. It is characterized by congenital stationary night
blindness and the Mizuo-Nakamura phenomenon, in which the retina exhibits a yellow-gray discoloration in the presence of light that disappears with dark-adaptation and reappears with new light exposure. Studies with animal models for Ogushi disease suggested that light exposure could be an important modifier, at least in patients with the arrestin mutation.27

Stargardt disease
Stargardt disease is an autosomal recessive disease caused by mutations in the ABCA4 gene (Figure 7). Animal models lacking the ABCA4 gene exhibit visual cycle abnormalities, which include high levels of A2E, which has been demonstrated to be retinotoxic.10,15,27 As A2E levels are modulated by light and rhodopsin activation, light restriction may have a role in this context.

In clinical practice, it may therefore be prudent to minimize retinal exposure to light during examinations, imaging and ocular surgery27 and to consider the use of ophthalmic lenses that filter blue-violet light. It should also be taken into consideration that some inherited retinal diseases are complicated by early cataracts so photoprotective measures may also be recommended after cataract surgery.66

Conclusion
In conclusion, phototoxicity is a current vision health concern and it has been demonstrated that UV and blue-violet light have deleterious effects on the eye. In the past, the exposure was limited to daily hours, but with artificial lighting and current technologies, exposure is around-the-clock, potentially putting the eye at a higher risk. Currently, there is still lack of consensus between the relationship of light exposure and retinal diseases, such as AMD, as well as in terms of progression after cataract surgery caused by increased transmission of short-wavelength light. However it has been well demonstrated that the use of photoprotective measures such as blue-blocking IOLs have no detrimental effects. Therefore, despite the ongoing debate, it can be considered reasonable to use IOLs or spectacles with lenses that filter blue-violet light, if not in all patients, at least in those at the highest risk and in younger patients with a longer pseudophakic life. Further controlled prospective studies are needed. •

KEY TAKEAWAYS
- UV and blue-violet light can have deleterious effects on the eye
- Blue-violet light sources include sunlight and light-emitting diodes (LEDs)
- With artificial lighting exposure is around-the-clock, increasing the risk of phototoxicity
- Light exposure may be associated with retinal diseases and age-related macular degeneration (AMD)
- Cataract surgery may be associated with AMD progression, although the literature is conflicting
- Photoprotective measures, such as blue-blocking IOLs or spectacles with clear ophthalmic lenses that filter UV and blue-violet light, do not have detrimental effects
- Therefore, the use of lenses with appropriate filters is reasonable despite the lack of clinical trial evidence, especially in advanced AMD cases or younger patients with a longer pseudophakic life
REFERENCES
The penetration of optical radiation deep within the eye is a paradox as light is an essential component for vision but it may also be a biohazard. Short wavelength blue-violet light is potentially harmful whilst longer wavelength blue-turquoise is essential for healthy living. Prof. John Marshall explains in this article the pathogenic power of light but also its fundamental requirement to circadian rhythms. In the human eye evolutionary development both mechanisms have been integrated to facilitate separation of health and hazard.

**Light: vision and biohazard**

The eye is the only organ in the body that has evolved to allow radiation to penetrate deep within it. In this process however the various ocular media, cornea, aqueous, lens and vitreous act as progressive wavelength selective filters such that ultraviolet B (280-315 nm)\(^1\) radiation is absorbed almost exclusively in the cornea whilst ultraviolet A (315-400 nm)\(^1\) may be attenuated by the cornea with almost all of the remaining radiation of this wavelength being absorbed in the lens\(^2\,4\) and only a very small amount passed to the retina (Fig 1). However optical radiation between 400 and 1400 nm made up of visible radiation or light (400-800 nm)\(^1\) and infrared A (800-1400 nm)\(^1\) not only passes through the various optical media to fall upon the retina but at the same time undergoes a concentration in irradiance of up to a hundred times between the cornea and the retina as a result of the refractive power of the cornea and to a lesser extent of the lens. It is this refractive property that concentrates the incident energy and converts for example the rays of the summer sun from the pleasantly warming sensation on the skin to a potential hazard to the eye if the sun is viewed directly. This penetration of optical radiation is the first paradox as radiation is a biohazard\(^5\,14\) but light is an essential component in the process we know as vision.

**Renewal processes of cells**

In all biological systems cells under stress are normally replaced on a periodic basis in order to combat environmental attrition. For example the cells of the skin are replaced by a never-ending cycle whereby new cells progressively move towards the surface of the body and are then shed, usually within five days of creation. This
process ensures that our skin is able to cope with the problems of both friction induced by touching things and of the effects of optical radiation. A similar process is found in the lining of the gut whereby renewal of cells overcomes the problem of focal trauma caused by the passage of food and the toxic chemical environment required for digestion. These renewal processes make the concept of ageing in biological entities an extremely difficult parameter to define as the cells of those parts of our body which are constantly dividing may be a few days or weeks old whilst those in other parts of the body which don’t divide may have been created shortly after conception or some time during the developmental processes in the womb.

Toxic blend of oxygen and light

Here then is a second paradox in that the retina may be considered as part of the brain as it develops early from the neural tube and as the cells develop they ceased to have the capacity to renew themselves. The photoreceptor cells, rods and cones, have a significant problem in that they are not only exposed to optical radiation throughout life but they have to transduce it in order to initiate vision. This process requires huge amounts of energy and as a consequence the cells have an extraordinary high oxygen demand being equipped with the highest concentration of mitochondria in any cells of the body. Thus this non-dividing cell system is simultaneously exposed to an environment with extremely high levels of both oxygen and radiation. This combination is known to be extremely toxic to biological systems because of the induced generation of superoxide and other free radicals.

The beauty of inverted retina

The next paradox is generated by the need to provide metabolic sustenance to the rods and cones in order to sustain their huge metabolic demand. This has been solved in all vertebrates by the evolution of the so-called inverted retina. At first sight it would seem strange that the cells that do the transducing of light are on the side of the retina furthest away from the incoming radiation. This apparent anomaly becomes understandable when the requirements for a blood supply to the light-sensitive cells is examined. If the photoreceptors pointed towards the incoming light then they would either have to have a large blood supply between them and the incoming light, thereby limiting transmission and resolution, or a large blood supply between the photoreceptors and the next layer of neurones thereby limiting neuronal processing. The structure of the inverted retina avoids these issues by allowing the photoreceptor cells to derive their blood supply from the innermost aspects of the choroid via an acellular membrane, Bruch’s membrane and the pigment epithelium. This anatomical arrangement also enables the retinal pigment epithelium to act as an anti-halation screen absorbing much of the unused radiation that has passed through the photoreceptor cells and thereby prevents scatter and degradation of the retinal image.

Auto-regenerative capacity of photoreceptors

The juxtapositioning of the photoreceptor cells and the retinal pigment epithelium also allows for a solution of the fourth paradox, that is how can non-dividing cells like the photoreceptors survive over a human lifetime in an environment of high oxygen and with the function of absorbing and transducer optical radiation. The solution is unique amongst neurones and that is throughout life
The light-sensitive portions of the photoreceptor cells are constantly renewed. This is an exquisite process which differs between rods and cones. Animal studies in rods demonstrated that 3 to 5 of the light sensitive membrane structures or discs are being manufactured daily on the innermost aspect of the light-sensitive outer segment and as new discs are added older discs are progressively displaced towards the retinal pigment epithelium. Typically a rod outer segment contains about 1000 discs and thus in theory the whole system is replaced in approximately two weeks. When the older discs reach the surface of the retinal pigment epithelium they are phagocytosed in a process that seems to be initiated by the onset of light on first awakening in the morning. Thus the spent products from our night-time photoreceptors, the rods, are phagocytosed first in the morning and then undergo “digestion” during the day by the action of lysosomes. By contrast it would appear that our daytime photoreceptor cells, the cones, are phagocytosed four hours or so into the sleep period and undergo degenerative processes during the night.

Biomarkers of ageing
This process of daily shedding of spent material from the photoreceptor cells presents a huge biomass to the retinal pigment epithelium and not surprisingly the system becomes compromised as a function of age. In most retinai, by the late 20s, persistent partially degraded debris becomes present within the retinal pigment epithelium and is seen as drusen. Drusen are also visible clinically and are identified as high-risk factors for AMD. As a result of further ageing processes debris begins to pass from the retinal pigment epithelium into the underlying Bruch’s membrane and may in some cases be seen in the six or seventh decade as focal excrescences termed drusen. Drusen are also visible clinically and are identified as high-risk factors for AMD. By contrast from the red region up through to the blue region of the spectrum individual photons have enough energy that by themselves they can induce photochemical changes in absorbing molecules. By contrast from the red region up through the infrared individual photons no longer have sufficient energy to act by themselves and damage mechanisms induced by radiation in these wavebands come about by multiple photons causing vibrational modes in absorbing tissues and damage resulting from
thermal processes. Thus in considering the potential hazards of any given light source, attention must be given to the spectral emissions and spectral radiances with blue rich sources being potentially much more damaging than those predominantly red or infrared.42, 45 This has interesting implications for man’s artificial light environment.

The dark side of blue-violet light
Interest in the potential damaging effects of light has extended over hundreds of years with claims that Galileo damaged his retina by viewing the sun through his telescope. This mythology is not sustained by detailed studies however many have damaged their vision by viewing the sun and systematic investigations began after the first explosion of the atomic bomb because of concerns about the associated flash. The real impetus however came in the early 60s subsequent to the development of the laser in 1960. Extensive military budgets were deployed to ensure that the potential for lasers to damage the retina were fully understood and ocular safety mechanisms were explored. Numerous studies at this time demonstrated that with short intense exposures lasers emitting blue light were a greater potential hazard than those in other regions of the optical spectrum.50-52 The peak of the “bluelight hazard” in a normal eye with a natural lens present was shown to be around 440 nm, although this peak moved into the ultraviolet in individuals that had undergone cataract surgery with the lens being removed. Recent research has confirmed the peak of blue light hazard at 435 nm, with an action spectrum from 415 to 455 nm. The blue-violet light hazard is treated as a special case worldwide in all codes of practice designed to protect people against lasers. Further work showed that with low-level irradiance but over very long periods, hours, days, months also resulted in retinal damage again highlighting that blue-violet light was more hazardous than other wavelengths.19, 54, 55 We now know that there are two mechanisms of light damage with slightly different absorption or action spectra but both peaking in the blue. For relatively low intensity and very long exposures we see what is described as type I damage which appears to result from absorption within the light-sensitive cells and short wavelength or blue cones seem to be the most sensitive.52, 56, 57 By contrast for relatively high intensity shorter exposures we recognize type II damage whereby the primary damage seems to occur in the retinal pigment epithelium and is thought to be associated with absorption by lipofuscin (Fig 2, 3).
FIG. 2] Two mechanisms of light damage: type I (chronic), type II (acute).

FIG. 3] Spectral dependence of phototoxicity with type I damage (chronic), type II damage (acute).
It is of interest that within the eye there are two naturally occurring systems which attenuate transmission of blue light. In the natural lens progressive yellowing occurs with age which serves to limit the passage of light towards the neural retina. At the centre of our vision, the macula there is a second pigment, the luteal pigment which is also yellow with an absorption peak at about 445 nm.\textsuperscript{59,60} Further at the centre of this region, the fovea, responsible for our highest acuity vision there are no short wavelength, or blue responding, photoreceptors, giving rise to the often forgotten phenomenon present in all of us, foveal tritanopia.\textsuperscript{61} These findings on the potentially damaging effects of ultraviolet and blue radiation led to the companies that manufacture intraocular lenses fitted subsequent to cataract surgery first introducing ultraviolet blocking systems in all intraocular lenses. This occurred without any significant clinical trials and in today’s parlance in the absence of “evidence-based medicine”. Nevertheless few if any cataract surgeons would now place an intraocular lens into a human eye that did not have UV filtration. It is of interest that over the past 15 years many companies have introduced intraocular lenses with blue attenuating filters. These lenses have an attenuation factor similar to that of a natural lens in its late 30s or early 40s. This has been a little more controversial because it arrived in the era of “evidence-based medicine”. The apparent unimportance of blue light for vision has recently been challenged in the greater forum of blue light for overall health. In a series of studies it has been demonstrated that blue light in the region of 470 nm (blue-turquoise light) is a fundamental requirement to initiate aspects of circadian rhythm.\textsuperscript{62-64} These studies have further demonstrated that a small percentage of retinal ganglion cells contain a pigment, melanopsin, whose absorption of blue light triggers a mechanism via the brain which regulates melatonin levels in the blood. When the retina is exposed to light with a blue component the absorption within melanopsin initiates a process whereby melatonin production is suppressed and the individual exposed “wakes up”. By contrast switching off absorption at night up regulates melatonin production and the individual goes to sleep (Fig 4). This process underlies the condition known as seasonally adjusted disorder (SAD) therefore it is obvious that longer wavelength blue-turquoise light around 470 nm is essential for well-being. Thus we have an apparent paradox whereby short wavelength blue-violet light at 441 nm is potentially harmful whilst longer wavelength blue-turquoise at 470 nm is essential for healthy living. It is of interest that the ganglion cells responsible for absorbing 470 nm are anatomically arranged such that they occur prior to the components of the retina that absorb the harmful 441 nm as light is transmitted from the cornea to the retina. Thus in our evolutionary development both mechanisms have been integrated to facilitate separation of health and hazard.

**The beneficial blue-turquoise light**

The apparent unimportance of blue light for vision has recently been challenged in the greater forum of blue light for overall health. In a series of studies it has been demonstrated that blue light in the region of 470 nm (blue-turquoise light) is a fundamental requirement to initiate aspects of circadian rhythm.\textsuperscript{62-64} These studies have further demonstrated that a small percentage of retinal ganglion cells contain a pigment, melanopsin, whose absorption of blue light triggers a mechanism via the brain which regulates melatonin levels in the blood. When the retina is exposed to light with a blue component the absorption within melanopsin initiates a process whereby melatonin production is suppressed and the individual exposed “wakes up”. By contrast switching off absorption at night up regulates melatonin production and the individual goes to sleep (Fig 4). This process underlies the condition known as seasonally adjusted disorder (SAD) therefore it is obvious that longer wavelength blue-turquoise light around 470 nm is essential for well-being. Thus we have an apparent paradox whereby short wavelength blue-violet light at 441 nm is potentially harmful whilst longer wavelength blue-turquoise at 470 nm is essential for healthy living. It is of interest that the ganglion cells responsible for absorbing 470 nm are anatomically arranged such that they occur prior to the components of the retina that absorb the harmful 441 nm as light is transmitted from the cornea to the retina. Thus in our evolutionary development both mechanisms have been integrated to facilitate separation of health and hazard.
Conclusion

Modern sources of artificial light have revolutionised our light environment with the potential to flood our individual places of work or homes at levels of illumination far beyond that experienced by our ancestors. Almost daylight levels of illumination can now be achieved at any time of the day or night by flicking a switch and governmental trends towards the use of low-energy lighting has seen the development of many blue rich sources, in particular LEDs. This takes modern man out of the evolutionary boundaries determined by solar radiation into a new era. It will be interesting to monitor the effects of our new environmental boundaries on the health of the outer retina further complicated by our increased life expectancy and the increasing prevalence of AMD. We have learnt from the dermatologists that light contributes to photoaging with UV and short wavelength visible playing a role. Given the increase in irradiance for any given exposure between the eye and the skin it seems sensible to limit our exposure to short wavelength radiation whenever possible. Most individuals use UV and light protection in high light environments by wearing so-called sunglasses. Depending upon the absorption spectra these can be very useful ocular protectors, if they attenuate ultraviolet and short wavelength blue-violet but they can also be less than useful if they transmit these wavelengths yet by reducing overall brightness cause the pupil to open and individuals to stay in the sun for longer. The recent introduction of UVA blocking systems and attenuation of short wavelength blue-violet for everyday wear clear lenses seems a sensible development in the face of our ever-changing light environment.

Key Takeaways

• The penetration of optical radiation deep within the eye is a paradox as light is an essential component for vision but it is also a biohazard.

• The inverted retina is designed to avoid scatter and degradation of the retinal image and to ensure an efficient neuronal processing.

• The light-sensitive portions of the photoreceptor cells are constantly renewed to survive, over a human lifetime, in an extremely toxic environment due to oxygen and light radiation.

• With ageing, the accumulation of lipofuscin in Bruch’s membrane may form drusen identified as high-risk factors for AMD.

• LEDs have high spectral emissions in the blue and at levels that may require attention over cumulative exposures during a human lifetime.

• Retinal phototoxicity has been demonstrated by several studies for high energy wavelengths, blue-violet light, ranging up to 455 nm.

• Blue-turquoise light, ranging from 465 to 495 nm, is a fundamental requirement for circadian rhythm and thus essential to maintain good health and well-being.

• UV and blue-violet light are both responsible for skin and ocular photoageing, therefore it is of great importance to limit our exposure to these radiations whenever possible.
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OCULAR LIGHT TOXICITY
AND THE REQUIREMENT FOR PROTECTION

POSITION PAPER

BOARD OF EXPERTS,
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OCULAR LIGHT TOXICITY AND THE REQUIREMENT FOR PROTECTION

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KEYWORDS:
UV, blue light, biohazard, phototoxicity, blue-violet light, blue-turquoise light, AMD, cataract, retina, ophthalmic lenses, sunlight, LED, blue-emitting screens, antioxidant supplements, vision health.
INTRODUCTION

In November of 2016, a panel of eight international eye health experts with a diverse range of professional and geographic backgrounds (North America and four European countries), met in Paris to discuss blue light toxicity. The panel captured a wide range of domains in eye health care, including academic and applied research in both the preclinical and clinical settings, as well as patient management in surgery and private practice. The meeting was driven by an increasingly pressing need to deepen knowledge among our eye health professionals on the biohazards of light exposure – and specifically on the dangers of and need for protection from blue light. The aim of the meeting was to share personal experience in this field, identify areas to focus on, and explore solutions for managing potential blue light toxicity. The discussion was held in the critical context brought to the table by Prof. Dot who opened the session with “the duality of blue light [...] raises the question of the need for selective protection – an issue which is currently not well known by ophthalmologists”.

Acknowledgments

The round table discussion was facilitated by Essilor International, who would like to thank all Experts for their active participation in the stimulating and fruitful discussion on this fundamental aspect of future eye health care, as well as their reactivity, frankness, and enthusiasm both during the meeting and the follow-up in the preparation of this position paper.
The electromagnetic spectrum covers a continuum of electromagnetic waves from radio waves through to gamma-rays, with increasing photon energy as wavelength decreases [Figure 1]. Within this range, UV radiation covers 100-380 nm and visible radiation covers 380-780 nm. High energy visible (HEV) light (380-500 nm), commonly known as blue light, accounts for 25 to 30% of the sunlight within the visible range. Blue light is located at the beginning of the visible spectrum and includes harmful blue-violet radiations (415-455 nm) as well as beneficial blue-turquoise radiations (465-495 nm), involved in normal metabolic functioning in humans (circadian rhythms and effective endocrine activity).

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**Figure 1:** Visible light (380-780 nm) in the electromagnetic spectrum. HEV, high energy visible; LEV, low energy visible.
BLUE LIGHT: OUR CHANGING ENVIRONMENT

While the sun is the major source of blue light, it is also found in increasing proportions in indoor lighting.

“Our exposure to indoor blue light is unprecedented in human history”, “we don’t know whether it’s bad or it’s good, but we need to be aware of it.” These statements by Dr. Tolentino reflect growing concern over the unknown nature of the long-term effects of blue light exposure on the retina. Driving these concerns is the fact that what we have on our hands today is a new and rapidly-evolving scenario; a large proportion of the first-world society (and to some extent developing countries) is experiencing dramatically increasing exposure to artificial blue light from both cold-white light emitting diodes (LEDs) and fluorescent light sources. The widespread success of these forms of lighting is a result of their enhanced performance compared to incandescent bulbs. Figure 2 highlights the changing blue light emission spectrum with the shift from incandescent lighting towards cold-white LEDs.

Figure 2: Emission spectrum of various light sources including cold-white LED (from Smick et al., 2013 ²).
Added to this changing exposure profile is a shift in the population’s habits with the widespread availability of various digital technologies, reflected in a move towards screen exposure at both increasingly younger ages and for increasingly longer time periods, both of which are affecting an increasing proportion of the world’s population. In the space of a single generation, we have gone from screen exposure being limited to watching a couple of television programs, to hours of professional, recreational and educational time spent staring at a screen. While data on the impact of LED blue light exposure are currently sparse, confirmation of the trend towards different and continually changing digital habits is emerging in the literature. Two recent surveys carried out by the US Vision Council and IPSOS evaluating more than 13 000 participants from Brazil, China, France, and the US, support the hypothesis that any issues arising from blue light effects will concern a large proportion of the population. One of the surveys (in over 4000 individuals) reported that more than 90% of individuals questioned use a computer or watch TV on a daily basis, while 70% of participants use a smartphone, almost all of whom use it daily. Furthermore computer use was intensive with approximately 60% of users spending more than 4 to 5 hours a day on their device. The US survey (in nearly 10 000 individuals) reported that almost 30% of participants spend over 9 hours a day. Reports also attest to a rapid change in habits towards increasing exposure, with 89% of the population spending more time on digital devices than 2 years ago and 65% looking at digital devices at a closer distance compared to 2 years ago.

Beyond these changing behavioral habits, long-term exposure is increasing as exposure to LED-backlit screens is starting from an earlier age. Combine this with the fact that not only have the older generations taken enthusiastically to the use of electronic devices, but in addition life expectancy is increasing with a gain of 1 year every 5 years, and it is evident that given these cumulative factors, our blue light exposure is expanding on all fronts.

**Pedagogy around blue light terminology**

An important issue was raised by the panel, highlighting the need for care in the choice of blue light terminology to ensure successful pedagogy. As highlighted by Prof. Dot, “the blue light range is dual, the beginning is harmful and the end is beneficial for physiological functioning”. For the wider public, the term “blue light” is increasingly associated with well-being and other health benefits, such as the use of blue light therapy to put manage depression, seasonal affective disorder, skin problems etc. Prof. Wolffsohn pointed out that, passing the message that ‘blue light’ is a source of harm may be doing the ‘push for protection’ a disservice as it may well lead to controversy in the eyes of the public “as some parties are telling us it’s good and some are saying it’s bad”. A terminology consensus was reached for the public education arena with the choice of ‘harmful blue-violet light’ versus ‘beneficial blue-turquoise’.
CURRENT CHALLENGES IN THE FACE OF INCREASING BLUE LIGHT EXPOSURE

What we don’t know about blue light exposure

Knowledge of the challenges we are facing in terms of the consequences of potential harm from blue light exposure is alarmingly inadequate. The potential problems associated with the current changing blue light exposure profile are fueled by the gap in our knowledge in several areas of this domain. As a very recent concern, the developed world is effectively a ‘living experiment’ as to the effects of blue light exposure. To assess the level of risk from blue-violet light exposure – and protect against it if necessary - we need more clinical data. In order to minimize the potential damage of blue light exposure in a large proportion of our population, our challenge is to get answers to these questions as soon as possible.

The essence of the problem is succinctly stated by the words of Prof. Wolffsohn, “it’s very difficult to measure exactly how much light we are receiving.” While the sun is the major source of blue light and “sunlight damages because of its pure intensity”, chronic exposure to artificial LED lighting is a very recent and rapidly-changing phenomenon. It is “a very different concept with lower intensity, longer duration and a very different balance across the spectrum”.

This was crystalized in a comment made by Dr. Tolentino, “How many people stare at the sun outside? …. It’s difficult to measure how much light we are receiving; the problem with screens is that we stare at them and the light goes straight to the macula. The power may be less (than the sun) but the time frame is much longer.” Given that “radiation exposure is a matter of power over time”, long-term exposure is a major issue being faced.

Other important parameters impacting exposure were raised by Dr. Orduna and Dr. Picaud. Night time viewing of screens and the intensity of LED lighting is likely to affect the extent of pupil dilation, which is important in terms of potential harm to the retina. With LED illumination, the turquoise light is much dimmer and affects the pupil reflex, meaning it dilates to a greater extent therefore allowing more harmful blue-violet light into the eye.

Further complicating the scenario is the fact that the spectrum of exposure is dynamic, varying considerably with age, personal, and professional environments, as well as with continuous technologic developments; as a result each individual has a personalized risk profile.

Pressing questions needing answers

In addition to the primary question of how do we evaluate how much blue light exposure we are getting, other important parameters include, what is the impact of different types of exposure (artificial lighting, screen viewing vs sunlight), of pupil adaptation (night-time screen viewing and blue/turquoise balance), light intensity, and proximity to source?

“We are experiencing much more blue light exposure and long-term exposure is not fully understood in terms of its potential for damage.” (Prof. Wolffsohn)
Clinical evidence linking blue light and retinal damage

Symptoms caused by blue light lead to a conundrum; on the one hand in routine clinical practice it is rare to see patients consulting primarily for short-term complaints due to screen exposure (i.e., computer vision syndrome) as Dr. Lamoureaux pointed out, while on the other hand if you ask the average person in the street if they have visual fatigue or dry eyes when using their computer or phone, the answer is almost systematically yes. In the clinic, symptoms are mostly reported by patients in the context of other pathologies. This raises the important issue of recognizing which short-term symptoms are specifically due to blue light exposure versus another ocular surface pathology. As Dr. Orduna pointed out, we need “to identify patients who have surface pathologies and distinguish which symptoms are caused by the ocular surface pathology versus those due to blue light”. This is no easy task as symptoms are likely to be a combination of several factors, given that when we look at screens we are receiving more blue light (compared to reading a book for example) and at closer proximity, we are staring, and blinking less; the difficulty is differentiating between these factors. Glare - which is related to visual fatigue - is an important aspect to take into account, with blue light being the main wavelength resulting in glare. In short, many factors contribute to discomfort, blue light being just one of them. Another issue increasingly reported in the literature is the disruption of sleep and circadian rhythms associated with increased nighttime blue light exposure. The implementation and widespread acceptance of the need for protection against UV was supported by solid clinical evidence that UV exposure is linked to damage of the anterior segment of the eye with crystalline lens pathologies. While it is generally agreed that there is strong molecular and functional preclinical evidence linking blue-violet light with ocular toxicity, this is yet to be shown in the clinical context.

Clinical data linking blue light and age-related macular degeneration (AMD) is currently limited to epidemiologic studies. Two studies attempted to analyze sun exposure in terms of blue light, the Chesapeake and the EUREYE. The Chesapeake study in 800 boatmen reported a borderline significant correlation between blue light exposure during the previous 20 years and development of severe AMD. The EUREYE study in 4763 individuals over the age of 65 years correlated blue light exposure and wet AMD for patients with lower antioxidant levels. Other epidemiological studies have evaluated the link between sunlight exposure and AMD. The Beaver Dam Eye Study followed 2764 individuals aged from 43 to 86 years for 10 years and found a significant association between the amount of time spent outdoors in the summer sun during their teens and 30s with the development of both early and late AMD. The Alienor Study, a population-based study of 963 residents of Bordeaux (France) aged at least 73 years, suggested that risk for early AMD is increased in
subjects exposed to high UV radiation, but also to low UV radiation, by comparison with medium exposures. Sui et al performed a meta-analysis of 14 epidemiology studies, 12 of which reported an increased risk of AMD with greater sunlight exposure, and six of which were significant.

A study based from the EUGENDA database (The European Genetic Database) demonstrated a correlation between past sunlight exposure with the development of early and late AMD. Exposure of more than 8 hours of daily outside life resulted in an increased risk of early AMD (odds ratio 5.54) and late AMD (odds ratio 2.77). Other risk factors such as smoking, age and gender were adjusted appropriately. Very few studies have addressed the issue of clinical symptoms associated with blue light exposure. One study in 52 patients compared clinical effects between eyes with intraocular lenses (IOLs) that filter short-wave blue light versus contralateral eyes with IOLs that did not. Improvements were seen for glare disability, heterochromatic contrast threshold, and recovery from photostress when blue-filtering was present. An anecdotal report in five patients also suggested higher blue light emission from LED backlit tablets caused more strain to the eyes.

The risk of AMD progression following cataract surgery lends further weight to the hypothesis that blue-violet light exposure has a role in AMD pathogenesis, with a three-fold increased risk of AMD progression directly attributed to a dramatic increase in blue light exposure.

**Limitations of current clinical knowledge**

Formal clinical data exploring blue light exposure is lacking. The few reports available are restricted almost exclusively to epidemiologic studies. By their retrospective nature, such studies are inherently limited in design, while survey-based questionnaires can introduce bias. Patient populations are often restricted and rarely uncontrolled, while statistical hypotheses (notably the number of subjects) can be questionable. For meta-analyses, the presence of confounding factors is a major weakness.

In the specific context of blue light, analysis of exposure to sunlight is difficult to quantify and vulnerable to error when collecting past exposure data. Furthermore, the rapidly changing blue light exposure profile (less sun exposure, more screen and LED lighting exposure) compared to even just 5 to 10 years ago, likely limits the relevance of conclusions drawn from older studies in the today’s setting.

In the current context of increasing artificial blue light exposure, there is an urgent need to design relevant long-term clinical trials. Planning well-controlled studies is a major challenge given that it is unfeasible to use control groups without access to technology and artificial lighting, as well as the difficulty of matching exposed with unexposed groups given the likely presence of confounding environmental factors.

But one thing is clear – if we don’t start now we will be facing the same dilemma in 10 years!

“We need more and new studies specific for today’s population in terms of food intake and screen behavior, along with the technology to examine eyes.” (Prof. Korobelnik)
The absence of LED regulations/recommendations

Concerns are increasingly being voiced regarding ocular safety in terms of LED usage. Unlike UV for which protective measures have been implemented for some time, with public education on the importance of protecting our eyes against the dangers of UV with protective filtering glasses, visors and sun avoidance, there are currently no blue light exposure recommendations nor any regulations relating to LED exposure. In France, the public committee the Agency for Food, Environmental and Occupational Health & Safety (ANSES), highlighted the potential of an as-yet undiscovered risk for chronic all-day, life-time exposure in light of the absence of any regulations concerning the blue light in LEDs 23. Along with the 2014 international SSL Annex (4E Implementing Agreement), they have urged for photobiologic safety assessments for all SSL devices (LED-based) using the joint CIE S009 / IEC 62471 standard. They have also called for the implementation of a regulatory framework to develop guidelines for protecting the population against potential blue-violet light-induced hazards in terms of the production, sale and use of LEDs. Dr. Picaud, who was a member of the ANSES group, emphasized the particular need for regulations for at-risk populations – notably the young and the elderly.

Learning from past experience (sugar intake and smoking)

An interesting parallel for managing the potential for damage with blue light exposure may well be found in the case of sugar, as Dr. Tolentino and Prof. Wolffsohn recounted. Some 30 years ago the potential dangers of high sugar intake were unknown – over the last few decades the quantities of sugar ingested have increased dramatically – as has the research in parallel. With extensive clinical and preclinical investigations, the tables have turned and we now aware of the dangers of high sugar intake – a striking example can be seen in the rapidly increased rate of diabetes in aboriginals, a population previously minimally exposed to processed sugar. The history of smoking tells a similar tale of unknown dangers identified only retrospectively. For cases where we do not know if potential dangers exist for something that has undeniable immediate advantages from the end-user’s perspective, we need to make the most of hindsight and learn from past errors, notably by exploiting the advantage of the addition of molecular biology and advanced cellular models to our set of research tools – something which we can use to provide solid supporting evidence of potential physiological dangers of blue light exposure.
A PRECLINICAL MODEL TO GUIDE THE CLINIC

While it is widely acknowledged that sunlight is a risk factor for AMD, identifying unequivocally the specific link to blue-violet light and ocular damage is yet to be shown in the clinic. However, our increasing strength in molecular biology research techniques has meant that preclinical research is providing strong supportive evidence of the potential for a link between blue light and ocular toxicity.

As Dr. Tolentino highlighted, molecular biology studies over the last two decades have allowed us to identify photo-oxidation as the main player in terms of the apoptosis and inflammation pathways involved in development of AMD. We know from in vitro and in vivo studies in retinal pigment epithelial (RPE) cells, that blue light from LEDs disrupts regulation of inflammatory markers (VEGF-A, IL-6, IL-8 and MCP-1) and pathological cytokine signalling, causes upregulation of oxidative products, such as lipofuscin, and DNA damage, as well as loss of photoreceptors and activation of apoptosis. However, while many preclinical studies have been performed, they are generally limited in terms of modelling chronic lifelong cumulative exposure damage from blue light exposure, nor do they distinguish between the effects of blue-violet versus blue-turquoise light.

In 2011, the Paris Vision Institute and Essilor teamed up to address these issues, developing an in vitro AMD model in primary swine RPE cells and incorporating innovative cell illumination protocols. RPE cells photosensitised with A2E, a by-product of the visual pigment, were exposed to 10 nm-wide illumination bands across the blue-green range (390-520 nm) then maintained in darkness for 6 hours before analysis. Irradiances were normalized to sunlight intensities reaching the retina under real-life conditions at each wavelength. Cell necrosis (reflecting acute light toxicity) and apoptosis (reflecting long-term cumulative light toxicity) were measured. The results confirmed earlier reports of in vitro and in vivo blue light toxicity studies. However, this study identified the specific range of wavelengths of 415 to 455 nm - corresponding to blue-violet light - as the most toxic band inducing cell apoptosis.

These data were subsequently fine-tuned to better understand the underlying mechanisms of toxicity. Researchers at the Vision Institute have shown that in response to blue-violet light (415 to 455 nm), reactive oxygen species (ROS) production increased, while antioxidant activity was inhibited, and mitochondrial stress was seen. Therefore, cell death is likely to occur under blue-violet light because the cell’s self-defense mechanisms are reduced in parallel with the increased ROS production.

While the question remains open as to the applicability of these in vitro results to the clinical setting, they are strongly supportive and provide guidance for protective strategies in terms of the blue-violet bandwidths to target for filters, along with the value of prescribing antioxidants, while also ensuring that beneficial blue-turquoise light reaches the retina. Further experiments are needed, this long-term in vitro chronic AMD preclinical model offers valuable information.

Phototoxic action spectrum on a RPE model of Age-Related Macular Degeneration (blue-violet light 415 – 455 nm)

Figure 3: Upper panel: blue-violet light toxicity in an in vitro model of AMD. Schematic drawing of RPE cell localization (left) and an image of RPE cells loaded with A2E (right) (A2E: green; nuclei: blue; cell contour: red). Lower panel: cell death (apoptosis) according to 10 nm band exposure showing greater toxicity in the blue-violet range (415-455 nm) despite lower irradiance levels (red diamonds) (Arnaud et al, 2013).
Eye health care advocates have their work laid out for them. Should we be promoting an approach to minimize exposure or pushing for protection? The reality is that reducing exposure is a minimally viable option – the pervasive role that technology has in the social, educational and professional contexts is unlikely to change, and if anything, our exposure will continue to increase.

However, increasing protection is clearly associated with a fundamental challenge given that we need to optimize the risk/benefit ratio of balancing the absence of convincing clinical data linking cumulative exposure and retinal diseases against the unknown potential cost of waiting. Many questions lie behind protection: What exactly should be proposed as protection? Should protection be standard practice? How should eye health professionals be educated and how should they educate their patients?

Preventative protection is up against the difficulty of communicating the value of a potential long-term benefit, without either solid clinical proof or any immediately perceptible benefit perceived by the user. Added to this is the unknown nature of transposing in vitro photobiology results into clinical evidence. Nonetheless, the beauty of blue light protection is that non-invasive no-risk solutions are available and already under exploration, as discussed by the panel and reported below.

**Figure 4: Challenging balance: minimizing phototoxic cumulative exposure and maximizing photo-protective measures**
One of the most accessible means of protecting our eyes is via the implementation of general good health habits. Several studies support the value of antioxidant supplements. The POLA study group demonstrated a protective role of xanthophylls, in particular zeaxanthin, for protection against AMD and cataracts. The AREDS study showed the value of AREDS-type supplements (vitamins C, E, and zinc with copper) for patients with intermediate risk/advanced AMD, while AREDS2 demonstrated that lutein/zeaxanthin intake with or without omega-3 slows progression to late AMD. Nonetheless, the use of antioxidants raised some contention amongst the panel, with prescribing practices varying. While most supported their use in specific at-risk populations - "I start antioxidant prescription when large drusen or advanced AMD are present" - there was debate over their value for the wider public.

Similarly, there is a growing body of convincing evidence emphasizing the importance of avoiding smoking given the strong correlation with the development of AMD, including an increased risk with passive smoking.

Among other blue light protective measures available, yellow IOLs are associated with a level of controversy. The debate dates back over a decade, with initial studies such as the Beaver Dam/Blue Mountains study supporting a link between cataract surgery and increased risk of AMD, whereas results from more recent studies refute these claims. What can be agreed upon is that current data have limitations in terms of interpreting blue light protection in this setting, namely that the studies performed historically were not statistically designed to validate this issue, many used old technology to evaluate the eyes, follow-up is insufficient, and the use of clear and yellow IOLs is not systematic.

The panel agreed that spectacles or contact lenses filtering blue-violet light are a safe choice as a non-permanent, non-invasive protective solution which can thus be renewed in line with changing technology. The point was emphasized that here also, randomized controlled trials are needed. The first encouraging signs that this is underway are seen with the anticipated prospective Japanese CLOCK IOL study which will compare clear vs blue-blocking IOLs with an impressive planned 20-year follow-up.
FILTERING LENSES: THE LATEST ARM AGAINST BAD BLUE-VIOLET LIGHT

A more proactive form of protection comes in the form of ophthalmic lenses filtering UV and blue-violet light. Lenses have been developed to reduce blue-violet light transmission to the eye. It was suggested by some of the experts from the panel that use of such lenses is appropriate for at-risk groups. For patients suffering from retinitis pigmentosa, the use of selective filters for blue light increases visual acuity and contrast sensitivity and decreases glare, which reduce visual fatigue. From a health perspective, ophthalmic lenses are pretty much guaranteed to be without a health cost.

Patients with high-risk genetic profiles for macular degeneration would benefit from the use of protective filtering lenses. Recent genetic testing has developed a genetic algorithm that determines lifelong risk of developing macular degeneration; “Those patients with a high-risk genetic profile should strongly consider blue light filtering protection” according to Dr. Tolentino.

Although opinions were mixed as to their usefulness in the general public (i.e. a population without any known risk), the tendency of the panel favoured their prescription with the clear message - there is no known downside to their prescription. ‘Wearing protecting eyewear filtering out blue light is the simplest solution - with the advantage that it is without toxicity’.
THE PRECAUTIONARY PRINCIPLE AND RECOMMENDATIONS

The precautionary principle is a strategy to cope with possible risks where scientific understanding is incomplete, and is applicable in situations requiring risk management. In the current scenario where the potentially damaging role of blue-violet light in ocular health is up for debate, the two main standpoints are that:

1) in the absence of solid clinical data, preventative measures are not implemented, versus

2) preventative measures are recommended based on supportive preclinical and epidemiological data which provide hints fueling personal convictions of the value of protecting in the context of ‘what is the possible danger of protecting?’ and ‘learning from past mistakes’.

An important question to ask is “Who should benefit from the precautionary principle?” The population at risk can currently be defined as including both the younger and older generations (kids and the elderly), vision-compromised patients, smokers and individuals with a poor diet in terms of oxidizing agents, and a more restricted group experiencing excessive blue-violet light exposure (such as high-level blue light exposure from outdoor activities. A more difficult, but very important, group to define includes individuals with susceptible genetic backgrounds.

**Based on the precautionary principle, the general recommendations of the expert panel were to protect against the modifiable risks with non-invasive solutions, by implementing:**

- **Good health habits (notably in high-risk patients):** quit smoking, implement an antioxidant-rich healthy diet.
- **Sun protection:** avoid high-level exposure (altitude, reflection of water and snow), sunglasses and visors.
- **Blue-violet light technology lens filters:** prescription of filter protection for selected populations (kids, elderly and potentially in retinally-compromised or high-risk genetics, for whom the benefits should still be clinically demonstrated).

Dr. Colombo raised an interesting point. The use of protection can have the added advantage of improving quality of life. “So we can think of lenses not only from a preventive point of view but also as a way to improve the quality of life.”
OCULAR LIGHT TOXICITY AND THE REQUIREMENT FOR PROTECTION

Promoting the precautionary principle: finding an equilibrium

While all experts agreed on the value of general preventative measures, including wearing sunglasses and a cap, a healthy diet and avoiding smoke exposure, convictions varied as to the value of systematic blue-filter lens protective measures, with a definite need restricted to specific settings such as high-level sun exposure, or specific populations such as young children or visually-compromised patients.

“I think there is probably no risk to starting prevention but maybe a risk to delaying” (Prof. Korobelnik).

“I believe everybody should have protective eye wear because there is no downside. I strongly believe that the general population should be educated about the potential dangers of blue light from computer/smart device screens and be given the choice to protect themselves.” (Dr. Tolentino)

“I believe we won’t be making a mistake. It’s a balance, we don’t want to create alarm over blue light, but it’s something we need to consider because there is strong evidence for the model” (Dr. Colombo).

The value of the precautionary principle in the setting of blue light exposure is clearly appropriate, in at least some, if not all, circumstances. But for it to work, it is essential that it is coupled with appropriate education of eye care professionals as well as the general public, using accurate communication.

Dr. Orduna took a more skeptical position about blue light protection with regard to macular degeneration and said “I still do not recommend specific blue light protection. We still don’t know what the specific weight of blue light is in the development of the disease compared to other risk factors. First the scientific community needs to clarify this point with specific research, and then I will talk about prevention in a specific population or prescribe a lens for everybody to protect them from blue light, supported by medical research.”
Complementing their recommendations, the panel also addressed areas of research to focus on in the quest for protective solutions. One of the primary domains mutually agreed on as critical to finding successful protective measures is the need to identify reliable genetic markers for identifying patients with a predisposition to eye damage (particularly for at-risk patients), with parallel implementation of widespread use of genetic markers by clinicians (currently mainly only used in clinical trials). Other tools that need to be identified are quantifiable objective parameters (eg the equivalent of cigarette pack smoking), and consideration should be given to confounding factors (environmental, social and wealth influences).

Prospective clinical studies with long-term follow-up need to be initiated now, along with the collection of specific epidemiologic data on exposure and protection. From a preclinical perspective, further studies in chronic exposure models should be promoted (i.e., furthering the Paris Vision Institute in vitro studies and moving into in vivo models).

In the clinical setting, eye health care providers need to be educated about potential dangers of chronic blue-light exposure and ongoing development of blue light filtering technology. A standard index of filtering/protection of ophthalmic lenses against blue light is likely to be useful in this context – as we have learnt with the public’s awareness of SPF protection against UV dangers. We also need to find the optimal method of educating the public and passing on recommendations for ‘erring on the side of caution’ with promoting proactive protection given the absence of any identifiable downsides to the precautionary principle.
KEY TAKEAWAYS

The November meeting allowed the international eye health experts to share knowledge and professional experience in the context of increasing exposure to harmful blue-violet light, the panel agreed that:

• The clinical consequences of chronic blue-violet light exposure in the current exposure conditions are unknown, and we urgently need to find ways of determining exactly how much blue light our eyes are being exposed to.

• There is a major gap in our clinical experience, notably the absence of well-designed clinical trials with adequate statistical strength in the current setting, and further preclinical and clinical research in this domain is essential.

• While multiple risk factors are incriminated in the pathogenesis of eye diseases such as AMD, the environmental factors (including cumulative exposure to harmful blue-violet light) are modifiable and raise interest for potential preventive measures.

• In the absence of known downsides, preventative protective measures of good health combined with use of lenses filtering blue-violet light (while allowing beneficial blue-turquoise light through) is the most reliable way forward today.

• Based on the precautionary principle, the general recommendations of experts are to apply the preventive / protective measures notably in selected populations (young children, elderly and potentially in retinally-compromised or high-risk genetics, for whom the benefits should still need to be clinically demonstrated).
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II. BLUE LIGHT AND DIGITAL ENVIRONMENT
The incidence of asthenopia is steadily increasing. The main culprit is the increasingly varied and intensive use of digital displays. This dual trend, however, is far from being a foregone conclusion. The observations and ideas for preventive solutions presented below were expressed during an interview with Dr. Marcus Safady, an ophthalmologist practicing in Rio de Janeiro and the 2013-14 president of the SBO - Sociedade Brasileira de Oftalmologia (Brazilian ophthalmology society).

**KEYWORDS**
Asthenopia, eyestrain, postural fatigue, glare, headache, dry eye, contrast perception, adaptation, comfort, posture, digital displays, ergonomics, e-reading, digital devices, connected life, computer, smartphone, tablet, Essilor® Eyezen™, ophthalmic lenses, protocol, eye examination.

**THE DIGITAL ENVIRONMENT AND ASTHENOPIA**

The incidence of asthenopia is steadily increasing. The main culprit is the increasingly varied and intensive use of digital displays. This dual trend, however, is far from being a foregone conclusion. The observations and ideas for preventive solutions presented below were expressed during an interview with Dr. Marcus Safady, an ophthalmologist practicing in Rio de Janeiro and the 2013-14 president of the SBO - Sociedade Brasileira de Oftalmologia (Brazilian ophthalmology society).

**Points de vue:** What are Brazilian ophthalmologists seeing during consultations?

**Dr. Marcus Safady:** We are seeing more and more patients suffering from asthenopias in our practice. Nowadays, symptoms such as dry eyes, red eyes, eye strain sensations, blurred near vision, headache, peri-, intra- or retro-ocular pain, and glare sensations are extremely common. The origins of these symptoms may be refractive (uncorrected or poorly corrected), accommodative or muscular, and clinicians must consider their true cause to treat them effectively.
What correlation do you see between asthenopia and digital displays?

If the patient is properly corrected and presents no particular abnormality in binocular vision, asthenopia symptoms are generally related to external causes. Foremost among them is the intensive use of digital devices, now ubiquitous in our daily lives. When we work in front of a screen our eyes blink less often, resulting in dryness of the ocular surface. The effort of accommodation and convergence is also more sustained due to the increased proximity of multiple displays (e.g. the smartphone and tablet are used at closer distances than the computer). Our eyes make an effort to focus and converge on more or less pixelated targets, whose quality and contrast vary, while remaining exposed to high screen brightness levels. The light emitted is characterized by a predominant dazzling white light that peaks in the blue at short wavelengths. An ophthalmic impact is unavoidable.

Does this type of disorder affect some populations more than others?

These displays exacerbate existing visual defects and also affect those who do not wear glasses. Studies show that 60% to 90% of people using digital displays have more or less troublesome symptoms of eye disorders, regardless of their visual correction. Ophthalmic consultations reveal this problem in adults, children and adolescents. In fact, young people, who often keep their eyes glued to video games, cell phones and computers all day long, even at school, are a particularly vulnerable population.
“Displays exacerbate existing visual defects and also affect those who do not wear glasses.”

**What are the most common solutions and recommendations?**

Patients may not be aware of the causes. When they consult, they usually come in for a refractive problem. They complain of eyestrain and subjective symptoms. Ophthalmologists need to be attentive and play an active role in the fight against this very real scourge. Recommendations are simple: a good visual examination (including visual acuity, binocular vision and accommodation), a refractive correction, ergonomic advice (i.e., best practices for the use of digital devices) and the prescription of a treatment (i.e., eye drops to relieve ocular dryness) or a preventive solution such as appropriate ophthalmic lenses.

**How is treatment for this problem handled in Brazil?**

In Brazil, as in the other countries, eye problems related to the ubiquity of digital displays are widespread. Vision care professionals are increasingly aware and a "standard" protocol is beginning to emerge. It is organized into four...
What are the desired characteristics for these preventive lenses?

They are two in number. The first is the provision of additional refractive power at the bottom part of the lens to relieve the eye's accommodative effort. A few fractions of additional diopter are invaluable when working for hours in front of a digital display. The second is the presence of a filter blocking blue light and the glare effect: a selective anti-reflective treatment reduces screen brightness and blocks harmful blue light.
The perfect ophthalmic lens must combine both features to fight effectively against asthenopia generated by digital device use.

These characteristics seem to be consistent with the ophthalmic lens offer called Eyezen and designed by Essilor research centers?

Absolutely!

“The central point of the prevention plan for asthenopia related to digital device use is the prescription of ophthalmic lenses adapted to the specificities and pervasiveness of digital displays”

- Intensive use of digital displays increases the incidence of asthenopia.
- The problem affects all age groups and as many people not wearing glasses as those with visual defects.
- In Brazil, an easy-to-use four-point protocol is helping to fight effectively against this type of disorder.
- Glasses combining additional refractive power in the bottom part of the lens and a blue light filter are the main preventive solution prescribed for asthenopia related to digital device use.
WILL “DIGITAL VISION” MEAN A BLURRY FUTURE?

Research points to the growing use of digital devices. In parallel, myopia is at epidemic levels in countries around the globe. Taking the longer view, this epidemic could have a negative impact on the lives of the myopic people, especially as they age, and will increase the economic burden that poor vision creates on the world around us.

Myopia widespread and growing; links to near vision demanding tasks and small digital screens

It’s been reported that of the approximately 7 billion people in the world, more have access to a mobile phone than a toothbrush.1

That astonishing statistic speaks to the power and pervasiveness of digital communication and information. Millions of people on this earth can use the technology to text or make a phone call, yet may not have running water and electricity in their residences.

Let’s admit that there is a hypnotic quality to the digital screens that inhabit our lives. Follow someone into an elevator as they are absorbed in what they’re reading on the phone. Stop to watch people on a busy street corner, exiting an office building or on public transportation – it’s a safe bet that a large number will have a smartphone or other digital device in their hands.

We are turning more and more of our daily routine over to our digital devices. From getting the news, to paying for coffee, to receiving directions to reminding us of appointments – digital devices have become the personal assistants for 21st-century lives.

We are living multi-screen lives and are more productive because of it. However, have we stopped to consider how spending so much time squinting at small screens is impacting our vision? Eye health professionals are increa-
singly worried about “digital vision” and the consequences resulting from spending so much time focused on small screens. In addition to failing eye sight, there are the related health issues and socio-economic impacts to consider. While users aren’t abandoning their digital screens, eye health professionals should be aware how to better advise them to be productive and retain their healthy vision.

Myopia increasing in Asia
In parallel, we observe a rise of myopia in developed and developing nations worldwide. It’s at epidemic levels. Eastern Asia, Europe and the United States have all seen a dramatic increase in the number of people who are experiencing shortsightedness.

Myopia is an elongation of the eyeball. While not being able to see distances can be frustrating, even dangerous when driving, it can be corrected with spectacles, contact lenses and refractive surgery. However, high myopia has been associated with a higher risk for ocular disorders, including retinal detachment and glaucoma.

According to researchers, rates of myopia have doubled, even tripled, in many eastern Asia countries during the past 40 years. Hong Kong, Singapore and Taiwan have experienced rate increases hovering around 80 percent. Professor Kathryn Rose of the University of Technology Sydney and Ian Morgan with the Australian National University mentioned the prevalence of myopia in East Asia as ranging from 82% to 96% depending on age groups and countries.\(^2\) Published studies confirm those figures:

<table>
<thead>
<tr>
<th>LOCATION</th>
<th>PERCENTAGE OF MYOPIA</th>
<th>AGE GROUP</th>
<th>YEAR OF THE STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seoul</td>
<td>96.5%</td>
<td>19 yo</td>
<td>2010(^3)</td>
</tr>
<tr>
<td>Taiwan</td>
<td>86.1%</td>
<td>18-24 yo</td>
<td>2010(^4)</td>
</tr>
<tr>
<td>Guangzhou, China</td>
<td>84.1%</td>
<td>17 yo</td>
<td>2007(^5)</td>
</tr>
<tr>
<td>Singapore</td>
<td>81.6%</td>
<td>17-29 yo</td>
<td>2009-2010(^6)</td>
</tr>
</tbody>
</table>

Since 1963, Chinese students have participated in a daily routine designed to relieve eye fatigue. While seated at their desks, they massage the pressure points around their eyes. It doesn’t seem to be working. Rates of myopia have been soaring in Chinese cities, nearing almost 90 percent in places.\(^2\)
According to researchers, rates of myopia have doubled, even tripled, in many eastern Asia countries during the past 40 years. In a news release about a King’s College London research project, Katie Williams from the university’s Department of Ophthalmology, said, “We knew myopia was becoming more common in certain parts of the world – almost 8 in 10 young people are affected in urban East Asia – but it is very interesting to find that the same pattern is being seen here in Europe. This has major implications for the future burden from this eye disease which can threaten sight in older age, particular in very shortsighted people.”

The same rise in myopia is happening in the United States. The American Academy of Ophthalmology estimates that the current rate of myopia has risen to 40 percent from 25 percent in the 1970s.7, 8

Link between myopia and education
Another interesting finding in several research studies is the association between level of education and the incidence of myopia. The research suggests that the more educated the person – regardless of where they live – the more likely they are to suffer from shortsightedness.

“Eye health professionals are increasingly worried about “digital vision” and the consequences resulting from spending so much time focused on small screens.”

Myopia prevalence in Europe
European countries have been experiencing the impact of digital vision and myopia as well. The European Eye Epidemiology (E³) Consortium has done an extensive study of meta-data associated with eye health research which estimates that refractive error affects more than half of the continent’s adult population – myopia being the leading type with 227.2 million people based on 2010 population estimates. Based on this study, the prevalence of myopia suggests that about 20.1 million Europeans are therefore at higher risk for associated complications such as retinal detachment.7

The E³ study also shows that younger people are more affected by myopia than their parents. According to the study, about one-half of younger Europeans are affected. After analyzing the data, the study uncovered that overall levels of myopia have increased about one-third for adults born after 1940 as compared to those born before that year.

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Myopia prevalence in Europe
European countries have been experiencing the impact of digital vision and myopia as well. The European Eye Epidemiology (E³) Consortium has done an extensive study of meta-data associated with eye health research which estimates that refractive error affects more than half of the continent’s adult population – myopia being the leading type with 227.2 million people based on 2010 population estimates. Based on this study, the prevalence of myopia suggests that about 20.1 million Europeans are therefore at higher risk for associated complications such as retinal detachment.7

The E³ study also shows that younger people are more affected by myopia than their parents. According to the study, about one-half of younger Europeans are affected. After analyzing the data, the study uncovered that overall levels of myopia have increased about one-third for adults born after 1940 as compared to those born before that year.

In a news release about a King’s College London research project, Katie Williams from the university’s Department of Ophthalmology, said, “We knew myopia was becoming more common in certain parts of the world – almost 8 in 10 young people are affected in urban East Asia – but it is very interesting to find that the same pattern is being seen here in Europe. This has major implications for the future burden from this eye disease which can threaten sight in older age, particular in very shortsighted people.”

The same rise in myopia is happening in the United States. The American Academy of Ophthalmology estimates that the current rate of myopia has risen to 40 percent from 25 percent in the 1970s.7, 8

Link between myopia and education
Another interesting finding in several research studies is the association between level of education and the incidence of myopia. The research suggests that the more educated the person – regardless of where they live – the more likely they are to suffer from shortsightedness.

“Eye health professionals are increasingly worried about “digital vision” and the consequences resulting from spending so much time focused on small screens.”
This is significant because it points to lifestyle factors as having a role in the rise of myopia.

The E³ analysis of studies, which looks at more than 60,000 people, shows that the rate of myopia is about twice as much higher in people with college degrees compared to those whose education stopped with primary school.9

One of the studies included in the E³ analysis was what is known as the Gutenberg Health Study from the University Medical Center in Mainz, Germany. By examining 4,685 people ranging in age from 35-74 without cataracts or refractive surgery, the results show that myopia increases as education increases.9

The question is then natural: Is there a link between myopia development and the use of digital devices? Although no study has shown a direct link, it has been shown that when using handheld video games, children adopt a closer working distance which in turn may favor Myopia onset and progression.7 Indeed, near work behavior appears to be highly linked to myopia prevalence. Epidemiological studies showed that higher amount of near work results in a high prevalence of myopia in children.10,11,12

The digital vision “antidote”
This rapid rise in myopia is alarming, especially as it affects younger people the most. Are we raising a global generation that will suffer from poor vision throughout their lives?

There is research that indicates that sunshine can be an antidote to digital vision. An Australian research project from 2003-2005 shows that time spent outdoors in natural light significantly affected the presence of myopia in children.13 Longer time of outdoor activity, such as sports and leisure activities, were associated with more hyperopic refractions and lower myopia rates in the 12-year-old students studied. Those who combined longer time of near work with shorter time of outdoor activity.
had the least hyperopic mean refraction, while the students who combined low levels of near work with high levels of outdoor activity had the most hyperopic mean refraction. The lowest odds for myopia were found in groups reporting the highest levels of outdoor activity.

Chinese schools are testing various methods to improve that country’s myopia epidemic. Some schools are experimenting with transparent classrooms – the walls and ceilings are constructed of see-through material to allow for as much light as possible – to determine if that helps improve the students’ eyesight.

Other schools are forcing children to be outside more during the day and away from near vision demanding tasks including small digital screens. Students are sent outside during lunch and recess with the doors locked to keep them there.14

The role of sunlight in our eye health is not completely understood as of yet. A theory suggests that the healthy wavelengths on the blue light spectrum from the sun (the good blue) releases dopamine in the retina which would prevent the eyeball from elongating, thus preventing from myopia. These wavelengths are also protective to vision and other health functions. And the cumulative effect of the damaging wavelengths of blue-violet light (the bad blue) has been linked to retinal cell death, and possibly to AMD. The sources such as artificial light (cold LED), computer screens and handheld devices are rich in harmful blue-violet light and may source potential risks.

In addition to good old-fashioned outdoor playtime for children, the importance of an annual eye examination by a trained vision professional can’t be over emphasized. With the increased use of digital devices and rising rates of myopia, an annual exam is the best way for parents to have poor vision diagnosed – and then corrected as needed – in their children.

Promising research
Promising researches from specialized centers in Australia and China do offer hope. The Vision Cooperative Research Center (Vision CRC) is a partnership between the Brien Holden Vision Institute at the University of New South Wales and the University of Houston College of Optometry.
“Let’s not give up the digital devices, but let’s be sure to take care of users’ eye health while advising both an annual comprehensive eye examination and frequent breaks from “digital vision” to take in a longer view.”

It has announced a new technology that slows the progression of myopia in children. Vision CRC has been conducting large-scale clinical trials in Australia and China designed to control in participating children the position of the central and peripheral retinal image points. Therefore, corrective lenses can be made to control myopic progression by changing the retinal image position at the periphery without affecting the image at the center of the retina. Professor Brien Holden (1942 - 2015) has been quoted saying, “What we need are treatments that effectively slow the progress of myopia which will significantly reduce the prevalence of high myopia. A reduction in the rate of myopia of 33% could produce a 73% reduction in myopia above 5.00 D.”

To strengthen research on myopia, Essilor International and the Wenzhou Medical University in China, opened in 2013 a joint research laboratory: the Wenzhou Medical University-Essilor International Research Center (WEIRC).

“What makes it all the more important is that the link between the severity of myopia and the risk of associated conditions is exponential. Slowing the development of myopia by only 50% reduces the risk of conditions that can lead to blindness (retinopathy, retinal detachment, etc.) by a factor of 10,” explains Dr. Björn Drobe, Essilor Group Researcher and Associate Director of WEIRC.

The laboratory works on three different approaches. The first is to gain a clearer understanding of the mechanisms that cause children to develop myopia. The second focus for research relates to the predictability of myopia, and more particularly involves a study conducted with a group of 1,000 children from urban and rural environments. Lastly, the laboratory is working to identify new ways of controlling the development of myopia through a clinical trial involving 210 children.

“Ultimately, the new knowledge gained will enable us to make our products more effective in terms of slowing the development of myopia with offerings that are suitable for all children and are attractively designed, as well as enabling the development of innovative solutions to counter the myopia pandemic,” summarizes Dr. Björn Drobe.

Socio-economic impact of myopia

Impaired vision is the most common disability in the world, affecting 4.3 billion around the globe. The good news is that 80 percent of those impairments can be avoided or cured. However, that much vision impairment comes with a price tag.

While the global direct socio-economic impact of myopia hasn’t been determined yet, the effect of poor vision on the global economy is well documented. A 2012 review by the Boston Consulting Group and Essilor found that:

- Approximately 33 percent of the world’s working population has uncorrected vision problems that result in a $272 billion loss of productivity to businesses globally.
Points de Vue - International Review of Ophthalmic Optics
Special Edition - Collection of articles from 2011 to 2017
www.pointsdevue.com

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The National Medical Research Council of Singapore commissioned a study on the economic cost of myopia. In 2009, the mean annual direct cost of myopia for school-aged children in Singapore was $148 (U.S. dollars), with the median cost at $83.33 (U.S. dollars) per student. It also showed that the cost of refractive surgery equaled the cost of buying and wearing contact lenses for 10 years. Beyond the cost for children, with a myopia rate of 39% in adults over 40, a 2013 study estimates the total cost of myopia for this population to be approximately SGD$959 (USD$755) million per year in Singapore.

**What it means for the future**
Research has indicated that myopia is rapidly rising in East Asia, Europe and the United States, especially among younger people. And research points to factors other than genetics, such as behavior and environment, as causing this epidemic. Is the common denominator among these the time spent using digital devices at near? The global use of these devices is only going to grow as we increasingly rely on them to connect with friends, get our news, make financial transactions, and simply make our lives easier and more productive. As a planet, we spend 3 billion hours a week playing video games. That means that we will spend more time in "digital vision" mode - fixated on small glowing screens using our eyes for near vision more often.

**There will be consequences.**
Yes, the majority of myopia cases can be corrected with spectacles, contact lenses or refractive surgery. And the research centers such as Vision CRC and WEIRC, as well as the technology development, give us hope for a better-seeing future. However, with so many young people dealing with shortsightedness, as they age the cost and impact of poor vision is likely to increase from such things as loss of productivity, motor vehicle accidents, falls, and social isolation. Add to that the significant increased risk people with high myopia have for related vision diseases.

Let’s not give up the digital devices, but let’s be sure to take care of users’ eye health while advising both an annual comprehensive eye examination and frequent breaks from “digital vision” to take in a longer view. •

**MARKET WATCH**

| • Poor vision slows the education of school-aged children, resulting in academic under-achievement and risk of reduced adult literacy. In fact, 30% of children worldwide need vision correction and don’t have it. |
| • Impaired vision is associated with 60 percent of driving accidents around the world. |
| • Globally, poor eyesight multiplies by seven the risk of falls and hip fractures in the elderly. |

| The good news is that 80 percent of those impairments can be avoided or cured. However, that much vision impairment comes with a price tag. |
| While the global direct socio-economic impact of myopia hasn’t been determined yet, the effect of poor vision on the global economy is well documented. A 2012 review by the Boston Consulting Group and Essilor found that: • Approximately 33 percent of the world’s working population has uncorrected vision problems that result in a $272 billion loss of productivity to businesses globally. |
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**GLOBAL**

**Global Scope of Poor Vision**

An estimated 2.5 billion people with vision problems do not benefit from corrective measures.

**Source:**

United Nations, Essilor
An epidemic of myopia is circling the globe, with Eastern Asia, Europe and the United States seeing rising rates of shortsightedness, especially in young people.

Research shows that there is a link between education level and myopia rates – those with more education are more likely to be myopic.

Corresponding to the increase of myopia is also an increase in near vision demanding tasks including the use of small digital devices as people rely on them more not only to communicate, but also to access news, information and entertainment.

“Digital vision” will likely have a socio-economic impact on the world, especially as young people with myopia grow older.

KEY TAKEAWAYS

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With its annual survey, *Hindsight is 20/20/20: Protect Your Eyes from Digital Devices*, The Vision Council monitors usage trends related to digital displays and their impact, as regards both eye strain and exposure to blue light. The report’s 2015 edition highlights the growing pervasiveness of digital displays in the United States and the stakes in raising awareness of the actors involved in the visual health sector like the general public.

**Mike Daley**  
Chief Executive Officer (CEO), The Vision Council, USA

**Dr. Dora Adamopolous**  
Medical advisor, The Vision Council, OD in Alexandria, USA

**Erin Hildreth**  
Marketing and Communication Manager, The Vision Council, USA

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Mike Daley began his optical career as an instructor with Ferris State University in 1975. He joined Essilor in 1976. With consolidated skills in sales, marketing, technical services, laboratory operations, he served as the President of Varilux Corporation (1989-1999). After 32 years with Essilor, he retired in 2008 as the President and CEO of the Lens Division of Essilor of America. Throughout his career, he has been recognized by his peers and has served in a leadership position for an impressive number of optical organizations including National Academy of Opticianry (NAO) Hall of Fame; Prevent Blindness America, Board of Directors; AOA Optometric Charity Board; SoloHealth Board of Directors; The Vision Council of America, Board of Directors, and past Vice Chairman. He holds Ferris State University Honorary Doctorate (2006).

Dr. Adamopolous graduated as a Doctor of Optometry from the New England College of Optometry in 1998. During her last few academic years, she had the opportunity to sharpen her clinical skills through a series of rotations in different types of medical settings on the East Coast. After graduation she worked in the private practice arena, treating and managing ocular pathology in a geriatric population. Today, she devotes her expertise to welcome and treat patients suffering from dry eyes, allergies, diabetes, cataracts and glaucoma. Involved in the development of visual health in the United States, she collaborates with the Vision Council as medical advisor.

Erin Hildreth has great past experience in communication, marketing and education. She served as the Education Manager for the Health Industry Distributors Association (HIDA), coordinating and providing contents for trainings. She led several editorial projects including advertising, content management and online development. Today, she is responsible for marketing and communication at The Vision Council. She develops and implements programs that educate consumers about eyewear trends, lens technology and health aspects. Keeping strong focus on eye health benefits, she works on UV awareness, protection and prevention necessity (including digital eye strain), aging and low vision.
Digital eye strain is more than a reality; it is a public health priority in the United States. This is the warning published by The Vision Council\(^*,\) which has just released its latest survey on this issue: Hindsight is 20/20/20: Protect Your Eyes from Digital Devices\(^1\). The document is based on an analysis of 9,749 questionnaires completed by a representative sample of adult U.S. residents. Its aim is to determine the broad outlines of behavioral changes with respect to digital displays, be they smartphones, tablets, computers, laptops or other electronic devices, such as game consoles. This state of play confirms the trend that has emerged in recent years: “From the moment people get up until the time they go to bed again – including when they are eating, exercising and reading – they are using one digital device after another and thus exposing themselves to risks related to prolonged exposure to light emitted by screens,” sums up Mike Daley, chief executive officer of the Vision Council. In concrete terms, more than 95% of American adults spend at least two hours a day in front of a screen and almost three out of ten spend over nine hours. Even though people working on computers are the most concerned by a potential “overdose”, the study stresses that one child out of four is exposed to screens over three hours a day. These constantly increasing figures can be explained by both new societal patterns (i.e. a decrease in physical activity, an increase in passive consumption and paperless contacts, etc.) and options made possible through innovation. “Digital technologies offer ever increasing options and opportunities to simplify consumers’ daily lives. This growing trend is not likely to be reversed any time soon. Nor are the related opthalmic problems,” Daley predicts.

**Screens as a source of eye strain**
The main effect of prolonged exposure (greater than two hours per day) to light emitted by screens is digital eye strain. Described as a passing discomfort, it manifests itself in different forms with symptoms such as red, dry or irritated eyes, blurred vision, pain in the neck, shoulders or back, headache, etc. “We blink 18 times a minute on average. However, staring at a screen for an extended period can result in less frequent blinking that could dry or even irritate the eyes\(^*\)”\(^*\), Erin Hildreth reminds us. The Vision Council’s marketing and communication manager relates that a recent study\(^3\) concluded that employees

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**Activities Associated with Digital Device Use:**

<table>
<thead>
<tr>
<th>Activity</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Work</td>
<td>44%</td>
</tr>
<tr>
<td>Waking up</td>
<td>38%</td>
</tr>
<tr>
<td>Recreational reading</td>
<td>43%</td>
</tr>
<tr>
<td>Travel</td>
<td>32%</td>
</tr>
<tr>
<td>Meal preparation</td>
<td>26%</td>
</tr>
</tbody>
</table>

\(^*\)From the moment people get up until the time they go to bed again – including when they are eating, exercising and reading – they are using one digital device after another and thus exposing themselves to risks related to prolonged exposure to light emitted by screens.”

M. DALEY

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“FROM THE MOMENT PEOPLE GET UP UNTIL THE TIME THEY GO TO BED AGAIN – INCLUDING WHEN THEY ARE EATING, EXERCISING AND READING – THEY ARE USING ONE DIGITAL DEVICE AFTER ANOTHER AND THUS EXPOSING THEMSELVES TO RISKS RELATED TO PROLONGED EXPOSURE TO LIGHT EMITTED BY SCREENS”

M. DALEY
working all day on a computer could present physiological changes of the lacrimal system similar to those found in dry eye syndrome. “This is not surprising when one considers that the work environment is often characterized by multiple or split screens, small fonts, poor posture and LED or fluorescent lighting.”

The blue light paradox
In addition to eye strain, overexposure to digital displays is linked to the issue of blue light. Eye doctor and medical advisor to the Vision Council, Dora Adamopoulos, recalls that “a great deal of research is currently underway to determine its precise impact on the eyes and vision. One thing is certain: the blue-violet spectrum (415-455 nm) is particularly harmful. It penetrates deeply and causes photochemical reactions likely to damage retinal cells, with a cumulative effect. The retina cannot be replaced; its alteration therefore leaves the eye vulnerable to harmful light and environmental factors, thereby increasing the risk of early development of ophthalmic disorders, such as AMD.” However, blue light is not an enemy that must be fought at all costs. The blue-turquoise spectrum participates in the regulation of natural circadian rhythms (i.e. sleep-wake cycles) among other things, and stimulates the pupillary reflex and such cognitive functions as alertness, memory and emotion regulation. “Blue light is both unavoidable and indispensable. So it is important to understand its repercussions on the organism and vision, and be familiar with the tools and recommendations for minimizing exposure, particularly from digital displays,” the expert advises.

“A QUESTIONNAIRE HANDED OUT PRIOR TO A CONSULTATION CAN HELP TO CLARIFY AT WHAT DISTANCE EACH SCREEN IS BEING USED, HOW THE OFFICE IS ORGANIZED, THE MOST COMMON POSTURAL POSITIONS ASSUMED AND SO ON, AND THIS INFORMATION CAN THEN SERVE AS A BASIS FOR DISCUSSING PROBLEMS AND POSSIBLE SOLUTIONS”

E. HILDRETH
Digital childhood and myopia

Prevention and protection are equally important for both adults and young people, who now use computers and smartphones in all aspects of their schooling and social life. The latest Digital Eye Strain report points to intensive screen use and a lack of data on the medium-term consequences. “The phenomenon is recent, so it is impossible to foresee the impact of emitted light on children’s eyes. But in our opinion, myopia is one the main risks that must be evaluated,” Erin Hildreth hypothesized. “The causes of myopia are related to a combination of genetic and environmental factors, and since the pervasiveness of digital devices stimulates ocular accommodation at very close reading distance, this could well be part of the problem.” The Vision Council therefore calls for vigilance and a complete eye exam every year to ensure the best possible development of children’s eyes. “A professional can evaluate symptoms or visual disorders resulting from the use of digital devices and suggest solutions and make recommendations,” she affirms. However, this approach comes up against one of the main findings of the study: the majority of parents are not worried about the effect of the digital environment on their offspring. 15% of respondents place no limits on the amount of time spent in front of screens, and 30% are not concerned about the potentially harmful impact of digital devices on the development of the visual system.

Think and act “awareness”

This finding of disregard for risk highlights one of the major challenges of the Vision Council’s action: public awareness. Its CEO confirmed this focus: “For us, education is the key. The transmission of information about the nature of digital eye strain, including risks related to exposure to digital displays and especially how to fight them, should be a major focus for mobilizing our sector.” To optimally publicize the issue, The Vision Council is diversifying its strategy and seeking to strengthen its communication in schools and during ‘key’ events: film releases, TV marathon broadcasts, new technology launches, or international trade fairs, including the celebrated CES (Consumer Electronics Showcase), an unmissable event for new technology fans. This is a good way to reach a large number of users and instill in them a desire to safeguard their eye health. And to facilitate the assimilation of prevention, the organization is relying on its flagship slogan: “20-20-20”. Every 20 minutes, look 20 feet in front of you (approximately 6 meters) for 20 seconds. This rule is easy for both adults and children to remember and use. “The Think About Your Eyes campaign (www.thinkaboutyoureyes.com) is also a great way to inform people about the benefits of an annual ophthalmic examination,” adds Daley, who sees in consumers’ appetite for connected information an excellent opportunity to use these media, including websites and social networks, and connect with other industry players about the importance of eye health in the digital environment.
“WE HAVE A DUTY TO EXPLAIN TO CONSUMERS THAT THEY DO NOT HAVE TO LIVE WITH DISCOMFORT OR PAIN WHILE USING DIGITAL DEVICES. CUSTOM GLASSES, WITH OR WITHOUT CORRECTIVE LENSES, CAN ALLEVIATE OR PREVENT SHORT-TERM SYMPTOMS AND PROTECT AGAINST LONG-TERM DAMAGE.”

D. ADAMOPOULOS

Vision professionals and new preventive measures
Eye care professionals have a big responsibility – and a good opportunity – to lead the fight against the deleterious effects of digital displays. In addition to the development of new health and technical solutions, Erin Hildreth encourages “ophthalmologists, optometrists and opticians to adopt simple and pragmatic measures to help their patients in their everyday activities.” Some ideas and recommendations include: promoting continuing education and keeping abreast of the latest findings in this area; taking an interest in public opinion and consumer perceptions; taking charge of consultations by systematically interviewing patients about their use of digital devices and finding out not only what type of devices are being used, but also how they are used and for how long. “A questionnaire handed out prior to a consultation can help to clarify at what distance each screen is being used, how the office is organized, the most common postural positions assumed and so on, and this information can then serve as a basis for discussing problems and possible solutions,” she suggested. This should be accompanied by some key preventive recommendations.

Preventive recommendations for the users of digital displays
1) Design your work space in such a way as to alleviate external stressors, with ideal lighting, an “eye-gonomic” setting (ergonomics for the eyes) and good posture.
2) Increase character size in relation to the device used.
3) Observe the 20-20-20 rule. Every 20 minutes, look 20 feet in front of you (about 6 meters) for 20 seconds.
4) Consult a health professional on a regular basis to obtain counseling and prescriptions for ophthalmic lenses designed for multiple screen use.

The importance of prevention
Advances in ophthalmic optics have already made possible a wide range of options for lenses capable of reducing glare and filtering out blue light. These two indispensable options to optimize visual comfort while using digital displays should encourage opticians to add them to prescriptions to more
closely meet their clients’ needs. “Many manufacturers also offer multifocal lenses for people who need to relieve eye strain and correct both near and far vision,” Dora Adamopoulos added. The medical advisor feels that “the optical/ophthalmic industry must continue to engage in research and development for new products, but also educate the community of vision care professionals and the general public. We have a duty to explain to consumers that they do not have to live with discomfort or pain while using digital devices. Custom glasses, with or without corrective lenses, can alleviate or prevent short-term symptoms and protect against long-term damage.”

It is more important than ever to disseminate this message, inasmuch as scientific advances are increasingly confirming the link between digital displays, eye strain, age-related eye diseases and the importance of prevention and protection.

“The new digital era is more stressful on our eyes and we must all adapt accordingly, professionals and users alike. The optical/ophthalmic industry has already identified the major issues raised by digital devices and during the last several years, we have witnessed a boom in innovation capable of reducing disorders related to the light emitted by screens. These products and technologies do much more than protect our eyes: they improve the quality and precision of our vision,” Mike Daley concluded.
Some key figures

- In 2015, 69% of American adults use a smartphone and 42.5% a tablet or e-book reader on a daily basis, versus 45% and 26% respectively in 2012.
- 60.8% spend more than five hours a day in front of a screen.
- 31.9% do not make any effort to reduce symptoms of digital eye strain.
- 72.5% are not aware of the potential damage caused by overexposure to blue light and do not know that digital displays emit blue light.
- 22% of parents say that they are concerned by the impact of digital device use on their children’s vision.
- 30.6% of the same parents allow children to use digital devices for over three hours daily despite their concern.

REFERENCES


KEY TAKEAWAYS

- Americans (both adults and children) are spending more and more time in front of digital displays on all types of devices.
- Disorders and risks related to light-emitting screens (i.e. eye strain and retinal pathologies) are either unrecognized or underestimated by the general public, the majority of whom neglect prevention and protection.
- Simple solutions exist to fight against digital eye strain and overexposure to blue light.
- The Vision Council recommends following the 20-20-20 rule (every 20 minutes, take a 20 second break while looking 20 feet away) and using ophthalmic lenses designed for screen use.
- Vision care professionals all have a role to play in terms of advocacy, awareness-raising and counseling.
THE CHALLENGES OF DIGITAL VISION IN A MULTI-SCREEN WORLD

In this new digital era, there are new risks for user eyes and new challenges for vision care professionals. Ten experts, optometrists, ophthalmologists and researchers have addressed this broad topic and offer us their experience and thoughts in the form of verbatim comments. This overview has been divided into three main thematic areas: risks and prevention, professional practices, and projections and expectations.

1. RISKS AND PREVENTION

What effects do digital displays have on health? The main risks, whether they are known, suspected or potential, primarily concern vision, but may also affect other functions. Experts are reassuring however: good visual hygiene, regular eye exams by professionals, appropriate optical solutions and enhanced public awareness provide effective prevention.

Impact of digital displays on vision

“Our visual system is biologically designed for distance vision. Near vision is only an accommodation reflex that helps us quickly identify objects close at hand. Our eyes are not designed to stare at screens for hours on end.”

José de Jesús Espinosa Galaviz

KEYWORDS
digital devices, connected vision, multi-screen environment, computer, smartphone, tablet, video games, blue light, ametropia, emmetropia, digital displays, posture, digital tools, connected life, eye strain, vision health, prevention, visual hygiene, accommodative effort, asthenopia, headaches, sensitivity to the light, diplopia, sleep, cortisol, melatonin, ergonomics, protection, child, myopia.
“OUR VISUAL SYSTEM IS BIOLOGICALLY DESIGNED FOR DISTANCE VISION. OUR EYES ARE NOT DESIGNED TO STARE AT SCREENS FOR HOURS ON END.”

JOSÉ DE JESÚS ESPINOSA GALAVIZ

“A reduction in the frequency of blinking during screen use increases the severity of such symptoms as dry eye or irritation and blurred vision. Smartphone users tend to hold their phones very close to the face, thus requiring an intense accommodative effort causing eye strain or headaches.”

Sebastian Marx

“In such rapidly developing cities as Singapore, we see concomitant growth in the number of people working in offices and cases of asthenopia, sensitivity to light, transient diplopia and so on.”

Koh Liang Hwee

“The increase in ophthalmic disorders is linked to the proliferation of screens and the time spent watching them: in the classroom (from primary school to postgraduate courses, including tablets, computers, electronic tables, etc.), but also at all ages via the social networks, television and e-books, which are becoming increasingly popular.”

Helen Summers

“No clinical study to date has demonstrated that overexposure to digital displays is the cause of early macular degeneration. However, blue light emissions are a reality and over time we are bound to see a clinical impact. Concerning the increase in cases of myopia, various studies point to the possible influence of digital displays used at ever closer distances. We still need to understand why certain subjects develop myopia and others don’t, even among twins.”

Sebastian Marx

“The main risk for the younger generation is myopia, perhaps not true myopia, but rather an ‘accommodative spasm’ (i.e. near point stress according to Skeffington), since the human eye and brain were not designed for extended near vision.”

Aravind Srinivasan
Consequences beyond vision

“In the medium and long term, digital displays affect people in different ways. The impact is not solely ophthalmic. The symptoms are varied, suggesting both physical disorders (neck and back pain, etc.) and psychological disorders (fatigue, irritability, poor concentration, memory problems and so on).”

Aravind Srinivasan

“Overexposure to blue light emitted by screens can disrupt the secretion of melatonin and thus affect the quality of sleep. Eye strain can also have an effect on productivity and lead to other disorders, such as stress, anxiety or mood swings.”

Koh Liang Hwee

“Ever more pervasive video gaming is associated with player immersion and strong screen flicker. These two situations can eventually stimulate systemic and endocrine functions, resulting in elevated cortisol levels. The main repercussions have been found to affect sleep, behavior, mood, motivation and learning.”

Helen Summers

Preventive solutions

“Consumer awareness campaigns are an important means of highlighting the risks and symptoms related to digital displays and offer an opportunity to stress the need for regular eye exams.”

Aravind Srinivasan

“OVEREXPOSURE TO BLUE LIGHT EMITTED BY SCREENS CAN DISRUPT THE SECRETION OF MELATONIN AND THUS AFFECT THE QUALITY OF SLEEP.”

KOH LIANG HWEE
“Every person consulting a vision care professional should be informed of the impact of digital devices and blue light, as well as the importance of good visual hygiene and the availability of optical solutions. A wide range of high-quality solutions are available; it is regrettable, however, that current prices limit their use primarily to adults rather than children.”

Helen Summers

“A new specialty, ergo-optometry, could be created. The ergo-optometrist would counsel patients on how to take better care of their visual health, explain what products to use to treat dry eye and provide personalized information with regard to lenses and frames, even for patients without refractive error. Overweight people can contact Weight Watchers. People with ophthalmic problems should be able to contact Eyes Watchers.”

Joachim Köhler

“We are not usually aware of our posture; our organism chooses the most appropriate position for a given situation, without worrying about potential physiological repercussions. It is essential to adopt good posture. For reading, I recommend the Harmon distance at a minimum; this is the distance from the tip of the elbow to the middle of the index finger.”

José de Jesús Espinoso Galaviz

“Good visual hygiene also includes: an ergonomic work space; good posture, a straight head and back; good lighting, with lower lighting for screens and adequate room lighting; breaks every 20 minutes; alternating between near and far screen distances, and suitable ophthalmic lenses.”

Helen Summers

2. PROFESSIONAL PRACTICES

How are digital devices influencing the everyday lives of vision care professionals? New consultation protocols, near vision refraction and control methods appropriate to digital displays, personalized counseling and more frequent continuing education are the main developments cited by experts. Many professionals are incorporating digital tools into their practices to better assess users’ needs. In the context of overexposure to digital devices, experts are also beginning to take more interest in children and emmetropic people (without refractive error).

Protocols and refraction

“Just a few years ago, protocols were established on the basis of the symptoms one should look for rather than on patients’ needs depending on their environment. This approach is now changing. Currently, in addition to patients’ histories, we are also interested in their concerns, expectations, environment and so on, and we are adapting protocols accordingly.”

Luis Ángel Merino Rojo

“For people who rely heavily on their near vision, I apply a protocol based on behavioral optometry. This approach is important when prescribing the best lenses for a particular type of activity.”

José de Jesús Espinoso Galaviz

“My approach? First I exclude ocular pathology and perform a refraction. Then I evaluate the patient’s visual faculties (accommodation, convergence, ocular mobility and sensory aspects such as stereoscopic vision, etc.). Once all these criteria have been evaluated, the treatment strategy can be defined.”

Elizabeth Casillas

“Far vision refraction is often performed using cycloplegic eye drops with a refractometer. Near vision is examined with trial frames equipped with interchangeable lenses to better evaluate posture, head position and reading distance in relation to a support, computer or digital device. Instruments such as ‘Capture I’ or ‘Visioffice®’ are used to measure frame parameters and such individual parameters as pupillary distance and the eye’s center of rotation.”

Helen Summers

“My staff has slightly modified their refraction methods to adapt to digital technologies. We placed a smartphone and tablet in the consulting room and, after the examination, we ask patients to read what is written on the screen. If they are unable to do so, we orient them towards specific lenses. Otherwise, all is well! By using digital devices to test near vision, we fit in more closely with our patients’ digital lifestyles.”

Joachim Köhler

Prescriptions and counseling

“There are several complementary approaches. The first involves optical correction, with high-tech lenses offering optimal vision quality and protection. The second approach involves training, consisting of various exercises designed to improve visual capabilities. The third approach involves education in visual hygiene (posture, breaks, a good work environment, etc.). The final prescription depends on the age and issues of each patient.”

Elizabeth Casillas
“WE HAVE A REAL ROLE TO PLAY IN THE TREATMENT OF DISORDERS RELATED TO DIGITAL DISPLAYS”

ELIZABETH CASILLAS

“The patient’s age affects the proposed treatment. People with presbyopia will be advised to wear progressive lenses, with a coating (i.e. a filter) suited to the specific issues posed by digital devices. For younger children, with or without a correction, lenses must primarily meet the objective of protecting their vision against the harmful effects of screens.”

Aravind Srinivasan

“We must be attentive to each of our prescriptions, always follow the same consultation protocol, compare feedback from each patient and keep a record of all results.”

Berenice Velázquez

“People working on computers are advised to have regular exams, in order to identify any symptoms of ophthalmic stress. The prevention aspect is particularly stressed for children, especially for children under 10.”

Helen Summers

“Information provided by researchers, universities, specialized societies, suppliers and the like, helps us stay on top of new developments and provide increasingly personalized solutions. We must make an effort to step out of the ‘comfort zone’ of standardized options and adapt them to individual needs.”

Sebastian Marx

“We have a real role to play in the treatment of disorders related to digital displays and must devote more time to informing and educating ourselves and to testing new solutions. In this regard, it could be useful to reinforce the sharing of experiences and dissemination of information through forums and professional networks.”

Elizabeth Casillas
“There is a paradox. On the one hand, we have more and more technological tools available to us (auto-refractometers, digital phoropters, photo and video sharing capability to improve diagnosis, etc.), but on the other hand, we have a new generation of professionals who no longer know how to perform an exam without these devices. The right balance must be found between the assimilation of new technologies and basic knowledge.”
José de Jesús Espinosa Galaviz

3. PROJECTIONS AND EXPECTATIONS

How do we anticipate future issues and respond to the realities of a multi-screen world? Between increased research efforts and the development of technological innovations that will facilitate customized products and services, the various ideas outlined offer a glimpse of the future of the ophthalmic optics sector, which is in a position to turn the digital challenge into a real growth engine.

Clinical studies and R&D

“Technological progress is making rapid headway, but the ophthalmic optics industry should be further ahead than it is if it is to adequately meet the health challenges associated with digital displays. It is important to invest more in health research in general and vision health in particular.”
José de Jesús Espinosa Galaviz

“The place of emmetropes

“My colleagues and I feel that emmetropes (i.e. people without refractive error) have been completely forgotten by our profession. During screen use, they are exposed to the same risks as glasses wearers. So it is important to educate them about the existence of simple solutions and practices to fight against asthenopia and other disorders related to digital devices.”
Luis Ángel Merino Rojo

“It would be useful to mount a major information campaign on the risks of overexposure to digital displays. And explain that vision care professionals have solutions to respond to these issues, even for emmetropes.”
Berenice Velázquez

Digital devices and professional practice

“For vision care professionals, digital technologies make it possible to share cases and experience, to the benefit of patients.”
Jaime Bernal Escalante

“Digital tools and certain applications can be used to take a number of different measurements: asthenopia, the quantity of blue light emitted by screens, etc. They can also be used to disseminate recommendations aimed at optimizing visual comfort and participate in the therapeutic education of users.”
Berenice Velázquez

“EMMETROPES HAVE BEEN COMPLETELY FORGOTTEN BY OUR PROFESSION. DURING SCREEN USE, THEY ARE EXPOSED TO THE SAME RISKS AS GLASSES WEARERS.”
Luis Ángel Merino Rojo
“ALL STUDIES FOCUSING ON THE EXACT RELATIONSHIP BETWEEN CONNECTED LIFE AND OPHTHALMIC DISORDERS SHOULD PROVE USEFUL.”
JAIME BERNAL ESCALANTE

“All studies focusing on the exact relationship between connected life and ophthalmic disorders should prove useful. And in my opinion, the development of shared databases would be a real “plus” for all vision health players.”
Jaime Bernal Escalante

Expected innovations

“More precise measuring equipment. The fact of having 20/20 (10/10) vision reveals nothing about the way patients’ use their eyes while watching a screen.”
Elizabeth Casillas

“Tools to measure the impact of luminous digital displays on the eye.”
Aravind Srinivasan

“New products, particularly ophthalmic lenses capable of protecting the eyes against technological ‘radiation’.”
Jaime Bernal Escalante

“The ideal lens: a product capable of integrating all treatments and filters on demand, based on the individual needs of each patient.”
Koh Liang Hwee

“A completely innovative approach, with ‘flexible’ smart lenses capable of adapting their optical properties to specific situations. A high level of modularity that could involve the use of electronic components.”
Sebastian Marx

Vision health in the future

“The multi-screen environment is part of daily life. This environment can potentially pose certain risks, particularly for the eyes, and it is up to us as vision care professionals to concern ourselves with these risks and provide some answers, either directly or via the Internet.

Indeed, technological and societal developments are opening up new fields of practice that offer our industry an opportunity to evolve! Personally, however, I prefer direct contact with patients, to show them that I am indispensable as a specialist.”
Joachim Köhler

“New visual needs concern a large number of everyday activities; therefore growth opportunities for the vision health sector can only increase. The solutions developed must provide added value: filters to prevent eye strain or blue light-related risks, lenses capable of stimulating peripheral areas of the retina to fight against myopia or stimulate amblyopic eyes and improve their performance. There are still many little exploited or untapped areas that will undoubtedly drive development in the future. The response to digital issues is part of this.”
Luis Ángel Merino Rojo
Conclusion

The new digital era is witnessing new societal, sensorial and behavioral transformations. This brief survey of the situation worldwide highlights the increased overall level of awareness of the ophthalmic optics sector confronted with the rapid, wide-scale changes driven by the emergence of digital technology and, more particularly, its impact on users’ vision and posture. From stronger prevention efforts to personalized treatment options, without forgetting projections for the future, the vision health sector is joining forces to adapt to developments, anticipate upcoming challenges and provide better performing solutions for ametropic and emmetropic patients of all ages.

Insights collected by Oliver Vachey, science journalist.

• The human eye is not designed for near vision over a long period. Spending too much time in front of screens results in asthenopia, dry eyes, red or irritated eyes and other ophthalmic symptoms.
• The medium-term impact on users’ general physical condition and behavior is correlated with overexposure to blue light and screen flicker.
• Preventive solutions exist for each situation, but public awareness needs to be improved.
• Professional practices are evolving and adapting with the goal of providing increasingly personalized treatment options designed specifically for users of multiple screens.
• Efforts are still needed in the area of clinical studies, R&D and innovation, to enhance the already substantial offer, provide new solutions and anticipate upcoming issues.
• The satisfactory integration of digital vision issues is a major factor affecting the growth and development of the ophthalmic optics sector.

KEY TAKEAWAYS
The World of Multiple Screens: A Reality That Is Affecting Users’ Vision and Posture

Just a few years after their market introduction, digital devices are abundantly present in people’s everyday lives. We now live in a multiple-screen environment and may use up to ten different devices with screens in a single day (laptop, desktop, tablet, console, digital TV, GPS, e-book reader, digital code device, smartphone or smartwatch).

Users today want to be connected at all times. However, these new media are affecting their vision and posture. To measure this impact, the Ipsos institute conducted a broad survey on four continents with four thousand people. The results show the growing challenges posed by this new digital reality to public health.

Cross-generational use of digital devices is accelerating

Today, digital devices have become an accepted part of everyday life, irrespective of age, social class or geographical area. After years of undisputed reign, the supremacy of television and computers has now been challenged by a massive influx of small screens – smartphones, tablets, e-book readers and game consoles – that have truly revolutionized digital practices. In less than ten years – the launch of the iPhone barely dates back to late 2007, and the tablet to 2010 –, these new media devices have emerged as essential everyday tools, generating new habits and new needs.

Survey

To measure the impact of the use of these new devices on users’ vision and posture, Ipsos conducted a broad survey on an international scale in four countries (Brazil, China, France and the United States), with four thousand people aged 18 to 65.

Sophie D’Erceville
Research director at Ipsos, Paris, France

After earning a degree in marketing and communication (Masters 2) from CELSA, Sophie worked for seven years in the quantitative research sector. She joined Ipsos in 2011, where she is responsible for numerous studies on marketing issues and trends for various sectors, including the optical sector. She has been assisting Essilor for a number of years with the implementation of international surveys aimed at better understanding and anticipating trends in the area of visual health and optics.

Keywords

The use of digital screens is now a daily reality for a very large majority of the population. Young and old alike use them several hours a day, and 29% of smartphone owners have their eyes riveted on their phone screens for more than four hours a day. Opportunities for use are varied and include reading, writing, watching videos, taking photos or videos and much more. Fig. 1.

Multiple-screen use is intensifying
Devices are no longer used just sequentially; they are increasingly used simultaneously. Combined, they exact a heavy toll on the eyes at any distance, whether viewed from afar or close-up: for example, 72% of people surveyed have watched television while using a smaller screen, such as a smartphone, tablet, e-book reader or game console, forcing them to constantly look back and forth from one screen to the other. 69% have used a computer while alternating with a smaller screen(s). This intensified use is reported by users themselves: 89% of them confirm that they seem to spend more time using screens, and 82% say they are watching screens for longer periods than two years ago. Fig. 2.

New digital uses are causing visual and physical discomfort
The increasingly intensive daily use of digital devices, particularly small screens – the smartphone is the most frequently used device on a daily basis –, involves a certain amount of discomfort, and users are well aware of this: 89% have felt discomfort or pain in their eyes, which they associate, at least in part, with their use of screens. But most of the time, their symptoms seem to be temporary and fairly harmless: they complain of eyestrain (74%), itchy eyes (50%), dry eyes (46%), rather than report that their eyes sting (34%) or hurt (35%).

Their eye symptoms, especially eyestrain (which 51% describe as moderately or highly bothersome) are considered just as uncomfortable as the bodily pain affecting the neck and shoulders (54%) or back (51%) Fig. 3.

In addition to these visual and physical symptoms, 46% of respondents report they have difficulty sleeping, including 35% for whom this is a real problem.

Even though these symptoms cause little or no concern on the part of users of digital devices, several factors should nonetheless alert healthcare professionals, leading them to monitor their development over time:
MARKET WATCH

Differences in habits with digital screens between now and 2 years ago

- **You spend more time** on digital devices now than 2 years ago: 59% (Total Yes), 89% (Yes, a lot more)
- **Whenever you use digital devices, you look at digital devices for a longer period of time** now than 2 years ago: 44% (Total Yes), 82% (Yes, a lot more)
- **You read text more often** on digital devices now than 2 years ago: 40% (Total Yes), 76% (Yes, a lot more)
- **You switch more often** from one digital device to another now than 2 years ago: 39% (Total Yes), 76% (Yes, a lot more)
- **You look at digital devices at close distances more often** now than 2 years ago: 32% (Total Yes), 67% (Yes, a lot more)
- **You look at digital devices at closer distances now** than 2 years ago: 31% (Total Yes), 65% (Yes, a lot more)

Base: All Respondents (n=4034)

**Question a:** If you had to compare the way you used digital devices 2 years ago to your actual habits, would you say that...

**FIG.2** | Intensification of multi-screen use

Level of discomfort experienced for each symptoms

- Tired eyes: 51% (Total Experienced the symptom), 74% (Total High/Medium level of discomfort)
- Neck and shoulder pain: 54% (Total Experienced the symptom), 70% (Total High/Medium level of discomfort)
- Back pain: 51% (Total Experienced the symptom), 66% (Total High/Medium level of discomfort)
- Headache: 39% (Total Experienced the symptom), 55% (Total High/Medium level of discomfort)
- Itching eyes: 29% (Total Experienced the symptom), 50% (Total High/Medium level of discomfort)
- Dry eyes: 31% (Total Experienced the symptom), 46% (Total High/Medium level of discomfort)
- Far blurred vision: 32% (Total Experienced the symptom), 46% (Total High/Medium level of discomfort)
- Difficulties to fall asleep: 35% (Total Experienced the symptom), 46% (Total High/Medium level of discomfort)
- Teary eyes: 25% (Total Experienced the symptom), 44% (Total High/Medium level of discomfort)
- Irritated eyes: 25% (Total Experienced the symptom), 41% (Total High/Medium level of discomfort)
- Close up blurred vision: 26% (Total Experienced the symptom), 40% (Total High/Medium level of discomfort)
- Red eyes: 21% (Total Experienced the symptom), 37% (Total High/Medium level of discomfort)
- Painful eyes: 21% (Total Experienced the symptom), 35% (Total High/Medium level of discomfort)
- Burning eyes: 20% (Total Experienced the symptom), 34% (Total High/Medium level of discomfort)
- Screen glare: 15% (Total Experienced the symptom), 34% (Total High/Medium level of discomfort)
- Dizziness: 16% (Total Experienced the symptom), 30% (Total High/Medium level of discomfort)

Base: All Respondents

**Question a:** Have you ever experienced these symptoms, even rarely?
**Question b:** How would you evaluate the level of discomfort when you experience these symptoms?

**FIG.3** | Body and visual discomfort linked to multiscreen uses (including difficulty falling asleep)
- There already seems to be a very strong link between intensity of screen use and the symptoms felt. In other terms, the longer and more frequently one uses digital devices, the more one is affected by ocular or physical symptoms. Small screens, especially those found on smartphones, tablets, or game consoles, seem to cause more problems for the eyes, due in particular to difficulty reading small type: people using these devices heavily (i.e. more than four hours a day) seem to feel that they have dry eyes more often than others (62% had already experienced this symptom, versus 46% for all users) or experience sore eyes more often (46% versus 35%). And as the use of digital devices continues to expand, it is likely that more and more people will face these symptoms in coming years.

- Moreover, more than half of those reporting one of these symptoms feel that their symptom(s) are worsening over time, and becoming increasingly troublesome.

- Users of digital devices also encounter the problem of blurred vision, when viewing them close-up (40%) or from afar (46%), which may be perceived as getting worse over time (31% for distance vision, and 29% for near vision).

- Despite these specific signs, few envisage spending less time viewing screens: over 40% of those surveyed state that they have simply not considered reducing the length of time of frequency they use their digital devices to relieve their symptoms, illustrating by this attitude their increasingly strong dependence on these everyday objects. Most of the time, users opt for quick, simple solutions, such as taking a break, changing position or looking away from the screen from time to time. It is also noteworthy that 60% have already tried to change the brightness of their screen, and that 40% wear dedicated eyewear during screen use.

**Everyone is concerned, particularly young people**

Since they use these devices for longer periods and more intensively than those over fifty, young people are the primary victims of damage related to digital device use, even before they become presbyopes, they now seem to suffer from a greater number of ocular and physical symptoms than their elders. Tired or sore eyes, headaches and blurred distance vision are felt far more frequently by those under forty year of age. These symptoms are also
accompanied by a greater awareness by those under forty of the link that may exist between the use of screens and visual discomfort.

Everyone is concerned by eye problems, including wearers of corrective lenses, and particularly contact lens wearers. A significant proportion of non-wearers are also affected: 61% of them have the impression that they must make more of an effort to see well when using digital devices (versus 66% of corrective lens wearers).

Finally, countries like Brazil and China, which are experiencing an unprecedented boom in the use of these new digital media, are also particularly exposed to this situation, due to their usage practices: in China, 45% of smartphone users say they use their phone over four hours a day (versus 29% for all countries), and for activities that are often more time-consuming than average (i.e. watching a film or a video, reading for long periods, etc.).

What are the potential risks of digital screen use and what solution(s) are available to prevent them?

Even though they are aware of being “addicted” to screen use, people still seem to be insensitive to the risks inherent in prolonged use of digital devices. For example, the danger to the brain of increased exposure to electromagnetic waves, supported by numerous scientific studies, is a topic that comes up regularly in the news without provoking much of a reaction from the public (in France, ANSES published reports in 2009 and 2013, that were widely reported in the press; and a law governing public exposure to electromagnetic waves was adopted on 29 January 2015).

Similarly, users of digital devices do not yet clearly perceive (or do not wish to perceive) the possible link between increased exposure to screens and a potential decline in their eye health. Regardless of the digital device used, those surveyed see the screen more as a source of eyestrain than as a potential danger for their eyes. For example, smartphones are considered by 27% as a device that could damage the eyes, while 39% consider instead that it is simply responsible for visual fatigue. Fig. 5.

Currently, sunlight and exposure to UV radiation are still considered the main risk for the eyes. As for blue light and its potential dangers, this remains an elusive concept for most people: only 47% consider spontaneously that they are familiar with the principle of blue light but, in fact, when it is explained to them, over half realize that they are not familiar with this phenomenon.

Awareness of the potential dangers of the intensive use of screens and the cumulative effect over time is more
MARKET WATCH

"The longer and more frequently one uses digital devices, the more one is affected by ocular or physical symptoms" accompanied by a greater awareness by those under forty of the link that may exist between the use of screens and visual health.

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Conclusion
With rapidly changing digital use practices, everyone is or will be concerned by the potential dangers represented by these screens. But increased awareness of the inherent risks is slow to develop; certainly, physical and ocular discomfort are increasingly felt by digital device users in their daily lives, but the long-term effects remain poorly understood. Healthcare professionals therefore have an important role to play in heightening people’s awareness and helping them protect themselves, in the face of this growing public health challenge.

KEY TAKEAWAYS

- The increasing use of digital devices is a transgenerational, global reality.
- 72% of respondents report that they use a combination of several different screen-based devices.
- The use of digital devices causes visual and physical discomfort (including difficulty falling asleep).
- Half of respondents consider their visual and physical symptoms bothersome.
- Half of respondents are bothered by strong screen brightness.
- Two out three people feel that they must make an additional visual effort when using screens.
- Three out of four people suffer from visual fatigue.
- Everyone is affected by this discomfort, particularly young people.
- 77% of users report that they are interested in purchasing dedicated eyewear to relieve this discomfort.
- Healthcare professionals have an important role to play in raising awareness and providing treatment.
III. POPULATIONS MOST AT RISK
Ocular phototoxicity in the mountains

Fototoxicidad ocular en la montaña

*The eye is born from light and for light* JW von Goethe

Although light is necessary for ocular physiology, notably for phototransduction, acute and chronic exposure can cause lesions to the eyeball.

The harmful effect of light has been suspected from antiquity; Socrates reported eye discomfort after watching eclipses. The consequences of light exposure on the retinal function were demonstrated experimentally in rats over 40 years ago, including at low intensity and over a long period of exposure. More recently, in vivo and in vitro models have demonstrated more specifically the role of blue light (BL) (380-480 nm) in the apoptosis of photoreceptors and of the cells of the retinal pigment epithelium. Light thus leads to photochemical reactions within ocular tissues. These require a chromophore, exposure time and a sufficient dose, releasing the free radicals involved in oxidative stress and the processes of eye ageing.

Ultraviolet rays and blue light which are of particular interest to us, belong to the vast range of electromagnetic waves. These are made up of photons, which are classified according to their wavelength with its own energy (inversely proportionate to their wavelength). We are familiar with UV rays particularly due to their action on the skin and the cornea (snow blindness) in our particular speciality. The ozone layer filters UV rays up to 290 nm, and the eye is therefore exposed to the remaining UVs, from 290 to 400 nm (UVB and UVA) and to the spectrum of visible light (which starts with blue light) in the absence of efficient protection. Intraocular transmission of the rays depends on their wavelength, but in fact UVs are mainly absorbed by the cornea and the crystalline. It is estimated that less than 2% of the initial UV dose reaches the retina in adult eyes, compared with 2 to 8% in children under the age of 10[1,2].

«El ojo nació por la luz y para la luz» JW von Goethe

Aunque la luz es necesaria para la fisiología ocular, especialmente para la fototransducción, una exposición aguda y crónica puede generar lesiones en la globo ocular.

Ya desde la Antigüedad se había sospechado el papel nocivo de la luz, Sócrates había mencionado una molestia ocular secundaria a raíz de la contemplación de los eclipses. Hace ya más de 40 años, en experimentos en ratones, se han demostrado las consecuencias de la exposición a la luz sobre la función retiniana, incluso a un bajo nivel de intensidad incrementando la duración de la exposición. Más recientemente, los modelos in vivo e in vitro han puesto de relieve, más particularmente, el papel de la luz azul (LB) (380-480 nm) en la apoptosis de los fotorreceptores y de las células del epitelio pigmentario de la retina[1]. La luz induce así reacciones fotoquímicas en los tejidos oculares. Estas necesitan un cromóforo, una cierta duración de la exposición así como una dosis suficiente para liberar radicales libres implicados en el estrés oxidativo y los procesos de envejecimiento ocular.

Los rayos ultravioleta y la luz azul, que nos interesa más particularmente, pertenecen al gran conjunto de las ondas electromagnéticas. Las ondas electromagnéticas están constituidas de fotones, clasificadas según su longitud de onda y contienen energía propia (inversamente proporcional a su longitud de onda). En nuestra especialidad, las radiaciones UV nos son familiares, particularmente por su acción en la piel y la córnea (oftalmia de la nieve). La capa de ozono filtra los UV hasta los 290nm, de esta manera, en ausencia de protección eficaz, el ojo queda expuesto al resto de los UV de los 290 a los 400 nm (UVB y UVA) y al espectro de la luz visible (que comienza con la luz azul). La transmisión intraocular de los rayos es función de...
Visible light (400 to 800 nm) provides us with the coloured sensation of our vision, whilst infrared light has mainly heat-related properties. The retina is exposed to the components of visible light due to their wavelengths, whence its potential danger. Sliney et al. estimate at 40% the fraction of blue light transmitted to the retina in adults aged 60 and still more in children, for whom 65% of blue light rays are transmitted.

Back in 1908, Hess discovered that the dose of cosmic rays increased with altitude during balloon travel. Thus, the dose of UVs received by the eye increases by 10% in levels of 1000m of altitude, by 20% on water, by 10% on sand and by 80% on snow. Mountain professionals are therefore a population who are overexposed to light (particularly UV and blue light) due to the combination of these elements.

Several large scale studies have been carried out amongst populations living in sunny plains (POLA, Sète, France[5,6]), Beaver Dam Eye study Wisconsin USA[7,8], Chesapeake Bay study, Australia[9]); these showed an increase in the prevalence of cataracts, notably cortical and, more controversially, of maculopathies [3,4,10,9].

To our knowledge, no study has been published on a population living at altitude and thus over-exposed. In our department we have carried out an original study on high mountain guides compared with a population living in the plains of the Lyon region (Etude enregistrée Eudract 2010-A00647-32, Promotor Essilor International, principal investigator Prof. Corinne Dot). This study highlights mainly the effects of the sun’s rays at altitude as well as under the more secondary conditions of the combined effects of the wind and low temperatures.

Study undertaken amongst high mountain guides in Chamonix[6]

Ninety-six high mountain guides (GHM) from the Chamonix valley aged over 50 and 90 control patients from the refraction department at the Desgenettes Hospital in Lyon, of comparable age, took part in this study.

A questionnaire was used to evaluate exposure at altitude (number and altitude of trips) and the means of protection used. Each of the patients was examined under dilatation by means of a clinical segment with retinal photography of the posterior pole. Statistical analyses used Student’s T test for the comparison of the 2 groups and a logistic regression to evaluate the risk factors. The results were as follows:

- Regarding surface pathologies, the mountain guides (GHM) presented statistically more dermatomalacia (28.1% compared with 4%, p<0.001), chronic blepharitis (52.1% compared with 10.2%, p<0.001) and abnormalities of the lachrymal points (33.3% compared with 4%, p<0.001). Their Break Up Time (BUT) is also statistically lower (4.55s compared with 7s, p<0.001). We also observed more pterygium (8.9% compared with 0%, p<0.001), pinguecula

su longitud de onda; sin embargo, los rayos UV quedan esencialmente absorbidos por la córnea y el cristalino. Efectivamente, se estima que menos del 2% la dosis de los UV iniciales alcanzan la retina en un ojo adulto, en contraste con el nivel del 2 al 8% en niños menores de 10 años.[7,8]

La luz visible (400 a 800nm) nos aporta la sensación de colores de nuestra visión mientras que los rayos infrarrojos poseen esencialmente propiedades calóricas. Por su parte, la retina está expuesta a los componentes de la luz visible debido a sus longitudes de onda, de ahí su peligro potencial. Sliney et al. estiman en un 40% la fracción de la luz azul que se transmite hacia la retina en los adultos de 60 años y ésta es aún mayor en los niños en los que más del 65% de los rayos de la luz azul se transmitiría.

En 1908, Hess descubrió, en el transcurso de vuelos en globo, que la dosis de radiaciones cósmicas aumenta con la altitud. De esta manera, la dosis de UV que recibe el ojo aumenta en un 10% por tramos de 1000m de altura, un 20% en el agua, un 10% en la arena y un 80% en la nieve. De esta manera, mediante la combinación de estos elementos, los profesionales de la montaña son un grupo de personas sobreexpuestas a la luz (especialmente a los UV y a la luz azul).

Se han realizado algunos estudios con grupos significativos entre los habitantes de planicies soleadas (POLA, Sète, France[5,6]) Beaver Dam Eye study Wisconsin USA[7,8] Chesapeake Bay study, Australia[9]). Los hallazgos de dichos estudios han puesto de relieve un aumento de la prevalencia de las cataratas corticales, en particular, y se discute si también favorece el desarrollo de las maculopatías[3,4,10,9].

En nuestro conocimiento, no se ha publicado ningún estudio sobre algún grupo de personas habitantes en gran altitud y sobreexpuestos. En nuestro departamento llevamos a cabo un estudio original sobre los guías de alta montaña comparándolos con una población que vive en una planicie de la región de Lyon (Estudio registrado en Eudract 2010-A00647-32, Promotor Essilor internacional, Investigador principal: Dr. Corinne Dot). Este estudio resalta principalmente los efectos de los rayos solares en altitud así como algunos aspectos más secundarios de los efectos combinados del viento y bajas temperaturas.

Estudio realizado sobre los guías de alta montaña de Chamonix[6]

Participaron en este estudio noventa y seis guías de alta montaña del valle de Chamonix mayores de 50 años de edad, así como 90 controles de edad comparable que acudieron a la consulta de refacción del Hospital Desgenettes en Lyon.

El cuestionario diseñado tenía como objetivo evaluar la exposición a la altitud (número y altitud de las excursiones) así como los medios de protección utilizados. Se examinó a cada uno de los pacientes bajo dilatación con un examen clínico del segmento anterior (clasificación LOC5, III, Lens Opacities Classification System III) completado por un análisis por cámara de shempflug (Oculyzer®, Alcon) y el segmento posterior asociado a una retinofotografía del polo posterior.
- Regarding the crystalline: the mountain guides had more abnormalities of the crystalline (42.4% compared with 16.2%, p<0.0001). They had mainly cortical opacities (30.8% compared with 10%, p<0.0001). The difference is also significant for cataract surgery (5.4% compared with 0%, p=0.02). The maximum average crystalline density measured by Oculyzer® was also greater in the mountain guides (22.5% compared with 20.2%, p=0.016). We also observed in the peripheral cortex of the guides round punctiform cortical micro-opacities in a significantly higher proportion (p=0.004) mostly located towards the nose.

- Regarding the macula, 30.2% of the mountain guides presented an abnormality of the macula area (including all abnormalities) compared with 18.9% in the control group (p<0.001). These abnormalities are mainly represented by drusen (28.7%) of a variety of sizes and numbers, and mainly seed-like.

- Significant risk factors identified are the high altitude (3000m to 5000m) and work in a snowy environment, a separate factor due to the scale of the reflection it generates.

- Protections used were photochromic lenses (OR=0.53 for crystalline opacities), the wearing of a visor (OR=0.37 for the crystalline, OR=0.4 for the macula) and the wearing of a ski mask (OR = 0.44 for blepharitis, OR = 0.5 for the crystalline, OR = 0.6 for arcus senilis).

Discussion

The mountain guides group presents more superficial pathologies, which are not described in literature as being linked to UV exposure, with the exception of pterygium. It is probable that this increase is due to a number of factors, combining UV action with weather conditions (cold and wind).

With regard to the crystalline, our results agree with the French POLA study and with those of the Chesapeake Bay study carried out amongst Australian fishermen and concerning the increased prevalence of crystalline cortical opacities. The crystalline would appear to behave like a real intraocular dosimeter of the UV rays received.

Fig. 2 Comparison of the prevalence of pathologies between the control group and the guides.

Fig. 3 Section showing anterior cortical micro-opacities using a Slit Lamp.
In terms of macular impact, results in literature are controversial. The POLA study does not find any difference in the population living in Sète. On the other hand the relative risk of showing signs of age-related maculopathy is 2.2 in the American Beaver Dam Eye study. The risk of developing AMD is also increased amongst Australian professional fishermen in Chesapeake Bay. In our study we also noted an increase in the prevalence of mainly seed-like drusen, which are a sign of macular ageing.

The low numbers in our population, along with the long-standing use by mountain guides of preventive equipment in the form of the wearing of sunglasses, certainly explains why we did not find more AMD, and underlines the relative efficiency of the means of protection used. However, the results of the questionnaire show that vigilance in terms of protection at medium mountain altitudes is lower, particularly when hiking and climbing. Yet exposure to UV rays is identical, whatever the weather, since UVs are not filtered by the clouds, whence insidious and chronic exposure, even at medium mountain altitudes.

Optimal ocular protection therefore involves the wearing of a visor with protective glasses: either sunglasses or photochromic lenses. This important data for professionals exposed to these rays (mountain and sea), should also be taken into consideration for children whose clear crystalline allows more rays to pass and for keen mountaineers and fishermen.

This data also underlines the importance of the latest technological progress made in terms of materials. Polycarbonate stops 100% of UVs (cut off at 385nm). For the visible spectrum, class 3 lenses halt 85% of visible rays and therefore allow 15% of rays through, to enable colour vision.

An interesting technological advance is the arrival of melamine-coated, class 3 brown sun lenses, which offer the “plus” of cutting out 100% of the start of blue light (cut out up to 425nm, preventing the absorption peak of ganglion cells at 480nm).

Our study underlines the protection offered by photochromic lenses. Photochromic lenses also exist mounted on curved frames, transiting from class 2 to class 4 shade, depending on outdoor conditions; these are also a good means of protection in the mountains.

Conclusion

Recent data confirms the harmful action of chronic sun exposure without protection. The increase in ocular surface pathologies, impact on the crystalline and impact on the macular, means that extraocular protection optimised by means of the new materials available should be advised, right from the youngest age.

This study on ocular phototoxicity in the mountains was the subject of a thesis for a Medical Doctorate, presented by H. El Chehab on 18th October 2011 in Lyon.

Conflicts of interest: Essilor International (promoter of the study)

Discusión

El grupo de guías desarrolla un mayor número de patologías de superficie que no han sido descritas como ligadas a una exposición a los rayos UV, con la excepción del pterigión. Es posible que este aumento sea multifactorial, a través de la combinación de la acción de los rayos UV y las condiciones climáticas (el frío el viento).

En cuanto al cristalino, nuestros resultados están en concordancia con el estudio francés POLA(4) así como con los obtenidos en el estudio Chesapeake Bay(5) en pescadores australianos sobre el aumento de la prevalencia de las opacidades corticales del cristalino. El cristalino parece comportarse como un verdadero dosímetro intraocular de los UV recibidos.

En cuanto a las afecciones maculares, los resultados en la literatura muestran una cierta controversia. En el estudio POLA no se encuentra ninguna diferencia con la población que vive en Sète. En cambio, en el estudio estadounidense Beaver Dam Eye Study, el riesgo relativo de presentar signos de maculopatía asociada a la edad es del 2,2. El riesgo de desarrollar una DMAE también aumenta entre los pescadores profesionales australianos de Chesapeake Bay. En nuestro estudio, también hemos observado un aumento de la prevalencia de drusas, principalmente miliares y que constituyen signos de envejecimiento macular.

La cantidad reducida de personas que constituyen nuestro grupo de estudio, así como la ya antigua tradición de prevención de los guías de alta montaña con el porte de protección solar seguramente explica por qué no se ha encontrado un mayor número de casos de DMAE y también pone de relieve una relativa eficacia de los medios de protección utilizados. No obstante, los resultados del cuestionario muestran que la vigilancia de la protección en media montaña es inferior, particularmente en la práctica de la escalada o senderismo. No obstante, la exposición a los rayos UV es idéntica cualquiera que sean las condiciones climáticas puesto que los rayos UV no son filtrados por las nubes, de ahí una exposición insidiosa y crónica incluso en media montaña.

La protección ocular óptima requiere el porte de una visera además de gafas protectoras: solares o fotocromáticas. Estos datos importantes sobre los profesionales expuestos (montaña o mar) también hay que tomarlos en consideración en el caso de los niños cuyo cristalino claro transmite más los rayos, así como los aficionados de montaña y pesca.

Estos datos subrayan también el interés de estos últimos avances tecnológicos sobre los materiales. El policarbonato bloquea el 100% de los UV (bloqueo a 385nm). En el espectro visible, las lentes de clase 3 bloquean el 85% de los rayos visibles y dejan pasar el 15% de los rayos para permitir una visión en colores.

Un avance tecnológico interesante es la llegada de las lentes melaminadas, gafas solares de categoría 3, con tinte marrón, que...
Acknowledgements

Marc Alexandre (Essilor International) for his work on ensuring that this original study was possible and could be undertaken in 2010, seventeen years after an initial study in 1993 covering a more limited number of guides.

Laboratoire Alcon, for the loan of the Oculyser® equipment, which permitted an original objective analysis of the crystalline.

Conclusión

Los datos recientes confirman el papel nocivo de la exposición solar crónica sin protección. El aumento de las patologías de superficie ocular, de la afección del cristalino y de la afección de la mácula, aboga por una protección extraocular optimizada acorde a los materiales que ahora están disponibles y desde la más temprana edad.

Este estudio de fototoxicidad ocular en la montaña ha sido objeto de una tesis Doctoral en Medicina, sostenida por H. El Chehab el 18 de octubre de 2011 en Lyon.

Conflicto de interés: Essilor international (promotor del estudio)

Agradecimientos

A Marc Alexandre (Essilor international) por la energía dedicada para que este estudio original fuera posible y realizable en el 2010, dieciséis años después de un estudio preliminar en 1993 en un número más limitado de guías.

Al laboratorio Alcon por el préstamo de material Oculyser® que ha permitido un análisis objetivo del cristalino inédito.

References - referencias


Ultraviolet damage to the cornea in the Tropics

Daños córneales por rayos ultravioleta en zonas tropicales

Summary

Ultraviolet radiation has been shown to cause harmful effects on the cornea, particularly in the tropics. This is further exacerbated by the depletion of the ozone layer. As UV-C is filtered by the ozone, acute photokeratitis is typically seen in eyes exposed to manmade implements such as during welding. Chronic exposure to UV-B can present with a plethora of corneal conditions, such as pterygium and pinguecula, climatic droplet keratopathy and ocular surface squamous neoplasia. Exposure of the cornea to UV-B during photorefractive keratectomy may predispose to the formation of subepithelial haze. It is therefore prudent to use personal protective devices to shield the eye from excessive UV radiation.

Ultraviolet (UV) radiation spectrum is classified by its wavelength: UV-A (315-380 nm), UV-B (280-315 nm), and UV-C (100-280 nm). While the ozone layer completely filters UV-C and 90% of UV-B from reaching the Earth’s surface, the remaining UV radiation is sufficient to cause damage to the eye, particularly so in the tropics where there is year-long exposure to strong sunlight. And this is further exacerbated by the losses of the stratospheric ozone of about 6% in the southern mid-latitudes and 4% in the northern mid-latitudes\(^1\). A 1% reduction in the ozone layer leads to an increase in radiation of 0.2% to 2% reaching the Earth’s surface.

The cornea absorbs most of the UV-B and all of the UV-C that reaches the eye. While the corneal epithelium and Bowman layer have significantly higher absorption coefficients than that of the stroma, the whole thickness of the corneal stroma absorbs 70-75% of the UV spectra shorter than 310 nm\(^2\).

The threshold for acute UV photokeratitis is found at a peak sensitivity of 270nm, which is only possible with manmade implements since the ozone layer blocks off UV-C. But it is possible to develop acute UV keratitis under natural sources such as solar eclipse burns\(^3\) and during skiing (commonly referred as “snow blindness”). Welders with acute photokeratitis may present with tearing, pain, photophobia, and is usually not apparent till several hours after exposure. It is akin to sunburn of the cornea and conjunctiva, though it is shown to be phototoxic rather than thermal injury to the corneal epithelium. Signs include superficial punctate keratopathy, conjunctival injection and chemosis.

Resumen

Se ha demostrado que la radiación ultravioleta tiene efectos nocivos en la córnea, especialmente en las zonas tropicales. Este fenómeno se ha acentuado con la desaparición de la capa de ozono. Dado que el ozono bloquea los rayos UV-C, la fotoqueratitis se observa más frecuentemente en ojos expuestos a herramientas fabricadas por el hombre como los aparatos de soldadura. En cambio, la exposición crónica a los rayos UV-B puede ocasionar un amplio abanico de trastornos de la córnea, como pterigión y pingüeula, queratopatía climática en gotas y neoplasia escamosa de superficie ocular (OSSN en inglés). La exposición de la córnea a los UV-B durante la queratectomía fotorrefractiva puede predisponer a la formación de opacidades subepiteliales (haze en inglés). Por lo tanto, es prudente utilizar dispositivos de protección personal para proteger al ojo de la radiación excesiva de los UV.

El espectro de radiación ultravioleta (UV) tiene diferentes clasificaciones según su longitud de onda: UV-A (315-380 nm), UV-B (280-315 nm), y UV-C (100-280 nm). Aunque la capa de ozono filtra completamente los UV-C y el 90% de los UV-B para que no alcancen la superficie de la tierra, la radiación de los UV restantes es suficiente para causar daños al ojo, especialmente en los trópicos donde la exposición a la luz solar es muy fuerte a lo largo del año. Esta exposición se acentúa con la desaparición del ozono estratosférico: 6% en las medias latitudes del sur y 4% en las medias latitudes del norte\(^1\). Una reducción del 1% en la capa de ozono conduce a un aumento de la radiación que llega a la superficie de la tierra de unos 0.2% a 2%.

La córnea absorbe la mayoría de los UV-B y todos los UV-C que llegan al ojo. Aunque el epitelio corneal y la capa de Bowman tienen coeficientes de absorción significativamente más elevados que el estroma, el espesor total del estroma corneal absorbe el 70-75% del espectro de UV inferiores a 310 nm\(^2\).

El umbral de fotoqueratitis aguda por UV encuentra su sensibilidad máxima en 270nm, lo cual sólo puede alcanzarse con herramientas fabricadas por el hombre puesto que la capa de ozono bloquea los UV-C. No obstante, es posible desarrollar queratitis por radiación UV aguda.
Climatic droplet keratopathy, also known as Labrador keratopathy, chronic actinic keratopathy, proteinaceous degeneration and keratinoid degeneration, is a spheroidal degeneration of the superficial cornea, found in areas of high UV exposure. A study of Chesapeake Bay watermen found a high odds ratio of 6.36 for average annual UV-B exposure in the upper quartile\(^5\). Histologically, the hyaline-like deposits are found in the Bowman’s layer and superficial stroma. The source of the deposits remains controversial. Fraunfelder\(^6\) believed that it is secreted by corneal and conjunctival fibroblasts, while others postulated that it is of plasma origin. Clinical findings are characterized by yellow, oily-appearing spherules in the subepithelium, within Bowman’s layer, or in the superficial corneal stroma (Fig.1). These spherules measure 0.1 to 0.4 mm, appearing at the limbus in the interpalpebral region in the early stages.

While there is strong association between UV-B exposure and squamous cell carcinoma of the eyelid\(^3\), the etiology and pathogenesis of ocular surface squamous neoplasia is multifactorial, including UV-B exposure, cigarette smoking, Human Papilloma Virus infection, exposure to petroleum derivatives and host susceptibility\(^5\). OSSN invariably involves the cornea at the sun-exposed interpalpebral region. Whether it is due to a greater propensity for malignant change in this zone, or environmental exposure remains unclear.

Excimer laser of different wavelengths can be produced with a combination of a noble gas and a halogen gas. The 193 nm excimer laser in the range of UV-C is utilized in laser refractive surgery such as photorefractive keratectomy (PRK) and laser in-situ keratomileusis (LASIK) for its precise etching abilities\(^9\). In vitro tests have shown a risk of carcinogenesis with the excimer laser, but its cell-damaging effects are less for lasers at 193 nm compared to the longer wavelengths. Furthermore, the short exposure of the cornea to the excimer laser mitigates this risk. The exposure of the stromal bed to

mediante exposición a los elementos naturales como las quemaduras causadas por los eclipses solares\(^3\) y mediante la práctica del esquí (comúnmente denominada “ceguera del esquiador”). Los soldadores con fotoqueratitis aguda pueden presentar signos como lagrimeo, dolor o fotofobia y habitualmente no se presentan hasta 4 horas después de la exposición. Es parecido a las quemaduras de la córnea y de la conjuntiva aunque parece ser fototóxico en vez de ser una herida térmica del epitelio corneal. Los signos incluyen queratopatía punctata superficial, inyección conjuntival y quemosis.

Se ha asociado la exposición solar crónica a múltiples trastornos de la superficie ocular como el pterigión, la pingüecula, queratopatía climática en gotas y la neoplasia escamosa de la superficie ocular (OSSN). Se ha observado que el pterigión ocurre comúnmente en los trópicos y toda una serie de estudios han demostrado su asociación con niveles elevados de UV-A y UV-B\(^4-5\). Sin embargo, el mecanismo mediante el cual la radiación por UV induce pterigión todavía queda por ser investigado.

La queratopatía climática en gotas, también conocida como queratopatía del Labrador, la queratopatía actínica, la degeneración proteinácea y la degeneración queratinoidal es una degeneración esferoideal de la córnea superficial que se observa en zonas de alta exposición a los UV. En un estudio realizado en marinos de la Bahía de Chesapeake, se encontró un coeficiente de alta probabilidad (odds ratio) de 6.36 de la media anual de exposición a los UV-B en el cuartil superior\(^5\).

Histológicamente, se encuentran depósitos hialinos en la capa de Bowman y el estroma superficial. La fuente de dichos depósitos sigue siendo objeto de controversia.

Fraunfelder\(^6\) pensaba que se trataba de una secreción de los fibroblastos corneales y conjuntivales mientras que otros postulaban que era más bien de origen plasmático. Los hallazgos clínicos han mostrado que éstos se caracterizan por estérulas amarillentas, de apariencia grasa subepiteliales, en la capa de Bowman o en el estroma corneal superficial (Fig.1). Dichas estérulas miden de 0.1 a 0.4 mm, apareciendo en la zona límbica de la región interpalpebral en las fases precoces.

Si bien existe una fuerte asociación entre la exposición a los UV-B y el carcinoma celular escamoso del párpado\(^5\), la etiología y patogénesis de la neoplasia escamosa de la superficie ocular es multifactorial, incluyendo la exposición a los UV-B, tabaquismo, infección por Virus de Papiloma Humano, exposición a derivados del petróleo y susceptibilidad del antígeno\(^5\). La neoplasia escamosa de la superficie ocular (OSSN) invariablemente incluye a la córnea en la zona interpalpebral expuesta al sol. Todavía no está claramente definido si se trata de una mayor proclividad de esta área a cambios malignos o si es una cuestión de exposición al entorno.

Se pueden utilizar láseres excimer de diferentes longitudes de onda con una combinación de un gas noble y un halógeno. El láser excimer de 193 nm en el rango de los UV-C se utiliza en la cirugía refractiva por láser como en la queratectomía fotorefractiva (PRK) y la queratomileusis in-situ por láser (LASIK) por sus capacidades precisas de grabado\(^9\). Las pruebas in vitro han mostrado un riesgo de carcinogénesis con el láser excimer pero los efectos nocivos en las células son menores en los láseres a 193 nm en comparación con las longitudes de onda más largas. Además, la breve exposición de la

Climatic droplet keratopathy, also known as ptérygium, pingüeula, climatic droplet keratopathy and ocular surface squamous neoplasia (OSSN). Pterygium commonly occurs in the tropics, and multiple studies have shown an association with increased levels of UV-A and UV-B\(^4-5\). However, the mechanism by which UV radiation induces pterygium remains to be investigated.

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Se ha asociado la exposición solar crónica a múltiples trastornos de la superficie ocular como el pterigión, la pingüeula, queratopatía climática en gotas y la neoplasia escamosa de la superficie ocular (OSSN). Se ha observado que el pterigión ocurre comúnmente en los trópicos y toda una serie de estudios han demostrado su asociación con niveles elevados de UV-A y UV-B\(^4-5\). Sin embargo, el mecanismo mediante el cual la radiación por UV induce pterigión todavía queda por ser investigado.

La queratopatía climática en gotas, también conocida como queratopatía del Labrador, la queratopatía actínica, la degeneración proteinácea y la degeneración queratinoidal es una degeneración esferoideal de la córnea superficial que se observa en zonas de alta exposición a los UV. En un estudio realizado en marinos de la Bahía de Chesapeake, se encontró un coeficiente de alta probabilidad (odds ratio) de 6.36 de la media anual de exposición a los UV-B en el cuartil superior\(^5\).

Histológicamente, se encuentran depósitos hialinos en la capa de Bowman y el estroma superficial. La fuente de dichos depósitos sigue siendo objeto de controversia.

Fraunfelder\(^6\) pensaba que se trataba de una secreción de los fibroblastos corneales y conjuntivales mientras que otros postulaban que era más bien de origen plasmático. Los hallazgos clínicos han mostrado que éstos se caracterizan por estérulas amarillentas, de apariencia grasa subepiteliales, en la capa de Bowman o en el estroma corneal superficial (Fig.1). Dichas estérulas miden de 0.1 a 0.4 mm, apareciendo en la zona límbica de la región interpalpebral en las fases precoces.

Si bien existe una fuerte asociación entre la exposición a los UV-B y el carcinoma celular escamoso del párpado\(^5\), la etiología y patogénesis de la neoplasia escamosa de la superficie ocular es multifactorial, incluyendo la exposición a los UV-B, tabaquismo, infección por Virus de Papiloma Humano, exposición a derivados del petróleo y susceptibilidad del antígeno\(^5\). La neoplasia escamosa de la superficie ocular (OSSN) invariablemente incluye a la córnea en la zona interpalpebral expuesta al sol. Todavía no está claramente definido si se trata de una mayor proclividad de esta área a cambios malignos o si es una cuestión de exposición al entorno.

Se pueden utilizar láseres excimer de diferentes longitudes de onda con una combinación de un gas noble y un halógeno. El láser excimer de 193 nm en el rango de los UV-C se utiliza en la cirugía refractiva por láser como en la queratectomía fotorefractiva (PRK) y la queratomileusis in-situ por láser (LASIK) por sus capacidades precisas de grabado\(^9\). Las pruebas in vitro han mostrado un riesgo de carcinogénesis con el láser excimer pero los efectos nocivos en las células son menores en los láseres a 193 nm en comparación con las longitudes de onda más largas. Además, la breve exposición de la
UV-B during PRK may cause prolonged stromal healing and subepithelial haze formation\(^{[10]}\). It has been suggested that the lower incidence of haze seen in laser-assisted subepithelial keratectomy (LASEK) may be due to less UV radiation causing cellular damage to the corneal stroma with the near intact epithelium\(^{[2]}\).

UV radiation has been implicated in the pathogenesis of multiple corneal disorders. Although further studies need to be done to ascertain the causal effect on these conditions, there is sufficient data to suggest such an association. With the depleting ozone layer, there is an increasing exposure of UV radiation, especially in the tropics. And personal protective devices such as hats and sunglasses, and life style changes can help to minimize exposure of UV radiation to the eye.

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**references- referencias**


THE INFANT’S VISION AND LIGHT – THE ROLE OF PREVENTION IN PRESERVING VISUAL CAPACITY

The eyes of infants are permeable to both ultraviolet radiation and blue light, and extremely sensitive to glare. Although light is essential for the proper development of visual function in children, surveillance and protection are particularly important in the first months of life. Moreover, medical surveillance and early screening help prevent risks associated with possible anomalies of the visual system.

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François Vital-Durand was Research Director at INSERM and the Ecole Pratique des Hautes Études, Scientific Attaché at Lyon-Sud Hospital and Associate Professor at the Montreal School of Optometry. He holds a Docteur ès Sciences degree and bachelor’s degree in psychology. He has taught at numerous universities in France and abroad. He researched the coordination between the eye and the brain in the kitten (at MIT, Boston) and the development of visual function in the newborn monkey (at Oxford). He founded the “Bébé Vision” clinic in Lyon, France, which accepts children as patients from the early months of life. He chairs the Lyon Committee of the Valentin Haüy Association serving the blind and visually impaired.

KEYWORDS
UV, blue light, prevention, protection, screening, surveillance, diagnosis, infant, baby, child

The newborn child can see from the moment of birth, and even before. When we laterally illuminate a pregnant woman’s womb around the sixth month of pregnancy, we can see under ultrasound that the foetus turns its head away from the light source. This is also the case after birth. If newborns quite often keep their eyes closed, it may be because they are asleep. But if they are awakened in a low-light environment, they will look at us. This enables us to measure their acuity, which is about 1/20. Depending on their complexion (very light or more pigmented), they are dazzled by light up to the age of about six months. For their comfort, strong light should be avoided. At the age of six months, their acuity reaches 2/10, and then 4/10 at one year and 10/10 at about five years of age, remembering that in preverbal children we measure detection acuity with acuity cards and then the more demanding morphoscopic acuity. The field of vision is complete at one year but the child needs to learn to use it, a process that will continue more or less successfully throughout life, depending on motivation. This is evidenced by the large proportion of motorists who do not use the rear-view mirror.

Colour and contrast vision is good at three months, but will continue to improve until adolescence. Stereoscopic vision appears at four months and rapidly becomes excellent. Focusing becomes precise at about eight months with the development of the fovea in which the cones are gradually concentrated, enabling a reliable orthoptic examination. Ocular motor control, including saccades and pursuit, is precise at one year, although latency or reaction time is characterized by a certain slowness up to the age of about ten. This relative slowness facilitates examination.

The role of light
Light plays a fundamental role in this visual performance. Numerous visual deprivation experiments conducted in baby monkeys and kittens show attrition of visual pathways due to vision deprivation, making it possible to define a “deprivation-sensitive period”. The existence of this sensitive period in humans is now widely recognized. A baby presenting early lens opacity should be operated on during the first months of life to prevent severe amblyopia. Fitting aphakic infants with contact lenses allows them to acquire normal vision. Similarly,
wandering attention and obesity due to lack of exercise and meals on the run.\textsuperscript{5,6} The two studies agree on recommending limited use. One may only wonder why no impact on the visual system was identified (or it was possibly ignored) in either report.

**Lighting levels**

The introduction of fluorescent lighting, driven by cost concerns and possibly comfort concerns, has significantly impacted luminous power in indoor environments. School children must receive 400 lux on their desks. But one wonders if is this for the teacher’s comfort or the children’s. Sensitivity to light develops rapidly in children, reaching its adult level in adolescence. It then gradually decreases with age. What teenager has not been reprimanded for reading in semi-darkness? But we forget that by age 35, we have already lost half of our sensitivity. “You’ll ruin your eyes… I know dad, speak for yourself.” With the introduction of fluorescent tubes in the 60s along with improvements in lighting and its use for longer periods, concern has spread about possible adverse effects that have not proven justified. So we use lighting generously. Night lights in children’s rooms calm their parents’ anxiety. Studies in this field are fraught with methodological problems. How do we isolate the factors responsible for the increase in the incidence of myopia or other pathologies in a rapidly changing world? Should we blame it on the increase in close work? That remains to be proven. Nutritional changes? Perhaps. On the other hand, the effect of relative confinement on the development of myopia has been clearly demonstrated in urban areas in Asia, among people living in confined, rather than open spaces. Lighting quality and quantity become important for comfort with age, and particularly with advanced age.

**The precautionary principle**

This term drawn from the fields of theology and law has been increasingly used in the medical field in recent times. It consists in establishing a practice on the basis of a body of knowledge, or even presumptions. It must be weighed against the constraints imposed by the practice, by estimating the benefit-cost ratio. A concern for optimizing living conditions, coupled with longer life expectations – one in two baby girls born today will live to be 100 – accounts for the widespread implementation of medical and health measures to reduce risk exposure. In the field we are dealing with, what constitutes a reasonable attitude?

**Sun protection**

One finds spectacles with flat coloured lenses as early as the 18th century, known as “conserves”, which were meant to shield the eyes from glare and protect vision (see the 1759 Richelet and 1902 Larousse dictionaries), but wide-brimmed hats and more recently caps have also come into wide use. Climbers have long worn protective sun goggles. In both cases, the aim was to shield the wearer’s eyes from glare and improve comfort. Sunglasses for babies are a recent development. They have a wide bridge, often featuring a non-slip design and wide temples that provide side protection. But most importantly, they have a wraparound design that covers the eyebrows. In the 1980s, I saw an albino child at the Bébé Vision clinic whose parents lived at a high elevation in the Alps. The optician prescribed the glasses shown in Figure 1. The side shields for adults were supplied by a manufacturer’s representative. A model for babies...
was not yet available. But times have long since changed. Opticians have developed a wide range of wraparound performance products, including side protection. Today, babies are well protected, if only for comfort reasons (Fig. 2, Fig. 3). But I was told by certain ophthalmologists that sun protection should only be resorted to in extreme situations in order to allow children’s retinas to develop defence mechanisms to prevent them from becoming dependent on glasses!

New parental attitudes
Parental behaviour has evolved considerably, and in a positive direction as regards protective gear for children. The increased average age of parents at the birth of their first child – 30 for women and even older for men – as well as the choice of family planning are factors conducive to a higher level of concern in the care of children. Just consider how often parents run to the doctor at the slightest concern. As for protective gear, parents are proud to show off their babies with sunglasses, helmets and knee pads on their toy scooters. This is quite commendable. Such prudent behaviour has given rise to investments by the protective equipment sectors, including the optical sector, in response to the emerging needs of children and echoing current information provided to paediatricians, general practitioners and, of course, ophthalmologists and opticians.

Screening, surveillance and diagnosis
The founding of the first Bébé Vision clinic in 1982 contributed to raising awareness in professional circles. The publicity given this initiative, devoted to the search for visual anomalies of all kinds, has raised awareness of the visual capabilities of the preverbal child. Infants see better than previously thought, and their vision deserves to be protected. The concept of a “sensitive period” led to recommending a first routine exam at the age of nine months. At this age, the examination is easy, and the child is cooperative and follows the treatment. Opposition begins to appear at approximately 12 months. Once an indication has been identified, whether it is due to heredity, prematurity or an apparent eye disorder – often an epicanthic fold – the medical profession recommends a check-up with a specialist (ophthalmologist or orthoptist). This practice has significantly reduced the number of surgical procedures for strabismus, since most of them can be avoided by early correction of refractive errors. The discovery of amblyopia during the health check performed at the entrance to the first year of primary school (at age 5) has become much less frequent. Consequently, treatment for the most serious ophthalmologic pathologies is provided earlier and remarkable progress has been made in therapeutic regimens.

Determining best practice: screening, surveillance or protection?
Routine screening of all children would be costly and unproductive, since it would inevitably be cursory. Nevertheless, in the absence of accurate statistics, it is estimated that nearly 15% of infants require follow-up, and more than half of these have a significant refractive error or a more serious disorder. Surveillance consists in referral to a specialist as soon as an indication or risk factor
“Although certain therapeutic ratios still need to be measured, all evidence indicates that protecting the eyes against cumulative sun exposure is the responsible attitude.”

has been identified. This is the current practice in France and it is paying off. It is through these visits, but also during visits to the paediatrician or general practitioner, that parents raise the issue of sun protection. Opticians have also contributed to raising parents’ awareness. The eyes should be protected from the sun for reasons of visual health and comfort from a very early age, and this practice must become routine. There are no contraindications. Although certain therapeutic ratios still need to be measured, all evidence indicates that protecting the eyes against cumulative sun exposure is the responsible attitude.

Conclusion
It is somewhat delicate to propose a hierarchy of factors that have contributed to the increasing acceptance of eye sun protection for infants and children. Much remains to be done, however, to encourage widespread acceptance of the need for children to wear protective eyewear. The dissemination of information on the dangers of exposure to high-energy visible light – particularly blue light and UV radiation – is gaining momentum, spurred on by the introduction of new lighting solutions. But better knowledge about vision in children, coupled with low birth rates, is focusing parents’ attention on their limited number of offspring, leading to a more protective attitude in their regard. Increased life expectancies are also making everyone more aware of the difficulties faced by the elderly as their sight begins to fail, irrespective of the origin of the pathology. Eyewear manufacturers have made a particular effort to provide appropriate solutions at a reasonable cost. Such products are even found in sporting goods stores.

Finally, the medical and paramedical professions have become aware of the need to protect the vision of infants and provide more comfort for this little toddler who is not yet able to express his or her discomfort. Protective eyewear for infants and children undoubtedly has a bright future.

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IV. HOW TO PREVENT
1. EXPERTS AND ECP INITIATIVES
Ultraviolet Radiation and the Eye: Complete Protection Requires Blocking Both Transmission and Backside Reflection

New research shows that effective protection from the serious hazards of ultraviolet radiation (UVR) requires that clear, photochromic, and tinted/polarized lenses protect wearers from both transmitted and reflected UVR.

Chronic exposure to the ultraviolet radiation in sunlight has been implicated in a number of serious ocular diseases, including pterygium, cataract, and climatic droplet keratopathy; and recent research has uncovered new, unexpected risks to the eyes. Unfortunately, the public remains dangerously under-informed about the nature and degree of this risk as well as the circumstances in which eye protection is most necessary.

Unexpected Risks

Recent research has shown that the time of maximum risk for UVR damage to the eyes is very different from the time of maximum risk to the skin. Risk to the skin is greatest when the sun is highest in the sky—ie, at solar noon and on the summer solstice (June 21st).

But because the eyes are deep set in the orbit, they are partially protected when the sun is high in the sky; so direct ocular UVR exposure is greatest when the sun is somewhat lower in the sky. For spring, summer, and fall, maximum ocular UVR exposure occurs between 8:00am and 10:00am, and between 2:00pm and 4:00pm. These, however, are not the times that people are most likely to wear sunglasses.

Side and Back Exposure

Even when the sun is high in the sky, the eye is exposed to a significant amount of UVR that comes from clouds or reflected by surrounding surfaces (Figure 1). This incidence index for skin care products, it measures the degree of protection provided by a lens. Unlike transmission data alone, however, the E-SPF measures total protection by integrating reflected UVR data with transmission data (see box).

What Patients Need

Knowing what we now do about sources of UVR exposure, it becomes apparent that for everyday protection, clear lenses and sunglasses must offer UVR blocking of both transmission and reflection. To address this need, a new generation of Crizal® antireflective lenses (launching in 2012) has been engineered to virtually eliminate UVR reflection from the backside, for a lens that truly maximizes UVR protection.

REFERENCES
Let’s start with the eye you are talking about. It all depends on the segment of the Points de Vue:

People exposed short-term to high-intensity solar radiation are conjunctival conditions usually located in the area of dust more readily develop pinguecula or pterygium, which treatment. It heals in three to four days with local vitamin er’s flash”. It heals in three to four days with local vitamin among welders, it is commonly called “arc eye” or “welder’s flash”. It heals in three to four days with local vitamin pain, photophobia and tearing. In the work environment, blindness”. This condition, clinically known as acute photic retinal injury caused by staring at an eclipse. UV retina is concerned, virtually all practi-

Dr. Sylvie Berthemy describes the most common clinical cases and identifies the most vulnerable population groups for Points de Vue. She also stresses the importance of including prevention in her clinical and medical practice.

Ultraviolet radiation, among other aetiological factors, is implicated in several ocular pathologies. Dr. Sylvie Berthemy describes the most common clinical cases and identifies the most vulnerable population groups for Points de Vue. She also stresses the importance of including prevention in her clinical and medical practice.

Dr. Sylvie Berthemy: It all depends on the segment of the eye you are talking about. Let’s start with the adnexa. Almost everyone has had eyelid erythema (sunburn) which can lead to the formation of an actinic keratosis. We could also mention UV radiation’s role as an aggravating risk factor in basal or squamous cell carcinoma or melanoma.

People exposed short-term to high-intensity solar radiation without protection may get what is known as “snow blindness”. This condition, clinically known as acute photokeratitis and common in ski areas, is accompanied by pain, photophobia and tearing. In the work environment, among welders, it is commonly called “arc eye” or “welder’s flash”. It heals in three to four days with local vitamin treatment.

In the long term, patients exposed to severe weather and dust more readily develop pinguecula or pterygium, which are conjunctival conditions usually located in the area of the medial palpebral fissure where the tissue is least protected by the eyelids. And we may encounter corneal degeneration or actinic or climatic droplet keratopathy, also called Bietti dystrophy, Labrador keratopathy (which affects 14% of the Inuit), elastic dystrophy, proteinaceous corneal degeneration or spheroidal corneal degeneration. When examined with a slit lamp, it resembles band shaped keratitis although, histologically, it is not the same. Despite the fact that ultraviolet radiation exposure appears to be the major aetiological factor, evidence of genetic origin has been demonstrated.

Take the iris next. Melanoma is increasing in frequency (approximately 6.5/10 million). Three-quarters of cases develop on the bottom part of light-coloured irises, and UV exposure may be a contributing factor. However, the link has not been definitely established.

As for the crystalline lens, the POLA study (evaluating age-related ocular pathologies) conducted on 2,600 inhabitants of Sète, France, showed that cataracts are three times more frequent and appear five to ten years earlier in people exposed to solar radiation (e.g. fishermen, guides, construction workers, etc.).

And as far as the retina is concerned, virtually all practitioners have been consulted by patients suffering from photic retinal injury caused by staring at an eclipse. UV exposure could also be a risk factor in the aetiology of AMD (Age-Related Macular Degeneration).

**KEYWORDS**

- eyelid erythema
- actinic keratosis
- carcinoma
- melanoma
- acute photokeratitis
- pinguecula
- pterygium
- keratopathy
- UV
- ultraviolet radiation
- cataract
- AMD
- tipofuscin
- melanin
- prevention
- Crizal® Prevencia®
In practice, what are the most frequent clinical cases of these diseases?

Pinguecula-type conjunctival lesions, UV keratitis and cataracts.

What groups of patients are particularly at risk?

Children, because their pupils are larger and their crystalline lenses more transparent; patients with a family history of retinal degeneration: too many patients still go outdoors without the protection of specific filtering lenses; patients with fair complexions and those who tend to be photophobic (with hypo-pigmented irises and choroids); people who work outside: gardeners, construction workers, farmers, fishermen, pilots, tour guides, etc.; those in contact with a source of radiation and heat: welders, glassmakers, users of UV therapy and researchers who work in contact with LEDs (Light Emitting Diodes) – not to mention the length of time people spend in front of computer screens or other devices; those who have had cataract surgery, although implants increasingly have UV protective filters; people with hypermetropia, whose convex lens acts as a magnifying glass, concentrating rays on the retina; and the elderly, who have developed lipofuscin, a pigment found in the RPE (Retinal Pigment Epithelium), which is made up of molecular residue. Lipofuscin increases with age and is responsible for the photoreactivity of RPE, resulting in the production of free radicals that promote AMD.

In the area of phototoxicity, are there any similarities between the eye and the skin?

Yes, they are subject to the same aging factors, both through the Joule effect – more prosaically known as heat – which can burn cells (causing erythema and keratinization) and harm the retinal pigment epithelium, for example, and through the photochemical effect, which is responsible for producing free radicals by breaking down cellular membranes, denaturing proteins or even attacking the nucleus. We know, for example that melanin (a pigment found in skin, hair and eyes) absorbs the $\epsilon$ (epsilon) of wavelengths ranging from 300–700 nm (nanometres) and curbs harmful photochemical reactions by trapping unstable particles generated by these reactions which would otherwise cause the accumulation of retinal cellular debris, thus slowing down premature aging of the retina. But our stock of melanin decreases with age.

In your opinion, at what age should we start talking about prevention?

As early as possible! We need to educate parents of young children about the risks involved and their greater vulnerability. Asking patients about their professional and leisure-time activities – a practice that is all too often neglected – is a natural lead-in to prevention counselling. We also need to take into account pathologies that weaken the eyes, such as diabetes (which affects the retina), glaucoma (daily eye drops: the conjunctiva and cornea) and so on.
What precautionary principles, recommendations and/or solutions should we prescribe to patients?

We need to advise them to protect their eyes by wearing headgear with a visor; to wear filtering glasses, or specific protective eyewear designed for the workplace; and to consult their ophthalmologist on a regular basis if they are exposed to radiation on a regular basis.

In families with a history of retinopathy in the broadest sense, we can recommend and prescribe transparent filtering lenses (Crizal® Prevencia®), and/or they should take advantage of a corrective lens prescription to add a filter. Depending on one’s own convictions and the patient’s sensitivity, we can extend this protection to everyone. Risk prevention is an integral part our mission as healthcare providers. Our counselling should also include diet and lifestyle recommendations for placing limits on tobacco and alcohol use, thus reducing oxidative stress and cell apoptosis.

Lastly, by working with opticians, we can adjust our recommendations to fit the specific needs of various types of patients.

In the coming years, what impact might preventive clinical practice (and the role of the ophthalmologist) have on the frequency of eye problems?

One hopes that preventive clinical practice – which, I repeat, is an integral part of our role as medical doctors – will impact eye problems by decreasing their frequency!

Interviewed by Annie Rodriguez

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**DEMO**

Dr. Sylvie Berthemy
Ophthalmologist

Ophthalmologist, court expert, hospital practitioner.

Postgraduate degree in Ophthalmological Genetics.

President of the Société de Contactologie des Alpes.

Head of the department of genetic diseases of the retina and optic nerve at Grenoble University Hospital.

CEA consultant, Institut Laue Langevin, ESRF, MBL, in school and sports medicine.

Lecturer at Joseph Fourier University in ophthalmological risk prevention for safety engineers and occupational physicians.

Numerous publications in ophthalmic journals dealing with preventive and occupational ophthalmic medicine (white rooms, LEDs, etc.).

Numerous articles on ophthalmology in mainstream magazines.

Participation in a number of reports: 2009 SFO and 2001 and 2005 SFOALC, coordinator of the 2013 report on myopia and contact lenses.

Author of five films: one on the tear film, three related to the use of lenses in children and one on myopia.

Speaker at many national and international conferences.

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**KEY TAKEAWAYS**

Those most vulnerable to the chronic effects of light exposure are:

- Children
- The elderly
- People with a family history of eye disease
- People with photophobia
- People who have had cataract surgery
- People with hypermetropia
- People who work outdoors
- People exposed to sources of radiation and heat
- People in prolonged contact with LEDs
- People with fair complexions
PREVENTION OF OCULAR PATHOLOGIES IN OPHTHALMOLOGY

In ophthalmology, the prevention of ocular diseases is gaining increasing prominence. Educational programmes, screening campaigns, early medical detection along with protective eyewear can all together reduce the incidence of ocular alterations and limit their social and financial implications. Eye phototoxicity (due to UV and blue-violet light) is one of modifiable risk factors and, as such, can be reduced by a photo-protective eyewear.

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Teacher of the ophthalmology specialization course of Sociedade Brasileira de Oftalmologia.

Head of the glaucoma department of Hospital Federal de Bonsucesso, Rio de Janeiro.

Currently chairman of the Sociedade Brasileira de Oftalmo(a)logia (S.B.O.).

There is a saying in Portuguese that goes: melhor prevenir que remediar, which means “prevention is better than cure”. Originating from the Latin praeventire (prae = “before”, venire = “to come”), “prevention” literally means “to anticipate, to perceive in advance”. In medicine, the great challenge of the public health programmes is precisely to prevent diseases or to diagnose them as early as possible. With the ageing of the world population, it is vital to create programmes for the prevention of Chronic Non-communicable Diseases (NCDs), responsible for 63% of deaths in 2008. The majority of deaths from NCDs are attributed to diseases of the circulatory system, cancer, diabetes and chronic respiratory diseases.

The principal causes of those diseases include modifiable risk factors such as smoking, harmful alcohol consumption, lack of physical activity and inadequate diet. Therefore, programmes for the prevention of these diseases must focus their actions on these aspects. In addition to the scientific aspects, prevention and early diagnosis promote better economic-financial indicators in health budgets, with less expenditure and better use of resources.

Ophthalmology and prevention programmes

In ophthalmology, the prevention of some diseases is gaining increasing prominence. An ophthalmological test carried out in pre-school children, in adults at around the age of 40 and in older people aged over 60, is capable of preventing changes such as refractive error amblyopia, diabetic retinopathy, glaucomatous optic neuropathy and age-related macular degeneration, amongst others.

We know that the cost of treating glaucoma is much higher than investments to prevent it. The increase in cases of blindness, with its social and financial implications, shows that the right thing to do is to adopt major screening campaigns for early detection of suspected cases.

Educational programmes in terms of control of cardio-circulatory diseases would greatly reduce cases of retinal vascular occlusion, a major cause of diminished visual acuity in adults. Several authors have already demonstrated the advantages of diabetic retinopathy control in telemedicine, with a reduction in the incidence of reduced visual acuity from diabetes.

The World Health Organisation re-
cently launched an Ocular Health and Prevention of Blindness programme, one of the most important chapters of which is cataract surgery.5

Ocular phototoxicity

The biggest cause of avoidable blindness throughout the world, cataract is perhaps one of the major examples of ocular alterations due to phototoxicity. (Fig. 1) It is already a well-known fact that the earliest appearance of presbyopia occurs in geographic regions with the highest incidence of UV rays. In Brazil, the age at which it appears in the north of the country may be up to 5 years lower, with patients presenting symptoms at age 38, whereas in the south this occurs at around 43 years. By clinical analogy, if UV rays are able to alter the flexibility of the lens and modify its accommodation capacity, continuity of that action would lead to the degeneration of its fibres and the onset of cataract. Continuing its intra-ocular pathway, part of the solar radiation reaches the retina and may be deleterious to the retinal tissue, causing age-related macular degeneration (AMD). The incidence of pterygium is also known to be higher in populations with greater daily exposure to sunlight.6,7,8 (Fig. 2) Another example of ocular phototoxicity is actinic keratitis in addition to peri-ocular cutaneous lesions.

Importance of ocular protection

All this clinical evidence has created awareness of the need for ocular protection against UV rays. Taking into account the fact that a clear eye lens lets more radiation through than a lens that is beginning to form a cataract, protection against UV rays is advisable from childhood. Long-term longitudinal population studies would be necessary to enable us to judge the reduction in ocular diseases caused by solar radiation in a population that used protective glasses from childhood, compared to a population without protection. But it is not only UV rays that are dangerous for the eyes. Recent studies have shown that blue light also has harmful effects on the retina. Known for its importance in relation to chronobiology, the blue light of the visible spectrum has a part of wavelength range that is harmful for the cells of the retina; the other part is beneficial to cognitive and chronobiology functions. This shows us that the concept of “selective” ocular protection against solar radiation is a reality that needs to be observed. The use of ophthalmic lenses with selective photo-protective features is a major advance in ophthalmology. •

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PUTTING THE MEDICINE IN THE LENSES: THE IMPORTANCE OF BLOCKING ULTRAVIOLET RADIATION AND BLUE LIGHT

Advancements in spectacle lens technology have given eyecare providers the ability to blend medical and refractive eyecare in our dispensaries. Lenses can do more than just provide amazing vision, they can be used to prevent or delay the onset of certain ocular disease states. Proper use of Crizal® UV, Crizal® Prevencia® and Xperio® UV will allow us to exceed our patient’s expectations by not only providing superior vision now, but by simply wearing lenses, protect their visual system for years to come.

My passion for medical optometry
When I think back on my fond memories of optometry school I can still remember the anticipation and excitement that came when it was time to grab that patient chart and get to work. The level of excitement was even more pronounced if the patient happened to be a medical case instead of just a boring refractive patient. So was born the great divide in my mind with my chosen profession, medical optometry vs. refractive optometry. As I now enter my tenth year of private practice I know my days will be filled with both medical and refractive cases and that is one of the many things that I believe makes the optometric profession a great one. Now that I have the chance to go lecture to many schools and colleges of optometry I stress to future optometrists that the medical aspect is exciting and challenging but not to forget about that all-important refractive part. The bulk of my day in private practice is filled with patients wanting to see as well as possible, which usually ends up with a refractive solution.

Prevention now is part of the discussion with patients
The technology wave in optometry seems to be ever-changing and it continues to improve the care that I can deliver to my patients. Thanks to companies like Essilor that understand the importance of re-investing in research and development, I now have optical products that intertwine medical and refractive optometry. Daily I am having conversations with my patients’ that discuss glasses and ocular disease in the same breath. These cutting-edge technological lens advancements are one reason why I have chosen to utilize Essilor products in my practice. These products are very exciting to me as an optometric physician because now instead of using products to simply just solve problems, I can use these products to actually prevent and delay the onset of certain conditions. As many other medical professions are having conversations with patients about prevention, I feel it is imperative that we have those same conversations. Due to medical advancements, patients are living longer and as we age, our natural
Light is both beneficial and harmful
How then as eyecare providers do we have the ability to prevent damage to the visual system? To understand that, it is important to remember what gives us sight: light, which is part of the electromagnetic spectrum (see Figure 1). While most of the electromagnetic spectrum is beneficial, parts of it are not, mainly UV radiation and high-energy blue light. We all know UV radiation is bad for the skin, but we often forget that it also damages the visual system as well. Only 2% of UV radiation reaches the retina, thanks to the cornea and crystalline lens. Since those structures absorb so much UV radiation, that is where the damage will occur, mainly in the form of cataract formation, pterygium formation, and some studies suggest dry eye issues. While these issues are many times not sight-threatening and fairly fixable, patients do not want to go through surgery or start dry eye therapy early in life. By educating patients that products exist that not only provide superior vision but limit UV exposure and may prevent ocular disease onset, we are addressing the medical need for proper eyewear.

The eye treats visible light differently than UV radiation. The visual system is adapted to focus visible light onto the retina, which gives us the ability to see. But not all visible light is good for the visual system. High-energy blue light, which sits next to UV radiation in the electromagnetic spectrum, causes damage to the eye, specifically the cone photoreceptors.

To understand how this occurs, we need to remember the chemical reactions that occur during the visual pathway (see Figure 2). We can see that in both the photoreceptor outer segment and RPE, blue light can cause oxidative stress. As we age, and based on our genetics, our antioxidant defense mechanisms decrease. We can’t change our genetics, we can take vitamins to help those defense mechanisms, but what about reducing our exposure to damaging blue light?

Dramatic increase in blue light exposure
I believe that since blue light exposure is cumulative, just like UV radiation, if we can limit exposure, we can prevent disease progression. To limit exposure we must know the sources of blue light. First is the sun. The blue light portion of daylight varies between 25% and 30%. Remember the sun also exposes us to UV radiation in addition to visible light. Blue light also comes from artificial light sources, which have been on a dramatic increase over the past few years. Compact fluorescent bulbs contain 25% harmful blue light. LEDs contain 35% harmful blue light; the cooler white the LED is, the higher the blue light proportion. Many of us have made the switch to these new energy-saving bulbs in our homes and offices, which will cause an increase in blue light exposure. If you still think you and your patients do not have increased blue light exposure, think again: smartphones, tablets and laptops are all sources of blue light exposure as well.

Blue-violet light, the most damaging band
We must then begin to understand blue light better. Thankfully, the Paris Vision Institute has done some groundbreaking research on that topic. In 2008 they began an eye health research project to better

“While most of the electromagnetic spectrum is beneficial, parts of it are not, mainly UV radiation and high-energy blue light.”

FIG. 1 Electromagnetic spectrum and zoom on visible light.
understand blue light. This was the first in vitro test in the ophthalmic industry to split the visible light spectrum into 10nm bands and see which bands of light caused the most damage on swine retinal cells. The results showed maximum cell damage occurs from 415-455nm, with a peak at 435nm +/- 20nm. This damaging band of light was termed blue-violet light. Cumulative exposure to blue-violet light will lead to retinal cell death and is one of the risk factors for macular degeneration. Compact fluorescent bulbs, LED sources and sunlight all emit these damaging wavelengths of light. Another important finding was that not all blue light is damaging. Blue-turquoise light ranges from 465-495nm and is essential for sleep/wake cycles, memory, mood, cognitive performance and pupillary constriction. Blue-turquoise light is also needed for visual acuity and color perception. Just because it’s blue does not mean it’s bad.

Limiting UV exposure with Crizal® UV and Xperio® UV
What can we do as eyecare providers to provide our patients with the clearest sharpest vision possible, while limiting exposure to the damaging part of the electromagnetic spectrum? This is where we can put the medicine or preventive medicine in the lenses. By utilizing the right products in our dispensaries, not only can we provide great vision, we can potentially delay and/or prevent onset of ocular disease. Let’s deal with UV radiation first. Typically when we talk about UV, we think of sunglasses. Sunglasses are important, but our patients receive up to 40% of their UV exposure when they are not in direct sunlight. The UV conversation is important on both the sun pair and everyday pair.
When I talk with my patients about UV prevention on their everyday pair, I talk about Crizal® UV non-glare lenses. Crizal® UV delivers amazing vision by fighting the five enemies of vision: glare, scratches, smudges, dust and water. In addition to these great features, Crizal® UV greatly reduces the amount of UV radiation that enters the visual system. When thinking about UV radiation, we have to think about both sides of the lens. UV entering the front side of the lens can either pass through the lens or be reflected/absorbed. By selecting the proper index of materials, polycarbonate and above, no UV radiation will pass through the lens. But that is only part of the equation; what about UV radiation reflecting off the backside of the lens? Studies suggest that up to 50% of UV exposure can come from reflections off of the back surface of the lens. Crizal® UV lenses reduce the backside UV reflections to 4%, giving it an industry-best eye-sun protection factor (E-SPF®) of 25 for clear lenses. In other words, wearing Crizal® UV lenses means you are 25 times more protected from UV radiation than not wearing any lenses at all. This built-in UV protection separates Crizal® UV from other non-glare lenses on the market and allows me to have medical-focused conversations about preventing ocular damage by wearing glasses.
What happens when the patients goes outside and is in direct intense sunlight? UV protection is of the upmost importance in this environment. My product of choice for sunwear is Xperio® UV. Similarly to Crizal® UV, Xperio® UV deals with UV radiation on both sides of the lens. The same rules still apply to UV coming through the front of the lens, so proper lens material selection is extremely important. The backside reflections are still present on sunwear and Xperio® UV reduces them to 1.5%, giving the highest E-SPF® available today of 50+. Not only does Xperio® UV limit UV exposure but it is also polarized, which means my patients get the maximum UV protection and best vision possible in their sunglasses. By
utilizing these products on my patients’ everyday pair and sun pair, I am protecting their visual system by preventing damage caused by UV radiation.

**Limiting blue light exposure with Crizal® Prevencia®**

What about reducing blue light? I have the blue light conversation with my patients that have a strong family history of macular degeneration, have signs of macular degeneration or have a high exposure to blue light. In my practice Crizal® Prevencia® is my lens of choice when it comes to reducing blue light exposure. This product is superior to the competition because it blocks more of the damaging blue-violet light and UV radiation than anything on the market today. It has patented LightScan™ technology that selectively filters out harmful blue-violet light and UV radiation, including backside reflections. It also allows beneficial blue-turquoise light to pass through the lens while maintaining excellent lens transparency. Crizal® Prevencia® No-Glare lenses are able to deflect 20% of the harmful blue-violet light that our patients are exposed to on a daily basis. The Paris Vision Institute showed that this amount of deflection reduced retinal cell death by 25%. On the surface, those numbers may not sound amazing, but it is greater than any other product on the market, and that 25% protection is very similar to what we expect when we discuss AREDS vitamin formulations with our patients. With Crizal® Prevencia® you can expect it to perform like the other Crizal® UV products. It has an E-SPF® of 25 and virtually eliminates backside UV reflections and features complete protection from the enemies of clear vision: glare, scratches, smudges, dust and water.

**Conclusion**

Our goal in the eyecare profession is simple, to provide our patients with the best vision possible. We strive to accomplish this goal daily by providing patients with the best medical eyecare they can get and/or by providing them with the best means of correcting their refractive error. This type of care has become standard and patients expect this when they visit their eyecare providers. How then can we exceed our patient’s expectations? I believe by using the technological advancements that are available in today’s spectacle lenses we can do more than we have ever been able to do before. We now have the power to blend the medical and refractive sides of optometry. We can do more than just deliver the best vision possible; we can now put the medicine in the lenses and have conversations with our patients about prevention of future ocular diseases by simply wearing the right lenses in the correct environment.

Essilor has given my dispensary the tools to do just that. I talk about Crizal® UV to all my patients on their everyday pair, Xperio® UV on their sun pair, and for those patients that have risk of macular degeneration or high exposure to damaging blue light, Crizal® Prevencia® is the answer. I encourage you to explore the benefits of these practice-changing products and begin having these same conversations with your patients and exceed their expectations.

**REFERENCES**


MARKET WATCH

CANCER COUNCIL AUSTRALIA’S INITIATIVES

Australia has the world’s highest rate of skin cancer. When it comes to preventing the threat of UV, the Cancer Council of Australia is probably the most experienced organisation in the world. Prof. Ian Olver, CEO of the Cancer Council of Australia, highlights the main initiatives implemented in the marketplace for Points de Vue, and gives his perspective on the future.

PROFESSOR IAN OLVER
CEO of Cancer Council Australia

Points de Vue: Professor Olver, please could you provide our readers with your perspective on the current scope of the activities of Cancer Council Australia and give them a little background on its origins and vision?

Prof. Ian Olver: Cancer Council Australia is the national body in a federated structure of state and territory Cancer Councils. We are funded by our member organisations to undertake national activity, such as advocacy for cancer policy to the Federal Government and national media. We help drive national prevention campaigns and activities, and working with our national committees, we produce the National Cancer Prevention Policy, which is updated by our committees according to the latest evidence available. We also produce clinical practice guidelines on a custom designed wiki to allow continual updating and wide dissemination. Our Supportive Care Committee co-ordinates patient information and we communicate to the public through social media and interactive sites such as iheard.com, which allows people to ask questions about cancer claims that they have seen on the web.

We promote cancer prevention lifestyle messages such as tobacco control, diet and exercise and sun protection, as well as early detection by encouraging participation in national cancer screening programs. On the fundraising side, Cancer Council Australia is responsible for national corporate partnerships and national promotion of major fundraising events such as Daffodil Day, Pink Ribbon and Australia’s Biggest Morning Tea. Cancer Council Australia’s vision is to minimise the threat of cancer to Australians, through prevention, best treatment and optimal support for patients with cancer and their families.

KEYWORDS
UV, skin cancer, ocular cancers, peri-ocular cancers, prevention, Crizal® UV
Overwhelmingly Australians have been made aware of the risk of skin cancer via the “Slip Slop Slap” campaigns of the Council. How important is this awareness of the dangers of UV exposure both locally and internationally? What is the impact of this on public health?

Protection against excessive UV exposure is a modifiable risk factor for skin cancer. In Australia skin cancers are a great economic burden. There are over 1 million GP consultations each year for non-melanoma skin cancer. Over 430,000 non-melanoma skin cancers are diagnosed each year and around 12,000 melanomas. We have over 2,000 skin cancer and eye cancer. Reducing the UV exposure translates into reduced risk of skin cancers, the most lethal, melanoma, has been curable signals the need for sun protection. With regard to skin cancers, when the UV level is three or above. The “Slip Slop Slap” campaigns have raised awareness of the importance of covering up the skin, as well as using sunscreen on exposed areas to reduce skin damage, and subsequent skin cancers, when the UV level is three or above. We know from measures like our National Sun Survey, younger Australians are starting to get the message. For example, in our 2004 survey, 60 per cent of adolescents said they would like to get a suntan, which had fallen to 45 per cent by 2011.

What have been the most significant developments in the scientific or clinical research in relation to UV exposure and cancer since you started with Cancer Council?

From the public health perspective the introduction of the UV index and its reporting in the press gives people an accurate picture of what times during the day in their geographic location the UV index will be 3 and above, which signals the need for sun protection. With regard to skin cancers, the most lethal, melanoma, has been curable if caught early and surgically removed, but fatal after it spreads. For the first time we have seen new targeted therapies developed that increase the survival time of widespread disease, because they target altered genes that are responsible for the growth of the cancer or target...
proteins that are preventing the body’s immune system from attacking the cancer. These drugs alone and in combination offer promise of vastly improved outcomes with less side effects than previous therapies.

In recent years Cancer Council has added “Seek and Slide” as required actions against UV exposure. What were the drivers behind this change, in particular with relation to ocular health?

Cancer Council added “Seek and Slide” to “Slip Slop Slap” to reinforce the message that seeking shade when the sun is most intense also helps to reduce the risk of skin damage. Sliding on sunglasses recognised the damage that UV exposure can do to the eyes, ranging from cataracts to retinal cancers and cancers on the skin surrounding the eyes. We have encouraged both children and adults to get used to protecting their eyes along with protecting the rest of the skin.

Sunglasses have had mandated standards and classification for UV protection for some time, but now Cancer Council is embarking on the endorsement of the “clear” prescription spectacle coating Crizal® UV. What role do you believe this will play in the further prevention of ocular and peri-ocular cancers?

Many who wear prescription spectacles have had difficulty adequately protecting their eyes. Devices such as clip on sun lenses are often bulky and address the UV that comes through the lens, but not that which comes in from the sides and reflects of the back surface of the lens into the eye. The Crizal® UV prescription lenses reduce both the UV coming to the eye through the lens and that being reflected onto the eye from the back of the lens, which will increase the protection of the eyes and skin around them. Reducing the UV exposure translates into reduced risk of skin cancer and eye cancer.

What other initiatives do you believe are required to improve the level of public safety and awareness to help reduce avoidable cancers, particularly in relation to the eyes?

There are target groups such as outdoor workers who are constantly exposed to UV as part of their regular job. They should be aware of the risks of sun damage to their skin and eyes and provided with protection or even have working hours changed to avoid the times of day when the UV index is high. Protection should include adequate eye protection.

Children are often thought of but also often overlooked when it comes to protecting their eyes. With their clear ocular media allowing greater UV transmission, what level of protection do they actually require?

Children can be very sensitive to sun damage. Avoiding sun exposure when the UV level is three or above is a good strategy for young infants. As a part of encouraging sun protection behaviour, children should wear hats, suitable clothing and have their eyes protected. Behaviours commenced in primary school are important. In addition schools should provide adequate shade cloth protected areas to allow shelter when the UV level is 3 or above.

In your opinion, what is the key role that ophthalmic clinicians (ophthalmologists, optometrists and optical dispensers) should be playing in prevention and protection against cancer?

The key role of ophthalmic clinicians in cancer prevention is in examining the eyes to ensure that they monitor pigmented lesions on the retina and detect early cancers in the skin around the eyes. Early detection of both is important to their outcomes. In terms of prevention, they should counsel patients to protect the eyes as well as wearing hats, appropriate clothing and using sunscreen when the UV exposure warrants it (a UV index of 3 or above).

“Reducing the UV exposure translates into reduced risk of skin cancer and eye cancer.”
Where to next? What are the key areas in the next decade for this important health issue?

Increasing public awareness of the need for sun protection, including the eyes, remains the major challenge to reduce the incidence of skin cancer. The UV coating of prescription lenses and the wearing of sunglasses each has a place in modifying this risk.

The advances in personalised medicine with more targeted therapies for melanoma will result in increasing survival rates for those with widespread disease. •

Interviewed by Tim Thurn

**B I O**

**Professor Ian Olver**  
Chief Executive, Cancer Australia

Professor Ian Olver, a highly respected medical oncologist and researcher both in Australia and overseas, is a member of the Advisory Council for Cancer Australia (the Australian Federal Government’s cancer control agency) and sits on the Council of the National Health and Medical Research Council. As head of the nation’s peak non-government cancer control organisation, he is a leading independent voice on evidence-based cancer control policy.

While Clinical Director at Royal Adelaide Hospital Cancer Centre, Professor Olver established the first oncology clinic in Alice Springs and pioneered a telemedicine link for multidisciplinary cancer care between Adelaide and Darwin. He is a leading campaigner for improved care of Indigenous Australians with cancer, following his groundbreaking work as a remote oncologist in Central Australia.

His books include *Conquering Cancer: Your Guide to Treatment and Research* and *Is Death Ever Preferable to Life?* He has also written 22 book chapters and over 240 research articles in journals.

Professor Olver’s perspective is informed by his unique mix of clinical/scientific experience and his work in the prevention and public health fields. He is also a well known researcher, whose current research focus is on psycho-social aspects of cancer.

Clinical Professor in the Department of Medicine at the University of Sydney, Ian Olver was awarded the Cancer Achievement Award by the Medical Oncology Group of Australia in 2008 and in 2011 became a Member of the Order of Australia.
What role do you think science and/or clinical practice should play in the prevention of ocular problems generated by UV and blue violet light?

The scientific and medical communities throughout the world actively contribute towards preventing ocular diseases. Reflecting on this important responsibility, Points de Vue recently interviewed several experts for their opinion on the role science and clinical practice should play in preventing eye diseases caused by exposure to UV and blue-violet light.

Recently, the relationship between light and health has gained interest,” points out Dr. Kazuo Tsubota. In this context, several scientists, clinical researchers and practitioners are paying growing attention to the harmful effects of chronic light exposure, primarily to UV and blue-violet light. All experts are looking at further research to elucidate individual risk factors and bring clinical evidence through quality solutions. The common view, shared by Dr. Rowena Beckanham, is that “prevention is critical to ocular health management” and it will remain critical in the future.
THE ROLE OF SCIENCE: the way ahead for research

The adverse affects of specific wavelengths of light, particularly UV, has been extensively covered in published research in the past decades. As Dr. David Sliney confirms, “there exists strong scientific evidence connecting the risks of cortical cataract and pterygium with UV (principally UV-B) radiation.” Although today, a significant body of work exists on both UV and blue light, there is further scope of scientific research in the days to come, especially on blue light. Encouraging researchers on this topic, Dr. Ralph Chou comments: “There is a lack of both basic and clinical scientific research into the ocular effects of optical radiation between 385 and 420 nm, and very little to follow up on the work published before 2005 on threshold exposures across the entire optical spectrum. We need a new generation of researchers in this field of study.”

At the same time, Dr. David Sliney emphasises the need for more epidemiological studies on the subject. “Although most laboratory studies demonstrate the retinal phototoxicity of blue light, many epidemiological studies do not confirm an increased risk of age-related eye retinal pathologies,” he says. “Further research is needed to clarify why this contradiction exists.”

In academia, the association of certain wavelengths of blue light with circadian system has also come under discussion. It is an area of great interest that needs extensive scientific investigation. Dr. Kazuo Tsubota sheds some light on this: “We know that light governs the circadian rhythm, but have more recently learned that it is blue light, specifically, which controls this task. Intrinsically photosensitive retinal ganglion cells (ipRGCs), a third class of photoreceptors found in the retina of the mammalian eye in 2002, primarily recognize blue light and send signals to the brain. In other words, the eye not only sees, but also functions as a clock. We believe that disruption of the natural circadian rhythm through protracted night time usage of computers and smart phones disrupts sleep and can lead to depression among other health problems, and scientists also consider that blue light may aggravate eyestrain and dry eye. I am more convinced than ever that further research is necessary in this area.”

THE ROLE OF CLINICAL PRACTICE: patient education and prescription

Prioritizing the patient’s health and best interests, even with the progressing nature of scientific and clinical evidence, practitioners recommend educating patients on possible risks of UV and blue light and prescribing products that protect against these. Dr. Randall Thomas observes: “It is difficult to know with scientific certainty, but there is a growing body of science that indicates a health benefit to diminishing human tissue exposure to certain, specific wavelengths of visible blue, and ultraviolet light. It is likely prudent that we as clinicians do all that is practical and reasonable to protect the eyes of our patients by recommending eyeglasses that limit the amount of these wavelengths. Most certainly, the aggressiveness of such interventions will continue to be shaped with on-going research.”

Dr. Sliney encourages patient education as well. He adds: “The reduction of excessive short-wavelength, blue-violet light is prudent as extra ‘insurance’ against potential delayed effects upon the retina. Clinical practice should serve an educational role in promoting UV protection for their patients, including peripheral (temporal) protection by frame design. Reducing short-wavelength light exposure may also be
beneficial – particularly in bright daylight and in some domestic light sources.” Dr. Walter Gutstein feels protection against UV and high-frequency violet light is going to become a standard in the future, “As far as the retina is concerned, we know that the blue receptor is always affected first. Unfortunately, if this receptor is damaged it would further lead to significant impairment. Not only this receptor displays blue and yellow but it is also majorly responsible for contrast regulation. Damage to this receptor is much more noticeable than to all other photoreceptors even though it varies from one person to another depending on several conditions. Evidently, protection from both UV and high frequency blue-violet light should become a standard in coming years.” When educating patients, attention should also be given to style choices and occupation. “Eye care practitioners have a fair amount of knowledge on the adverse effects of UV radiation and blue-violet light on the eye,” explains Dr. Ralph Chou. “They should educate their patients on how occupational and lifestyle exposure to optical radiation can be reduced or modified to prevent future ocular health problems, and prescribe appropriate eyewear as well.”

“PROTECTION FROM BOTH UV AND BLUE-VIOLET LIGHT SHOULD BECOME A STANDARD IN COMING YEARS”

NEXT STEPS: clinical evidence for preventive eyewear

Clinical evidence of eyewear performance helps convince patients greatly of their benefits. As a practitioner herself, Dr. Rowena Beckanham strongly urges the need for this. She argues: “As practitioners we need a strong evidence base to show consumers the benefits of new coatings and lenses to enable performance in a fast changing digital world. We need clinical trials published in reputed journals that stand up to the rigor of the scientific community to show the risks of increasing blue light exposure:

a. risks to macular health;

b. the use of digital technology and visual fatigue;

c. interference with sleep patterns in sleep deprived teenagers with overuse of digital technology.”

In practice, there is still very little known on the eyewear options and the role of the lens coating in filtering out UV and blue-violet visible light. “Although it is generally understood that sunglasses provide ocular protection from potentially harmful ultraviolet (UV) radiation, what is less known is the importance of the lens coating quality, filtration properties and fitting geometry. When treating patients, eye care professionals should explain the potential consequences of short- and long-term exposure to UV, as well as offer well-fitting treatment options that adequately filter UV and other potentially harmful short-wavelength visible light,” says Bret Andre. From his perspective, “further research isolating visible light wavelengths that cause ocular damage will assist lens designers to optimize protective lenses without sacrifice to visual function.”

Survey conducted by Anwesha Ghosh

KEY TAKEAWAYS

• UV radiation and blue-violet light can have adverse effects on the eye.

• The eye not only sees, but also functions as a clock.

• Protracted night time usage of computers and smart phones disrupts sleep and can lead to depression, among other health problems.
Macular degeneration (AMD) and cataracts play a major role in the United States health care system and global effort directed to the prevention of these conditions now is part of optometry initiatives. To benefit society both from a financial and a productivity perspective, optometrists focus on four areas in clinical practice: protective lenses, nutraceuticals, genetic testing and periodic examinations.

Introduction to disease prevention
In February 2014, approximately 24 optometrists gathered at the University of Houston for the first ever Ocular Surface Disease Wellness Conference. The subject of “wellness” and disease “prevention” were addressed in this historic two-day meeting. Prior to this meeting, most specialized gatherings by optometrists addressed disease diagnosis and treatment, rather than prevention. The concept of disease prevention is unique in the vision care arena. One possible exception is in the area of myopia where efforts to retard progression have been tried using bifocal eyeglasses, contact lenses (orthokeratology) and pharmacological agents (atropine).¹ A follow-up meeting has been scheduled for December, 2014 in Dallas, Texas.

Two conditions that lend themselves to prevention discussions by optometrists in the United States include ocular surface disease (OSD) and ocular damage from high-energy visible light (HEV) as well as damage from ultraviolet light (UV). Macular degeneration (AMD) and cataracts play a major role in the United States health care system and any effort directed to the prevention of these conditions will benefit society both from a financial as well as a productivity perspective. Cataract surgery is the most commonly performed surgical procedure in the United States today. The average cost of cataract surgery today is $3,230 per eye², and it is rising because of the use of new technology (laser cataract surgery and multifocal IOLs). Estimates of the global cost of visual impairment due to age-related macular degeneration is $343 billion.³

Specific to AMD prevention, U.S. optometrists now focus on four areas of preventative steps. Those areas include nutritional supplements, genetic testing, specialty lens coatings to block selective wavelengths of blue light, and periodic dilated fundus examinations with OCT studies. While there are several OCT models available, we have personally been pleased with our newer Cirrus™ HD-OCT instrument. Genetic risk assessment for age-related macular degeneration is becoming commonly employed in the United States for those patients with risk factors. Steve Arshinoff⁴ writes: “Previously we considered the phenotypic appearance of
the eye, macular pigment levels and patient-related non-genetic factors to determine AMD risk.” 4 Genotyping with commercially available genetic testing (Macula Risk™, RetnaGene™) now allows us to predict with 90 percent accuracy an individual’s 2-, 5- and 10-year risk for progression to advanced AMD. Following the reporting of the Age-Related Eye Disease Study 2 (AREDS2) (NEI) results, we now have definitive information on AMD prevention and progression using particular nutritional supplements, although further research is necessary.

**Pathophysiology and economics**

The number of people living with macular degeneration is similar to that of those who have been diagnosed with all types of invasive cancers.5 As many as 11 million people in the United States have some form of age-related macular degeneration. The number is expected to double to nearly 22 million by 2050. Most researchers believe that blue light exposure has a role in the pathogenesis of AMD. According to Margrain et al.6: “Laboratory evidence has demonstrated that photochemical reactions in the oxygen-rich environment of the outer retina lead to the liberation of cytotoxic reactive oxygen species (ROS). These ROS cause oxidative stress, which is known to contribute to the development of AMD. The precise chromophore that may be involved in the pathogenesis of AMD is unclear but the age pigment lipofuscin is a likely candidate.” They continue: “Studies in human macular pigment density and the risk of AMD progression following cataract surgery lend further weight to the hypothesis that blue light exposure has a role in the pathogenesis of AMD but the epidemiological evidence is equivocal. Blue-violet light has a twofold effect on lipofuscin. It causes an increase in production and also activates its phototoxic components (free radicals), causing the death of RPE cells. On balance the evidence suggests but does not yet confirm that blue light is a risk factor in AMD.”

Research by the Schepens Eye Institute (Harvard University) suggests that a low density of macular pigment may also represent a risk factor for AMD by permitting greater blue light damage.

**Science**

Existing artificial light sources are basically of two types: incandescent (includes halogen) and luminescent (fluorescent and LED). Incandescent lights are becoming difficult to find in the typical home repair stores in the United States as the newer LED light sources begin replacing them. These newer light sources are much more energy efficient, have a longer lifetime and the government has decreed that this exchange takes place. It is thought that by 2020, 90% of all light sources worldwide will be based on solid state lighting products and LEDs. These newer light sources give off a greater proportion of blue light than the older incandescent bulbs. We know that the sun is the standard light source. The blue light proportion of our daylight in the entire visible spectrum varies between 25% and 30%. We know that blue light is vital to a number of physiological processes and interfering with it may have adverse effects. A recent study by Gray and colleagues in the *Journal of Cataract and Refractive Surgery* found that patients with blue-light filtering IOLs performed significantly
better under driving conditions with glare compared with similar patients who had clear IOLs. Dr. Henderson and her colleagues see no harm posed by blue filters, at least in visual parameters: they “feel that the potential protection against AMD is worth it.”

Clinical practice
Several groups around the world have studied the potential health risks of products using LEDs. Basically three high-risk populations have been identified: (1) children and aphakes who receive a higher blue light proportion on the retina, (2) those individuals suffering from ocular photosensitive pathologies or using photosensitive drugs (light-sensitive agents used in photodynamic therapy such as Verteporfin used to ablate blood vessels in the eye when treating wet macular degeneration), and (3) those individuals who are daily exposed to LEDs while using short viewing distances.

1. AMD AND PROTECTIVE LENSES
As a practical matter, optometrists and ophthalmologists in the United States have begun the process of utilizing electronic medical records (ObamaCare). During the early part of the patients’ examination they are asked several questions by the technician and those individuals who fall into one of the above three groups are then counseled on the particular risks they face and are prescribed spectacle lens treatments which will help protect them from the increased threats offered by increased blue light presence. Because optometrists are the guardian of good vision, it is important for us to counsel patients about modifiable risk factors. Two of those risk factors include smoking and cumulative light exposure, especially UV and HEV blue light.

We have found at the Clayton Eye Center in Morrow, Georgia that the best results are achieved when the doctor himself/herself initiates the conversation about blue light protection in the examination room and then the dispensing optician reinforces the message. We specifically prescribe the new Crizal® Prevencia® lens treatment in order to selectively filter out only the dangerous wavelengths while allowing the good wavelengths to pass through. We know that blue wavelengths are the most potent portion of the visible electromagnetic spectrum for circadian regulation. Because the timing and quantity of light and darkness both affect sleep, evening use of amber lenses to block blue light might affect sleep quality. We have found over the past several months that our patients appreciate the fact that we are protecting their eyes with these discussions and we feel that to not educate our patients would be a great disservice. We reference data from the Beaver Dam and Blue Mountain studies, which implicate blue light as a factor for age-related macular degeneration, particularly following cataract surgery. Our clinic performs more than 3,000 cataract surgeries a year and each post-op visit emphasizes the potential risk of blue light. Our IOLs are blue blocking for added protection. Our practice recently became involved with an accelerated program emphasizing doctor-directed dispensing. Each of our nine optometrists now prescribes various lens products and coatings to each patient when indicated and outlines the specific products on a specially designed form and reviews these products with the patient. The patient is then escorted to the optical department from the clinical area by a technician or the doctor and the form is presented to the dispensing optician. Products such as AR, Transitions®, digitally surfaced progressive designs (such as the Essilor S Series™) and Crizal® Prevencia® coatings have increased substantially as a result of this new process. Several lens manufacturers have become involved with blue blocking technology; however, so far only Essilor has designed a coating that blocks specific wavelengths. VSP’s Unity BlueTech lens, Hoya’s Recharge™, PFO’s iBlucoat™ and Signet Armorlite’s BlueTech (Indoor and Outdoor) all block HEV (high energy visible light) and offer improved contrast sensitivity. However, they also block the blue-turquoise range, which has been demonstrated to be “good” light and necessary for other functions, including increased contrast sensitivity and mood regulation.

“The concept of disease prevention is unique in the vision care arena.”
2. AMD AND NUTRACEUTICALS
The Age-Related Eye Disease Study 2 (AREDS2) was a multi-center, randomized trial designed to assess the effects of oral supplementation of macular xanthophylls (lutein and zeaxanthin) and/or long-chain omega-3 fatty acids (docosahexaenoic acid [DHA] and eicosapentaenoic acid [EPA]) on the progression to advanced age-related macular degeneration (AMD). While the results of the study left several questions unanswered, it also led the way to changes with respect to the prescribing of nutritional supplements for those patients with early macular degeneration and those at risk. Optometrists in the United States now routinely encourage their patients to take these nutraceutical supplements as a matter of procedure and this author suspects that this practice will become standard of care in only a matter of time. There are several commercial products on the market to choose from: Bausch and Lomb’s Preservision Eye Vitamin AREDS 2 Formula Soft Gels are probably the most commonly used. This particular product is beta-carotene free, which is a positive for current/former smokers. Another product that I have frequently used is Science Based Health’s Macula Protect Complete, which is also beta-carotene free. The study demonstrated that there was a 25% overall risk reduction of progression to exudative AMD. The role of macular pigment (MP) is also acknowledged and many optometrists now measure macular pigment and dose supplements accordingly. The U.S. diet is known to be low in lutein and zeaxanthin. The third carotenoid, Meso-Zeaxanthin, is a key carotenoid in the macula and even lower in the U.S. diet. We know that smokers are at high risk for AMD. In smokers and former smokers, beta-carotene has been associated with an increased risk of lung cancer.12, 13, 14

3. AMD AND GENETIC TESTING
Genetic testing has progressed in several areas of medicine over the past ten years. One area that has enjoyed the benefits of continuing research is AMD. We can now make a prognosis to within 90% accuracy of how a patient’s eye disease will progress.15 Several research projects have demonstrated that those patients subjected to testing have better outcomes than those without. At the 2013 American Society of Retina Specialists Annual Meeting, Dr. Peter Sonkin, a retina specialist from Tennessee Retina, presented the results from an analysis of the impact of genetic testing in their practice over a five-year period. The data revealed that patients who had Macula Risk testing and were subjected to a stratified surveillance schedule as well as a patient education program had better visual acuities on presentation compared to those patients without genetic testing. The November 2013 Issue of Ophthalmology highlighted an article titled “Prediction of Age-Related Macular Degeneration in the General Population – The Three Continent AMD Consortium”, which is a study evaluating AMD prognostics using three prospective population-based studies: the Rotterdam Study, the Beaver Dam Eye Study, and the Blue Mountain Eye Study. The non-genetic model which included age + sex + BMI + smoking + AMD status has a 78% predictive accuracy, while the genetic model, which included genetics with the above criteria, had an 82% predictive accuracy. Using all available information I have now come up with a formula for what the primary care optometrist should now do to prevent vision loss. This protocol is used by our doctors and many others and is a compendium of existing good practices.

“The number of people living with macular degeneration is similar to that of those who have been diagnosed with all types of invasive cancers”
4. THE CLAYTON EYE CENTER VISION LOSS MODEL IN AMD
- Diagnose AMD
- Perform genetic testing on each AMD patient
- Increase monitoring frequency including OCT testing
- Prescribe the appropriate nutraceuticals
- Prescribe selective blue-blocking spectacle lenses
- Counsel patients on diet, smoking, exercise and weight (BMI).

Optometrists in the U.S. have embraced genetic testing for AMD much in the same way other physicians have embraced genetic testing for cancer and several other diseases. There are over 2,000 tests available. Screening embryos for disease is becoming more frequent.

Conclusion
In summary, AMD is on the rise. As individuals continue to live longer, the optometrist is going to diagnose increasingly larger numbers of cases. Prevention is a must. We now have several tools that will allow us to aid in our preventative efforts. Government-mandated lighting changes will expose us to larger doses of potentially harmful HEV blue light. Computer usage continues to be on the rise and these tools as well as electronic tablets, smart phones and other games used at closer near point distances will also increase our exposure. By prescribing spectacle lenses that can aid in filtering out the noxious wave lengths, we may be able to prevent many individuals from acquiring this dreadful disease down the road. By adding genetic testing and nutraceutical supplements to our armamentarium, we may be doing the world a tremendous favor. Our job is vision preservation, and this is one way to accomplish that task. To not implement the above protocol but rather take a “wait and see” approach may be doing your patients more harm than good. The Optometric Oath, which made its debut in 1987, states, “When I have been called on to provide protection, Crizal® Prevencia® lenses will be prescribed.”

The above approach fulfills my responsibility.

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AMD: CLINICAL PROTOCOL, PREVENTION AND OUTLOOK

AMD is the leading cause of legal blindness in industrialized countries. It has two forms, atrophic and exudative, and a multifactorial pathogenesis. To cope with the ever-increasing incidence of AMD, retinal specialists resort to three strategies: primary prevention, patient management in clinical practice and prospective medical research aimed at finding new therapies.

Age-related Macular Degeneration (AMD) is a chronic degenerative disease of the retina. It selectively affects the central portion of the retina called the macula and causes the degeneration of retinal visual cells. AMD is the leading cause of legal blindness in industrialized countries. The pathophysiology of AMD is still poorly elucidated, but the implication of intoxication processes leading to the death of retinal pigment epithelial cells has been established in recent years. During aging, these cells may present functional impairment related to the accumulation of proteolipid complexes, known as lipofuscin granules, in lysosomes. These granules are formed gradually by the accumulation of undegraded protein and lipids from the external photoreceptor fragments phagocyted by the pigment epithelium. Lipofuscin also contains cytotoxic derivatives derived from the visual cycle such as A2E. Under the effect of blue light, A2E oxidizes and induces protein, lipid and DNA oxidation, causing significant oxidative stress in the cells of the retinal pigment epithelium during aging and resulting in the death of the latter.

Pathogenesis
Age-related degeneration of the macula has a multifactorial pathogenesis. The primary factor is, of course, age, since the disease appears after age 50 and its prevalence increases rapidly after age 75. There is also a genetic predisposition to the disease: the risk of developing AMD is four times greater if a parent or sibling has it. Several genetic polymorphisms associated with the disease have been identified. Among them, variants of the gene coding for complement factor H or the gene encoding HTRA1 (a protease) are implicated. Since 2005, a total of 19 loci have been identified as being related to AMD. They involve a variety of biological functions, including the regulation of the innate immune system.

Pathophysiological mechanisms
The exact pathophysiological mechanisms of AMD are still poorly elucidated, but the implication of intoxication processes leading to the death of retinal pigment epithelial cells has been established in recent years. During aging, these cells may present functional impairment related to the accumulation of proteolipid complexes, known as lipofuscin granules, in lysosomes. These granules are formed gradually by the accumulation of undegraded protein and lipids from the external photoreceptor fragments phagocyted by the pigment epithelium. Lipofuscin also contains cytotoxic derivatives derived from the visual cycle such as A2E. Under the effect of blue light, A2E oxidizes and induces protein, lipid and DNA oxidation, causing significant oxidative stress in the cells of the retinal pigment epithelium during aging and resulting in the death of the latter.

KEYWORDS
AMD, macula, maculopathy, retinal pigment epithelium, lipofuscin, photoreceptors, A2E, oxidative stress, phototoxicity, blue light, in vitro, photoreceptors, anti-VEGF, drusen, prevention, antioxidants, photo-protection, Crizal® Prevencia®
system, maintenance of cellular structure, growth and permeability of blood vessels, lipid metabolism and atherosclerosis. The simultaneous presence of three variants (factor H, HTRA1 and CC2-FB) in the same individual can increase the risk of developing AMD by a factor of up to 250.

Smoking is strongly associated with AMD: it increases the risk of developing the disease by a factor of three. Numerous research studies have shown that a diet low in vitamins, trace elements and antioxidants can predispose to the disease.

Retinal phototoxicity related to blue light is also implicated in the pathogenesis of AMD. The wavelengths responsible for this toxicity in the presence of lipofuscin were recently elucidated in vitro revealing a spectrum of blue-violet light ranging from 415nm to 455nm with a highly toxic peak at 435nm. This toxicity increases in proportion to the amount of lipofuscin in the retina, but a slight toxicity remains, even in the absence of lipofuscin. These wavelengths are, of course, present in the solar spectrum, but can also be found in the radiation of certain light-emitting diodes.

Obesity also doubles the risk of AMD. Hypertension, cardiovascular diseases and cholesterol have been implicated as well, but their role remains uncertain.

The forms of AMD

AMD has two different forms, atrophic and exudative. The atrophic or “dry” form is related to atrophy of the macula, characterized by the progressive degeneration of the retinal pigment epithelium and the neurosensory retina. There is no known cure for this form of the disease, which progresses slowly. The exudative or “wet” form is characterized by the abnormal development of blood vessels below the macula. This ocular neangiogenesis is also known as choroidal or subretinal neovascularization, thus providing another name for this form: neovascular AMD. These malformed new blood vessels are fragile and porous, and are therefore subject to vascular hyperpermeability. They also destroy the normal architecture of the retina and its functioning.

The neovascular form has various subtypes depending on the type and location of neovascular relative to the pigment epithelium. The photoreceptors suffer and ultimately scar tissue develops, permanently destroy-

"Age-related degeneration of the macula has a multifactorial pathogenesis."
ing the macula. This is the most aggressive form of the disease and may represent two-thirds of the forms of AMD. Choroidal neovascularization is due to the phenomenon of angiogenesis, in which vascular endothelial growth factor (VEGF) plays a significant role. VEGF has therefore been the target of new therapeutic strategies developed in recent years, leading to the development of anti-VEGF treatments. These treatments are now the gold standard for treating the disease and are administered via intravitreal (i.e. intracocular) injections, which are repeated every other month on average. The early stages of AMD are characterized by the presence of small yellowish white spots on the fundus in the macula region called drusen and/or alterations of the pigment epithelium. This feature defines age-related maculopathy or pre-AMD.

**Exudative AMD: treatment protocol**

The severity and speed of development of the exudative form, along with the efficacy and cost of the treatments developed to date, make it a real public health concern and a diagnostic and therapeutic emergency. Ophthalmologists/retinologists who treat AMD patients must be able to see them on very short notice (within a week at the most) if they present with scotoma (dark spots in their central vision) or macular syndrome: a decrease in visual acuity or difficulty reading; metamorphopsia (distorted perception of images and straight lines). In the presence of these symptoms, eye exams must be conducted promptly, including visual acuity assessment via an ETDRS chart; a fundus examination and/or retinogram; an optical coherence tomogram (OCT). Fluorescein angiography may be performed if necessary. Another injection must be given after the first three injections, if persistent or recurring signs of continued activity of the neovascular lesion are clinically detected via a fundus examination and/or optical coherence tomography.

If the diagnosis of subfoveal exudative AMD is confirmed by these examinations, it is recommended that anti-VEGF treatment be initiated as early as possible, irrespective of the initial level of visual acuity. Anti-VEGF treatments must be administered by intravitreous injection. Extra- and juxtafoveal choroidal neovascularization with subfoveal exudative manifestations should be considered a subfoveal location of AMD.

In the current state of science, the most commonly adopted treatment protocol is as follows: one anti-VEGF injection per month during three consecutive months (the interval between the two injections must be at least four weeks), followed by a monitoring phase. During the monitoring phase, patients must be examined every four weeks as follows: a visual acuity assessment using an ETDRS chart; a fundus examination and/or retinogram; an optical coherence tomogram (OCT). Fluorescein angiography may be performed if necessary.

Another injection must be given after the first three injections, if persistent or recurring signs of continued activity of the neovascular lesion are clinically detected via a fundus examination and/or optical coherence tomography.
tomography. On average, patients receive six or seven injections per year. AMD is a bilateral disease. After the first eye is affected, there is an increased risk of bilateralization (about 10% per year). In the presence of functional symptoms (i.e. visual impairment, metamorphopsia, scotoma, etc.) concerning the fellow eye during follow-up, the patient should be seen on an emergency basis. It is recommended that surveillance examinations for AMD be performed on both eyes to screen for an asymptomatic incipient lesion in the fellow eye.

Atrophic AMD: patient management
Unfortunately, the same therapeutic advances are not available for patients with atrophic AMD as for those with the exudative form. Although it progresses more slowly, the long-term prognosis remains poor and possible complications involving neovascularization warrant regular monitoring (i.e. self-monitoring using the Amsler grid, leading to a rapid consultation if there is any change in functional symptoms).

When the decrease in visual acuity becomes debilitating, management of patients with advanced AMD involves rehabilitation and the use of low-vision-support magnifying optical systems to mobilize unaffected areas of retina to improve vision.

Patients presenting with age-related maculopathy must be educated about self-monitoring methods, via the Amsler grid in particular. Patients at very high risk with large confluent drusen and RPE alterations must be examined frequently to detect the possible appearance of neovascularization amenable to treatment. It is essential that patients receive a clear diagnosis. Ensure that patients know...
the name of the disease that is the cause of their declining visual acuity and whether they are suffering from an early or advanced stage of the atrophic or exudative form of the disease. Explain to patients that this is a chronic condition that can be treated but cannot be cured, and that it does not lead to total blindness (since peripheral vision is preserved). Regular follow-ups are essential.

Inform patients about their visual prognosis, the risk of involvement of the second eye and the risk of progression from the atrophic form to the exudative form.

Primary prevention
The constantly increasing incidence of AMD fully justifies strong primary prevention efforts to combat this condition. Primary prevention is based on combating the risk factors for the disease. For example, simple lifestyle and dietary preventive measures can be recommended to all, such as: combating tobacco use; combating obesity, lipid disorders and hypertension; practicing a physical activity on a regular basis; and adopting a diet rich in the macular pigments lutein and zeaxanthin (found in fruits and vegetables) and omega 3s (oily fish like salmon, tuna, etc.).

Based on the results of conclusive research on the toxicity of blue-violet light radiation, a partnership with a manufacturer of eyeglass lenses has led to the development of lenses (Crizal® Prevencia®) capable of reflecting a fraction of this toxic radiation and preventing it from penetrating the eye. It is therefore logical to recommend that as many people as possible wear this type of photo-selective protection, particularly those with genetic risk factors.

Protection from solar radiation via photo-protective lenses is still called for out-of-doors from an early age in view of the transparency of the crystalline lens in children. The prevention of complications for patients with precursor lesions is also currently necessary. This is accomplished primarily through the prescription of food supplements, on the basis of evidence provided by the publication of a number of large epidemiological studies. Supplementation with antioxidants (zinc and vitamins C and E) decreases the risk of progression and worsening of AMD by 25% in at-risk patients. Supplementation with 10mg of lutein and 2mg of zeaxanthin in addition to antioxidants would reduce the progression of advanced AMD by 18%. The value of omega 3 is not as clear-cut, but studies have shown that taking large doses of DHA could reduce the risk of developing neovascular AMD in high-risk patients.

The prescription of food supplements whose composition is consistent with data from these studies is therefore recommended for at-risk patients and patients known to have AMD.
New therapeutic avenues

A number of new therapeutic avenues for AMD are currently being explored. Some new molecules should soon be available in association with anti-VEGF treatments for exudative AMD, including anti-PDGF (Platelet derived growth factor) agents, also administered via intraocular injection. Numerous other molecules are also being tested, including complement factor inhibitors and anti-TNF (tumor necrosis factor) molecules.

Gene therapy is also being studied in exudative AMD with the goal of producing an anti-VEGF agent directly in the retina by introducing a gene directly into retinal cells by a viral vector. This would free patients from risks related to repeated injections.

Cell therapy is a new avenue being explored for atrophic AMD. The idea is to implant stem cells or autologous RPE cells in the retina to renew the supply of functional cells and stop the degenerative process. Lastly, for visually impaired patients at a very advanced stage, an artificial retina is also under development. An implant is placed in the retina that will receive images via an eyeglass-mounted camera.

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FIG. 4 Example of a very-high-risk patient with untreated AMD in one eye and drusen and pigmentary alterations in the other eye.

“The constantly increasing incidence of AMD fully justifies strong primary prevention efforts to combat this condition.”

CLINIC

AMD has a multifactorial pathogenesis:

- Age
- Genetics
- Smoking
- Diet low in vitamins, trace elements and antioxidants
- Retinal phototoxicity (blue light)
- Obesity
- Hypertension

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KEY TAKEAWAYS

1. New therapeutic avenues
2. Cell therapy
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The environment in which we use our eyes now is different than it was 10 years ago. New indoor lighting types and a drastic increase in use of digital devices has caused a dramatic increase in blue light exposure. Current research shows that blue light causes retinal cell death. It is essential for us as eye care experts to understand blue light and how we can manage it in the clinical setting. Fortunately lens technology is evolving so we can deal with this change to our visual environment and better protect our patients.

Technological advancement is all around us. These advances aim to make life more convenient - and whether you love them or hate them, they are here to stay. The music industry has gone from records to 8-tracks to cassette tapes to compact discs and now to digital files. What about when we travel, Uber and Lyft have changed the way we get a ride. Because of these changing technologies, the customer’s demands change, and industries must in turn evolve and adapt, creating new up-to-date solutions to meet and exceed their customer’s needs – or risk being left behind.

What about lenses? Have they evolved? Certainly, over the last 50 years we have gone from bifocals to progressives, glass to polycarbonate to high index, thick to ultra-thin lenses and most recently from front side only UV protection to front and back side UV protection. All these changes are worthwhile, however our patients continue to change the way they use their eyes, while the environment in which they use their eyes is also rapidly changing. It is essential as eye care providers we employ technology that evolves with our patients. These changes are all around us, but they are so common place that many times we do not recognize the impact they have on vision.

PHOTOGRAPH NOTES
Dr. Ryan L. Parker, OD, is in private optometry practice in Ardmore, Oklahoma. USA.

KEYWORDS
Ocular disease prevention, UV radiation, age related macular degeneration, Smart Blue Filter™, evolution, harmful blue light, embedded protection, Transitions®, Crizal® Prevencia®, Xperio UV™.
“Since the damaging effects of blue light are cumulative, it is important that we take into account our exposure from all sources and craft solutions to address exposure from those sources.”

Blue-Violet light
One major change is blue light. Blue light is not new – it is part of the visible spectra [Figure 1]. The sun has been the single biggest source of blue light since the beginning of time with an exposure outdoors 500 times greater than indoors. The change in blue light comes with our knowledge of its effect on the visual system. Thanks to the research done by the Paris Vision Institute and Essilor, we now know that most swine retinal cell death occurs when these cells are exposed to the blue-violet light bands between 415nm-455nm, with a peak at 435nm [Figure 2]. This is considered damaging harmful blue light and has no benefit to the visual system. Blue-violet light has always caused damage to the visual system – but this is not the change we are referring to...... the change that is at the forefront of our profession is our patient’s level of exposure to this blue light.

Increasing blue light exposure
Our visual environment is different. Not only is damage occurring outdoors, but with the recent changes in light sources, damage can also come from indoors. The incandescent light-bulbs of yesteryear only emitted around 3% of damaging blue-violet light. New energy-efficient LED light sources emit around 35% damaging blue-violet light. By the year 2020 84% of our indoor light sources will be LED. Our patients are exposed to this light each and every day. And as if that is not enough, blue light exposure from our technology revolution is also changing our visual environment. We are a “plugged-in” society, 30% of adults spend 9 or more hours daily on a digital device, 1 in 4 children spend 3 plus hours a day on a digital device and many adults check their phones close to 100 times per day. The technology revolution is causing increased blue light exposure because the vast majority of these devices that we are now attached to, use LED light sources. Since the damaging effects of blue light are cumulative, it is important that we take into account our exposure from all sources and craft solutions to address exposure from those sources. There is no doubt our visual environment is evolving - the way in which we use our eyes is different now than it was 10-15 years ago. The amount of blue light we are exposed to is rapidly increasing. From our new more energy-efficient light sources to the exponential availability of digital devices, to the sunlight, blue light is everywhere. To protect our patient’s visual system our lenses must evolve.

Lens evolution and patient education
We are currently at the beginning of a new lens technology evolution. Over the past few years the major lens companies came to market with new technologies protecting against blue light. Whenever a profession is in the early stages of technology evolution, it is essential that experts in the field truly understand the new knowledge and products. In the optical field we are no different. As the optical experts, our customers and patients rely on us for our knowledge. In 2015, the Vision Council reported that 72% of adults were unaware of the dangers of harmful blue light. That means in my clinic almost three-quarters of the people I see have no idea that damage is occurring daily nor are they aware where this damage is coming from. This damage is cumulative, making prevention critical for maintaining ocular health.

What does current research tell us about blue light?
As with any new technology, research can be slow to surface. In fact when I travel the country and lecture about the harm caused by blue light, one statement I hear from time to time is “that makes sense, but there has not been...
Since current research shows that blue light causes retinal cell death, specifically 415nm-455nm, blue-violet light has been linked to causing age-related macular degeneration. Many other factors determine a patient’s risk for this disease including age, race, genetics, diet, smoking status and weight. Blue light is a key ingredient in this mix because all other risk factors are difficult to modify in my patient population, but I can easily reduce the amount of blue light my patients are exposed to, thus reducing their risk factor for retinal disease.

What optical solutions do we have to reduce blue light exposure?

The first evolution involved lenses containing pigment to absorb blue light, mainly melanin. Melanin does a good job of absorbing a wide range of blue light, both harmful blue light and good blue light. Putting melanin in a lens causes it to have a yellow tint, which can be considered cosmetically unacceptable for some patients. The next technological wave was non-glare (i.e. anti-reflective) lenses, which deflect a percentage of blue light away from the lens. Nearly 80% of patients accept this difference if it is explained to them properly.

enough research yet.” Honestly that statement stops me in my tracks. Do you really need more research or is it just that you are resistant to change? I agree that research is essential, but what we have to date is very solid. Let’s review the research I mentioned earlier. In 2008, The Paris Vision Institute in conjunction with Essilor set out to explore which wavelengths of visible light caused the most retinal cell death. They were able to split the visible light spectrum into 10nm bands and focused the energy on swine retinal cells. Swine retinas were used because the size of the eye and cone density very closely matches the human eye. It took 4 years of research to identify that peak cell death occurs at 435nm, with a danger zone of +/- 20 nm. This means visible light from 415nm-455nm [Figure 2], blue-violet light, caused the most retinal cell death. In 2015, the researchers confirmed this specific toxic light action spectrum with oxidative stress biomarkers. This research evidenced that blue-violet light is a defense inhibitor in addition to a strong stress inducer.

It is very important to note that not all blue light damages the retina. Blue-turquoise light from 465nm-495nm is in fact very beneficial to our sleep/wake cycles and hormone balance. Not all blue light is harmful so we must understand which wavelengths we need to block and which ones are good. These are the findings that I use in my clinic when discussing blue light and blue light reducing products with my patients.

**FIG. 2** Toxic action spectrum of light on apoptosis for A2E-loaded RPE cells (40 µM A2E). ***p<0.001 as compared to control cells maintained in darkness.
When comparing different products it is important to keep in mind the percentage of blue-violet light from 415nm-455nm that a lens blocks, not just the total amount of blue light. Moreover, if the lens blocks light above 465nm that is actually beneficial light and that does not lead to advanced retinal cell death. Some lenses on the market today have big claims regarding the amount of blue light they block, but it is not just the amount, but also the specific wavelength that is important.

**Essilor’s solutions**

In 2013, Essilor launched Crizal® Prevencia® as the first selective clear lens on the market to provide protection from both UV radiation and harmful blue light. This was provided as a non-glare lens treatment that deflected part of the harmful blue-violet light while allowing patients’ eyes to receive beneficial blue-turquoise light. This technology works on the principle of deflection and helps to block up to 20% of harmful blue light. Thanks to the innovative research done by the Paris Vision Institute & Essilor’s R&D team, this was a great evolution in non-glare lenses, and so far over 3 million patients are wearing it worldwide.

Fast forward 3 years and what has changed. In 2016, Essilor is poised to be the leader in blue light protection. Another evolution in technology was needed to make this happen. Crizal® Prevencia® is an impressive product, but like other non-glare blue light reducing lenses on the market, increased costs and the blue-violet hue off the front of the lens were barriers for some patients. Thanks to Essilor’s outstanding R&D team they once again raised the bar and created an evolution in lenses, Smart Blue Filter™.

Smart Blue Filter™ is embedded protection in the lens. The molecules in Smart Blue Filter™ absorb 20% of harmful blue-violet light from 415nm-455nm while allowing beneficial blue-turquoise light to pass through the lens. It is clear in color and appearance. The Smart Blue Filter™ removes the previous barriers to blue blocking lenses. It is automatically integrated into digital Varilux® lenses, Transitions® lenses and Essilor’s new enhanced single vision lens Eyezen™+ when you order these products.

**Essilor’s commercial range**

With the evolution of lens technology we can begin to deal with blue light much more effectively. As an optometric physician I can now talk with my patients about blue light exposure and their family history of retinal disease along with their personal modifiable risk factors and prescribe a lens that is best for them. The exciting part now is the ability to mix and match products and customize different levels of protection to create the Eye Protect System™. The research shows that blue light causes retinal cell death and I believe that an essential level of protection is important for every patient. This would include a Smart
Conclusion

It is clear that the environment to which our eyes are exposed is different today than it was 10 years ago. Blue light exposure continues to increase from numerous sources not only inside, but outside as well. In response to research showing that certain wavelengths of blue light damage the retina our industry has responded with an evolution in lens technology to manage blue light. With our understanding gained from current research we can compare how different products perform. Lens prescribing will be an exciting area with the ability to combine different technologies and create an Eye Protect System™ customized to our patient's specific risk factors. We can not only protect everyone’s eyes by using lenses that have dramatically evolved over recent years, but also have the potential to prevent devastating sight threatening macular disease.

Blue Filter™ product (Varilux® Digital, Eyezen™+, Transitions® Signature® VII) paired with a Crizal® non-glare lens, providing a 20% reduction of damaging blue-violet light and an E-SPF® of 25 [Figure 3]. If the patient has more risk factors, advanced protection may be needed. Using a Smart Blue Filter™ product and pairing it with Crizal® Prevencia® would provide a 30% reduction in harmful blue-violet light and an E-SPF® of 25 [Figure 2]. The ultimate level of protection would include Transitions® XTRActive® or Transitions® Vantage® paired with Crizal® Prevencia®. This combination will block at least 45% of harmful blue light and provide an E-SPF® of 25 [Figure 3]. I use this ultimate level of protection for my patients who have early signs of AMD or a strong family history along with other risk factors.

At this point we have only addressed part of the problem, which is blue light coming from indoor light sources and digital devices. One thing is certain, our patients all spend time outside and we must consider outdoor blue light exposure because the sun emits a lot of blue light. When Transitions® lenses “activate” outside they provide between 85%-88% protection against harmful blue light and if paired with a Crizal® Product an E-SPF® of 25 is achieved [Figure 4]. An even higher level of outdoor protection would involve prescribing Xperio UV which provides an amazing 92% reduction of harmful blue-violet light and provides an industry leading E-SPF® of 50 [Figure 4].
• Blue-violet light from 415-455 nm has been evidenced as a strong oxidative stress inducer and a defense inhibitor, thus one of the most harmful forms of light for the retina.

• The potential risk linked to the increasing blue light exposure can be modified thanks to the latest ophthalmic lens technology such as the Smart Blue Filter™.

• Patient education is critical to raise awareness about both the harmful effects of blue light and existing preventive solutions.

• Blue light is composed of a harmful (blue-violet) and beneficial (blue-turquoise) radiations. It is essential that an ophthalmic lens blocks the former and lets through the latter.

• When comparing different optical solutions for blue light filtering it is important to keep in mind that not only the amount of blue-violet light blocked is important but also the wavelength bands blocked.
New ophthalmic lenses filtering harmful blue-violet light were introduced into the marketplace a few years ago. Since their launch, Crizal® Prevencia® lenses have gained widespread commitment among optometrists, dispensing optician lens experts and wearers. Jean Oldbury, owner of an independent practice in Macclesfield (UK), reveals for Points de Vue her experience-based learning and know-how in terms of prescribing Crizal® Prevencia®. In this interview we discuss how to deliver eye care excellence while growing business effectiveness.

Points de Vue: Could you provide our readers with a little background on the current scope of your activity and vision that drives your practice towards effective patient care?

Jean Oldbury: Oldbury and Cruickshank is an independent optometrist practice that was established over 25 years ago. We have an active patient base of over 15 000, we run 4 to 5 clinics at any one time and have over 20 staff. The benefit of the expansion of our practice and the extra space it provided for meeting and training rooms has given us the edge in terms of training for our entire staff. I have invested a lot of time in the training and development of the team because I know that confidence and passion from a team contribute to developing a business. Over the years we have developed our niche of offering top quality service and products and have been at the forefront of using the latest innovative technology to support our brand. Our ethos is simply to offer the best visual solutions to each and every patient to suit their lifestyle needs.

As a business partner, which kind of support can you expect from your main supplier and how does it translate into results?

We have been supporting and supported by Essilor UK for most of our 25 years in business. In 2009 we were part of the Needs Based Solution project (NBS) from Essilor UK. This was a fast and furious learning curve for our team.

Jean Oldbury BSc (Hons)
Jean Oldbury is an optometrist who graduated from Aston University in 1981, and is a member of The College of Optometrists and The Association of Optometrists (AOP) in the UK. She has been an optometrist and partner at Oldbury and Cruickshank Opticians in Macclesfield, Cheshire, England for over 25 years. The practice is recognised as a centre of excellence for optometry. Jean has a special interest in paediatric optometry. In addition to her clinical optometry practice, Jean has developed skills in business management, human resources and marketing.
"Our ethos is simply to offer the best visual solutions to each and every patient to suit their lifestyle needs."

However the techniques we learnt then have paid dividends over the past 7 years. Our business grew by 25% in the year we implemented NBS. Since then we have been part of Optometrist Led Recommendations (OLR), Reception Perfection and multiple lens dispensing projects, more widely known as the Optical Dispensing Program (ODP). All of which have played a tremendous part in the growth of our business. We have found that skills learnt from ODP can be applied to other aspects of our business.

**Positioning your product offer at the forefront of the latest technology, how did you implement an innovation such as Crizal® Prevencia®?**

The launch of Crizal® Prevencia® is a great asset to Essilor’s portfolio. We were excited to learn about the new features this lens will deliver to our patients, and we felt that the health benefit, in combination with our best recommendations, could be communicated in the handover process.

On its initial launch, we researched information about “bad” blue light and discussed the benefits in depth with the lens experts along with the optometrists, and ensured all were comfortable and understood why Crizal® Prevencia® was a new benefit to the patient. Thanks to ODP we applied our terminology of “new protective no-glare” lens to Crizal® Prevencia® which is now used consistently by all the team.

**Who is the primary target for Crizal® Prevencia®?**

We are aware that many patients are concerned about the effects of digital devices on their eyes, such as headaches, visual fatigue and dryness. We also have patients who may initially fail to make the connection between their symp-
<p>“We recognise the measures we need to put into place with children, and as a consequence we make talking about Crizal® Prevencia® an integral part of our conversations with parents and children.”</p>

“BEST PRACTICE”

OCT examination
© Adam Schofield | Cheshire Photography Co.

toms and their use of digital devices, and so we educate them on these effects if need be. The launch of Crizal® Prevencia® was an ideal product for us to recommend to support and reassure the many concerned patients. We are increasingly finding that parents in particular are preoccupied about their children’s frequent daily use of digital devices both at home and at school, and the benefits of Crizal® Prevencia® go some way to giving parents peace of mind. We recognise the measures we need to put into place with children, and as a consequence we make talking about Crizal® Prevencia® an integral part of our conversations with parents and children. We feel it’s very important to educate the public on points like the fact that children’s eyes are more sensitive than adults’ eyes due to the crystalline lens. In a young eye, the lens is more transparent, allowing more harmful rays to pass through and reach the retina. As the lens ages, it becomes less transparent, making it a little more difficult for shorter, more energetic rays to pass through. Children have exposure to an increasing amount of blue-violet light through the introduction of more and more LED internal lighting. Given that the damage is cumulative and that we can hope our children will live longer, over time these wavelengths are likely to be harmful to the eye. People with a family history of AMD are generally at greater risk, making this an important area to explore. From our older patients, we often hear comments such as “I wish I
had known that when I was younger” or, “I wish I had looked after my eyes when I was younger”. For some people it is too late to put measures into place, however they listen all the more attentively when we talk about their children and grandchildren. We want to ensure that they are aware that the world around us is becoming increasingly digital, yet their eye care choices may not reflect by the same ratio. Sometimes we need to simply reassure parents that it is okay for their children to be on the devices and this usually helps them to be more open to preventative measures. We are not here to judge what their children are doing, just to ensure that we are doing our job by recommending the best visual solution for them.

What are the drivers for successful selling process of Crizal® Prevencia®?

It helps at the dispensary to have samples to show patients along with a helpful leaflet provided by Essilor to endorse the patient’s decision. All our team wear Crizal® Prevencia®, reinforcing professional belief in the product. The optometrists, dispensing opticians and lens experts are confident about the product and believe it is their duty of care to inform and advise their patients of the latest innovations in protective lenses in this digital age.

Protection when outdoors is essential for long-term eye health, and we thus strongly advocate for it. Sunlight is
the most critical source of harmful blue-violet light. We thus focus our attention on understanding our patients’ lifestyles - not just what they do on a day-to-day basis but also how they live in general. We have invested significant time to re-visit the questions we ask our patients to achieve this. The handover from the optometrist to the lens expert consistently involves a recommendation of “new protective no-glare” from the optometrist. All of our optometrists are very comfortable in recommending this product, which really helps the lens experts expand their use of Crizal® Prevencia®. Our team have connected with Crizal® Prevencia® themselves and have taken the time to understand – and hence believe in - the key benefits to our patients. Crizal® Prevencia® is a lens which is up-to-date and in line with current living. Our team understands that Crizal® Prevencia® works by blocking 20% of the harmful blue violet light which in turn reduces retinal cell death rate by 25% without reflecting the blue-turquoise part of blue light. It is particularly innovative that this level of protection is available without completely undermining the cosmetic appearance of the lenses.

We are very aware that patients can become overwhelmed if we give them too much information. Careful thought has been given to the key words and phrases that are used, notably “protection, possible prevention” and “filtering out the dangerous blue light which can damage your macula”. Mentioning the sources of the harmful blue-violet light includes “sunlight and low-energy bulbs such as..."
LED” (which peak in blue part of the spectrum). As we work hard to build a relationship of trust with all our patients, lens experts report that most people are happy to go ahead without knowing the more complicated details. Our patients often say “Yes!! Because you say so!!”. Understanding the product and benefits ourselves and then adapting the conversation to suit patients has been a key part of the process. The team are confident that we can elaborate on facts about Crizal® Prevencia® and the harmful blue-violet light for those patients who want to know more.

What new learning around Crizal® Prevencia® dispensing have you uncovered?

We have been dispensing Crizal® Prevencia® within our Premium Lens package for the last couple of years and are now in a position to ask our patients for their feedback when they return for their next prescription. There has been very little decrease in uptake on future dispense of Crizal® Prevencia®, moreover many patients comment on their visual improvement when driving at night and that they like the blue hue to the lens. That said, there has been some less positive feedback, with the most common complaint being comments from friends or family about the blue hue. Recently, one patient reported that while she initially felt that she didn’t like that a friend had negatively commented on the blue hue, she knew she could see better, especially with night driving so she ignored her friend’s comment, and admitted to enjoying wearing them especially in lower lighting, with confidence that they are protecting her eyes.
Conclusion
I am confident that the product is essential for everyone involved with Crizal® Prevencia®. The passion from optometrists and lens experts over the benefits of this lens, is a driver for selling this product. When we began the launch a couple of years ago within our practice, we thus worked on giving as much support and information to our entire team about the advantages to the wearer. While this initially took some time, following regular practice sessions, 121 training sessions and support from Essilor, we now offer Crizal® Prevencia® to all our patients and it is applied to approximately 60 to 70% of all our coated lenses. This figure continues to grow as we increase the number of patients using Crizal® Prevencia® - as does our confidence in the Crizal® Prevencia® lens. A great lens innovation!

Key Takeaways
- The commitment of eye care professionals, training programmes from lens suppliers as well as patient education, are fundamental to delivering eye care excellence and business effectiveness.
- In addition to sunlight, the availability of new types of artificial lighting and digital screens increases cumulative exposure to harmful blue-violet light and may contribute to potential eye damage.
- Practice-based experience shows that Crizal® Prevencia® is an excellent asset in product mix improvement, patient care and satisfaction.
- Besides well-known photoprotective benefits of Crizal® Prevencia®, new patient knowledge shows that these lenses may help to improve dim light vision, especially when driving.
BLUE LIGHT: FROM SCIENTIFIC EVIDENCE TO PATIENT CARE

Blue light exposure, whether outdoors from the sunlight or indoors from LED backlit screens, can have deleterious effects. Based on a literature review, Victor Molina describes numerous ocular complications that blue-violet light exposure can induce. He also elaborates on preventive measures, specifically blue light filters, that eye care professionals have at their disposal, and describes who may benefit the most from these solutions.

Victor Molina is a qualified optometrist who graduated from Complutense University in Madrid with a Master of Optometry at the Centro Boston of Optometry in Madrid in 1998. He has managed the Divisions of Optometry and Contactology at the Spanish company Tu Visión (S.L) for the last 23 years.

He extended his expertise in the field of contact lenses with a Specialist Training in Contact Lenses at the Centro de Optometría Internacional of Madrid in 2000. He subsequently developed business-oriented skills by following a course in Executive Education in Company Training and the Corporate Program for Management at the ESADE Business School in Barcelona.

He has lectured in Clinical Optometry and was a professor in the Master’s program of Contact Lens Fitting at the European University of Madrid (UEM), as well as a lecturer of Contactology at the Universidad Nacional Autónoma de Managua in Nicaragua. He is currently responsible for Continuing Education at Tu Visión.

Victor has reported on ocular health via various media (television, radio, press) since 1993, and maintains an interest in Military History.

The digital revolution and the widespread use of smart mobile devices (telephones, tablets, e-readers, video display terminals [VDTs], etc.) provide on the one hand innumerable benefits, but have led to the rise of a variety of novel visual problems. One such problem is exposure to blue light. Blue light is mostly emitted outdoors by the sun, but is also found indoors in “cold-white” light emitting diodes (LEDs) present in the lighting systems of most backlit screens, televisions, etc. in an increasing number of homes, workplaces and shops. LED lighting has replaced the traditional bulbs and fluorescent tubes as the most efficient form of lighting in terms of energy.

Blue light is the part of the visible spectrum closest to ultraviolet radiations, and is composed of short wavelengths ranging from 380 to 495 nm corresponding to the highest energy of visible light. It has been linked to various ocular problems that can be classified into five major groups: visual fatigue, dry eye, discomfort from glare (these three elements being closely linked), retinal cell damage, and alteration of physiological cycles (sleep, tiredness, depression, irritability). Scientific evidence for the link between blue light and these problems is reviewed here.

1. Blue light and visual fatigue
Reading or working with LED backlit screens increases visual fatigue which manifests as tensinal and ocular symptoms with respect to other visual media or to written text. It is not yet clear if the high intensity of the blue light emission by these digital screens is the unique cause of such eyestrain or if it is a cumulative factor added to ocular accommodation, convergence and postural...
problems, which are also affected by working on computers.5,6 In any case, there has been a spectacular increase in recent years in the number of consultations related to computer visual syndrome (CVS). CVS is defined as “the set of visual and ocular problems related to close work caused by the use of computers,”7, to which we should no doubt add “and with the use of digital mobile devices.”

2. Blue light and dry eye
The sensation of dry eye and its many associated symptoms is closely linked to CVS.8,9 Dry eye in VDT users has been associated with reduced tear film break-up time.10 Regardless of whether it is productive or evaporative dry eye, the symptoms worsen when carrying out close-up activities with any type of digital screens equipped with blue light-emitting LED lighting.11,12,13,14 A close link has been demonstrated between tear film stability and visual function under blue light exposure.15 Patients with dry eye experience fluctuations in visual acuity17, which worsen when performing continuous close-up activities, particularly with screen use.18 Consequently, limiting exposure to short-wave blue light (in time and intensity or by filtering) would help reduce visual difficulties for patients suffering from dry eye with low break-up time.16

We also need to take into account the fact that complaints by these patients of a sensation of glare or halos increase when working with sources of back lighting which emit blue light, a far stronger source of light scattering than longer wavelengths.16

3. Blue light and glare
Glare may be defined as the loss of contrast in the retinal image resulting from or triggered by light scatter or stray light, due to entopic phenomena -intraocular light dispersion (ILD)- or by the presence of an object in the visual field or between the light source and the eye or in the presence of a source of intense light.19,20 There are several types of glare; disability glare such as oncoming car headlights when driving at night, adaptation glare with traumatic mydriasis, dysphotopsia referring to halos observed after photorefractive or cataract surgery (or a similar phenomenon due to lack of media transparency or instability of the tear film), and discomfort glare e.g. induced by a window in an improperly fitted office or reflections on a tablet screen.19, 21 Irrespective of the type of glare and its origin (external or entopic), blue light - and thus LED lighting - is a cumulative factor.

FIG. 1 Superficial Punctate Keratitis from a patient suffering of Sjögren’s Syndrome. © Víctor Javier García Molina

FIG. 2 Cataract, besides progressive low vision a great source of intraocular glare. © Víctor Javier García Molina
LED lights are found in both backlit screens as well as environmental lighting (offices, shops, televisions...). They provide more luminosity more efficiently than other types of lamps, and a large proportion of their spectrum is short wavelength, increasing the visual field and peripheral vision (explaining their widespread use in the car industry). However they result in a greater sensation of discomfort from glare than other types of lamps, with increasing discomfort associated with increasing blue light in the light source. And related to entopic phenomena, blue light is directly associated with increased intraocular light diffusion, which assumes increased significance when there is a lack of media transparency, such as with keratitis resulting from dry eye (Fig. 1) or in the case of age-related cataracts, which, as they progress, increase the patient’s sensitivity to light intensity and glare. (Fig. 2) This is also true in cases of macular dysfunction where the retinal image is formed with difficulty (age-related macular degeneration [AMD], central serous chorioretinopathy, etc.).

4. Blue light and retinal cell damage
Age, smoking, alcoholism, skin type (I, II, III, IV), exposure to ultraviolet (UV) radiation, genetics, refractive and nutritional factors (antioxidant deficiency, vitamin-poor diets, etc.), and systemic disorders (diabetes, high blood pressure, etc.), have habitually been cited as risk factors for the most common visual diseases and non-refractive ocular complications, AMD and cataracts. Added to this is exposure of the eye to the effects of visible light, specifically to short wavelengths of blue light having potential link with AMD. (Fig. 3 & 4)

In vitro studies have linked prolonged blue-violet light exposure to various kinds of retinal (photochemical) damage due to oxidative processes and the generation of free radicals. It increases production of reactive oxygen species (ROS) such as hydrogen peroxide and superoxides. These ROS are highly toxic and are related to photoreceptor cell death promoted by oxidative stress, their generation also induces Mytogen Activated Protein Kinases (MAPK) activation which modulates inflammatory mechanisms, stress and cell death too, including retinal photoreceptors cells.

These in vitro processes have been linked to blue LED light exposure which, compared to green and white LED light, increases ROS production and causes photoreceptor-derived cell damage. Nonetheless, the wavelength-dependent effect and its influences on white LED light-induced retinal degenerations is not yet fully understood. The role played by exposure to the visible light spectrum in potentially damaging the retina and its pigment epithelium, and of photochemical damage involved in the development of AMD was established in the late sixties.

Several studies have demonstrated the mechanisms leading to increased ROS production. The cumulative absorption of blue-violet radiations of the electromagnetic spectrum by visual pigments (melanin, melanopsin, flavoproteins or lipofuscin) increases ROS production which is in turn linked to the development of AMD. Nonetheless, the role of blue-violet light in the pathogenesis of AMD remains controversial mostly because of the limitations of the past in vitro studies.

5. Blue light and physiological cycles
As daytime animals, human physiology is in part regulated by light. Blue-turquoise light ranging between 465 and 495 nm, plays a critical role in linking our internal biological cycles, the circadian rhythms, to the external environmental conditions. Dysfunctioning of these cycles is linked to a wide range of psychological disturbances and...
systemic diseases: sleep disorders, depression, anxiety, obesity, diabetes, heart disease and stroke, cancer, etc. Among the various mechanisms that have been described to explain this connection, light, in particular blue-turquoise light, is the principal agent regulating our biological cycles and rhythms. The gradual opacification of the crystalline lens due to ultraviolet over-exposure, leading to the development of cataracts causes a reduction in light transmission, especially short wavelengths.44, 45 After a cataract operation, not only are visual capacities recovered, but quality of life improves with less depression, recovery of sleep routine, etc.46

Although outdoors blue light exposure may be effective in the treatment of various disturbances (affectional, cognitive, behavioral; increased reaction thresholds) and has a possible role in regulating refractive errors47, the fact remains that, continuous use of blue light-emitting LED backlit screens and exposure at times corresponding mainly (but not only) to night-time cycles, causes sleep alterations and behavioural disorders (irritability, asthenia, etc.) due to disrupted circadian rhythms.44, 48

6. A preventive armamentarium for eye care professionals
In the face of this wealth of scientific evidence, eye care professionals are asking the question as to how best we can assist our patients in preventing these eye complications. The initial approach for tackling this issue will require precise refraction and analysis of the binocular system to avoid asthenopic load induced by refractive, accommodative or convergence problems. Providing patient advice and guidelines for visual and postural hygiene to reduce exposure times to blue light is also critical, in order to reduce the CVS load.49, 50, 51

The use of antioxidants such as N-acetylcysteine or NAC to avoid the production of ROS induced by blue-violet light52, is a potential tool to address some of these retinal complications. Without a doubt however, the use of selective filters to filter blue-violet wavelengths has proven to be the most complete solution to date.

The idea that selective filters can increase visual capacity, or reduce glare, light scattering and harmful effects associated with blue-violet wavelengths of the visible spectrum is not new. Many vertebrates (including humans) have intraocular filters which minimize the noxious effects of light. For humans this is the crystalline lens which filters ultraviolet light and the macular pigment that might provide some protection against the hazards of short wavelength visible light53. In 1933, the studies of Walls and Judd54 established a link between the presence of these natural intraocular filters which block blue light and the phenomenon of light scattering associated with the short wavelengths of the visible spectrum. This has the effect of increasing visual capacity, reducing glare and increasing object-background contrast.54, 55

Cataract surgery results in partial loss of our natural protection. Numerous studies have been carried out to assess the beneficial effects of intraocular lenses (IOLs) equipped with blue light filters under real life conditions compared to those without a filter. These studies report less discomfort from glare, improved visual quality, increased contrast sensitivity function, increased chromatic differentiation or heterochromic contrast capacity, and reduced visual response time.55, 56, 57, 58, 59

A similar link has been established with AMD, where scientific evidence has linked the role of blue light to the pathogenesis of AMD60 via the degenerative oxidative processes taking place in the retina.60, 61, 62

A similar situation may apply to blue light filters designed by the optical industry for this purpose, although irrespective of the data and proofs obtained in the laboratory the fact that the subjective sensation of visual benefit may vary significantly between individuals under real-life conditions should be taken into consideration, especially when evaluating subjects with visual pathologies.

Photo protective filters should block high energy radiations that can damage the eye and reduce glare that can be

« In vitro studies have linked prolonged blue-violet light exposure to various kinds of retinal (photochemical) damage due to oxidative processes and the generation of free radicals. »
uncomfortable or debilitating. Many studies have concluded that blue light filters are more efficient when they block wavelengths similar to those naturally filtered by the crystalline lens (under 450nm). At wavelengths below 460 nm, sensitivity to light and photophobia increases as the wavelength decreases. However they should not filter radiations higher than 480 nm in order to preserve critical physiological functions dependent on blue-turquoise light and the protective pupillary reflex against over-exposure to light. It is thus important that ophthalmic lenses filters selectively blue-violet radiations without filtering blue-turquoise light being then more efficient against glare and improving the CSF.

Visual acuity tests are inadequate to actually assess visual behavior and degree of comfort of our patients in real-life situations. Colour and good contrast sensitivity, both black/white and polychromatic, are a fundamental aspect of the evaluation given the increasing time spent looking at documents, web pages, video games, etc. Thus filters that increase contrast are an important tool for eye care professionals. They are particularly of value in the presence of glare and/or lack of media transparency which reduce image quality for patients suffering from cataracts, chronic keratitis and dry eye, pupillary defects, post-surgery refraction, etc. In such cases, measurement of contrast sensitivity function is essential to ensure optimisation with ophthalmic filters.

7. Who benefits from blue light filters?
The most obvious answer would be to cite the entire list of ocular conditions previously mentioned. These are all conditions in which glare and loss of contrast sensitivity function and the disorders induced by them play an important role in visual discomfort. Similarly, patients with AMD are at higher risk, since blue-violet light exposure may contribute to their disease as is anyone exposed to sources of blue light or an environment dominated by cold-white LEDs.

At this point we can ask, who is not exposed to these conditions? From the digital dad who shops on the internet, to the digital mom who surfs web pages, the grandmother who talks on Skype, the international businessman who conducts business on a tablet, the child who chats via smartphone, the teenager who spends hours on a video console. All of us, whatever our age, are liable to experience some of the visual complications associated with increasing exposure to blue light.

However, in a recent survey of Spanish visual health professionals, only 40% cited the 0 to 15-year-old age group as a range for whom lenses filtering blue-violet light should be prescribed and, likewise for patients aged 65 and up, only 30% of the respondents considered the prescription of such lenses necessary. This is ironic given that 65 and up it is an age range most likely to experience pathological retinal changes, where media transparency makes the use of filters that provide adequate contrast sensitivity function and protection against glare even more important; and children are one of age groups more exposed to the widespread use of digital led-light devices (Fig. 5). Moreover and surprisingly, only 54% of respondents believed that any user will benefit from using a blue light filter, while the remaining 46% considering them only necessary for patients requiring them for professional activities (truck drivers, computer technicians, office jobs, etc.), or for ocular diseases.
Conclusion
Blue light is ubiquitous and is associated with a dichotomous effect, being both beneficial (blue-turquoise light) as well as noxious (blue-violet light). Some symptoms due to blue-violet light exposure may be cumulative, however everyone is concerned by this risk. We should raise awareness on the need to protect our eyes from the potential risks of cumulative exposure to blue-violet light, starting with professionals as well as our patients. So let’s forge ahead with the “digital” era, but in a responsible manner! •

KEY TAKEAWAYS

• Investigations into cumulative exposure to blue-violet light support a link with various visual conditions and pathologies.

• The literature implicates blue light exposure in numerous visual symptoms ranging from visual discomfort induced by glare to alterations of physiologic pathways resulting in retinal cell death.

• Reducing ocular exposure and absorption of blue-violet light improves critical visual functions such as contrast sensitivity and limits alterations like visual fatigue, glare or dry eye.

• Lenses filtering blue-violet light designed by the optical industry are useful to reduce blue light exposure and help eye care professionals to address a range of problems associated with retinal damage and offer a simple, effective solution to their patients.
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2. INDUSTRY SOLUTIONS
SUNGLASS and Rx STANDARDS - UV protection

Normas de gafas de sol con (Rx) y sin graduación - protección contra los UV

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Leader of Australian Delegation at ISO TC172/SC7, Liaison officer between ISO TC172/SC7 & ISO TC94/SC6
Australia

En un gran número de países existen normas para las gafas de sol, existe también una norma ISO para las gafas de sol graduadas y, próximamente, se publicará una norma ISO internacional de Gafas de Sol. Dichas normas tratan de los requisitos y métodos de prueba para limitar la transmittancia de los UV.

1/ ¿Cual es el motivo que impulsa esta dinámica de normalización sobre los límites de los uv?

Los profesionales de la atención ocular, los consumidores y los pacientes son cada vez más conscientes de los riesgos para la salud de la piel y los ojos de los rayos UV cuando uno se expone al sol.

En cuanto a la protección de la piel, se siguen observando mejoras significativas en la utilización de ropa protectora y pantalla solar así como una mayor concienciación e información sobre los riesgos de daños graves debido a la exposición solar.

No obstante llevar gorra o sombrero sólo brinda protección parcial a los ojos, especialmente si tomamos en consideración la radiación UV que se refleja desde el suelo (UVR).

Por lo tanto, las gafas de sol son la única manera efectiva para reducir significativamente la exposición de los ojos a los UV para llegar a niveles seguros además de reducir el deslumbramiento.

La demostración de los riesgos así como la concienciación del público a la exposición de los rayos UV desde el suelo (UVR) constituyen el motor principal de la elaboración de normas que pueden constituir pautas confiables para reglamentar la fabricación y el uso de las gafas de sol.

2/ Los riesgos de la exposición a los rayos uvr para el ojo

El gran número de estudios sobre los efectos nocivos de la exposición a los rayos UVR para los ojos han logrado que se acepte de manera generalizada que existe una relación significativa entre los niveles de exposición ocular a los rayos UVR y la incidencia y gravedad de toda una serie de trastornos incluyendo las cataratas, pterigión, ceguera del esquiador, degeneraciones maculares, tumores palpebrales así como el envejecimiento acelerado de la piel periorcular.

También hay riesgos inesperados. Se conoce que los efectos nocivos para la piel son máximos cuando el sol está en su punto más elevado.
And the exposure levels are not the same everywhere. People living in equitable climates, in latitudes closer to the equator, especially in the Southern Hemisphere, and in places with high number of sunny days per year receive up to 15% more exposure to UVR than the average.

The experts contributing to formulation of the eye protection standards interpret these risks, the science, and use data from recognised authoritative sources to formulate UV limits.

It is important that standards hold wide margins of safety to deliver the confidence in the sunglasses to protect well in all circumstances and location of wear.

3/ Consumer awareness and expectations

In a recent consumer survey of purchasing behaviour across all major markets, the following result was obtained.

These sunglasses:

- Protect your eyes from bright light
- Protect your eyes efficiently from UV and harmful rays
- Allow you to relax your eyes
- Allow you to have a good quality of vision
- Has high quality
- Respect the perception of the environment
- Allow you to have the frame that you want
- Are recommended by an eye care professional
- Allow you to look good
- Allow you to have a dark tint
- Are technologically innovative
- Are adapted to a specific activity
- Are made by a reputable / wellknown lens brand
- Are made by a reputable / wellknown frame brand

For Eyeglass wearers who wear plano sunglasses (measure of importance to consumers when purchasing sunglasses, scale 1-10).

Los ojos, al estar ubicados dentro de sus órbitas, se encuentran parcialmente protegidos cuando el sol está en su punto más alto. En cambio, en la mayoría de las estaciones, la máxima exposición ocular a los UV solares ocurre entre las 8 y 10 de la mañana y entre las 2 y 4 de la tarde, lo cual no era pereceivable.

Además, los niveles de exposición no son los mismos en todas partes. Las personas que viven en climas cálidos, en latitudes más cercanas al ecuador, especialmente en el hemisferio sur, y en lugares con un gran número de días soleados al año, reciben hasta el 15% más de exposición a los rayos UV que la media.

Los expertos que contribuyen a la formulación de normas de protección ocular interpretan estos riesgos así como los elementos científicos y utilizan datos de fuentes reconocidas para formular los límites de los UV.

Es importante que las normas contengan márgenes amplios de seguridad para que el portador confíe en las gafas de sol y pueda gozar de una buena protección en todas circunstancias y lugares.

3/ Concienciación del consumidor y expectativas

En una reciente encuesta de consumidores sobre sus comportamientos de compra, realizada en todos los mercados principales, se obtuvieron los resultados a continuación.

Estas gafas de sol:

- Protegen tus ojos de la luz brillante
- Protegen tus ojos eficazmente de los UV y los rayos nocivos
- Permiten que tus ojos se relajen
- Permiten una buena calidad de visión
- Son de buena calidad
- Respetan la percepción del entorno
- Te permiten utilizar la montura que desees
- Te dan una buena apariencia
- Te brindan la posibilidad de tener un buen bronceado
- Son de alta tecnología
- Están adaptadas a una actividad específica
- Fabrican una marca conocida de lentes
- Están a la moda
- Fabrican una marca conocida de monturas

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For Eyeglass wearers who wear plano sunglasses (measure of importance to consumers when purchasing sunglasses, scale 1-10). Para los portadores de gafas de sol no graduadas (medición de la importancia para los consumidores cuando compran gafas de sol, escala 1-10).
This indicates that UVR protection is very important for consumers. Standards provide a reference for regulation against which the performance of products can be determined. The standards set a benchmark for performance based on the best scientific information available.

Good standards are an agent to prevent the sale and use of sunglass products which perform badly or give poor protection.

Confidence created by the active use of standards generates increased sales of sunglasses. Sales volumes are underpinned by standards which guarantee good performance.

In parallel with the increased awareness for plano sunglass consumers, we also see an increase in the use of prescription tinted lenses or Rx sunglasses. The eye care professionals and their patients are becoming more aware of the protective benefits from providing a second pair of Rx sunglasses for times when exposure to the sun may be higher than usual.

4/ The standards for sunglasses

PLANO POWER

AUSTRALIA: AS/NZS1067:2003 (with amendments)\(^2\),

Australia published the first general purpose sunglass standard in 1971 which is the only sunglass standard enacted in law. (Australian Federal Government Trade Practices Act).

Compliance is assessed and enforced by the ACCC (Australian Competition and Consumer Commission). It is therefore mandatory.

Australia has a combination of a geographic location much of which is close to the equator, with a high number of sunny days/year, and is influenced by the fact the earth is always nearer to the sun in the Australian summer than during the Northern Hemisphere summer. In addition, the air is cleaner in the Southern Hemisphere than in the North, so more UVR reaches the earth’s surface. In addition, the lifestyle is very much outdoors-oriented in Australia. The combination of these effects means that Australians receive approximately 15% more solar UVR than those living in equivalent locations in the Northern Hemisphere.

It explains why Australia’s standards have a very strong focus on protecting its citizens by maintaining tough UV protection requirements for sunglasses, and enforcing that by law.

Australia maintains 400 nm as its defined upper limit of the range considered to be UV, while the other sunglass standards use 380 nm.

Regulation in the Australian sunglass industry imposes large fines, and non-compliant sunglasses banned from sale – sometimes involving big brand names.

USA: ANSI Z80.3:2010 Non-prescription sunglasses and fashion eyewear\(^1\)

This standard was created and is regularly updated by an ANSI-accredited committee of experts, and the Sunglass Association of America is the chair for the committee.

Esto indica que los consumidores consideran la protección contra los rayos UVR algo muy importante.

Las normas son una referencia para la elaboración de normativas y la eficacia de los productos puede determinarse con arreglo a las mismas. Las normas establecen un comparativo de eficacia basado en la mejor información científica disponible.

Las buenas normas son el medio adecuado para evitar la venta y la utilización de gafas de sol que tienen poca eficacia o baja protección.

La confianza que se genera a través de la utilización activa de las normas también aumenta las ventas de las gafas de sol. Los volúmenes de venta se ven potenciados por normas que garantizan una buena eficacia.

Paralelamente a la mayor concienciación de los consumidores de gafas de sol no graduadas, también se observa un aumento en la utilización de lentes tintadas con prescripción o gafas de sol Rx. Tanto los profesionales de la atención ocular como sus pacientes son cada vez más conscientes de los beneficios de protección que brinda un segundo par de gafas de sol graduadas para aquellos momentos en los que la exposición solar pueda ser más elevada de lo habitual.

4/ Las normas para las gafas de sol no graduadas

LA IMPORTANCIA DE LAS GAFAS DE SOL NO GRADUADAS

AUSTRALIA: AS/NZS1067:2003 (y sus anexos)\(^2\),

Australia publicó la primera norma de gafas de sol de uso general en 1971 y constituye la única norma de gafas de sol promulgada en ley. (Australian Federal Government Trade Practices Act).

La Comisión Australiana para los Consumidores y la Competencia ACCC (Australian Competition and Consumer Commission) es el organismo a cargo de evaluar la conformidad y de asegurar su aplicación. Esta norma es, por lo tanto, de obligado cumplimiento.

Australia combina una ubicación geográfica cercana al ecuador, con un número elevado de días de insolación al año; además, la Tierra siempre se encuentra más cerca del sol en el verano australiano con respecto al verano del hemisferio norte. Cabe añadir que el aire es más limpio en el hemisferio sur que en el hemisferio norte, de manera que hay una mayor cantidad de radiación UVR que alcanza la superficie terrestre. A esto hay que añadir que, en Australia, el estilo de vida está muy orientado hacia actividades exteriores. La combinación de estos factores significa que los australianos reciben aproximadamente el 15% más de radiaciones solares UVR que las poblaciones que viven en sitios equivalentes en el hemisferio norte. Esto explica por qué las normas australianas hacen un fuerte hincapié en la protección de sus ciudadanos al exigir niveles de protección muy exigentes contra las radiaciones UV en las gafas de sol e incluso han sido incorporados en la ley.

Australia mantiene los 400 nm como límite superior del rango considerado UV, mientras que otras normas relativas a las gafas de sol llegan a los 380 nm.

La normativa que rige la industria de las gafas de sol australiana impone multas considerables y las gafas de sol no conformes quedan prohibidas a la venta. Ya se han visto algunos casos, incluso de grandes marcas.
The standard is not mandatory, but relies on voluntary manufacturer-regulation. However, nonprescription sunglasses are classified and regulated by FDA as Class I devices in accordance with Title 21 of the Code of Federal Regulations (CFR). Sunglasses that are imported into the US must comply with country of origin marking requirements in the United States Tariff Act. Manufacturers and initial importers/distributors must register their establishments with the FDA annually and foreign manufacturers must also designate a U.S. Agent.

Nonprescription sunglasses are generally marketed as “Over The Counter” medical devices and are subject to general labeling and OTC labeling requirements outlined in Title 21 CFR Part 801 - Labeling.

There are 4 classifications in Z80.3-2010 used to define the UV transmittance and traffic signal recognition requirements.

Sunglasses which comply with the traffic signal recognition requirements are categorised as cosmetic (luminous transmittance \(Tv >40\%\)), or General Purpose (Tv from 8 to 40%). If sunglasses in these two categories don’t meet the traffic signal recognition requirements, they must be labeled “not intended for driving”.

**EUROPEAN UNION: EN1836:2005 + A12007 Sunglasses and fashion spectacles**[^3]

Sunglasses cannot be sold in Europe without the CE mark. The CE mark is a claim of compliance with the PPE EU Directive 89/686/EEC. The normal way to comply with the Directive is to comply with the EN1836:2005 standard.

Compliance is by self-declaration and there is little evidence of surveillance of compliance.

EN1836 has 4 transmittance or tint categories requiring different UV transmittance limits.

The standard has means to verify claims about UV transmittance (and absorption) for Solar UV, UV-A, UV-B and for blue light.

While a study in UNSW Australia found that 17% of CE marked sunglasses did not comply with the EN1836 standard, only a small 1.8% failed for the UV requirements.

This is a vast improvement on past surveys, and indicates the sunglasses did not comply with the EN1836 standard, only a small 1.8% failed for the UV requirements.

**EEUU: ANSI Z80.3:2010 Gafas de sol sin prescripción y gafas de moda**[^1]

Una comisión de expertos acreditados por la ANSI elaboró esta norma que es objeto de actualización regular. La Asociación de Gafas de Sol de EEUU (Sunglass Association of America) preside dicha comisión.

Esta norma no es obligatoria pero se basa en un cumplimiento voluntario por parte de los fabricantes. No obstante, las gafas de sol no graduadas son estandarizadas y reglamentadas por la FDA como dispositivos de Categoría 1 y conformidad con el Título 21 del Código Federal de Regulación (CFR). Las gafas de sol que se importan en territorio EEUU deben estar conformes con los requisitos de marca del país de origen como lo estipula la Ley de Aranceles de EEUU (United States Tariff Act).

Los fabricantes y distribuidores/importadores iniciales deben registrar su establecimiento con la FDA anualmente y los fabricantes extranjeros deben, además, designar un agente en EEUU.

Las gafas de sol no graduadas son generalmente comercializadas como dispositivos médicos en venta libre (“Over The Counter”) y están sujetas a etiquetado general y, por lo tanto, a los requisitos de etiquetado OTC incluidos en el Título 21 CFR Sección 801 - Etiquetado.

Existen 4 categorías en Z80.3-2010 que se utilizan para definir la transmittancia de los UV y los requisitos de reconocimiento de las señales de tráfico.

Las gafas de sol conformes a los requisitos de reconocimiento de las señales de tráfico son clasificadas como dispositivos cosméticos (transmitancia luminosa \(Tv >40\%\)), o de Uso General (Tv de 8 a 40%). Si las gafas de sol de estas dos categorías no cumplen con los requisitos de reconocimiento de las señales de tráfico, éstos deben tener la mención “no utilizar para conducir”.

**UNIÓN EUROPEA : EN1836:2005 + A12007 Gafas de sol y filtros solares**[^2]

Las gafas de sol no pueden venderse en Europa sin el marcado CE. El marcado CE significa que las gafas son conformes a la Directiva PPE EU 89/686/CEE. La manera normal de cumplir con esta Directiva es cumplir con la norma EN1836:2005.

La conformidad se determina mediante auto-declaración y existen pocas pruebas de comprobación de dicha conformidad.

La EN1836 tiene 4 transmisionces o categorías de tinte con diferentes límites de transmittancia de los UV.

La norma está dotada de los medios para comprobar la afirmación sobre la transmittancia (y absorción) de los UV solares, UV-A, UV-B y la luz azul.

Mientras que un estudio de la UNSW de Australia reveló que el 17% de las gafas de sol con marcado CE no cumplían con la norma EN1836, sólo un pequeño 1.8% no cumplier con los requisitos relativos a los UV. Esto constituye una gran mejora con respecto a estudios anteriores y es una indicación de que los fabricantes de gafas de sol han respondido bien a la demanda de una buena protección contra los UV.

[^1]: The standard has not been published in China so it doesn't have a number or year of publication.
[^2]: La norma no ha sido publicada en China y, por lo tanto, no tiene un número que corresponda al año de publicación.
ISO 12312.1 Eye and face protection – Sunglasses and related eyewear


5/ UV requirements in the major standards

The following is an informative Annex in the ISO12312.1 Sunglass standard.

“The eyes have a natural aversion response to bright light that limits outdoor filter exposure when one is not wearing sunglasses. This aversion response that provokes squinting limits filter exposure greatly, but sunglasses without side shields may permit peripheral exposure of biological significance due to the Corneo effect: The analytic characterization of ultraviolet skylight, as adapted for calculating corneal irradiance show that the largest influence on filter exposure in temperate regions is the seasonal variation of solar irradiance as adjusted by ground reflectance and the time from solar noon. Diffuse sky radiation decreases with increasing altitude, and corneal irradiation varies significantly with lid opening and ground cover.

The adopted transmittance limits are based on calculations of the biologically weighted exposure doses. The ultraviolet transmittance limits for sunglasses will keep these doses below a recognized safe limit even for exceptional daily exposure except over snow. Further margins of safety to account for tropical conditions or walking over snowfields in late spring have been incorporated. This has been done by adding additional safety factors to those implicit in the exceptional exposure experiences at mid-latitudes over normal terrain.

The specification of spectral (instead of average or weighted) transmittance limits provides a further very large increase in the margin of safety.”

There are some differences in the way the UV requirements are defined in the various sunglass standards. Some specify spectral transmittance limits for specified wavelength bands, while others set integrated transmittance limits.

But in practice, studies show that the number of sunglasses passing one standard and failing another is exceedingly small. UV protection is almost guaranteed with modern sunglass lens materials.

China (RPC) GB xxxx-1-20xx¹ Protección de los ojos y del rostro - Gafas de sol y dispositivos asociados - Sección 1 Gafas de sol de uso general².

Este es un nuevo borrador de norma recién redactada y que está en espera de aprobación antes de su publicación. Va a sustituir a la Norma de la Industria Gafas de Sol y se basa en la EN1836. Se ha adaptado para acercarse a la ISO12312.1 pero con requisitos netamente más exigentes relativos a los UV.

ISO 12312.1 Protección de ojos y rostro - Gafas de sol y dispositivos asociados³


5/ Requisitos relativos a los uvr en las normas principales

El texto siguiente es un Anexo informativo de la ISO12312.1 sobre la norma de gafas de sol

“El ojo tiene una respuesta natural de aversión a la luz brillante lo que limita la exposición del filtro en exteriores cuando uno no lleva gafas de sol. Esta respuesta de aversión que provoca la necesidad de entrecerrar los ojos limita ampliamente la exposición al filtro, pero las gafas de sol sin protectores laterales pueden permitir exposición periférica de significación biológica debido al efecto Corneo: la caracterización analítica de la luz ultravioleta del cielo, adaptada para el cálculo de la irradiancia córnea, muestra que la mayor influencia de la exposición del filtro en regiones templadas es la variación estacional de la irradiancia solar ajustada a la reflectancia del suelo y el tiempo transcurrido desde el mediodía solar. La radiación difusa del cielo disminuye con mayor altitud y la irradiación corneal varía significativamente con la apertura de los párpados y el recubrimiento del suelo.

Los límites de transmittancia adoptados se basan en cálculos de las dosis biológicas de exposición ponderadas. Los límites de transmittancia ultravioleta para las gafas de sol mantendrán estas dosis por debajo de un límite seguro reconocido incluso en una exposición diaria excepcional, salvo cuando hay nieve. Se han incorporado márgenes de seguridad adicionales en caso de condiciones tropicales o de marcha en campos nevados hacia finales de la primavera al añadir factores de seguridad adicionales a los implícitos en las experiencias de exposición extraordinaria en latitudes medias en terrenos normales. La especificación de los límites de transmittancia espectrales (en vez de una media o un valor ponderado) brinda un aumento significativo adicional en el margen de seguridad”.

Existen algunas diferencias en la forma en la que se definen los requisitos UV en las diversas normas de gafas de sol. Algunas especifican los límites de transmittancia espectral para las bandas de longitud de onda específicas, mientras que otras establecen límites de transmittancia integrados.

Pero en la práctica, los estudios muestran que el número de gafas de sol que aprueban una norma y fallan en otra es ínfimo. La protección contra los UV es prácticamente garantizada con los materiales modernos de las gafas de sol.
Comparison of the major sunglass standards for UV requirements.

The categories

Generally sunglasses and Rx sunglasses are categorised according to the luminous transmittance.

Category 0 is \(0\% \leq Tv \leq 90\%

Category 2 is \(43\% \leq Tv \leq 80\%

Category 3 is \(18\% \leq Tv \leq 43\%

Category 4 is \(3\% \leq Tv \leq 18\%)

\[
\begin{array}{|c|c|c|c|}
\hline
\text{EN 1836: 2005} & \text{AS/NZS 1067: 2003} & \text{ANSI Z80.3: 2001} & \text{ISO DRAFT 12312.1} \\
\hline
280 - 315 \text{ nm} & T_{\text{UVB}} \leq 0.125Tv & T_{\text{UVB}} \leq 0.125Tv & T_{\text{UVB}} \leq 0.05Tv \\
280 - 315 \text{ nm} & T_{\text{UVB}} \leq 0.05Tv & T_{\text{UVB}} \leq 0.05Tv & T_{\text{UVB}} \leq 0.05Tv \\
315 - 380 \text{ nm} & T_{\text{UVA}} \leq 0.5Tv & T_{\text{UVA}} \leq 0.5Tv & T_{\text{UVA}} \leq 0.5Tv \\
315 - 380 \text{ nm} & T_{\text{UVA}} \leq 0.5Tv & T_{\text{UVA}} \leq 0.5Tv & T_{\text{UVA}} \leq 0.5Tv \\
315 - 400 \text{ nm} & T_{\text{UVA}} \leq 1\% & T_{\text{UVA}} \leq 0.25Tv & T_{\text{UVA}} \leq 0.25Tv \\
\hline
\end{array}
\]

Tv is the luminous transmittance

\(T_f(\lambda)\) is the spectral transmittance

\(T_{\text{UVB}}\) is the solar UVB transmittance

\(T_{\text{UVA}}\) is the solar UVA transmittance
Claims for UV transmittance or absorption

All sunglass standards have the means to verify claims for a specific % transmittance or absorption.

For example, the ISO standard has “In the case where it is claimed that a filter has x % UV absorption, the solar UV transmittance of the filter TSUV shall not exceed 100.5 - x %.”

So for a sunglass claimed to absorb 99% UV, the solar UV transmittance shall not exceed 1.5%.

Prescription Sunglasses

ISO 8980.3:2003 Transmittance for finished uncut spectacle lenses is the international reference for prescription tinted lenses and Rx sunglasses. It was formulated and maintained by ISOTC172/SC7/WG3.

The UV requirements are not as tough as for plano sunglasses.

UVB – for category 0, TSUVA shall be ≤Tv, for categories 1 to 3, TSUVA ≤0.125Tv, and category 4 TSUVA ≤1% absolute

UVA – For categories 0 to 2, TSUVA shall be ≤Tv, and for categories 3 and 4, TSUVA ≤0.5Tv

For photochromic lenses the UV requirements must be met both in the dark and light states.

There is a constant challenge for the committees in ISOTC94/SC6 eyeprotection and ISOTC172/SC7/WG3 spectacle lenses to ensure that the requirements in the sunglass standards are not in conflict with those in the prescription transmittance standard.

6/ How is UV performance measured?

There are some differences between the standards but the most up-to-date methodology is in the latest draft of ISO12311 Test methods for sunglasses.

Measurement is permitted with spectrophotometric equipment capable of measuring spectral transmittance with specified uncertainties.

Measurements are made normal to the surface of the lens.

The spectral values are measured at no more than 5nm intervals and the solar UV values calculated by integrating over the specified range of wavelengths taking into account the spectral distribution of sunlight and the spectral sensitivity of the eye. Data is provided in the standard to calculate

• Luminous transmittance Tv
• Solar UV transmittance TSUVA
• Solar UV-A transmittance TSUVA
• Solar UV-B transmittance TSUVB

7/ How do the standards define UV?

Since spectacle lens and sunglass standards define 380 nm as the upper limit of the UV range, there is opportunity for manufacturers to make claims like “UV400” – or similar for another wavelength.

Afirmaciones de la transmittancia o absorción de los UV

Todas las normas de gafas de sol deben contar con los medios para comprobar las afirmaciones sobre un porcentaje específico de transmittancia o absorción. Por ejemplo, en la norma ISO: “En el caso de que se afirme que un filtro tiene x % de absorción de los UV, la transmittancia UV del filtro TSUV no debe exceder el (100.5 - x) %.”

Es decir, para unas gafas de sol de las que se afirma que absorben el 99% de los UV, la transmittancia de los UV solares no debe exceder el 1.5%.

Gafas de sol graduadas

La ISO 8980.3:2003 sobre la transmitancia de lentes de gafas sin cortar, acabadas, es la referencia internacional para las lentes tintadas graduadas y las gafas de sol graduadas. Fue formulada y mantenida por la ISOTC172/SC7/ZG3.

Los requisitos sobre los UV no son tan exigentes como en el caso de gafas de sol no graduadas.

UVB – para la categoría 0, TSUVA deberá ser ≤Tv, para categorías 1 a 3, TSUVA ≤0.125Tv, y categoría TSUVA ≤1% absoluto

UVA – para categorías 0 a 2, TSUVA deberá ser ≤Tv, y para categorías 3 y 4, TSUVA ≤0.5Tv

Para las lentes fotocromáticas, los requisitos deben cumplirse tanto en estado claro como en estado oscuro.

Se plantea un reto constante a las comisiones en la ISOTC94/SC6 sobre protección ocular y en la ISOTC172/SC7/WG3 de lentes de gafas para asegurarse de que los requisitos en las normas de gafas de sol no estén en conflicto con los requisitos de la norma de transmittancia de lentes graduadas.

6/ ¿Cómo se mide la eficacia contra los uv?

Existen algunas diferencias entre las normas pero la metodología más actualizada se encuentra en el último borrador de la ISO12311 sobre los métodos de prueba para las gafas de sol.

Se permite realizar las mediciones con equipo espectrofotométrico capaz de medir la transmittancia espectral con ciertos márgenes especificados.

Normalmente, las mediciones se llevan a cabo en la superficie de la lente.

Los valores espectrales se miden en no más de 5nm de intervalo y los valores de los UV solares se calculan integrando en un rango específico de longitudes de onda tomando en consideración la distribución espectral de la luz solar y la sensibilidad espectral del ojo. En la norma se proporcionan los datos para realizar el cálculo de:

• La transmittancia luminosa Ty
• La transmittancia de los UV solares TSUVA
• La transmittancia de los UV-A solares TSUVA
• La transmittancia de los UV-B solares TSUVB

7/ ¿Cómo se definen los uv en las normas?

Puesto que las lentes de gafas y las normas de gafas de sol definen los 380 nm como el límite superior del rango UV, se deja cabida para que los fabricantes afirmen “UV400”, o algo similar en otra longitud de onda.
Because it wasn’t possible in the ISO forums to agree a definition for UV400, it was decided to write a Technical Report called “Short Wavelength Visible”. This is intended to explain and educate those who are interested in the effects on the eye in this interface range and how lenses attenuate these effects.

The Technical Report is currently in formulation with contributions from experts around the world.

8/ A challenge for manufacturers

Increased public awareness of UV and the harmful effects has driven manufacturers to eliminate more and more UVR, to create sharper cutoff lenses, and to cut more and more of the blue light region.

The consequence of removing blue light is a possible yellowing of clear lenses and failure to comply with the coloration limits for traffic signal recognition.

The challenge is to create superior products recognising these limitations.

9/ ISO Sunglasses standard 12312.1

Since 2004, ISO committee TC94/SC6/WG3 has been developing the sunglass standard ISO12312.1 and its supporting test methods standard ISO12311. These standards are intended to be published simultaneously.

The UVR values in EN1836 were used as a starting point for 12312.1. The spectral values were replaced by integrated values, which are tougher, and the UVA requirements have been tightened also.

The ISO standards for sunglasses are close to completion and are expected to be published during 2012.

When the ISO standards are published, EN1836 will be withdrawn and the ISO standard will become the reference for claiming conformity to the EU Directive, allowing CE marking and sale in Europe.

10/ Trends for the future

The most significant event in the near future will be the publishing and adoption of the ISO sunglass standards.

Countries will need to decide about adoption of these new standards to replace their national standards – where they exist.

If there is a wide acceptance of the ISO standard that will benefit those engaging in cross-border trade in sunglasses since only one international standard will need to be respected.

Further in the future it is expected that UV protection requirements will become tougher. We can also expect labelling requirements to increase which will better inform consumers at point of sale about the protective level of products.

Puesto que no ha sido posible, en el marco de la ISO, llegar a un acuerdo de definición de UV400, se adoptó la decisión de elaborar un Informe Técnico denominado “Radiación Visible de Longitud de Onda Corta” (“Short Wavelength Visible”). Este informe tienen como objetivo explicar e instruir a aquellos que estén interesados en los efectos en el ojo de este rango de interfaz y cómo las lentes atenuan dichos efectos.

Actualmente, el Informe Técnico ha sido elaborado con contribuciones de expertos de todo el mundo.

8/ Un desafío para los fabricantes

La mayor concienciación del público en general sobre los efectos nocivos de los UV ha conducido a que los fabricantes eliminen cada vez más los UVR, a que fabriquen lentes que bloqueen más nítidamente y que bloqueen cada vez más los rayos de la zona de la luz azul.

Como consecuencia de la eliminación de la luz azul, se puede producir un posible amarilleo de las lentes transparentes y no cumplir así con los límites de coloración para el reconocimiento de las señales de tráfico.

El desafío consiste pues en elaborar productos superiores a la vez que se cumplen dichas exigencias.

9/ La norma ISO 12312.1 para gafas de sol

Desde 2004, la comisión ISO TC94/SC6/WG3 ha venido elaborando la norma ISO12312.1 para las gafas de sol y su norma de métodos de prueba ISO12311. Se tiene la intención de que dichas normas sean publicadas simultáneamente.

Se utilizaron los valores UVR de la EN1836 como punto de partida para la 12312.1. Los valores espectrales fueron sustituidos por valores integrados, más estrictos, y los requisitos UVA también son más exigentes.

Las normas ISO para las gafas de sol están a punto de ser terminadas y se espera su publicación en el transcurso del año 2012.

Cuando se publiquen las normas ISO, se retirará la EN1836 y la norma ISO se convertirá en la referencia para afirmar la conformidad con la Directiva EU, que permite el marcado CE y su venta en Europa.

10/ Tendencias futuras

El acontecimiento más significativo en el futuro próximo será la publicación y la adopción de las normas ISO de gafas de sol.

Los países tendrán que decidir sobre la adopción de estas nuevas normas para sustituir sus normas nacionales, en donde éstas existan.

Si existiera una amplia aceptación de la norma ISO, esto acarrearía beneficios a los que realizan actividades comerciales transfronterizas de gafas de sol puesto que sólo será necesario respetar una norma internacional.

En un futuro más lejano, se espera que los requisitos de protección de UV serán más exigentes. También es de esperarse mayores requisitos de etiquetado que puedan informar mejor a los consumidores en el punto de venta sobre el nivel de protección de los productos.
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9. ISO4007-2010 Eye and face protection - Vocabulary
Risk of UV exposure with spectacle lenses

Riesgo de exposición a los UV con las lentes de gafas

Anti-reflection (AR) coating is an excellent spectacle lens option for increasing luminous transmission of the lens, reducing glare, and enhancing the cosmetic appearance of the wearer. It is recommended for virtually all types of eyewear, including clear lenses for general, everyday use as well as nighttime driving; photochromic lenses for patients who frequently move between indoors and outdoors throughout the day; and occupational near and intermediate lenses for computer users indoors. AR coating should be applied to both lens surfaces, since it will decrease the direct and internal reflections that can occur at each surface. This will reduce glare from light sources both in front of and behind the wearer.

By the very nature of how AR coatings work, they will generally increase reflection of non-visible wavelengths, notably ultraviolet (UV) and infrared (IR). At typical levels in the natural environment, IR from sunlight gives the sensation of warmth on the skin, but poses little risk to the structures of the eye. On the other hand, short exposure of several hours to normal UV levels, or brief exposure to high levels of UV, can cause immediate and painful problems such as sunburn to skin and keratitis. Continued long-term exposure over months and years can cause or exacerbate conditions such as premature aging of the skin, cancer, pterygium, cataract, and macular degeneration.

For an AR coating applied to the front surface of a lens, the coating provides additional protection beyond the UV-absorbing properties of the lens itself. Different AR coatings can reflect 25% or more UV, depending on wavelength. By comparison, lenses with scratch-resistant coatings usually reflect no more than about 5% of any UV wavelengths, what would be expected of a typical uncoated ophthalmic material. Thus, with an AR coating on the front lens surface, harmful UV radiation now will be reflected back into the environment and away from the wearer’s eye. But the same AR coating on the back surface of the lens can actually increase the amount of UV incident at the eye. In addition, this will happen under viewing conditions and times of day when the wearer is least likely to be aware of any danger.

Many patients are familiar with the risk of sunburn in mid-day hours, from about 10 AM to about 2 PM, especially during summer months. However, Sasaki et al. demonstrated that most of the direct exposure...
of the eye to UV will occur mid-morning (before 10 AM) and mid-afternoon (after 2 PM) throughout the year, when the sun is lower in the sky and close to the wearer’s horizontal viewing plane. The potential risk of UV exposure is present either from the front, if the lens does not adequately block UV, or from the side, if the combined lens and frame do not provide appropriate coverage of the wearer’s face\textsuperscript{[6,9,14]}. With the consideration of possible UV reflection from the back surface of the lens, the risk is also greatest at these hours, but now when the wearer actually faces away from the sun! A recent study demonstrates that the UV reflection risk is greatest when the wearer is about 145 degrees from the sun, that is, with sunlight coming from behind the wearer, just over his or her shoulder\textsuperscript{[6]}. Figure 1 demonstrates eyewear that leaves the wearer’s eye exposed from the side and from behind.

The various international standards for prescription and non-prescription lenses address UV exposure only in terms of limiting or minimizing transmission through the lens\textsuperscript{[1,2,7,10,11,3]}. None of the standards address UV exposure caused by a lens that does not adequately cover the eye, thus leaving the eye exposed from the side or above.

Also, none of the standards address UV reflection from the back surface of the lens, which will depend not only on the AR coating but the size, curvature, wrap (faceform) angle, and vertex distance of the lens. This could leave the patient – and the practitioner! – with the mistaken impression that UV transmission through a finished lens is the only hazard that needs to be considered.

What can eyecare practitioners do to provide the best possible UV protection for their patients? In addition to minimizing visible wavelength reflection, the AR coating applied to the back surface of all prescription lenses intended for daytime use outdoors should minimize UV reflection, down to the wavelengths expected from sunlight in the natural environment at about 290 nm. A new index, the Eye-Sun Protection Factor (E-SPF)\textsuperscript{[6]}, informs the practitioner and the wearer about the UV protection provided by such a lens. It that takes into account UV transmission through the lens and UV reflection from the back surface of the lens, as well as the varying sensitivity of the cornea to different wavelengths within the UV spectrum. Technically, E-SPF can be determined empirically by measuring the UV incident at the eye first without and then with the lens in place, or it can be estimated by calculating the inverse of the sum of the UV transmittance and reflectance\textsuperscript{[6]}.

E-SPF is an index similar to that used for sunscreen products (see Urbach, 2001\textsuperscript{[17]}, for an excellent historical review) and ultraviolet protective clothing (see Gambichler et al., 2006\textsuperscript{[6]}, for a review of the development of the European standard, EN 13758), in that a higher category value indicates greater UV protection. The category value specifies the approximate multiple units of time necessary to receive a given exposure dosage: for example, with an E-SPF 25 lens, it would take about 25 minutes to receive the equivalent total dosage as 5 minutes for an E-SPF 5 lens.

Muchos pacientes están familiarizados con el riesgo de quemaduras de sol en las horas alrededor del mediodía, desde las 10 AM hasta las 2 PM aproximadamente, especialmente durante los meses de verano. No obstante, Sasaki et al.\textsuperscript{[15]} han demostrado que la mayoría de la exposición directa a los UV del ojo ocurran a media mañana (antes de las 10 AM) y a media tarde (después de las 2PM) a lo largo del año, cuando el sol está más bajo en el cielo y cerca del plano de visión horizontal del portador. El riesgo potencial de la exposición a los UV está presente ya sea de frente, si la lente no bloquea de manera adecuada los UV, o por los lados, si las lentes combinadas con las monturas no proporcionan la cobertura adecuada del rostro del portador\textsuperscript{[16,9,14]}. Si tomamos en consideración el hecho de que se reflejen los UV desde la superficie posterior de la lente, el riesgo también es mayor en estas horas y además ¿cuando el portador está dándole la espalda al sol? Un estudio reciente demuestra que el riesgo de recibir reflejos UV es mayor cuando el portador se sitúa a 145 grados con respecto al sol, es decir, cuando la luz solar viene por detrás del portador, justo por encima de su hombro\textsuperscript{[6]}. La figura 1 demuestra algunas gafas que dejan al ojo del portador expuesto lateralmente y por detrás.

Las diversas normas internacionales para las lentes de prescripción y las no prescritas abordan el tema de la exposición a los UV sólo en términos de limitación o reducción al mínimo de la transmisión a través de las lentes\textsuperscript{[1,2,7,10,11,3]}. Ninguna de las normas abordan la exposición a los UV ocasionada por lentes que no cubren adecuadamente el ojo y, por lo tanto, dejando al ojo expuesto lateralmente o por encima de las gafas.

Del mismo modo, ninguna de las normas trata del reflejo de los UV desde la superficie posterior de la lente, lo cual dependerá no solamente del tratamiento antirreflejante sino del tamaño, curvatura, ángulo de envolventamiento del rostro y la distancia al vértice de la lente. Esto puede dejar al paciente -y al profesional- con la impresión errónea de que la transmisión de los UV a través de las lentes finalizadas es el único riesgo que cabe tomar en consideración.

¿Qué pueden hacer los profesionales del cuidado ocular para suministrar la mejor protección posible a sus pacientes contra los UV? Además de minimizar el reflejo de longitudes de onda visibles, el tratamiento antirreflejante aplicado a la superficie posterior de todas las lentes de prescripción cuya utilización es de día y en exteriores, debería minimizar la reflexión de los UV, hasta longitudes de onda correspondientes a un día soleado en un entorno natural en aproximadamente 290 nm. Un nuevo índice, el Factor de Protección Solar (E-SPF)\textsuperscript{[6]}, informa al profesional y al portador sobre la protección que proporciona dicha lente. Este toma en consideración la transmisión de los UV a través de las lentes y los UV reflejados desde la parte posterior de la lente, así como la sensibilidad variable de la córnea a diferentes longitudes de onda dentro del espectro de los UV. Técnicamente, se puede determinar empíricamente el E-SPF al medir los UV incidentes en el ojo, primero sin la lente y luego con la lente colocada, o puede estimarse calculando lo inverso de la suma de la transmittancia y reflectancia de los UV\textsuperscript{[6]}.

El E-SPF es un índice similar al utilizado para los productos de protección solar (véase Urbach, 2001\textsuperscript{[17]}, para una excelente reseña histórica) y la rapa protectora contra los ultravioleta (véase Gambichler et al., 2006\textsuperscript{[6]}, para una reseña del desarrollo de la norma europea, EN...
The eyecare practitioner also should make appropriate frame recommendations to the patient, and adjustments to any dispensed eyewear, all of which derive from the proper positioning of the lens with respect to the eye[16,9,14]. This is especially relevant for over-the-counter non-prescription sun eyewear for contact lens wearers and patients who otherwise do not need a prescription. The best protection will be provided by a frame that is contoured with sufficient faceform and pantoscopic angles to fit closely to the wearer’s face and head (see Fig. 2). Such a frame often requires that the lens have a steep base curve, usually 6 D or greater. This may not be possible or practical for certain prescription powers.

If the frame has a relatively flat front, or when a high faceform angle is not possible or desirable, then it should have a wide temple or sideshield. But the frame horizontal dimension should not extend significantly past the side of the wearer’s face or head, even if the temple is wide. The frame vertical dimension should be enough to sufficiently cover the eye and extend upward to cover the brow, thus minimizing direct exposure of the eye from above. Finally, nosepads should be correctly chosen or adjusted to minimize the vertex distance.

Eyewear can be fashionable and functional. For patients who spend much of their time outdoors, it also needs to be protective. An appropriate AR coating on each lens surface, indicated by a high E-SPF value, as well as proper frame choice and fitting techniques, will contribute to the patient’s long-term eye health. ❑


8. Gambichler T, Laperre J, Hoffmann K. The European standard for sun-protective clothing: EN 13758), in the median in the que a un valor de categoría superior indica una mayor protección contra los UV. El valor de la categoría determina las unidades de tiempo aproximativas necesarias para recibir una dosis de exposición dada, por ejemplo, con una lente de E-SPF 25, serán necesarios unos 25 minutos para recibir la dosis total equivalente de 5 minutos con una lente E-SPF 5.

El profesional del cuidado ocular también debe dar las recomendaciones adecuadas al paciente sobre la montura y realizar los ajustes correspondientes a cualquier tipo de gafas prescritas, relativas al posicionamiento adecuado de las lentes con respecto al ojo[16,9,14]. Esto es particularmente pertinente para las gafas solares sin prescripción, para los portadores de lentes y los pacientes que no necesitan una prescripción. La mejor protección será proporcionada por una montura cuyo contorno se ajusta lo suficientemente bien a la forma del rostro y con ángulos pantoscópicos para ajustarse bien al rostro y cabeza del portador (Fig. 2). Una montura de este tipo supone que la lente tenga una base con una curva pronunciada, habitualmente de 6D o superior. Esto puede no ser posible o práctico en algunas potencias prescritas. Si la montura tiene una parte frontal relativamente plana, o cuando no es posible o deseable un ángulo elevado de contorno del rostro, entonces debería tener patillas anchas o protecciones laterales. No obstante, las dimensiones horizontales de la montura no deberían extenderse más allá de la parte lateral del rostro o cabeza del portador, incluso si las patillas son anchas. La dimensión vertical de la montura debe ser lo suficientemente grande para cubrir el ojo y extenderse hacia arriba para cubrir la ceja, por lo tanto, disminuyendo al mínimo la exposición directa del ojo desde arriba. Finalmente, se deben seleccionar cuidadosamente los soportes nasales o ajustarlos para minimizar la distancia al vértice.

Las gafas pueden ser funcionales y estar a la moda. Para aquellos pacientes que pasan una gran parte de su tiempo en el exterior, éstas también deben ser protectoras. Un tratamiento antirreflejante adecuado en la superficie de cada lente, indicado por un valor E-SPF, así como una selección adecuada de la montura y técnicas de ajuste, contribuirán a la preservación de la salud ocular del paciente a largo plazo. ❑

References- referencias


8. Gambichler T, Laperre J, Hoffmann K. The European standard for sun-protective clothing: EN 13758), in the median in the que a un valor de categoría superior indica una mayor protección contra los UV. El valor de la categoría determina las unidades de tiempo aproximativas necesarias para recibir una dosis de exposición dada, por ejemplo, con una lente de E-SPF 25, serán necesarios unos 25 minutos para recibir la dosis total equivalente de 5 minutos con una lente E-SPF 5.

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UV danger to the eyes

Chronic exposure of the eyes to UV radiation is a widely established public health problem (cortical cataract, pterygium, pinguecula, eyelid cancers…), and over 40% of our exposure to UV occurs during low to moderate sunshine situations\(^1\), in which we can wear our colourless spectacle lenses comfortably. However, due to the lack of information regarding the dangers of UV radiation and in the absence of a recognised protection factor for lenses which could help in their choice, it is still rare for consumers to take protection of their eyes into consideration when purchasing lenses for their spectacles.

Indeed, the foremost expectation expressed by spectacle wearers is clarity of vision. Therefore, to meet this requirement, anti-reflective lenses have gradually become the standard lenses offered.

What level of UV protection is really offered by the lenses which are currently on the market?

Organic materials, which absorb UV rays, offer near-complete protection against all frontal UV exposure. But recent studies\(^2\) show that the UV rays arriving from the sides and back of the lens, where they are reflected strongly by the anti-reflective treatment on the inner side, can represent up to 50% of the UV exposure suffered by the eye and its surroundings.

Indeed, although the anti-reflective lenses on the market are designed to be efficient against the reflection of visible light, they reflect on average 25%\(^2\) of the ultraviolet spectrum!

Crizal UV lenses were therefore created from the need to develop a new AR treatment ensuring protection for the wearer against UV light arriving on both sides of the lens!

1. See the articles in this issue referring to the dangers of UV for the eye and its surroundings.
2. Read the article in this issue by Karl Citek.
Designing the first anti-reflective lens that protects against UV
Spectral considerations: The Thelen formula

The foremost function of an anti-reflective treatment is to improve the transparency of the spectacle lens, reducing reflection from both sides of the lens.

Anti-reflective lenses, as designed and made in the ophthalmic industry, are based on the laws of interference. The principle consists of alternating layers of low index and high index materials in order to create destructive interference and therefore reduce as far as possible the level of reflection for the desired spectral range. Optimisation to wavelengths close to the visible involves depositing thin layers, the thickness of which is around a few tens of nanometres.

The main parameters used to improve the efficiency of anti-reflective treatment are now well known in the business. There is a mathematical formula, defined empirically by Thelen\(^3\), which shows their respective impact on the average reflection level of a stack of anti-reflective layers. In this formula it appears that reflection is an exponential function of the spectral band width on which one is seeking to optimise an anti-reflective coating. This shows that it is all the more difficult to reduce average reflection because it has to be optimised across an extended spectral range.

In the case of Crizal UV, the aim is specifically to achieve reduced UV reflection whilst maintaining the optimal level of transparency that characterises Crizal, Essilor’s premium range of anti-reflective lenses.

To achieve this we have succeeded in identifying a limited number of groups of multi-layer stacks characterised by highly specific combinations of thicknesses of these layers. Identification of these groups of stacks has resulted in an application for an international patent.

Geometric considerations

In addition to spectral considerations, optimisation of the performances of Crizal UV also meets considerations of a geometric or angular nature.

Figure 1 clearly illustrates that the share of light coming from behind the wearer and reflected by the rear side of the lens is contained in a solid angle of between 30° and 45°. This angular range has been defined by measurements made in experimental conditions representative of real life wearing conditions, and corresponds to the values given in scientific literature\(^4\)–\(^5\).

In summary, Crizal UV is a multilayer anti-reflective stack whose optical performance meets a twofold requirement, spectral and angular. This product is characterised by an optimal level of visual transparency in the direction facing the wearer, typically between 0° and 30° and by minimum reflection in terms of UV light arriving on the rear surface of the lens, at an angle of between 30° and 45°.

In order to explain and demonstrate the innovation brought by Crizal UV, we have designed a new demonstrator, which has been made available to the group’s various subsidiaries (see Fig 2).

Diseño de la primera lente antirreflejante que protege de los UV
Consideraciones sobre el espectro: fórmula de Thelen

La función principal de un tratamiento antirreflejo consiste en mejorar la transparencia de las lentes de gafas, disminuyendo la reflexión en ambas caras de la lente.

Los tratamientos antirreflejo diseñados y realizados en la industria oftálmica se basan en las leyes de la óptica interferencial. El principio consiste en alternar capas de materiales de índice bajo y de índice alto con el fin de crear interferencias destructivas y, por lo tanto, bajar así al máximo el nivel de reflexión en el tramo espectral deseado. La optimización en longitudes de onda cerca de lo visible implica depositar capas finas cuyo espesor es de algunas decenas de nanómetros de magnitud.

Los parámetros principales que permiten mejorar la eficacia de un antirreflejo son actualmente bien conocidos por los profesionales del oficio. Existe una fórmula matemática determinada empíricamente por Thelen\(^3\) y que muestra su impacto respectivo en el nivel de reflexión media de una superposición de antirreflejos. Según esta fórmula, la reflexión resulta ser una función exponencial de la longitud de banda espectral en el que se trata de optimizar un antirreflejo. Esto demuestra que es mucho más difícil disminuir la reflexión media en la medida en la que éste debe optimizarse en un tramo espectral extendido.

En el caso de Crizal UV, el objetivo es precisamente conseguir disminuir la reflexión de los UV a la vez que se mantiene el nivel de transparencia óptima que caracteriza a Crizal, la gama de lentes antirreflejos premium de Essilor. Para conseguirlo, hemos podido identificar un número limitado de familias de superposiciones multicasas caracterizadas por combinaciones muy específicas de espesores de capas finas. La identificación de estas familias de superposiciones ha dado lugar a una petición de patente internacional.

Consideraciones geométricas

Además de consideraciones sobre el espectro, la optimización de la eficacia de Crizal UV responde también a consideraciones geométricas o angulares. En la figura 1 se ilustra claramente la proporción de la luz que llega por detrás del portador, la que la cara interna de la lente refleja y que está contenida en un ángulo sólido entre 30° y 45°. Este tramo angular fue determinado por mediciones en condiciones experimentales representativas de las condiciones de porte en la vida real y que corresponde a los valores que también menciona la literatura científica\(^4\)–\(^5\).

En resumen, Crizal UV es una superposición de capas anti-reflejantes cuyas eficacias ópticas responden a una exigencia doble, espectral y angular. Este producto se caracteriza por un nivel de transparencia visual óptima en la dirección delante del portador, típicamente entre 0° y 30° y por un nivel de reflexión mínima de la luz UV que llega a la superficie de la cara interna de la lente entre 30° et 45°.
UV reflection factor

The requirement for low UV reflection implies being able to quantify it properly, taking account of the health risks associated with UVA radiation (315 nm - 380 nm) and UVB radiation (280 nm - 315 nm) on the human eye. To do this we used the existing international standard (standard ISO 8980-3 :2003) which proposes a calculation of the UV transmission factor applied to ophthalmic lenses. In this standard, the UV performance calculation is carried out using a weighting function $W(\lambda)$ (Fig. 3) which depends on:

- the direct sun radiation spectrum $ES(\lambda)$ received at the Earth’s surface – small amount of UVB compared to UVA, due to absorption by the ozone layer of rays between 200 and 300 nm,
- the relative efficiency spectral function $S(\lambda)$ or ‘function of UV risk’, which shows that UVB is more dangerous than UVA. This latter function $S(\lambda)$ expresses the biological risk linked to photochemical deterioration of the cornea, when it is exposed to UV.

We have therefore applied this function to evaluate reflection $R(\lambda)$ in UV, using the formula:

This factor is used in the calculation of the E-SPF$^3$ which is used to evaluate the level of UV protection offered by ophthalmic lenses (Fig. 3).

Characterisation of performances

The development of Crizal UV has required new characterisation methods. Firstly in the R&D phase, spectral ellipsometry and variable angle spectrophotometry, in both UV and visible, were used to characterise all materials, from the substrates to the thin layers. Measurement methods based on the same principles were adapted and deployed at production sites in order to guarantee the performance levels of this new product, from both a spectral and an angular point of view.

The UV protection provided by low level UV reflection, $(R_{UV})$, from 5 to 10 times less than that measured on the anti-reflection coated lenses of the main manufacturers$^4$, thus means an E-SFF protection factor of 25 for colourless Crizal Forte UV lenses, and 50+ for Crizal Sun UV sun lenses.

1 Read the article in this issue by Karl Citek.

2 Best UV protection for Crizal Forte UV lenses according to the E-SFF factor compared with colourless anti-reflective lenses in equivalent materials with the best anti-reflective properties produced by other main manufacturers on the market. Lens performance measurement only: the E-SFF factor does not include UV radiation that enters the eye directly without interaction with the lens, which depends on external factors (the wearer’s morphology, frame shape, wearing conditions, …). E-SFF measurements: independent body, USA, 2011.

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Para explicar y concretar la innovación que Crizal UV aporta, hemos diseñado un nuevo dispositivo de demostración que ponemos a disposición de las diferentes filiales del grupo (véase la Fig 2).

Factor de reflexión UV

La exigencia de un nivel bajo reflexión en los UV supone poder cuantificarla de manera pertinente tomando en consideración los riesgos de salud asociados a las radiaciones UVA (315 nm - 380 nm) y UVB (280 nm - 315 nm) en el ojo humano. Para ello, nos hemos apoyado en la norma internacional existente (norma ISO 8980-3 :2003) que propone un cálculo del factor de transmisión en los UV aplicado a las lentes oftálmicas. En esta norma, el cálculo de la eficacia contra los UV se realiza utilizando una función de ponderación $W(\lambda)$ (Fig. 3) que depende de:

- del espectro de la radiación solar directa $ES(\lambda)$ recibida en la superficie terrestre - pocos UVB con respecto a los UVA, debido a la absorción de la capa de ozono entre 200 y 300 nm,
- de la función espectral relativa de eficacia $S(\lambda)$ o ‘función de riesgo UV’ que muestra que los UVB son más peligrosos que los UVA. Esta última función $S(\lambda)$ expresa el riesgo biológico asociado al deterioro fotocromático de la córnea, cuando ésta está expuesta a los UV.

Por lo tanto, hemos aplicado esta función para ponderar la reflexión $R(\lambda)$ en los UV según la fórmula siguiente:

Se utiliza este factor en el cálculo del E-SFF$^3$ que permite evaluar el nivel de protección UV de las lentes oftálmicas (Fig. 3).

Caracterización de las eficacias

El desarrollo de Crizal UV ha requerido nuevos medios de caracterización. Primero, en la fase de I&D, la espectrofotometría y la espectrofotometría de ángulo variable, en los UV y en lo visible, han sido útiles para caracterizar el conjunto de los materiales, desde los substratos hasta las capas finas. Se han adaptado y desplegado medios de medición basados en los mismos principios en los medios de producción con el fin de garantizar la eficacia de este nuevo producto, tanto de un punto de vista espectral que angular.
The usual optical characterisations confirm that anti-reflection efficiency remains unchanged in the visible spectrum for Crizal Forte UV lenses compared to previous generations of Crizal lenses.

**Conclusion**

Associated with organic materials, Crizal UV lenses bring to the market for the first time protection against UV radiation incident at the back of the lens, whilst ensuring optimum visual clarity for the wearer.

Crizal Forte UV colourless lenses are associated with an E-SPF protection factor of 25\(^5\), the best on the market.

In sun lenses, Crizal Sun UV offer an even higher level of protection, with an E-SPF factor of 50+.

With a complete offer available and based on an E-SPF factor that is explicit for consumers, vision professionals can convey an important prevention message and help wearers to make the right choice in terms of protection for their vision health.

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La protección UV aportada por la baja reflexión UV (R\(_{UV}\)), de 5 a 10 veces inferior a la medida en las lentes anti-reflejantes de los fabricantes principales\(^4\), se traduce así por un índice de protección E-SPF de 25 en las lentes transparentes **Crizal Forte UV** y 50+ en las lentes solares **Crizal Sun UV**.

Los métodos de caracterización óptica habituales confirman la eficacia antirreflejo sin modificación en lo visible de Crizal Forte UV con respecto a las generaciones precedentes de lentes Crizal.

**Conclusión**

Las lentes Crizal UV, asociadas a materiales orgánicos, aportan por primera vez en el mercado una protección contra los rayos UV que llegan por detrás de la lente, a la vez que se garantiza una mejor claridad de visión para el portador.

Las lentes transparentes Crizal Forte UV contienen un índice de protección E-SPF de 25\(^5\), el mejor del mercado.

En las gafas de sol, Crizal Sun UV aporta un nivel de protección aún superior con un índice E-SPF de 50+.

Al disponer de una oferta completa y basándose en un índice E-SPF explícito para el consumidor, los profesionales de la visión pueden transmitir un mensaje importante de prevención y ayudar a los portadores a realizar la mejor decisión de protección para su salud visual.

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\(^1\) E-SPF of 10 for Essilor Orma\(^®\) Crizal Forte UV lenses

\(^2\) E-SPF de 10 en las lentes Essilor Orma\(^®\) Crizal Forte UV

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Maximizing Protection from Ultraviolet Radiation Hazards: Assessing the Risks; Finding Solutions

Exposure to the ultraviolet component of sunlight causes damage to ocular tissues that can accumulate over a lifetime. This chronic ultraviolet radiation (UVR) exposure has been associated with pterygium, cataract, climatic droplet keratopathy, and other serious ocular conditions. As a result, many spectacle lenses now offer effective blocking of UVR transmission. However, recent work by Karl Citek, OD, PhD, and others has found that UVR can be reflected from the backside of clear, photochromic, and tinted/polarized lenses; and that No-Glare (antireflective, or AR) technology actually increases the level of backside UVR reflection. Maximum protection from UVR requires that all lenses—including clear lenses intended primarily for indoor wear—effectively shield wearers from both transmitted and reflected UVR. This is now possible with Essilor’s new Crizal UV lenses with patented Broad Spectrum Technology; these lenses maximize long-term eye health by shielding eyes from exposure to transmitted and reflected UVR.

**INTRODUCTION**

Sunlight is the primary source of ultraviolet radiation (UVR) to which humans are exposed. Although a portion of the sun's UVR is absorbed by the ozone layer of the atmosphere, significant UVR penetrates the ozone to strike the surface of the earth. For most people, the total amount of UVR received (the cumulative dose) increases linearly over time.\(^1\)

Documentation that cumulative sunlight exposure causes irreversible eye damage has been part of the medical literature for more than 100 years, but public awareness of the need to protect eyes from sunlight has lagged far behind. And new findings about the nature of solar UVR hazards underscore the importance of continuous UVR protection, which for eyeglass wearers is possible only if every pair incorporates effective UVR protection.

For example, we now know that a significant portion of the solar UVR incident on the cornea comes from indirect sources, including UVR striking from the side rather than the front (called “albedo”) and UVR reflected by the backside of spectacle lenses. (All types of spectacle lenses can reflect UVR, including clear, photochromic, and tinted/polarized lenses). As this paper will document, the hazard to eyes from UVR reflected by the backside of spectacle lenses is a serious problem that until now has had no solution.

This paper will further document that the problem of reflected UVR is not limited to sunglasses wearers. Rather, studies have found that, on average, people receive over 40% of their annual UVR dose at times of day when they are unlikely to wear sunglasses; and up to 23% of people never protect their eyes from the sun at all (Table 1).\(^2\) Clearly, eyeglass wearers need both their everyday glasses and their sunglasses lenses to provide complete UVR protection.

**CHRONIC UVR EXPOSURE AND LONG-TERM EYE HEALTH**

Although acute photokeratitis can occur from a single very high dose of UVR (eg, from skiing without eye protection) most UVR damage is cumulative—it is chronic UVR exposure and the lifetime UVR dose that are of greatest importance in UVR-associated diseases. This is as true with eyes as it is with skin, where solar UVR is known to contribute to aging and the development of cancer.

UVR that reaches the eye can cause serious damage. Epidemiologic studies have linked chronic UVR exposure with serious ocular pathology, including climatic droplet keratopathy, pterygium, cortical cataract, and pinguecula (Table 2). Although the relationship has not been definitively proved, solar UVR exposure has also been implicated in the development of age-related macular degeneration (AMD).

**TABLE 1  Sources of UVR Exposure**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Sunlight Exposure (Lx)</th>
<th>Percent of Annual UVR exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indoor</td>
<td>500</td>
<td>~7%</td>
</tr>
<tr>
<td>Cloudy sky</td>
<td>5,000</td>
<td>5%</td>
</tr>
<tr>
<td>Clear sky</td>
<td>25,000</td>
<td>30%</td>
</tr>
<tr>
<td>Summer sky</td>
<td>100,000</td>
<td>58%</td>
</tr>
<tr>
<td>TOTAL</td>
<td></td>
<td>100%</td>
</tr>
</tbody>
</table>
UVR EXPOSURE AND THE EYE

It was once thought that ocular UVR exposure rose and fell in parallel with the intensity of ambient UVR — as is true of skin exposure — but this is not the case. Set deep within the orbit, the eye is effectively shaded by the brow and upper lid when the sun is directly overhead. Thus, when the sun reaches its zenith at solar noon (and ambient UVR peaks), only a fraction of this radiation reaches the eye.3

Sasaki and colleagues demonstrated this relationship between solar angle and the quantity of solar radiation striking the eye by using a specially designed mannequin with UVR sensors installed on both the top of the head and within the eye socket at the position of the cornea. As expected, UVR exposure at the top of the head rose and fell with solar angle, but the in-eye sensor registered the highest levels of UVR in the mid-morning (from 8:00AM to 10:00AM) and mid-afternoon (2:00PM to 4:00PM), leading these researchers to conclude that UVR exposure in the eye peaks at times other than solar noon and suggests a need for all-day UVR protection.4

Ocular anatomy has other effects on UVR exposure. The human skull is configured to allow a large temporal field of vision. As a result, a significant amount of sunlight can strike the eye from the side. This exposure to oblique light creates a particularly significant hazard due to the peripheral light focusing (PLF) effect, also known as the Corneal effect.5,6

![Figure 1](https://example.com/figure1.png)

Figure 1  Focused peripheral light reaches the nasal limbus.

In PLF, light incident from the side is refracted by the peripheral cornea, which focuses it on the nasal limbus where the corneal stem cells reside (Figure 1). Although the limbal stem cells are protected by the sclera from direct UVR exposure, PLF bypasses this protection and concentrates sunlight (including its UVR component) at the nasal limbus, increasing exposure there as much as twenty-fold.5 Epidemiologic evidence indicates that this concentrated sunlight plays a critical role in the development of pterygium.7

### Table 2 Ophthalmic conditions associated with UVR/sun exposure

<table>
<thead>
<tr>
<th>Condition</th>
<th>UVA</th>
<th>UVB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eyelid</td>
<td>Basal cell carcinoma; squamous cell carcinoma; wrinkles; sunburn; photosensitivity reactions</td>
<td></td>
</tr>
<tr>
<td>Ocular Surface</td>
<td>Pterygium; climatic droplet keratopathy; photokeratitis (&quot;snow blindness&quot;); pinguecula; dysplasias and malignancies of the cornea and conjunctiva</td>
<td></td>
</tr>
<tr>
<td>Crystalline Lens</td>
<td>Cortical cataract</td>
<td></td>
</tr>
<tr>
<td>Uvea</td>
<td>Melanoma; pigment dispersion; uveitis; blood/ocular barrier incompetence</td>
<td></td>
</tr>
<tr>
<td>Vitreous</td>
<td>Liquifaction</td>
<td></td>
</tr>
<tr>
<td>Retina</td>
<td>Age-related macular degeneration (not definitely proved)</td>
<td></td>
</tr>
</tbody>
</table>

### Backside UVR Reflections: A Newly Recognized Hazard

Antireflective lens technology (sometimes referred to as “No-Glare” or “AR” technology) is widely used in

#### Contribution of Light Scattering and Reflection

Short wavelengths of solar radiation, including UVR, are scattered by clouds and by particles in the atmosphere — this scattering of blue wavelengths makes the sky appear blue. High levels of UVR can also be reflected from surfaces such as sand, snow, water, and grass (see Table). This reflected and scattered UVR accounts for more than half of the UVR that strikes the cornea.8

At times close to solar noon, when the brow and upper eyelid shield the eye from direct sunlight, scattered and reflected sunlight becomes the primary source of ocular UVR exposure. This scattered and reflected UVR can strike the eye from any direction, including directions that bypass spectacle frames and lenses (see Figure). (The exception is full-wrap sunglasses and goggles.)

If the individual is wearing glasses, a significant portion of this UVR can be reflected directly into the eye by the backside of the lenses. This is true whether the lenses are clear, photochromic, or tinted/polarized.

Depending on the geometry of the lens, the frame, and environmental conditions, up to 50% of UVR exposure comes from the back and sides of the lens. Thus, even if the lens is capable of blocking 100% of UVR transmission, the eye can still receive a substantial dose of UVR due to side and back exposure. This light scattering (which enables UVR to come at the eye from the side and behind) and reflection from the backside of spectacle lenses must be taken into account in any consideration of UVR protection.
s spectacle lenses to enhance the cosmetic and optical performance of the lens by increasing light transmission and eliminating visible reflections and glare. Unexpectedly, Antireflective treatments have recently been found to increase the reflectance of UVR. While clear lenses without antireflective treatment reflect approximately 4% to 6% of UVA (380-315 nm) and UVB (315-280 nm), antireflective lenses reflect an average of 25% of most UVR wavelengths. And some AR lenses reflect close to 90% of individual UVR wavelengths. This high level of UVR reflectance makes scattered and reflected UVR a particular concern since they can strike the back surface of a spectacle lens and be reflected into the eye (Figure 2). UVR reflected by the backside of a lens can enter through the central cornea. It can also reach the temporal limbus and do harm through the PLC mechanism. Heretofore, backside reflection could be prevented only by goggles or high-wrap frame designs that allow little or no light to strike the back surface of the lens. Current photochromic, sun lenses, and clear lenses do not address this particular hazard. Some clear lenses (eg, those made from polycarbonate) and all photochromic lenses block transmission of 100% of UVR that is directly incident on the front of the lens; materials that do not inherently absorb UVR can be treated to block UVR transmission. However, the backside reflection of UVR remains the Achilles heal of UVR protection and safer vision.

**NEW CRIZAL UV LENSES**

**ELIMINATE BACKSIDE UVR REFLECTION**

To address the significant hazard of backside UVR reflection, Essilor has developed new Broad Spectrum Technology that extends the superior Crizal antireflective lens efficacy from the visible light spectrum to the ultraviolet spectrum (Figure 3). Essilor’s new Crizal Forte UV and Ophtog™ with Crizal UV lenses are the first to feature this new technology for clear, everyday lenses, in which UVA and UVB reflections from the backside of the lens are reduced — without loss of the other benefits of Crizal AR lenses. This means that Crizal UV lenses not only maximize visible light transmission for enhanced visual clarity, they also provide protection from reflected UVR — in addition to resisting and repelling scratches, smudges, dust, and water (Table 3).

**PUTTING UVR IN PERSPECTIVE**

Long-term exposure to solar UVR causes cumulative damage to ocular tissues that can harm eye health. Environmental factors like depletion of the ozone layer will increase levels of UVR on the surface of the earth for decades to come, and prevention of UVR-associated eye diseases will become correspondingly more important.

Recent studies show that reflection of UVR from the backside of spectacle lenses represents a significant source of ocular UVR exposure. Other investigations have found that peak times of ocular UVR exposure are mid-morning and mid-afternoon — times when indi-

![Figure 2](image2.png)
Figure 2  Antireflective lenses that are not Crizal UV reflect UVR off the backside, so significant UVR can strike the cornea (even if the lens protects against transmitted UVR). Today, only Crizal UV lenses (Crizal Forte UV, Ophtog with Crizal UV, Crizal Sun™ UV, Ophtog Sun™ with Crizal UV) protect against reflected UVR.

![Figure 3](image3.png)
Figure 3  Crizal UV extends effective blocking of UVR reflections deep into the UVR spectrum, while within the visible light spectrum Standard AR and Crizal UV provide virtually identical reflection blocking.

---

**TABLE 3 Benefits of new Crizal UV and Crizal Sun™ UV (designed specially for sunwear)**

<table>
<thead>
<tr>
<th>Superior visual clarity</th>
<th>Glare reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Improved contrast sensitivity and visual acuity</td>
</tr>
<tr>
<td></td>
<td>Resists scratches and smudges</td>
</tr>
<tr>
<td></td>
<td>Repels dust and water</td>
</tr>
<tr>
<td></td>
<td>Easy cleanability</td>
</tr>
<tr>
<td>Maximum UV protection</td>
<td>Blocks transmission of UVR (100%) through the lens*</td>
</tr>
<tr>
<td></td>
<td>Reduces backside UVR reflection</td>
</tr>
</tbody>
</table>

*when paired with a photochromic or higher-quality lens material.
THE EYE-SUN PROTECTION FACTOR

People purchasing sunscreens know exactly how much UVR protection they are getting because sunscreens all carry a number on the label. This provides a precise indication of the sun-blocking strength of a given sunscreen and makes it easy to compare one sunscreen with another. Although UVR protection is as critically important to eyes as it is to skin, until now nothing like that has existed to indicate the UVR protection offered by specific spectacle lenses.

With this in mind, Essilor has worked with independent experts to develop the Eye-Sun Protection Factor (E-SPF). Defined as the ratio of UVR at the cornea with and without lenses in place, E-SPF measures the amount of protection provided by a lens as compared to no protection at all. (The ratio is weighted to take into consideration the impact of UVR at different wavelengths on the cornea.)

Calculation of E-SPF takes into account both transmission of UVR through the lens and backside UVR reflection. By integrating these two aspects of UVR protection, E-SPF provides a readily understandable measure of the UVR protection offered by a given lens. As with the reference index of the skincare industry, higher values of E-SPF indicate better UVR protection. For example, new Crizal® UV lenses, with minimal backside UVR reflectance, have higher E-SPF values than competitive antireflective lenses (see Table).

Perhaps the most important aspect of the E-SPF is that it gives eyecare professionals a simple way to tell patients how they can maximize protection—without lengthy, complex explanations or recommending specific products. Now telling patients how to protect their eyes is as straightforward as telling them how to protect their skin. Pick the highest number for the best protection. It’s that simple.

This is now possible for clear, everyday lenses with the patented Broad Spectrum Technology in Essilor’s Crizal Forte UV and Optifog™ with Crizal® UV lenses; and for sunwear with Crizal Sun® UV and Optifog Sun™ with Crizal UV. These lenses reduce backside UVR reflection to offer the most complete protection possible against ocular UVR exposure.

One of the reasons that eye protection from UVR has lagged behind skin protection has been the lack of an easy way for eyecare professionals to talk about it. The new Eye-Sun Protection Factor (E-SPF) takes care of this problem. Now, ECPs can explain that patients receive the most protection with the highest E-SPF (see box). Choosing the most complete eye protection becomes as simple as choosing a sunscreen: just look at the numbers.

REFERENCES


<table>
<thead>
<tr>
<th>E-SPF of different AR Lenses</th>
<th>Main AR Lenses</th>
<th>E-SPF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crizal Sun UV</td>
<td>50+</td>
<td></td>
</tr>
<tr>
<td>Optifog Sun with Crizal UV</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Crizal Forte UV</td>
<td>Competitor A</td>
<td>3</td>
</tr>
<tr>
<td>Optifog with Crizal UV</td>
<td>Competitor B</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Competitor C</td>
<td>5</td>
</tr>
</tbody>
</table>

Individuals are not likely to wear sunglasses. Hence, to achieve the goal of minimizing ocular UVR exposure, spectacle-wearing patients should be well protected in every pair of glasses they have, whether the lenses are clear, photochromic, or tinted/polarized.

Today’s higher quality lens materials provide 100% blocking of UVR transmission, but the AR technology on the back surface of a lens can reflect unexpectedly high levels of UVR and significantly increase the eyes’ dose of UVR. The most complete solution for everyday UVR protection, thus, is lenses that protect against both UVR transmission and reflection.
WE ARE LIVING BETTER AND FOR LONGER

We gain an extra three months life expectancy every year, around 6 hours per day... one in every two little girls born today in France will reach the age of one hundred. Progress in the health field, although unequally distributed throughout the regions of the world, means overall that we are living better and for longer. But what about ocular health? Is the human eye prepared for working in good health for over 100 years?

THE PREVENTION OF EYE DISEASES IS A MAJOR PUBLIC HEALTH CHALLENGE

With the increase in life expectancy, some eye diseases and afflictions such as cataract or age-related macular degeneration (AMD) are unquestionably rapidly on the increase. Today the number of people affected by cataract is estimated at 250 million worldwide, with 100 million suffering from AMD, and these figures are set to double over the next 30 years Fig. 1 Within this context, the importance of preventing eye diseases becomes clear and the main objective is to minimise the risk of diseases occurring by taking direct action on the causes. (Fig. 1)

UV AND HARMFUL BLUE LIGHT ARE INVOLVED, AMONGST OTHER CAUSES, IN THE APPEARANCE OF CATARACT AND AMD

Age, tobacco smoking, diet and environmental factors such as prolonged exposure to ultraviolet rays are extensively noted in scientific literature as being risk factors in the occurrence of senile cataract.

In addition to UV, visible light can also have a cumulative impact on ocular health and particularly play a part in the development of AMD. In fact, in addition to age, genetic factors or tobacco smoking, several epidemiological studies, including the „Beaver Dam Eye Study“ and the „Chesapeake Bay Study“ conclude that the risk of AMD is greater in the case of cumulative exposure to visible blue light [3].

And yet, within blue light, which is in wavelengths of between 380 and 500 nanometres (nm), it is important to distinguish the BAD blue from the GOOD blue [4]. The combined work of the Essilor International and the Vision Institute recently resulted in definition of the precise spectrum of retinal phototoxicity [5] and concluded that it is Blue-violet light, which is the closest to UV and centred at 435nm, which is the most harmful for the retina. This „bad blue“ can be of solar or artificial origin. Several independent studies undertaken by health agencies are...
now looking at the risks linked to new sources of artificial light, such as electroluminescent diodes or LED [48], because the latter have an emission peak situated in the „bad blue” range (Fig. 2).

On the other hand, Blue-Turquoise light, at wavelengths of between 465 and 495 nanometres (nm) is known as the „good blue” because it acts on many non-visual functions that are essential for the body to function well [10].

Research programmes aimed at discovering new solutions to prevent or treat AMD must take account of this distinction between good and bad blue and attack harmful rays in a selective manner.

SELECTIVE PHOTO-PROTECTION USING INTERFERENCE FILTERS

Various products offer protection against Blue-Violet light, such as therapeutic filters and sun lenses. Although the protection level is high, their colour can be an obstacle to permanent everyday wear (distortion of colours, appearance, vision in low indoor light) and also they necessarily cut out both bad and good blue light, making no selection between the two.

In order to offer selective photo-protection and a high degree of visual comfort for everyday wear, the use of interference filter technology would seem to be the ideal solution for a clear lens. It cuts out the Blue-Violet light that is harmful for the retina, whilst maintaining optimal transmission of the Blue-Turquoise light in the neighbouring spectral band.

Twenty years of expertise in anti-reflective treatments and two years of research have enabled Essilor to achieve the design of the Crizal® Prevencia™ lens, an interference filter that reflects light in order to:

1. Filter out harmful rays, the Blue-Violet that contributes to AMD as well as UV rays which play a part in the appearance of cataract

   The various anti-reflective coatings on both sides of the Crizal® Prevencia™ lens filter out harmful light selectively:
   - 20% of Blue-Violet light, [400-450 nm], is cut out thanks to optimised reflection of these wavelengths on the front side. The residual colour of the reflection proves its efficiency in the Blue-Violet range.
   - On the back side, the interference layers have been created to minimise the reflection of UV rays into the eye.

   This unique combination today offers the most complete eye protection available in a clear lens.

2. Allow beneficial blue light to pass through Crizal® Prevencia™ transmits 96% of Blue-Turquoise light, [465-495 nm], thus preserving visual functions as well as some non-visual functions such as:
   - stimulation of the pupil reflex, the retina’s natural protection against over-exposure to light, centred at 480 nm,
   - synchronisation of the biological clock (waking/sleep cycles, hormonal cycles, memory, cognitive performance, etc.) centred on a 30 nm bandwidth, [465-495 nm].

3. Whilst guaranteeing excellent lens transparency Crizal® Prevencia™ ensures optimal vision clarity with overall visual transmission of 98%. This lens also retains the benefits offered by former generations of the Crizal range: the most efficient dirt-resistance on the market as well as excellent resistance to scratching, dust and water. (Fig.3)

CRIZAL® PREVENCIA™, EFFICIENCY PROVEN IN VITRO.

Essilor and the Vision Institute carried out an experiment on the retinal pigmented epithelium (RPE) cells involved in the macular degeneration process, in order to model the protection offered by the Crizal® Prevencia™ lens.

These retinal cells were photosensitized and exposed for 18 hours to narrow 10 nm bands of illumination in the blue light spectrum range between 400 nm and 500 nm, in the physiological conditions of sunlight on the retina. Photobiological work showed an average reduction in cell mortality by apoptosis of 25% compared to the naked eye in the spectrum range [400 nm, 450 nm]. Figure 4 shows the comparative levels of apoptosis between the naked eye (grey) and Crizal® Prevencia™ (purple) for each of the bands of blue illumination. This level of protection would therefore mean a reduction in the long term in the cumulative risk linked to harmful blue light and therefore the onset of AMD.
After the age of 45, the eye’s defence system weakens and the eye becomes more highly exposed to UV rays and Blue-Violet light. The retinal cells are therefore more vulnerable to harmful light to penetrate more deeply. The retinal cells are therefore particularly vulnerable populations such as children and adults aged over 45.

Before the age of 10, the eye’s extreme transparency allows bands of harmful light to penetrate more deeply. The retinal cells are therefore more highly exposed to UV rays and Blue-Violet light.

After the age of 45, the eye’s defence system weakens and the sensitivity of retinal cells increases. This means that the risk of eye disease increases.

This preventive lens is for everyone, and specifically for more particularly vulnerable populations such as children and adults aged over 45.

- Before the age of 10, the eye’s extreme transparency allows bands of harmful light to penetrate more deeply. The retinal cells are therefore more highly exposed to UV rays and Blue-Violet light.
- After the age of 45, the eye’s defence system weakens and the sensitivity of retinal cells increases. This means that the risk of eye disease increases.

Crizal® Prevencia™ can also be combined with a photochromic technology used to obtain optimal protection for outdoor activities whilst offering the desired transparency for indoor wear.

- When activated the photochromic lens is tinted and protection is then at its maximum, at over 80% whatever the material used.

Crizal® Prevencia™ is the ideal preventive solution against the dangers of harmful light, which are still relatively unknown amongst the general public. The role of vision professionals and Essilor is therefore key in creating awareness and recommending this product.
PROTECT CHILDREN’S EYES EVERY DAY: CRIZAL® PREVENCIA® FOR KIDS

Annual exposure to solar radiation is three times higher in children than adults. Moreover, because of their physiology, children’s eyes are more vulnerable and require special protection against UV rays and blue-violet light. Designed for children as well as adults, the new Crizal® Prevencia® lenses are completely transparent, providing optimal photo-protection from day to day. The use of sunglasses will ensure additional protection in direct sunlight.

The harmful effects of chronic exposure to ultraviolet radiation and the blue-violet component of visible light are now clearly established as factors in the development of ocular diseases such as cataracts and AMD. The cumulative effect of this exposure over a lifetime contributes to accelerating onset of these serious conditions. And this process begins in early childhood: children are doubly exposed to the risks posed by these harmful light rays.

The primary risk factor in children: overexposure
To begin with, children spend three times longer outdoors than adults, which increases their exposure to the most powerful source of UV rays and blue light: the sun. LED screens (tablets, smartphones, computers, etc.), which are new sources of blue-violet light, intensify this exposure further, as children come to use them more and more frequently and at a signifi-
cantly earlier age. In the UK, the use of tablets at home among children aged 5 to 15 trebled between 2012 and 2013 (from 14% to 42%). One quarter (28%) of three- and four-year-olds use a tablet at home.² Nearly 20% of French children aged 7 to 12 were using a tablet in 2013³ – a figure three times higher than in 2012 (Fig 1).

All these devices are undeniably tools for enhancing cognitive development, improving awareness and teaching children to master the digital world. However, they can also foster an addiction to virtual environments and lead to difficulty sleeping. Their use needs to be limited and supervised through parental controls on the content and length of each child’s daily use. The growing number of screens that are backlit with cool white LEDs, which are known to generate potentially harmful blue-violet light, may increase the risk of chronic photo-toxicity over time.

An additional risk factor in children: the permeability of the visual system

In early childhood, the crystalline lens is much more permeable to harmful UV and blue-violet rays, a significant portion of which can reach the retina (Fig. 2). Retinal exposure to UV radiation may lead to rapid growth in the concentration of lipofuscin during the early years of life⁴ (Fig. 3); lipofuscin can subsequently prove toxic to the retina when subjected to blue-violet light.

The importance of risk prevention and education

It seems appropriate, then, to create solutions for preventing this risk and protecting children from a very young age. The parallel with skin should serve as a warning: according to the WHO, excessive exposure to the sun in childhood can contribute to skin cancer later in life.⁵ Although there is growing knowledge of the need to protect children’s skin from the sun, and a wider range of specially designed protective sun cream (SPF 50+) is available for their use, the same cannot be said for eye protection in children.

Adults, however, protect themselves better than their children: a U.S. study showed that just 48.4% of the parents surveyed use sunglasses to protect their children’s eyes.⁶ A separate study in France revealed that 84% of parents own at least one pair of sunglasses, compared to 68% for their children.⁷ But even among children who have sunglasses, the nuisance of using them means they are worn far less often than circumstances require. You need merely visit the beach in summertime to discover that the number of children wearing sunglasses remains quite small.

FIG. 1 The spread of personal devices among children (ages 7 to 12) and teenagers (ages 13 to 19) in France (Source: Ipsos).

FIG. 2 Total transmittance of clear ocular media of aging human eye. Fitted from the CIE 203:2012 data. Does not take into account cataract surgery beyond 60 years old.
FIG. 3 | Rapid increase in lipofuscin concentration between the ages of 0 and 10.

Source: Adapted from (Wing et al., IOVS, 1978), ex vivo, in the total RPE.
For the vivo, at fovea and 7° temporal to the fovea, see (Delori et al., IOVS, 2001), faster increase with age.

“Crizal® Prevencia® Kids is a daily-wear lens that is especially tailored to the needs of children.”

Crizal® Prevencia® Kids: the solution offering everyday protection for children

For children who already wear corrective lenses, there are increasingly effective solutions for daily protection. Up until recently, the only consistent way to filter out both blue light and UV rays was to wear tinted filters (yellow, orange) inside and/or sun lenses outside. This solution already represents a significant burden for older patients; the idea that this approach could be used with children on a daily basis, purely for protection, is unthinkable. Moreover, these filters completely eliminate blue light, distorting our color perception and potentially depriving the eye of the benefits of the blue-turquoise component of the visible spectrum (465-495 nm), which regulates our biological clock and in particular our waking and sleeping phases. It was in response to this need for a simple and effective form of prevention that Crizal® Prevencia® lenses were designed, for use by both adults and children.

These antireflective lenses come with a new interferential filter that provides selective protection (Fig. 4). Harmful light rays are filtered so as to reduce the effects of UV rays and blue-violet light (415-455 nm) on the crystalline lens and retina.

The blue light that is beneficial to our bodies is maintained. Crizal® Prevencia® allows 96% of blue-turquoise light to pass through. The lens offers guaranteed transparency, with transmission of more than 98% of visible light to ensure optimal vision.

Proven in vitro effectiveness

Crizal® Prevencia® lenses mark the culmination of lengthy research conducted in cooperation with Paris Vision Institute (IDV), considered one of Europe’s premier integrated research centres specializing in eye disease. To demonstrate the efficacy of these lenses in protecting retinal cells, the IDV conducted an in vitro
experiment which revealed that in retinal pigment epithelium cells protected from blue-violet light by Crizal® Prevencia®’s interferential filters, the rate of cell death through apoptosis fell by up to 25% in comparison with unprotected cells.1, 10 The most visible proof of the protection offered by Crizal® Prevencia® lenses is the color of the residual reflection produced by its filter: blue-violet (Fig. 6). When the lenses are exposed to the harmful component of blue light, these rays are partially reflected, and that distinctive reflection — which can be shown to future wearers when they purchase the lens — is a reliable indicator that the eye is being protected.

**Certified UV protection (E-SPF™ 25)**

When it comes to protection against UV rays, Crizal® Prevencia® Kids lenses offer the same level of protection as all the other untinted lenses in the Crizal® range, certified by an E-SPF® (Eye-Sun Protection Factor) of 25. Coupled with the Airwear® material that prevents any UV light rays from passing through, Crizal® Prevencia® lenses include a filter on their inner surface that virtually eliminates UV reflection into the eye.

“Children spend three times longer outdoors than adults, which increases their exposure to the most powerful source of UV rays and blue light: the sun.”

**FIG. 5**

Comparative results between Crizal® Prevencia® and the naked eye of RPE cell death by apoptosis, exposed for 18 hours in vitro to normalized sunlight for a 40 year old human eye.
This UV exposure from the rear of the lens can be quite significant, accounting for up to half of all UV exposure for unprotected eyes. Before the introduction of the most recent Crizal® lenses, anti-reflective lenses on the market still reflected a substantial amount of UV radiation. The E-SPF® index, developed by Essilor, is the only international rating that measures the protection offered by a given lens on both its outer surface (for light transmission) and its inner surface (for reflection back into the eye). A factor of 25, currently the highest on the market for an untinted lens, indicates that the eye receives 25 times greater protection than it would otherwise (the sun lens offers an E-SPF® of 50+). The E-SPF® index gives eye care professionals a standard they can use with children who wear lenses and their parents, who are already familiar with the SPF index used for sun creams.

A lens designed for children

In order to provide greater overall protection against harmful light rays, Crizal® Prevencia® Kids is a daily-wear lens that is especially tailored to the needs of children. Its effective anti-reflective treatment ensures perfect transparency, which means

"In early childhood, the crystalline lens is much more permeable to harmful UV and blue-violet rays, a significant portion of which can reach the retina."
vision quality and comfort, notably for classroom learning and when viewing a screen.

When used with Airwear® material, Crizal® Prevencia® lenses are the most shock-resistant on the market, 12 times more resistant than standard lenses – which will reassure parents of even the most daredevil children. They also have the advantage of being 30% lighter and 20% slimmer to suit fragile noses, so children are more likely to accept them. In addition, Crizal® Prevencia® lenses are treated to provide maximum resistance to two things much feared by parents: scratches and smudges. These lenses – easier to clean than any other lens on the market – are ideal for children whose lenses quickly become dirty.

**Conclusion**

We need to take steps as early as possible to protect eye health and prevent the risks posed by the harmful effects of UV rays and blue-violet light, because young children are especially vulnerable to the damage they can do. All children’s eyes need to be protected in the outdoors with appropriate equipment that provides good coverage against the sun when sunlight is strongest, alongside the proper precautions for protecting their skin: sun cream, a wide-brimmed hat, avoiding exposure when the sun is at its most intense.

For children who wear eyeglasses at all times, vision health professionals can now recommend the kids’ version of Crizal® Prevencia® lenses.

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**Front side protection**

Cut 20%(1) blue-violet light and 100%(2) UV

**Back side protection**

Backside reflection virtually eliminated (≈ 4%)

(1) The blue-violet light cut may slightly differ depending on lens material.

(2) For any Crizal® Prevencia® lens material other than clear 1.5 plastic.

**CORRECTIVE LENSES TO PROTECT AGAINST UV**

**CLEAR LENSES**

For everyday protection against the cumulative effects of exposure to UV rays, lenses with protection factor E-SPF™ 25 offer the highest level of protection available for clear lenses. Crizal® lenses were the first in this category to offer this level of protection. They are available in an extensive range for all wearers, both children and adults (Crizal® Kids UV, Crizal® Prevencia®, Crizal Forte® UV, Crizal® Alizé® UV, Crizal Easy® UV). Associated with materials that absorb UV, Crizal® lenses benefit from technology that considerably reduces the eye’s exposure to UV due to reflection from the inner side of the lens.

**CORRECTIVE SUN LENSES**

For optimal protection from the sun, Crizal Sun® UV lenses have protection factor E-SPF™ 50+. They offer the essential level of protection when conditions demand the wearing of sun lenses (strong sunlight, altitude, beach, etc.). Crizal Sun® UV can be associated with tinted lenses or Xperio® polarizing lenses.
They are clear, completely transparent lenses that provide maximum protection against harmful UV and blue-violet rays, both outdoors and in the presence of harmful light from new sources such as LED screens. They also include all the necessary features for meeting the special needs of children and helping them learn to live with their eyeglasses at all times: clear and comfortable vision; thin, lightweight equipment; lenses that are easy for parents to maintain and that hold up better against the rough and tumble life that children sometimes lead.

**AWARDS / HONORS / SUCCESSES**

**Crizal® Prevencia® lenses received numerous honors worldwide in 2014:**
- in Canada: they were voted “Product of the Year” (the most innovative product of 2014 in the Optics category) by a panel of experts and consumers
- in France: Essilor’s R&D team accepted an award for technological innovation – the “Prix Fibre Innovation 2014” – given to Crizal® Prevencia® lenses at a daylong event hosted by OpticsValley, an optics trade group, at the Université Pierre-et-Marie-Curie in Paris
- in Australia: the entire range of Crizal® UV treatments won certification from Cancer Council Australia, an organization that is unmatched worldwide for its experience in preventing risks from UV radiation. This is the first seal of approval of its kind for an interferential treatment in the history of ophthalmic optics

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SCIENTIFIC QUEST FOR PERSONALIZED RISK PREVENTION

Light is suspected of being a risk factor for major vision-threatening diseases. Yet an equal light exposure can unequally affect people. Multiple intricate factors are responsible for a distinct personal risk profile. The scientific quest in understanding both eye phototoxicity and individual risk profiles can set a turning point towards personalized prevention in the future.

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Coralie joined Essilor in 2011, after a physics/optics engineer degree from Institut d’Optique Graduate School ParisTech and two Master degrees with honours from Paris XI in fundamental physics and in optics for new technologies. Her research is centred on photobiology of the eye, photometry and interferential physics for new ophthalmic healthcare lenses.

As Vice President R&D Disruptive at Essilor, Denis cultivates innovations, covering many technical domains and leveraging academic partnerships. With a Master in Quantum Mechanics from Ecole Normale Supérieure and an MBA from INSEAD, after ten years’ experience with BCG, Denis joined Essilor ten years ago.

Thierry joined Essilor in 2007 to develop biomedical collaborative research, especially with Paris vision Institute: photobiology, low vision and visual neurosciences are among his active topics. Thierry is a French engineer (ESPCI ParisTech) who specialized in medicinal chemistry (PhD, Pierre & Marie Curie University, Paris). He spent 20 years in the pharmaceutical and biotech industry in R&D and business development positions. Thierry also has an MBA from HEC.

KEYWORDS
prevention, eye phototoxocity, UV radiation, blue light, in vitro, in vivo, cataracts, pterygium, conjunctivitis, pinguecula, AMD, retinitis pigmentosa, glaucoma, diabetic retinopathy, oxidative stress, photo-ageing, risk profile
E ach day, our retina absorbs millions of billions of photons with an expected increased magnitude due to our new light exposure behaviors. Day after day, these streams of photons can induce irreversible eye damage and contribute to the onset or development of debilitating eye diseases. The phenomenon is aggravated by the accelerated ageing of the world population, since an ageing eye is more photosensitive along with altered defense.

A better understanding of the pathogenesis of vision-threatening diseases, a sharp analysis of light/eye interactions, and an individual risk profiling for these eye conditions are now urgent to provide appropriate and personalized eye photo-protection solutions, starting with eyewear, for efficient and long-term prevention.

1. EYE PHOTOTOXICITY

While light is necessary and beneficial to visual and non-visual functions, any optical radiation might potentially be hazardous to the eye if it is received and absorbed by eye tissues at doses capable of causing photomechanical, photothermal or photochemical reactions. On the one hand, brief and extreme bright light exposure may induce mechanical or thermal permanent and rapid eye injuries. On the other hand, moderate light exposure for an extended period of time may result in progressive biochemical changes and ultimately induce irreversible cell death. For this chronic lifelong eye light-damage, the spectral specificity of light is critical. In particular, UV radiations and high-energy visible light are pointed out as high risk spectral bands respectively for the anterior eye and the retina.

UV radiations and the anterior eye

Chronic eye exposure to solar UV radiations has been progressively associated with the pathogenesis of numerous cornea and crystalline lens diseases. If additional photobiology studies would be of interest to better dissect the intricate link between UV and the eye, sufficient in vitro, in vivo and epidemiology data confirm the contributory role of UV in numerous diseases of the anterior eye, such as cataracts, pterygium, conjunctivitis, pinguecula, climatic droplet keratopathy, ocular surface squamous neoplasia, etc. (for more details, see Points de Vue no. 67).

In 1956, Kerkenezov observed an early clinical indication of the role of UV in pterygium. Later, Minas Coroneo evidenced that peripheral light focusing by the anterior eye to the sites of usual locations of pterygium and cataract is involved in the pathogenesis of these eye conditions. The Chesapeake Bay study reported a significant correlation between the spatial zone affected by the climatic droplet keratopathy and the average annual UV exposure. Corinne Dot et al. evidenced that mountain professionals are at higher risk for cataracts. The POLA, Beaver Dam Eye and Chesapeake Bay epidemiology studies revealed a higher prevalence of cortical cataracts in populations living in bright sunny plains.

Public awareness has rapidly been high on the UV eye hazard since skin UV protection has now long been encouraged and normalized (SPF factors).

Blue light and the retina

Since UV radiations are totally absorbed by the cornea and the crystalline lens after the age of 20, the most energetic light reaching the retina is blue light.

Photobiology studies on blue-light eye damage started half a century ago, with the landmark paper of Noell evidencing blue retinal phototoxicity in rodents exposed to white fluorescent lamps. In 1972, Marshall, Mellerio and Palmer observed blue light damage in the pigeon cones. Since then, with the advent of lasers, the number of photobiology studies on blue light has soared. Ophthalmologists themselves have been encouraging such phototoxicity and exposure threshold studies for their patients’ exposure when conducting laser surgery (for retinal procedures, also for refractive surgery) or for themselves considering

“Light protection should be part of a personalized prophylactic program instructed by eye care practitioners.”
the light intensity of ophthalmic instruments (slit-lamp and others). More recently, in the 1990s, the IOL industry has funded phototoxicity research to support the benefits and safety of the blue-light filtering IOLs implanted during cataract procedures. 

In vivo experiments revealed that photochemical damages to the retina exhibit lower dose thresholds in the blue range compared to green and red as evidenced in monkeys, rats, and rabbits. Blue light hazards were further studied on the outer retina (photoreceptors and retinal pigment epithelium (RPE) (Fig. 1), on immortalized RPE cells loaded with either oxidized photoreceptor outer segment, purified lipofuscin or synthesized A2E. A greater toxicity of blue light was demonstrated by exposing human RPE loaded with lipofuscin during 48 hours upon violet-blue-green light (390 nm – 550 nm, 2.8 mW/cm²) and yellow-red light (550 nm – 800 nm, 2.8 mW/cm²). This cell death was mediated by apoptotic processes involving caspase-3 and p-53 activation. Many of these studies suffer limitations such as not being precise enough on the light dose sent, or illuminating with very high irradiances that trigger acute light-toxicity mechanisms rather than lifelong cumulative exposure damage. Moderate irradiances and longer exposure should be sought when studying the pathogenic mechanisms of Age-Related Macular Degeneration (AMD) or diabetic retinopathy. Under the supervision of Professor Sahel and Dr. Picaud, Paris Vision Institute and Essilor researchers joined skills to go a step further from a photometry standpoint. By developing innovative cell illumination protocols and systems, we together have studied various phototoxic action spectra involved in the pathogenesis of severe vision-threatening diseases (AMD, retinitis pigmentosa, glaucoma, etc.). For instance, we have evidenced the precise phototoxic action spectrum of RPE within the blue-green range in sunlight physiological retinal exposure on an established in vitro model of AMD. The 415 nm - 455 nm narrow spectral range was highlighted as the greatest phototoxic risk to RPE cells (Fig. 2).

In vitro and in vivo studies have progressively revealed a strong scientific rationale for cumulative blue toxicity on the outer retina. The understanding of cell mechanisms involved has provided crucial inputs on the pathogenesis of outer retina diseases, in particular AMD. First, cumulative exposure to blue light favors the accumulation of all-trans-retinal in the photoreceptor outer segments (POS). All-trans-retinal interacts with blue-violet light with a decreasing profile between 400 nm and 450 nm. Its blue photo-activation induces oxidative stress within the POS. This stress is normally compensated by retinal antioxidants and enzymes, but age progressively reduces anti-oxidative defenses, thus failing to compensate for the oxidative stress. The POS progressively oxidize, and their renewal into the RPE is more challenging as their membrane components are difficult for the RPE to break down. Thus, intracellular digestion is incomplete and generates an accumulation of residual lipofuscin in the RPE. Lipofuscin is sensitive to blue-violet light. Blue photoactivation may generate reactive oxygen species. When the number of these species exceeds cellular defence capacity, RPE cells die by apoptosis. Deprived of these support cells, the photoreceptors deteriorate in turn, contributing to the
loss of vision diagnosed in patients suffering from AMD. Age and light-related accumulation of lipofuscin in the RPE are major pathogenesis features of AMD. Numerous epidemiology studies confirm the correlation between blue light exposure and AMD.26, 27, 28, 29, 30, 31, 32 The EUREYE study found significant association between blue light exposure and neovascular AMD in individuals having the lowest antioxidant level. In the Chesapeake Bay study performed on 838 watermen, AMD patients – compared with age-matched controls – were significantly higher exposed to blue over the preceding 20 years but equally exposed to UV, suggesting that blue light exposure is related to AMD. The Beaver Dam Eye Study reported a correlation between sunlight and 5-year incidence of early AMD changes. Leisure time spent outdoors while persons were teenagers (13 – 19 years) and in their 30s (30 – 39 years) was significantly associated with the risk of early age-related macular changes. A recent meta-analysis led by Sui et al. interestingly concluded that light is a risk factor for AMD.33 Beyond the outer retina, photobiologists have recently suspected that high energy visible light could also affect inner layers of retina, such as retinal ganglion cells (RGC). Specific blue light may be absorbed by chromophores located in mitochondria. As an abundance of mitochondria are localized in RGC and these cells are involved in the degenerative processes of glaucoma, we suspect that blue light is a risk factor for glaucoma as well. In ageing retina, where functional mitochondria are no longer in an optimum homeostatic state34, blue light might dramatically precipitate the onset of glaucoma and other optic neuropathies. It could even contribute to accelerating glaucoma once diagnosed.35, 36

**Light, a risk factor for major vision-threatening diseases**

Light is suspected of being a risk factor in many debilitating eye diseases. For cataract and AMD, it is now well established: UV radiations accelerate the cataract onset while in AMD, blue-violet light exposure is a precipitating factor. For other diseases such as diabetic retinopathy or glaucoma, photobiologists suspect cumulative lifetime exposure to blue light contributes to the oxidative stress of specific retinal cells. In all cases, the contribution of light among other pathogenic factors grows with age and when the defence and repair mechanisms against photochemical damage are less effective, which is the case when the eye disease is already diagnosed (e.g. antioxidant enzymes such as SOD-2 or catalase are less effective).

**Normative data**

European and ISO standards for sunglass (EN 1836 and ISO 12312-1) and for tinted ophthalmic lenses (ISO 8980-3) have, for many years, used a relative spectral effectiveness weighting function S(λ) to characterize UV hazards. This was originally published in ICNIRP guidelines 1989 and is derived from an action spectrum for skin erythema. A sister function in the blue range was proposed later, B(λ), derived from the seminal work by Ham et al. for the acute hazard on aphakic monkey eyes. B(λ) was defined by multiplying the spectral values of Ham et al.’s research with the spectral transmittance of the human lens. Nevertheless, there is no standard on cumulative
blue light toxicity. New identification of phototoxic action spectra should be advantageously used to create and/ or revise normative data on phototoxicity.

**From in vitro and in vivo... to clinical data**

*In vitro* phototoxicity studies bring valuable and robust information on the light action spectrum as well as on the light-associated specific cell and disease biomarkers. The Essilor and Paris Vision Institute study with 10 nm step illuminations is a good illustration. Animal models (*in vivo*) are interesting living and integrative models of a disease. They make it possible to study the role of a specific gene (transgenic knock-out animals), the involvement of a specific toxicity pathway (e.g. inflammation, oxidative stress) or a disease target. They are essential in correlating disease with imaging, biology testing, immunohistochemistry or behaviour. But they have limitations: pathogenic mechanisms can differ from humans (e.g. using rodents in AMD models while rodents do not have a macula); light illumination is more intense and of a shorter duration than in real life. While *in vitro* and *in vivo* experiments raise the understanding of a disease-specific light action spectrum and pathogenic mechanisms, the only clinical evidence of light-associated eye diseases is brought by longitudinal epidemiological studies. It is therefore necessary, when studying real-life eye chronic phototoxicity, to find light-specific markers of disease early-signs and disease progression.

2. **INDIVIDUAL RISK PROFILING**

Now that cataract is being better treated in eastern and southern countries, AMD, glaucoma and diabetic retinopathy are becoming the three major vision-threatening diseases. This threat is largely worsened by the world ageing and by lifestyle risk factors such as poor antioxidant diet. From a public health perspective, considering that it is now urgent to optimize the disease management for these three eye conditions, all efforts should be paid to raise awareness in the general population, especially in the sub-populations considered more at risk, to develop and select the most relevant tests for early diagnosis and disease profiling, ultimately to monitor the disease progress and to apply the most appropriate therapeutic strategy. This sounds obvious but we believe we have now reached a more mature stage of understanding of the disease mechanisms associated with the availability of a set of new diagnostic tools including eye imaging, biological testing of biomarkers and psychophysical methods. In order to manage effectively a multifactorial disease, there is an utmost need of disease profiling and monitoring and if applicable of multi-facetted therapy – aiming at multiple mechanistic targets – as well as prevention. Individual risk profiling is defined by multiple intricate factors, source-, patient- and environment-dependent (Fig. 3).

**Individual light exposure profile**

Our own light exposure profile is defined by the correlation of the number of light sources, their localization, their spatial distribution, their radiance including directivity, but, critically, also their spectral distribution, the exposure duration and repetitions.

There is no doubt that solar radiations are the most harmful ones, since sun radiance is more than 100 times higher than the radiance of standard artificial lighting and since daylight is rich in UV and blue light. The physical environment (ground reflectances, altitude, latitude, etc.) significantly modifies the amount of light received by the eye. The eye UV dose increases by 10% every 1,000 metres. While sand reflects 10% of UVB, water reflects 20% and snow more than 80%. Therefore, populations exposed to bright sunlight in high ground reflectance environments (mountain professionals, etc.) or living in very sunny plains are at higher risk of UV and blue-light related eye damage, including cataracts and AMD.

In addition to daylight, in our ageing and connected digital world illuminated by new solid-state lighting, our light exposure profile is rapidly and dramatically evolving. Starting at increasingly younger ages of our existence, our eyes are subjected to longer and simultaneous exposures, at shorter distances, with higher radiance and higher energy than with former incandescent sources. Since they produce light with much lower energy consumption, these new solid state lighting sources have be-
come the dominant domestic lighting technology. In Europe, by 2016, no traditional incandescent light sources will be available. The European lighting industry estimates that over 90% of the total luminaires world market will be based on solid state lighting products by 2020. Beyond domestic lighting, the LED compactness plus the wide spectral range they can cover (monochromatic LEDs) have generated many new lighting applications, for mobile phone and tablet back lighting or even for toys and clothes.

New LED-based light sources may emit more blue than former incandescent lamps. Current white LEDs are combining a blue pumped LED with a phosphor emitting at higher wavelengths. For mass production of white LEDs, blue diodes based on InGaN or GaN crystals are combined with a yellow phosphor (YAG:Ce or similar); they produce “cold-white” with a color temperature CCT equal or higher than 5500 K. They may emit up to 35% of blue light within the visible range, much more than incandescent lamps (< 5%). To produce “warm-white” with a CCT <3200 K, with less than 10% of blue, an extra layer of phosphor emitting red light is needed, which significantly reduces the luminous efficacy of the LED.

At retinal level, received irradiance is directly proportional to the radiance of the light source. By having a small light emission area, LEDs have a higher radiance, which makes them brighter, even for the same irradiance level.

Worldwide initiatives have been launched to conduct a health risk assessment on systems using LEDs. A task group was for instance mandated by the French Agency for Food, Environmental and Occupational Health & Safety (ANSES) in 2008. They concluded that a photochemical blue light risk could exist, consecutive to prolonged white LED exposure. Risky light exposure profiles may be identified and related to high-risk populations (for more details, see Points de Vue no. 68):

- the daily adjustment and testing of high power cold white LEDs, by lighting installers, operators in lighting manufacturing facilities, show technicians and collectors, dentists, surgeons, etc.;
- the use of toys with LEDs, since children have a crystalline lens more transparent to blue light;
- automotive LED daytime running lights, when activated near children or photosensitive persons (aphake, pseudo-aphake eyes, people suffering from ocular photosensitive pathology or using photosensitive drugs, etc.);
- some directional LED lamps sold for home applications, if viewed at distances equal or shorter than 200 mm;
- the prolonged and repeated use of cold white LED-based devices by children and teenagers, especially in the evening, etc.

**Individual characteristics**

Each person is unique. We do not respond to equal light exposure the same way. Genetics, morphology, ethnic, gender, age, behaviors (smoking, diet, etc.), squinting effects, eye protection (eyewear, shadow cap, nutra-ceuticals, etc.) are all contributors to a distinct personal risk profile. Age, for instance, is largely involved in the progressive deterioration of visual functions such as dark adaptation.

**Fig. 3** | Individual risk profiling is defined by multiple intricate factors.
number of photosensitizers is rapidly increasing in the retina, particularly in the RPE where the lipofuscin age pigment builds up. This increase is partly due to blue photo-ageing processes. Age plus cumulative blue light exposure may irreversibly alter the classical visual cycle in the outer retina, progressively leading to AMD.

**New tools for personalized early diagnosis**

In the case of AMD, Erica Fletcher et al. have interestingly reviewed the new means of detecting the signs of early-stage disease.46, 47 AMD has genetic and environmental risk factors. Genetic testing is now readily available using a combination of 16 genes to help predict the risk profile of an individual. Among the genes associated with an increased risk of developing AMD is complement factor H (CFH), the mutations of which contribute to explain immune dysregulation. Retinal imaging, Hogg et al. have recently highlighted that a particular form of drusens called reticular pseudodrusen, at a subretinal level, is a risk-factor for progression to late-stage disease.48 A research team in the Netherlands has developed a software to analyze color fundus photographs so as to quantify and characterize drusens and determine a risk assessment. Using fundus autofluorescence (FAF), the RPE cell dysfunction can be detected through the accumulation of lipofuscin. Some scientists report that certain patterns in FAF imaging can help predict the evolution of AMD towards the choroidal neovascularization (CNV) late-stage form. Psychophysical methods can also help define if a subject is at greater risk of AMD or, later, of progressing to late-stage disease. Next, it would be helpful to monitor the light exposure in cohorts of patients and try to associate disease progression. Therefore, a comprehensive clinical assessment and biomarker testing, including questionnaires (family history, lifestyle, etc.), visual examination and specific psychophysical methods, imaging, genetic testing and light exposure profiling can define an individual profile risk and help detect and characterize AMD at an early stage as well as monitor disease progression.

**3. PERSONALIZED PREVENTION**

For most eye diseases, an individual can undergo a complete assessment of his or her risk profile specifically related to a disease and then if needed, once informed and educated, decide to adopt a more thorough and frequent medical and self-surveillance so as to detect early enough the onset and progression of a disease and to take personalized prophylactic actions.

Light protection should be part of a personalized prophylactic program instructed by eye care practitioners. If a patient is profiled at risk of one of the vision-threatening diseases, and if the precise light spectrum incriminated as a risk factor is known (such as blue-violet light for AMD), then it is in the patient’s interest to protect himself selectively, especially when a portion of visible light needs to be filtered out as part of the preventative measure. Contrary to UV radiations, which can be fully blocked with no compromise on vision, filtering out visible light is always a trade-off with color vision and other physiological functions such as chronobiology or scotopic vision. Fortunately, the ophthalmic optics industry, in recent years, has fostered or benefited from the emergence of innovative narrow-band filtering technologies and has now started to develop photoselective and photoprotective ophthalmic filters. These new lenses offer practitioners an effective complementary tool to the armamentarium of prophylactic solutions available to their patients.

**4. CONCLUSION**

Major eye diseases, such as cataracts, AMD, glaucoma or diabetic retinopathy, have a tremendous impact on patients’ quality of life but also significant implications on the cost of healthcare. The threat is enhanced by the accelerated ageing of the world population and by evolving behavioral and environmental factors. Photobiology research has progressively identified light as a risk factor for these multifactorial conditions. Further photobiology studies, in vitro and in vivo, with well-calibrated light conditions, along with epidemiology studies with proper spectral light exposure quantification, are now necessary to identify significant correlations between light action spectra and pathogenic mechanisms. As initiated in RPE and RGC models, Essilor and the Paris Vision Institute are pursuing their efforts in developing accurate photometric tools (devices and protocols) for controlled cell illu-
mination. A better understanding of the pathogenesis of eye conditions would interestingly be complemented by a better disease management, including real-life phototoxicity studies to find light-specific early markers of a disease and individual risk profiling. In our digital world with huge technical tools in image analysis and miniaturized sensors, the personalized and early follow-up of an eye disease is now made possible and paves the way for relevant personalized prevention. In the meantime, technology breakthroughs in the ophthalmic optics industry are meant to provide new effective solutions to design efficacious and personalized photo-protective and photo-selective eyewear.

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**INTRODUCTION / ABSTRACT**

Ultraviolet radiation (UVR) potentially damages the skin, the immune system and structures of the eye.

Today there is no reliable and universal method to assess and compare protective properties of lenses.

Sunglasses as well as clear lenses can reduce transmission of UV effectively, however, an important share of the UV burden is attributed to reflection from the backside of the lenses.

To provide reliable labelling of the UV protection offered by lenses, an Eye-Sun Protection Factor (E-SPF™) has been developed by Essilor, encompassing both transmission and reflection.

A group of experts: ophthalmologists, optometrists and dermatologist, from 5 European countries reviewed existing literature on UV dangers and evaluated the relevance of E-SPF™.

Scientific articles have been submitted to Clinical Journal in Ophthalmology and Dermatology (1,2).

**1- THE EYE AND UV EXPOSURE**

**UV definition**

Ultraviolet radiation is defined by the wavelengths 100 to 400 nm. UV-C (100-280nm) is essentially absorbed within the atmosphere. Of the UVR reaching earth UV-B (280-315nm) accounts for 5% and UV-A (315 nm and above) up to 95%.

The shorter the wavelength, the more spectral energy increases, and the higher the potential damage. The potential biological damage at 300 nm is 600 times greater than at 325 nm for example (Fig. 1).

**Sources of UV**

The main source of UVR is sunlight. Artificial lighting contributes to a lesser extent but may increase with the advent of energy efficient light sources (3).

**Ambient UV: direct radiation, scatter, and reflection**

Direct sunlight only partly contributes to ambient UV. Under average conditions, more than 50% of ocular exposure comes from scattering and reflection from clouds and the ground.

The average annual UV dose is estimated to be 20,000 to 30,000 J/m² for Americans, (4) 10,000 to 20,000 J/m² for Europeans, and 20,000 to 50,000 J/m² for Australians. Vacations can add more than 30% to the UV dose.

**Fig. 1** | Radiation as a function of increasing photon energy.

**Fig. 2** | Hourly average of UV intensity in the eye when facing towards and away from the sun (Watt/m²).

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UV AND EYE PROTECTION

**EYE-SUN PROTECTION FACTOR.**

**A NEW UV PROTECTION LABEL FOR EYEWEAR**

CHRISTIAN MIEGÉ

Director Professional Relations

Essilor Europe, France

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2- Absorption and transmission within the eye

Identifying absorption and transmission of UVR within structures of the eye is key to understanding potential damage [9].

UV transmission is strongly dependant on age. Below 9 years of age, a larger portion (2-5%) of UVA is transmitted by the cornea and the lens. Significant inter-individual differences have also been shown [10].

3- UV hazard to eye structures

Acute and chronic damage to the eye by UV and visible light has been extensively studied, including epidemiological studies, with greater significance on chronic exposure [11].

Cornea

The cornea is most exposed, with the greatest level of UVR absorption from direct irradiation (Fig. 4). In addition oblique rays are reflected across the cornea and anterior chamber into the limbal area leading to elevated pathologies in this area. Most common diseases: Pterygium, pinguecula, climatic droplet keratopathy.

Cortical cataract

It is known that UV light induces cataracts [12] with a damage threshold at 350 nm of 60 mJ/cm². With growing and aging populations and other changing demographic factors the incidence and prevalence of cataracts will increase. Reducing the risks that can lead to cataracts is therefore important.

Dry eye, premature presbyopia, AMD

Decreasing tear film production linked to ageing, reduces UV absorption and antioxidant production by tears.

The association between UVR and AMD remains controversial. Blue light is a more significant contributor to development of AMD.

UV related skin aging and diseases of periorbital skin

The acute response of the skin to UV is inflammation (sunburn). Clinical symptoms include erythema, swelling, pain and pruritus [13].

Chronic effects include photoaging and photocarcinogenesis. Some clinical signs of photoaged skin include dryness, irregular pigmentation, lentigines, wrinkling and inelasticity. The delicate periorbital skin is particularly susceptible to effects of photoaging [14].
Mitochondrial DNA is a chromophore for UVA and UVB and subject to damage by UVR. DNA deletions are increased by up to 10-fold in photoaged skin compared to sun-protected skin of the same individual.\(^{[11]}\)

Photocarcinogenesis includes the development of actinic keratosis, squamous cell carcinoma, basal cell carcinoma, and malignant melanoma (Fig. 5). 5% to 10% of skin cancers are appearing on the eyelids.\(^{[17]}\)

**4. THE NEED FOR EYE PROTECTION**

**Populations at risk**

With the increase of life expectancy and cumulative effect of UVR exposure during all the life, the protection of the eye against UVR concerns everyone, and should start at the earlier stage. As already indicated, UV transmission to the retina is greater in children.\(^{[11]}\)

For those spending time at higher altitudes, outdoor workers, and those spending more leisure time outdoors, ocular UV exposure is greater.\(^{[16]}\)

Photosensitising drugs, such as psoralenes, non-steroidal anti-inflammatory drugs, antiarrhythmics, tetracyclins, and chloroquine increase susceptibility to UVR damage.

**Eye-Sun Protection Factor**

The clothing industry employs UPF (Ultraviolet Protection Factor) to measure garment UV transmission.\(^{[10]}\) While in the skincare industry, UV protection is defined by SPF (Sun Protection Factor) and is applied to sunscreens and some daily creams (European Standard EN 13758).

Essilor developed an Eye-Sun Protection Factor for lenses taking into account: transmission, reflection from the back surface, protection of structures of the eye and periorbital skin.

An accepted E-SPFTM used by manufacturers, eye care professionals, and consumers will enable identification and comparison of the UVR protective properties of lenses. This includes clear prescription lenses, contact lenses and sunglasses (prescription or non-prescription).

**E-SPFTM figures are calculated using the following formula:**

\[
E – SPP^{TM} = \frac{1}{T_{UV} + R_{UV}^{145}}
\]

**E-SPFTM was defined taking** into account transmission and reflection of UV and visible light at angles from 0° for light coming through the lens and from 145° incidence for light coming from the backside of the lens. It gives a clear understanding of its intrinsic ability to protect the eye.

**Table 1 shows that E-SPFTM values are similar to SPF labeling for sunscreens and for the consumer this familiarity could help them easily understand the level of protection provided by spectacles and sunglasses.**

**Additional factors also** play a role such as the spectacle frame, anatomical features of the individual, solar angle, and UVR which might enter the space between the frame and the eye.

**CONCLUSION**

With increasing life expectancy and changing lifestyles, the cumulative effects of UVR in the periorbital region (malignancies), at the cornea and conjunctiva (pterygia) and the crystalline lens (cataracts), are of increasing relevance to public health.

**UV protection for the eye** and the periorbital area is often inadequate and not well defined.
UV and eye protection

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Table 1 | Tuv = Transmission of UV, Ruv = Reflection of UV, E-SPF™ = Eye-Sun Protection factor.

This paper proposes an E-SPF™ to deliver a unified, easily understood index of UV protection for lenses. Lens manufacturers are encouraged to adhere to a shared standard.

REFERENCES

NEW OPHTHALMIC LENSES FOR A CONNECTED LIFE: EYEZEN™ FOR AMETROPE AND EMMETROPE, AND VARILUX® DIGITIME™* FOR PRESBYOPE

The advent of digital technology has not only changed methods of communication and information management, but also the visual and postural-motor habits of users. To respond to these emerging needs, the ophthalmic industry has taken the path of innovation in the area of physical chemistry and optical design. This article presents a twofold technological breakthrough, which gave rise to a new category of ophthalmic lenses for a connected life. Designed by Essilor, a world leader in ophthalmic optics, these lenses are dedicated to users of all types of digital devices.

NEW OPHTHALMIC LENSES FOR A CONNECTED LIFE: EYEZEN™ FOR AMETROPE AND EMMETROPE, AND VARILUX® DIGITIME™* FOR PRESBYOPE

The last ten years were marked by the emergence of digital devices, such as smartphones and tablets. Already indispensable to our daily lives, they are revolutionizing the way we communicate, learn, stay informed, work, entertain ourselves and relax. With an average of four different devices (computer, smartphone, tablet and TV), we tend to switch between them more frequently and at times even use them simultaneously (Fig. 1). The time spent using these devices has increased significantly and continues to grow: indeed, nine out of ten people state that they spend more time using them today than they did two years ago. Our daily lives have been turned upside down by this digital revolution, but everything has also changed for our eyes.

Specific needs
Indeed, the use of these devices creates new visual and postural behaviours and modifies our light environment. Ten years ago, there was only one reading distance: the distance at which we held a book or newspaper. On paper, characters have always been fixed in size and highly contrasted. Today, in addition to reading books, we also read on smartphones, tablets and computers at different distances (some of them quite short) and in various postures, as a study undertaken by the Essilor R&D has shown (page 22). On screens, characters are becoming smaller and more pixelated.

Céline holds a French engineering degree from Ecole Centrale Marseille and Paul Cézanne University in Marseille (where she obtained her Master in Optics and Photonics). In 2010 she defended her thesis in optics in collaboration with Essilor International and Charles Fabry laboratory. After working one year as a research engineer at ONERA (a French aerospace research center), she definitively joined Essilor International’s R&D Optics department in 2011. Céline develops designs to meet the emerging needs of wearers.

Marie holds a French engineering degree from Chimie Paris Tech and a Masters in marketing from Essec Business School. She joined Essilor International in the Global Marketing team in 2011. Marie is in charge of projects targeted at lens wearers with specific needs, such as pre and emerging presbyopes. She develops innovative marketing offers, such as the new ophthalmic lenses aimed at users of digital devices.

KEYWORDS
Eyezen™, Varilux® Digitime™, mid-distance lenses, Crizal® Prevencia®, power distribution technology, light filtering technology, blue light, blue-violet light, blue-turquoise light, UV, AMD, LED, anetropia, emmetropia, presbyopia, pre-presbyopia, digital screens, posture, digital devices, connected life, eyestrain, photobiology.

* Product availability date may vary depending on the country
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**NEW OPHTHALMIC LENSES FOR A CONNECTED LIFE:**

**EYEZEN™ FOR AMETROPES AND EMMETROPES,**

**VARILUX® DIGITIME™**

*PRODUCT*

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R&D Optics, Essilor international, France.

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Marie Jarrousse

Global Marketing, Essilor International, France.

**KEYWORDS**

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Furthermore, our light environment has changed. Ten years ago, our eyes were exposed to natural light or artificial light emitted by incandescent lamps. Now, our eyes are constantly exposed to the bright light of screens and other light sources, such as LED or CFL bulbs, which strongly emit of diffusing and potentially harmful blue-violet light.

As a result, our eyes must focus more intensely and more often to adjust to the varying distances of use and to small pixelated characters found on screens. This not only causes eyestrain, but also postural aches and pain. In fact, a study conducted in 2014 by the Ipsos institute on four thousand people in France, the United States, Brazil and China revealed that:

- Two out of three people feel that they must make an additional effort in front of screens to see well,
- Three out of four people suffer from eyestrain,
- 70% complain of neck and shoulders pain
- Over one out of two people are bothered by the strong brightness of their screens.

Lastly, the harmful blue-violet light emitted by screens can contribute to premature aging of the eyes.

This study showed that all users of digital devices – regardless of their age, the type of device used and the frequency of use, feel the same discomfort, related to the difficulty of reading small characters and screen brightness. However, the level of discomfort varies depending on the user’s age. In fact, for younger users, the main discomfort is screen brightness, while for older users, it is the effort required to decipher the small characters.

A complete range designed for connected life
To meet these new visual needs, Essilor designed a new category of lenses for a connected life. This all-new range of ophthalmic lenses provides a correction for each reading distance required by digital devices, relaxes users’ eyes and protects them against the potential dangers of blue-violet light:

- Advanced single-vision lenses combined with Crizal® Prevencia® coating, called Eyezen™, for young adults (aged 20-34), pre-presbyopes (aged 35-44) and emerging presbyopes (aged 45-50), available for all prescriptions, and even for emmetropes,
- Mid-distance lenses combined with Crizal® Prevencia® coating Varilux® Digitime™, for presbyopes (aged 45 and over), specifically designed for digital devices use.

Eyezen™ lenses are meant to replace standard single-vision lenses for wear by ametropic patients, but are also intended for occasional wear by emmetropes during their on-screen activities.

There are three different products optimized for three user profiles depending on their age (Fig. 2):

- For young adults (aged 20-34): Eyezen™ 0.4
- For pre-presbyopes (aged 35-44): Eyezen™ 0.6
- For emerging presbyopes (aged 45-50): Eyezen™ 0.85
### The New Single Vision Lenses as Primary Pair for Ametropes or in Plano for Emmetropes.

With 3 Optimizations Depending on Profiles:

<table>
<thead>
<tr>
<th>Young Adults</th>
<th>Pre-Presbyopes</th>
<th>Emerging Presbyopes</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 - 34 years old</td>
<td>35 - 44 years old</td>
<td>Without near vision correction 45 - 50 years old</td>
</tr>
</tbody>
</table>

**Eyezen™ 0.4**

- **Standard Correction**
- **With +0.4 D**
- To relax eyes of **20-34 YO** from digital stress

**Eyezen™ 0.6**

- **Standard Correction**
- **With +0.6 D**
- To relax eyes of **35-44 YO** from digital stress

**Eyezen™ 0.85**

- **Standard Correction**
- **With +0.85 D**
- To relax eyes of **45-50 YO** from digital stress

Protection from UV & Harmful Blue-Violet Light emitted by Screens. Reduced screen glare & improved contrasts.

**Thanks to Crizal® Prevencia®**

---

Fig. 2] Eyezen™ lens range for 20- to 50-year-olds, segmented by age (advanced single-vision lenses combined with Crizal® Prevencia® coating)
### PRESBYOPES
With near vision correction 45 - 65 years old

<table>
<thead>
<tr>
<th>KEEN ON SMARTPHONES &amp; TABLETS</th>
<th>KEEN ON COMPUTERS</th>
<th>KEEN ON LARGE SCREENS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Varilux® Digitime™ NEAR</strong></td>
<td><strong>Varilux® Digitime™ MID</strong></td>
<td><strong>Varilux® Digitime™ ROOM</strong></td>
</tr>
</tbody>
</table>

**Extended Vision**
- ○ ○ ○

**Intermediate Vision**
- ● ● ○

**Near Vision**
- ● ● ●

**New Ultra Near Vision**
- □ RELAXED VISION FOR SMARTPHONE

---

**Protection from UV & Harmful Blue-Violet light emitted by screens. Reduced screen glare & improved contrasts.**

**Thanks to Crizal® Prevencia®**

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**Notes:**
- The Varilux® Digitime™ mid-distance lens range for presbyopes aged 45 and over, segmented according to digital device use (occupational lenses, for occasional wear during on-screen activities or other activities requiring near or intermediate vision correction, combined with Crizal® Prevencia® coating).
In addition to providing perfect correction for ametropia, Eyezen™ lenses also provide the necessary accommodative support to relieve eyes from stress while using digital devices, which differs depending on age. On top of that, they protect eyes from the harmful light emitted by screens, reduce glare and improve contrast thanks to their Crizal® Prevencia® coating.

Lastly, the mid-distance lenses are intended for occasional wear by presbyopes during their on-screen activities or during any other activity requiring near or intermediate vision correction.

There are three different products optimized for three different categories of presbyopes, depending on the device they use most frequently (Fig. 3):

- For presbyopes keen on smartphones and tablets: Varilux® Digitime™ Near
- For presbyopes keen on computers: Varilux® Digitime™ Mid
- For presbyopes keen on large screens (TV or video projectors): Varilux® Digitime™ Room

Varilux® Digitime™ Near mid-distance lenses are optimized for smartphone or tablet use with wide near vision fields, but they also provide an intermediate vision field suitable for computer use. The minimum guaranteed depth of field is 80 cm, regardless of the prescription.

Varilux® Digitime™ Mid mid-distance lenses are optimized for computer use with wide intermediate vision fields, while also providing a near vision field suitable for smartphone or tablet use. The minimum guaranteed depth of field is 100 cm, regardless of the prescription.

Finally, Varilux® Digitime™ Room mid-distance lenses are optimized for large screens use with wider extended vision fields (delimited by room size) and offer intermediate vision and near vision fields suitable for computer, smartphone or tablet use respectively. The minimum guaranteed depth of field is 220 cm, regardless of the prescription.

Response to emerging needs in the design of this new range of lenses for a connected life
To respond to the emerging needs of wearers, Essilor brings its expertise in two areas: first, in optical lens design, to provide a perfectly suited correction, and secondly, in the area of protective lens coatings, to protect the eyes against the potential dangers of the blue-violet light emitted by screens. The range originated with a twofold technological breakthrough.

A unique technology of power distribution
Essilor R&D conducted a study on the emerging visual and postural behaviors engendered by the use of digital devices. The study revealed that the average reading distance is nearer on these devices than when reading on traditional paper media (33 cm for smartphones and 39 cm for tablets versus 40 cm for paper). Researchers also noted an average increase in the eye declination angle while reading on a smartphone (25° for smartphones versus 18° for a reading task on paper). These data (Fig. 4) reveal the need for a new Ultra-Near Vision field.

![Comparison of reading distances (D, D’) and eye declination angles (Ed, Ed’) between a paper medium (newspaper) and a digital screen (smartphone)](image-url)

FIG. 4 | Comparison of reading distances (D, D’) and eye declination angles (Ed, Ed’) between a paper medium (newspaper) and a digital screen (smartphone)
A unique power distribution technology was designed to respond to emerging needs. This technology distributes the power over the lens, including additional power in the bottom part of the lens to support the eye's accommodation effort when using digital devices, according to the physiological needs of each identified group of wearers.

It is important to point out that this additional power respects the physiological functioning of the visual system, without inhibiting the accommodative function of the wearer’s eyes.

How is this technology managed on Eyezen™ lenses?
The additional power values selected are related both to the fact that the objective amplitude of accommodation decreases with age² (Fig. 5), and that accommodative power drops after sustained and prolonged near-vision work³. For example, a drop in accommodative power of 0.4D was observed after 20 minutes of near-vision work for a traditional reading task³. For this reason, the additional refractive power provided is 0.4D for the 20-34 age group, 0.6D for the 35-44 age group, and 0.85D for the 45-50 age group.

How is this technology implemented on Varilux® Digitime™ mid-distance lenses for presbyopes?
Most near-vision exams are performed at a distance of 40 cm for reading tasks. This data is taken into account in the design of the new ophthalmic lenses for presbyopes connected life, so that wearers can effortlessly find their near-vision zone while reading. However, when using their smartphone, they bring it closer and naturally lower their gaze. At that point, their eyes encounter the additional power under the near-vision zone, thus relieving accommodative excess.

This technology creates an additional field of vision: the Ultra-Near Vision zone, which provides additional power. This zone, located under the near-vision zone, allows users to lower than gaze more than when they are reading on paper media. This additional power allows users to use their device at closer distances.

The additional power provided by the lens reflects users’ specific characteristics, as well as their prescriptions and the widths of the fields of view of the target product. So the additional power will not exceed 0.50D depending on the chosen lens and prescription.

Finally, the near-vision zone is not impacted by the additional power. The eye health practitioner’s prescription is therefore always respected.

Since using a smartphone at a distance of less than 40 cm (Fig. 6) is likely to generate a substantial degree of discomfort and difficulty in focusing (i.e. blurred vision), the additional power under the near-vision zone provides support for accommodative effort, giving the wearer’s vision more clarity.

What are the benefits of this technology for the wearer?
The additional power helps to reduce visual fatigue for the wearer, even during prolonged smartphone use. It also improves the readability of small characters. Finally, it allows presbyopes to adopt a more natural posture when using their smartphone.

Ergonomics of visual field positioning on Varilux® Digitime™ mid-distance lenses for presbyopes
Positioning of visual zones on mid-distance Varilux® Digitime™ lenses. The ultra-near, near, intermediate and
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**FIG. 5** Change in the amplitude of objective accommodation as a function of age

<table>
<thead>
<tr>
<th>Wearer’s age (in years)</th>
<th>Objective accommodation amplitude (in Diopters)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>8</td>
</tr>
<tr>
<td>30</td>
<td>7</td>
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<td>40</td>
<td>6</td>
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<td>80</td>
<td>2</td>
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<tr>
<td>90</td>
<td>1</td>
</tr>
<tr>
<td>100</td>
<td>0</td>
</tr>
</tbody>
</table>

**FIG. 6** Benefits of the Ultra-Near Vision zone provided by additional power. Smartphone use without additional power (above). With additional power (below).

Additional power in the lower part of mid-distance lenses located under the near vision zone.
extended visual zones are positioned optimally – in view of design constraints (fields of view width, prescription, minimal guaranteed depth of field, etc.) natural gaze lowering of the wearer and average use distances for each specific digital device (Fig. 6). With the exception of the ultra-near vision zone, which includes a progressive zone followed by an area of stable power, all zones are stable in power, therefore improving wearer comfort.

The positioning of these zones is customized to the prescription to follow the wearer’s natural convergence and provide good binocular vision. In this regard, the zones are properly positioned in the lens, vertically and horizontally, to minimize visual fatigue for the wearer.

**Characteristics of the intermediate vision zone.** Between the near vision zone and the intermediate vision zone, or extended vision zone depending on the case, there is a variation in power known as degression (Fig. 7). This helps to ensure a given minimum depth of field (Fig. 3). The value of this degression is set according to the prescribed addition value, the desired minimum guaranteed depth of field and the wearer’s subjective accommodation.

**Customization of intermediate vision on Varilux® Digitime™ mid-distance lenses**

The average distance of use for a computer is 63 cm, but a wide variation has been observed: 95% of people use a computer at distances between 38 and 88 cm. It is therefore recommended to customize intermediate vision for each individual.

To customize intermediate vision, Essilor has taken into account a new parameter known as “Screen Distance”, which corresponds to the distance between the eye and the computer screen. When calculating the lens, this
parameter is used to customize the depression and the horizontal positioning of the intermediate vision zone relatively to the near vision zone. Taking the “Screen Distance” into account does not change the vertical position of the intermediate vision zone in the lens. The length of the depression is therefore fixed. A general illustration of customized depression as a function of “Screen Distance” is given in Fig. 8 for Varilux® Digitime™ Room mid-distance lenses.

The default “Screen Distance” value is set to 63 cm which is the average distance of use for a computer (in the event that the optician do not indicate this parameter when the order is placed). It may range between 35 and 99 cm (as a reminder, 95% of people use their computer at a distance ranging between 38 and 88 cm).

**The benefits** provided by this parameter are a natural posture in front of the computer and maximum comfort for intermediate vision use.

**New light environment**
In addition to the optical design of lenses, it is essential to define an appropriate treatment for new light environments and for the spectral characteristics of the light emitted by screens.

**The role of light is essential, but can also be harmful at times**
Visible light plays a crucial role in our everyday lives. It is essential, in particular, for the perception of colours,
PRODUCT

Protection provided by Crizal® Prevencia®

Photobiological studies demonstrated an average 25% reduction in cell death by apoptosis comparatively between Crizal® Prevencia® lenses and exposure without a filter over the spectral range [400 nm ; 450 nm]. Fig. 10 illustrates the comparative levels of apoptosis between the naked eye (grey) and a Crizal® Prevencia® lens for each blue light bandwidth. This level of protection over the long term would mitigate the risk related to harmful blue light, and therefore the onset of AMD.

Blue-violet light is everywhere

Today, our eyes are confronted with potential new dangers, both at home and at work. Several independent studies conducted by health agencies are now taking an interest in risks related to new sources of artificial light, such as light emitting diodes (LEDs), since the latter, particularly cool white LEDs, present an emission peak in the harmful blue-violet band (Fig. 11) and have a more elevated luminance/brightness than traditional sources. Yet, LEDs are now present in most modern lighting systems and in a large number of screens, especially computer, tablet and smartphone screens.

A unique light filtering technology and wearer benefits

Concerned about the amount of time we spend in front of screens, Essilor has made protection one of its priorities for its new range of lenses designed for a connected life. To protect our eyes, Crizal® Prevencia® coating, the product of a unique light filtering technology, was therefore integrated into the entire range.

This technology selectively filters out harmful blue-violet light, emitted especially by screens, while allowing beneficial light (including blue-turquoise light necessary to the proper regulation of circadian rhythms) to pass through. This technology also provides the best protection against reflected light, smudges, scratches, dust and water for optimal vision and lasting transparency.

Identification by Essilor and the Paris Vision Institute of the toxicity of blue light on the retina

Joint research conducted by the Paris Vision Institute (see inset) and Essilor International led to the identification of the most dangerous part of the blue light spectrum for retinal cells involved in the development of AMD. The light inducing the highest mortality rate in retinal cells corresponds to a narrow band of 40 nm centred on 435 nm. These wavelengths correspond to the blue-violet light bordering on the blue-turquoise light that is essential to our health (Fig. 9). The research was conducted on retinal pigment epithelium (RPE) cells, the first cells to degenerate in AMD. These cells were photosensitized, to form a model of aging and AMD, and exposed to narrow 10-nm bands of illumination in the blue-green spectral range, between 390 nm and 520 nm, under physiological conditions of solar radiation on the retina (with a control band centred on red at 630 nm).

Chronic exposure to light also presents some risks for our visual health. Blue light is the highest energetic light to reach the retina, since ultraviolet radiation (UV), which is even higher in frequency, is blocked by the anterior ocular media. There is a rich literature on the harmful effects of blue light on the retina, the first articles dating back over forty years. But it was only recently that the precise spectrum of toxic action of this light on a cellular model of AMD was demonstrated.

Contrast and for visual acuity. Besides vision, certain frequencies of the visible light spectrum are even more important for our health. For example, blue-turquoise light, around 480 nm (465-495 nm) is known as “good blue” light because it is responsible for synchronizing our circadian rhythms (our biological clock) in charge of regulating our waking and sleep cycles, as well as our body temperature and mood, among other things.

The benefits of Eyezen™ lenses and mid-distance lenses

Varilux® Digitime™ Near, Mid and Room Distance...
The benefits of Eyezen™ lenses and mid-distance lenses combined with Crizal® Prevencia® coating have been confirmed by numerous tests. When evaluated under actual conditions of use, 89% of wearers reported that they experienced reduced glare and better contrast during screen use. In addition, in vitro tests have shown that blocking 20% of blue-violet light would reduce the rate of retinal cell (RPE) death by apoptosis by 25%. This should contribute to longer-term health benefits, and particularly to the prevention of premature aging of the eyes.

**Recommendations concerning the mode of prescription**

For the prescriber, the new lens range stands out for its simplicity of implementation. We have made no changes to the prescriber’s usual practices. He or she conducts the eye exam to obtain the prescription in the usual manner.

- For Eyezen 0.4, 0.6, 0.85 lenses, the only prescription required is for distance vision: the prescriber does not need to worry about selecting additional power.

- For Varilux® Digitime™ Near, Mid and Room mid-distance lenses, the distance vision prescription and addition is required: the prescriber does not need to worry about selecting the design.

As for the optician, Essilor can provide a “Screen Distance” measurement tool for in-store use so that the customer can also enjoy the benefits of a customized version of mid-distance lenses. All that the optician need do, when placing the order, is indicating the “Screen Distance” value obtained via this measurement tool in addition to the customer’s prescription.
Conclusion
By continually placing end users at the heart of its innovation process, Essilor closely studied how new digital devices and their use are impacting vision and posture, thus making it possible to identify and characterize new visual needs.

This in-depth understanding of the users of digital devices led to a combination of two cutting-edge technologies of power distribution and light filtering. This patented technology alliance (several patent applications have been filed and are currently under consideration), the basis of both Eyezen™ lenses for ametropes and emmetropes and of Varilux® Digitime™ mid-distance lenses for presbyopes, is a perfect fit with our new connected lifestyles.

These new lenses underwent performance testing under actual conditions of use and prescription before they were placed on the market. This approach was adopted to confirm their benefits and measure wearer satisfaction.

Institut de la Vision de Paris - is linked to Pierre & Marie Curie University, the Paris Vision Institute is considered one of Europe’s foremost integrated research centres of excellence on eye diseases and vision. 200 researchers and medical doctors and 15 industry players work together for the discovery and clinical evidence of new therapeutic approaches and preventive solutions, as well as innovative compensatory technologies for vision impairments.

www.institut-vision.org
YOUR PATIENTS SPEND A LOT OF TIME LOOKING AT SCREENS.

Smartphones, tablets, computers and TV are now an indispensable part of their lives, to socialize, inform, learn, educate, work, play, relax and see the world.

4 DIFFERENT DIGITAL DEVICES ARE USED ON AVERAGE FOR WORK, EDUCATION AND LEISURE*.

2 OUT OF 3 PEOPLE DAILY USE A SMARTPHONE.

64% OF PEOPLE SPEND 4 HOURS OR MORE ON A COMPUTER PER DAY.

* Consumer quantitative study conducted in 2014 among 4,000 individuals in France, Brazil, China and the US by Ipsos for Essilor.

REFERENCES
1. Ipsos, Digital devices users: behaviours and needs, May 2014. Quantitative consumer survey conducted in France, United States, China, Brazil, 4,000 respondents.

KEY TAKEAWAYS
• Connected life has changed our light environment and visual and postural behaviours.
• Essilor internal and external research centers have achieved a detailed comprehension of these needs.
• The new range of lenses for a connected life is the result of this research and was designed to respond to these new needs.
• It is available in several products to specifically meet each user’s needs.
• They are suitable for all users starting at age 20.
• The range was tested and approved by wearers before it was placed on the market.

www.pointsdevue.com
THE NEW RANGE OF EYEZEN™ LENSES: WHAT ARE THE BENEFITS PERCEIVED BY WEARERS DURING SCREEN USE?

With recent technological advances, ophthalmic lenses can now offer more than just good everyday vision. They are also aiming to meet emerging needs arising from connected life. Innovations are put to the test by specialized research institutes to measure user satisfaction and the effects of lenses on postural and visual fatigue during screen use. The new Essilor® Eyezen™ lenses were tested in an independent study before they were placed on the market. This article describes the results obtained with a population of ametropic patients wearing single vision lenses.

Essilor® Eyezen™ lenses were defined according to Essilor’s R&D programme: LiveOptics. This programme includes four major steps for the introduction of a new design. Tests conducted with consumers known as “wearer testing” constitute the fourth part of this programme.

Because the best evidence is provided by the wearer, it was essential that this new class of Eyezen™ lenses be tested and approved by the final consumer.

To ensure the impartiality of this type of test and lend credibility to the key role assigned to the wearer in the quality process, testing protocols are validated by independent research institutes. The latter, which hold large consumer databases, are in charge of implementing questionnaires for the purpose of evaluating the objective and subjective performance of Essilor® products.

As a product testing specialist, Brieuc de Larrard is contributing in large measure to the development of Eurosyn’s Research department and to the introduction of sensory testing in numerous business sectors. Today, he actively participates in the development and validation of innovative product testing methodologies on a national and international scale.

Keywords: Eyestrain, postural fatigue, glare, headaches, dry eye, contrast perception, adaptation, comfort, posture, digital screens, ergonomics, e-reading, digital devices, connected life, computer, smartphone, tablet, Essilor® Eyezen™, Crizal® Prevencia®, ophthalmic lenses, wearer test, protocol.
“To ensure the impartiality of this type of test and lend credibility to the key role assigned to the wearer in the quality process, testing protocols are validated by independent research institutes.”

INTRODUCTION
To focus attention on the consumer benefits of the new class of Eyezen™ ophthalmic lenses, Essilor contacted Eurosyn, a French market research institute specializing in sensory analysis. In cooperation with this institute, Essilor established a test protocol for the purpose of validating the performance of Essilor® Eyezen™ lenses with a target group of ametropic subjects.

The purpose of this study was to verify whether or not an effortless transition from standard single-vision lenses to Essilor® Eyezen™ lenses was possible for persons with refractive error. More specifically, the goal was to determine the perceived benefits of this new range of lenses during digital device use.

METHODOLOGY
The Essilor® Eyezen™ range comprises three new products: Essilor® Eyezen™ 0.4, Essilor® Eyezen™ 0.6 and Essilor® Eyezen™ 0.85. These three additional refractive powers were all tested during this study. Thin lenses (n = 1.67) equipped with Crizal® Prevencia® coating were chosen for this study. The lenses were optically centred on the height of the pupil, taking into account the measurement of the pupillary distance for far vision. Before beginning the wear period, each of the testers answered a quantitative questionnaire to assess their general satisfaction and the level of visual fatigue experienced (if any) with their usual eyeglasses (standard single-vision lenses). Each tester was asked to wear Essilor® Eyezen™ lenses instead of their main pair of usual eyeglasses for four weeks. These tests were performed “blind”, i.e. the subjects were given no information on the type of lenses being tested, and the prescription for these lenses was exactly the same as the prescription for their previous pair of glasses, to avoid any bias related to the new refraction.

At the end of the wear period, the consumers evaluated the performance of the Essilor® Eyezen™ lenses by completing an online questionnaire. This questionnaire was used to quantify wearers’ satisfaction in terms of visual comfort during the performance of everyday tasks, and more specifically, during tasks related to the use of digital devices.

POPULATION
Inclusion criteria were: 1/ Be between 20 and 55 years of age. 2/ Be a user of digital devices (for at least 6 hours a day). 3/ Alternate between different screens. 4/ Present symptoms of visual fatigue and/or postural discomfort. 5/ Not be a wearer of progressive lenses. Exclusion criteria were: 1/ Associated strabismus and amblyopia. 2/ Anisometropia greater than 1.5 dioptre. 3/ Diabetes, glaucoma or other eye diseases. Wearers were recruited online by Eurosyn. The institute then contacted each subject to discuss their availability for appointments: the first to select the test frame and take all necessary measurements; and the second, to be fitted with the eyeglasses to be tested. During this appointment, visual acuity testing for distance vision (Monoyer scale) and near vision (Parinaud) was performed. For this wearer test, the sample population was divided into three groups, depending on the wearer’s age: The first group of 25 wearers, aged 20 to 34, were equipped with Essilor®
Figure 1 shows that ametropia distribution is representative of non-presbyopic wearers of single-vision lenses.

Cylinder distribution is shown in Figure 2. It indicates a high percentage of low astigmatism values with 75% of the sample having a cylinder of less than 0.5 dioptre.

RESULTS

The results concern the entire Essilor® Eyezen™ range, including all three additional refractive powers (0.4, 0.6 and 0.85). They are consolidated over the complete sample. Seventy-six wearers tested the Essilor® Eyezen™ ophthalmic lenses for four weeks.

In this type of test, the first criterion to be verified is adaptation. The wearers reported that adaptation to these new Essilor® Eyezen™ lenses was easy (“fairly easy” to “very easy”), and 83% were satisfied, all additional refractive powers combined. As regards rapidity of adaptation, 79% rated it as rapid (“fairly rapid” to “very rapid”).

The testers wore the Essilor® Eyezen™ lenses on a continuous basis throughout their activities (including during their use of digital devices). In fact, 94% of them wore the
Essilor® Eyezen™ lenses more than four hours a day and over one out of two testers wore Essilor® Eyezen™ all day long (see Figure 3).

A first observation was made on visual fatigue symptoms and postural pain felt by subjects. All wearers recruited for the test previously experienced ocular or postural discomfort during screen use.

At the end of the wear period, all wearers reported that they felt less visual and postural discomfort while using their digital devices. They stated that this discomfort was less frequent and less intense with the test lenses (Figure 4 and 5).

The remainder of the analysis was aimed at identifying the benefits perceived by the subjects while wearing Essilor® Eyezen™ lenses.

Due to the additional refractive power provided at the bottom of the lens, they are perfectly suited to wearers who make demands on their near vision throughout the day, particularly while using digital devices. Figures 6 and 7 highlight the performance of Eyezen™ lenses: At the end of the day, 90% of wearers state that their eyes are less tired (‘somewhat less tired’ to ‘much less tired’), in comparison with how they felt with their old eyeglasses (standard single-vision lenses).
In addition, 91% of wearers who tested Essilor® Eyezen™ lenses felt that they had less difficulty reading small characters, particularly during smartphone use (see Figure 7: “somewhat better” to ‘significantly better’).

Moreover, 90% of wearers reported that light from screens caused less glare, as indicated in Figure 8.

It is also noteworthy that 89% of wearers had improved perception of contrasts with Essilor® Eyezen™ Crizal® Prevencia®, in comparison with their previous eyeglasses (see Figure 9).

Visual comfort = outcome of benefits

In addition, the study evaluated visual comfort during the use of digital devices, as well as the general satisfaction level.

Indeed, 91% of wearers reported having comfortable vision during screen use with Essilor® Eyezen™ lenses (see Figure 10). 83% of wearers were very satisfied with the level of on-screen comfort provided by these new lenses. On average, 72% of wearers were satisfied with their visual comfort using a computer compared to their previous eyeglasses.

The final result, at the end of four weeks of testing, indicates that 91% of ametropic wearers were satisfied with Essilor® Eyezen™ (see Figure 11), with a satisfaction level ranging from 7-10 on a scale of 10. It is also noteworthy that 78% of wearers reported being very satisfied with the new Essilor® Eyezen™ lenses (with a score of 8-10 on a scale of 10).

This demonstration of performance over the entire Essilor® Eyezen™ range was verified for each of the additional refractive powers (0.4, 0.6 and 0.85). All three ophthalmic lenses provided an equivalent level of satisfaction for the individual testers, as indicated in Figure 12.
In addition, 91% of wearers who tested Essilor® Eyezen™ lenses felt that they had less difficulty reading small characters, particularly during smartphone use (see Figure 7: “somewhat better” to ‘significantly better’). Moreover, 90% of wearers reported that light from screens caused less glare, as indicated in Figure 8. It is also noteworthy that 89% of wearers had improved perception of contrasts with Essilor® Eyezen™ Crizal® Prevencia®, in comparison with their previous eyeglasses (see Figure 9).

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Q: With these new lenses, does your ability to read small characters seem to be...

FIG. 7
Lesbarkeit kleiner Schriftzeichen

Q: With this new pair of glasses, the glare sensation facing screens is:

FIG. 6
Reduction in feelings of eye fatigue

Q: With this new pair of glasses, contrast perception is:

FIG. 9
Improved contrast perception

Q: With these new lenses, are your eyes...

FIG. 8
Glare during digital screen use

Q: What was your general level of satisfaction with this new pair of lenses? (1= Highly unsatisfactory to 10= Highly satisfactory)

FIG. 10
Visual comfort before and after using Essilor Eyezen lenses
(1= Highly uncomfortable to 10= Highly comfortable)

Q: What was your general level of satisfaction with this new pair of lenses? (1= Highly unsatisfactory to 10= Highly satisfactory)

FIG. 11
General satisfaction

Q: What was your general level of satisfaction with this new pair of lenses? (1= Highly unsatisfactory to 10= Highly satisfactory)

FIG. 12
General satisfaction relative to additional refractive power
CONCLUSION

Essilor® Eyezen™ lenses were tested and approved by ametropic wearers, who previously wore standard single-vision lenses, with very good results. Indeed, 91% of them were satisfied with the new Essilor® Eyezen™ lenses, regardless of their additional refractive power.

Throughout the testing, we observed that wearers preferred Essilor® Eyezen™ lenses to their previous eyeglasses by a wide margin. They reported that their eyes were less tired and that they had less difficulty reading small characters. Finally, during on-screen use, their impression of glare also seemed to have decreased while their perception of contrast increased.

In addition, this new type of lens can completely replace a standard single-vision lens, throughout the day for all types of activity. In fact, 94% of wearers, all prescriptions combined, wore these new ophthalmic lenses for a minimum of four hours a day.

In conclusion, Essilor® Eyezen™ lenses, combined with Crizal® Prevencia® coating, were truly appreciated by wearers. Today, 93% of them continue to wear their new eyeglasses, and 88% would recommend this new type of ophthalmic lens to their families and friends.

KEYTAKEAWAYS

- Essilor® Eyezen™ ophthalmic lenses combined with Crizal® Prevencia® coating were approved during a wearer test conducted by an independent institute.
- The results showed a reduction in all symptoms of visual and postural fatigue.
- A reduction in glare and improved contrast were demonstrated during the use of digital devices.
- 91% of wearers reported having comfortable vision during screen use, and 91% expressed satisfaction with Essilor® Eyezen™.
- Essilor® Eyezen™ lenses are proving to be an appropriate solution for the emerging constraints arising from connected life. They are a suitable replacement for standard single vision lenses.
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FROM RESEARCH TO HARFMUL LIGHT FILTERING

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INTRODUCTION

Light is a driving force of life, from the most basic function of producing cellular energy to permitting highly sophisticated processes in intelligent life forms. Essential to visual functioning, it brings an unexpected dichotomy to the eye, concomitantly conferring both beneficial and harmful light. Irreversible eye damage from noxious light exposure, which is exacerbated in our currently aging population, has become a preoccupying public health issue.

The major source of light is the sun, emitting harmful ultraviolet (UV) and blue-violet light as well as beneficial blue-turquoise light. Added to this, the development of new sources of artificial light is altering our light exposure profile, increasing exposure to harmful light, with eyes increasingly subjected to potential risks of cumulative retinal damage.

Studying light-induced eye damage is invaluable for designing effective light filtering solutions as part of the preventive tools armamentarium. One of the challenges facing the ophthalmic optics industry is to find the balance between protecting our eyes from noxious light while simultaneously allowing essential light to reach the retina, for both visual and non-visual functions. A better understanding of the biology behind retinal damage is essential for developing refined solutions for adequately protecting our eyes.

In this White Paper we review the current state of research and development, focusing on the role of oxidative stress in retinal photaging. We present the new lens solutions put forward by Essilor through their collaborative research with the Paris Vision Institute.

KEYWORDS

harmful blue-violet light, sunlight, light emitting diodes, oxidative stress, ROS, retinal damage, retinal pigment epithelium, phototoxicity, UV, E-SPF®, prevention, Eye Protect System™, Smart Blue Filter™
LIGHT AND THE VISUAL CYCLE

The electromagnetic spectrum and light transmission to the eye

The electromagnetic spectrum covers a continuum of electromagnetic waves, from radio waves, microwaves, infrared, visible and UV radiations, through to X-rays and gamma-rays, the photon energy increasing with decreasing wavelength [Figure 1]. Sunlight is composed of 5-10% UV radiation (100-380nm), -40% visible radiation (380-780nm), and 50-55% infrared radiation. These are either absorbed or transmitted by the successive layers of the eye, modulating the light reaching the retina1.

UV waves are harmful to the anterior part of the human eye. In a healthy adult’s eye no UV radiations actually reach the retina. UVC (100-280nm) from sunlight are filtered by the atmosphere, while most UVB (280-315nm) are absorbed by the cornea. Residual UVB and most UVA (315-380nm) are then absorbed by the crystalline lens. In contrast, visible light reaches the retina in high proportions2.

In addition to allowing us to perceive the world around us in terms of shape, contrast and colour, visible light also plays an important role in various non-visual functions of the body, controlling many rhythmic biological functions. High energy visible light (380-500nm), commonly known as blue light, accounts for ~25 to 30% of the sunlight within the visible range. It includes both harmful blue-violet radiations (415-455nm) which can be damaging to the retina, but also beneficial blue-turquoise radiations (465-495nm), essential for normal physiological functioning during the day. Although transmission of blue light to the retina decreases with age, as a protection, it nonetheless remains present at significant levels.

Fundamentals of the retinal visual cycle

To reach the retina, light passes first through the cornea, the aqueous humour, the crystalline lens and then the vitreous humour. From here, it crosses the retinal ganglion cells and then several cell layers before reaching the outer retina. The outer retina is composed of retinal pigment epithelium (RPE) cells plus the outer segments of the visual photoreceptors (rods and cones) [Figure 2]. The discs of the photoreceptor outer segments (POS) contain visual pigments formed by covalent binding between 11-cis-retinal (a photosensitive derivative of vitamin A) and a transmembrane opsin signalling protein.

Absorbed photons transmit energy to the photoreceptors via the opsin, triggering isomerisation of the 11-cis-retinal which causes a conformational change to all-trans-retinal [Figure 3]. The all-trans-retinal is released from the activated opsin into the cytoplasm.
and is then rapidly reduced to its non-oxidised form all-trans-retinol\textsuperscript{3-5}, in a healthy retina. This crosses the sub-retinal space and enters the RPE where it is converted back to 11-cis-retinal which returns back to the photoreceptors, binding with opsin, and completing the visual cycle [Figure 3]. The RPE plays a critical role in vision; in addition to the constant renewal of 11-cis-retinal, it is also responsible for the phagocytosis of the POS discs and providing nutrients and oxygen to the photoreceptors. The visual cycle is the fundamental basis of our vision, and its dysfunction triggers irreversible retinal damage.

Figure 2. Visual pigments in photoreceptor outer segments

Figure 3. The visual cycle in rods
Eye damage and focus on retinal pathologies

Chronic eye exposure to solar UV waves is associated with the pathogenesis of numerous diseases of the anterior part of the eye, such as pterygium and pinguecula. It is also associated with crystalline lens pathologies, in particular the development of cataracts.

While the visual cycle can be progressively disrupted with ageing, this process is known to be accelerated by light. Retinal damage can originate from photomechanical, photothermal or photochemical reactions. Optical radiation (UV, visible and infrared) has the potential to cause photomechanical and photothermal damage from brief and extreme exposure, while photochemical damage is more commonly due to cumulative and prolonged exposure and is also wavelength-dependent being blue-violet light specific for the outer retina. The cumulative harmful effect of light on the retina depends on the irradiance it receives (i.e. the power received on a given surface per unit area). Retinal irradiance is in turn dependent not only on the light source radiance (i.e. the power of the light source per unit area per unit angle), but also on pupil size (decreases with age and brighter light) and anterior ocular media transmittance.

Among known retinal pathologies, the most preoccupying is age-related macular degeneration (AMD). Along with age, genetics, smoking and diet, blue-violet light is known to contribute to accelerated ageing of the outer retina and is thus a risk factor for AMD. AMD involves the degeneration of RPE cells and then the photoreceptors, and is associated with chronic inflammation and oxidative stress. In developed countries, it is the leading cause of irreversible visual impairment, with 17.8 million cases in the US and estimated as 265 million worldwide over the next 30 years. Prevention of retinal damage caused by blue-violet light via photoprotection is an important aspect of optimising retinal health management.

The changing profile of light exposure

Light exposure profiles vary considerably among individuals, integrating a multitude of factors; the type and number of light sources, their localisation, spatial distribution, as well as radiance, spectra exposure duration and repetitions.

Exposure to UV and blue light from the sunlight varies depending on the time of day, geographic location, season, etc., but is also affected by social influences (skin cancer awareness, sunglasses’ quality, and social norms relating to skin tanning).

Artificial light sources also contribute to retinal light exposure, altering the light exposure profile with more light sources, longer and repetitive exposure, higher radiance and energy, and at shorter distances. Exposure is occurring in people of all ages, and at increasingly younger ages. Solid-state lighting (SSL) now dominates domestic lighting, with incandescent bulbs being phased out this year, and the European Lighting Industry estimating that over 70% of light sources will be based on SSL by 2020. Current cold-white light emitting diodes (LEDs) include up to 35% of blue light within the visible range, compared to incandescent lamps which have less than 5%. Warm-white light has less than 10% of blue light but also has lower luminous efficacy. Thanks to their compact form and wide spectral range, LEDs are now extensively used in everyday self-illuminating applications including mobile phones, tablets, computers, TVs and even in toys and clothes. Radiance from LEDs can be up to 1000 times higher than that of traditional incandescent lamps. Combined with the fact that the chronic toxic effect of a light source depends strongly on exposure duration and repetition, this could make LEDs a potential contributor to long-term retinal damage.
Impact of UV on the anterior part of the eye

In the healthy adult’s eye, UV radiation is almost completely filtered out by the cornea and the crystalline lens and do not reach the retina. In vitro, in vivo, and epidemiological data demonstrate that chronic eye exposure to UV radiation is associated with the pathogenesis of numerous corneal and crystalline lens pathologies. The role of UV in corneal damage was shown as early as the mid-1950’s when Kerkenezov reported its involvement in the development of pterygium. Since then, numerous in vivo and in vitro studies using corneas and crystalline lenses from several species (including humans) have demonstrated the higher the wavelength, the higher the UV light damage threshold and thus the lower the toxic effect. Weighting the UV hazard spectrum by the sunlight spectral distribution, the greatest danger of UV is in between UVA and UVB with a maximum at around 315 nm.

The mechanisms behind blue-violet light retinal damage

Photochemical damage is mainly associated with long-term and repetitive exposure to moderate irradiances, arising when a photosensitive molecule or chromophore undergoes physico-chemical changes after photon absorption. Damage is dependent on the balance between light exposure and the body’s retinal repair systems which manage oxidative stress. These systems are affected by age, genetic and/or environmental factors that can decrease their efficiency.

In the presence of oxygen, high-energy photons can react with photosensitive compounds to produce photochemical reactions and then reactive oxygen species (ROS) including singlet oxygen (O₂*), superoxide anion (O₂⁻), hydrogen peroxide (H₂O₂) and hydroxyl radicals (HO·). These ROS are highly toxic and can cause protein oxidation, lipid peroxidation, mutagenesis, etc. They are naturally derived from numerous intracellular sources including the mitochondria, enzymatic systems or photosensitizers and can occur as a result of exogenous influences such as light, smoking or diet poor in antioxidants.

As one of the highest oxygen-consuming structures in the body, the retina is extremely susceptible to oxidative stress. Combined with an abundance of photosensitizers in the outer retina, prolonged visible light exposure and a high energy demand, this gives fertile ground for oxidative stress. The two major photosensitizers in the retina are 11-cis-retinal in the outer segments of the photoreceptors and lipofuscin, a “wear and tear” pigment which accumulates with age in RPE cells. Other photosensitive molecules which may also play a role include cytochrome C, flavins and flavoproteins.

Three major natural antioxidant systems supporting retinal health are superoxide dismutase (SOD), catalase and glutathione. SOD alternately catalyses the partitioning of the (O₂⁻) radical into “safe” (O₂) or (H₂O₂). (H₂O₂), which is also dangerous, is in turn converted into water (H₂O) and (O₂) by the catalase enzyme or by the glutathione peroxidase enzyme which also converts reduced glutathione (GSH) into oxidized glutathione (GSSG).

When exposed to blue-violet light, all-trans-retinal (which accumulates in the POS), is highly photoreactive and induces oxidative stress, with decreasing sensitivity between 400 and 450 nm. In the absence of sufficient antioxidant activity, the POS progressively oxidises and their renewal within RPE becomes more challenging, generating accumulation of residual lipofuscin in the RPE. Lipofuscin contains a photosensitizer with a maximum absorption in the blue-violet spectral range at 440 nm. Accumulation of lipofuscin in the RPE is a key feature of ageing and AMD. The RPE cells become progressively clogged with age-related waste products, ultimately resulting in apoptosis. Deprived of their support cells, the photoreceptors deteriorate, leading to permanent retinal damage.

Literature review on retinal blue-violet light damage

Retinal damage by blue light has been studied for a half century, starting with the landmark paper publi-
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Figure 4. Simplified normal processing of ROS. ROS in red, antioxidant defenses in green (adapted from Jarrett et al., 2012)

shed by Noell et al. describing blue retinal phototoxicity in rodents exposed to white fluorescent lamps. In vitro studies on immortalised RPE cells loaded with purified lipofuscin showed lower toxicity thresholds with violet-blue-green light (390-550 nm) versus yellow-red light (550-800 nm). Similarly, human RPE cells loaded with A2E (a well-characterised chromophore in lipofuscin) were approximately 7-fold more sensitive to blue light than to green light. Phototoxicity was not observed without any photosensitizer, and increased with increasing photosensitizer concentrations. This was confirmed in several animal models. The role of broadband blue light in oxidative stress was shown in cultured human RPE cells causing lipofuscin-dependent protein oxidation, lipid peroxidation, mitochondrial DNA damage, lysosomal changes and cell death.

Supportive data are found in numerous epidemiological studies suggesting a correlation between blue light exposure from the sun and AMD; in a recent meta-analysis of 14 epidemiology studies, 12 reported an increased risk of AMD with greater sunlight exposure, six of which were significant. Studies of human macular pigment density and the risk of AMD progression following cataract surgery lend further weight to the hypothesis that blue light exposure has a role in AMD pathogenesis, with a three-fold increased risk of AMD progression directly attributed to a dramatic increase in blue light exposure after surgery.
In vitro modelling of blue light toxicity on the outer retina (cell death)

While these studies leave little doubt that the outer retina sustains photochemical injury from blue light mediated by the visual pigment for the photoreceptor outer segments and by lipofuscin in the RPE cells, many of the published in vitro studies in this field suffer limitations. These include a lack of precision in terms of the light dose and/or use of very high irradiances that can trigger acute light-toxicity mechanisms rather than reflecting lifelong cumulative exposure damage which is more accurately represented by moderate irradiances and longer exposure, particularly in the context of AMD.

In 2011, a fruitful collaboration was developed between researchers at the Paris Vision Institute and at Essilor to address these issues. A well-established in vitro AMD model and innovative cell illumination protocol and device were used to evaluate the precise phototoxicity action spectrum (cell apoptosis) occurring under conditions mimicking physiological retinal exposure to sunlight.

Primary swine RPE cells were cultured in the absence of any photosensitizer, then photosensitised with A2E and finally exposed to 10 nm-wide illumination bands across the blue-green range (from 390 to 520 nm in 10 nm increments) plus an additional band centred at 630 nm for 18 hours, using an innovative LED-based fibered device. After light exposure, cells were maintained in darkness for 6 hours then analysed. Moderate irradiances (< 1.6 mW/cm² for 630 nm and < 1.3 mW/cm² below 460 nm) normalised to the daylight spectrum reaching the retina after being filtered by the ocular media were used. Cell necrosis (reflecting acute light toxicity) and apoptosis (reflecting long-term cumulative light toxicity) were measured.

What they found was that firstly, none of the light exposures evaluated altered the necrosis rate compared to cells maintained in darkness, supporting that moderate light irradiance is not associated with acute toxicity. Secondly, decreased cell viability was detected with very low A2E concentrations at 420, 430 and 440 nm, corresponding to blue-violet light. Finally, apoptosis was significantly induced between 415-455 nm [Figure 5], and increased with increasing A2E concentrations. These findings delivered a very precise definition of action spectrum.
Investigating oxidative stress

Working from the premise that blue-violet light induces apoptotic death in RPE cells, Essilor and the Paris Vision Institute took a step towards fine tuning the understanding of the link between blue light and AMD via accumulation of oxidative stress and modulation of cell defence mechanisms. To identify the role of blue-violet light in accelerating ageing of the outer retina, and more specifically to understand the early stages in this process, they used their cell and light models to address two key questions; does blue-violet light induce ROS and does it inhibit antioxidant defence mechanisms? ….. or both?

As previously described, A2E-loaded RPE cells were exposed to 10 nm-wide light bands within the blue range (390-520 nm) and also at 630 nm with a normalised retinal sunlight spectrum. A shorter exposure time was used (15 hours) given that oxidative stress is related to earlier and more sensitive biomarkers. Well-characterised biomarkers implicated in oxidative stress were analysed. Two main ROS, (H₂O₂) and (O₂⁻), were used as a measure of change in the balance between ROS generation from light exposure according to their elimination by antioxidant mechanisms [Figure 3]. Blue-violet light exposure is associated with high levels of (H₂O₂) and (O₂⁻), significantly higher than control levels (in darkness) with the greatest effect observed between 415 nm and 455 nm [Figure 6], corresponding exactly to the toxic spectral band associated with cell apoptosis [Figure 5].

To answer the complementary question as to the effect on oxidative stress repair processes, researchers used a three-pronged approach, evaluating the effects of blue-violet light on glutathione conversion, as well as SOD and catalase activity and also mRNA expression levels of SOD 2 (mitochondrial SOD), catalase and glutathione peroxidase (GPX1) (enzyme that catalyzes the conversion of GSH to GSSG). GSSG concentrations, the oxidised form of GSH and as such an indicator of oxidative stress, increased following blue light exposure at 400, 440 and 480 nm, suggesting that glutathione antioxidant mechanism tries in the first place to compensate the high quantity of (H₂O₂) in these light conditions, especially for blue-violet light [Figure 7]. In addition, mRNA expression levels of the enzyme glutathione peroxidase (GPX1) decreased significantly, showing that even though glutathione is more active just after blue light exposure, the defence mechanism will be progressively disrupted (a drop in mRNA expression induces a drop in protein synthesis and thus a decrease of...
glutathione peroxidase production, preventing from GSH and (H₂O₂) to be converted to GSSG, (H₂O) and (O₂)).

SOD activity was also significantly increased by blue-violet light at 440 nm suggesting SOD is first strongly activated in an attempt to compensate the increased (O₂⁻) production. SOD2 mRNA levels were significantly decreased, highly suggesting the defense enzyme tends to be disrupted and decreased, and thus less functional [Figure 8]. There was also an almost complete reduction in catalase activity with blue-violet light at 430 and 440 nm (with or without A2E), suggesting a reduced antioxidant capacity.

The significant increase of GSSG quantity and in SOD activity just after exposure to 400-480 nm wavelengths reflect the cell’s attempts to rapidly defend itself against the increased ROS production, however this increased activity failed to compensate the increased (H₂O₂) and (O₂⁻) accumulation only in the blue-violet range (more in between 415-455nm).

The significant reduction of mRNA expression levels of SOD 2, GPX1 (glutathione peroxidase) and cata-

Figure 7. GSSG concentrations (A) and GPX1 mRNA expression levels (B) in 20 µM A2E-loaded RPE cells after 15h light exposure.

The lowest the p-value, the highest the significance. (0.01<=p<=0.05) = * (0.001<=p<0.01) = ** (0.0001<=p<0.001) = *** (p<0.0001) = ****

p-value as compared to control cells maintained in darkness.

Figure 8. SOD activity (A) and SOD 2 mRNA expression levels (B) in 20 µM A2E-loaded RPE cells after 15h light exposure.

The lowest the p-value, the highest the significance. (0.01<=p<=0.05) = * (0.001<=p<0.01) = ** (0.0001<=p<0.001) = *** (p<0.0001) = ****

p-value as compared to control cells maintained in darkness.

Do not disclose, do not copy without written authorization. Paris Vision Institute & Essilor R&D results.

Submission of scientific paper in progress. Results presented in ARVO 2015, 2016 (posters)
lase in the blue range supports the hypothesis of a progressive strong decrease in protein synthesis and thus, at the end, a strong decrease of the 3 antioxidant mechanisms.

Finally, further proof of oxidative stress with blue-violet light exposure (at 420, 430, and 440 nm) was obtained in terms of its effects on the mitochondria, with a modified cellular distribution restricted within the perinuclear area [Figure 9], along with an altered morphology (from tubular to globular). A statistical drop in the mitochondrial respiration rate impacting ATP production was also seen at 440 nm (compared to 400 and 480 nm), reflecting a major metabolic defect under blue-violet light exposure.

Researchers thus confirmed that blue-violet light results in increased ROS production in RPE cells. Also, blue-violet light decreases cell’s self-defence systems, making them inadequate to compensate ROS increase.

A need for blue-violet light protection

With clear experimental data confirming that blue-violet light acts as a strong inducer of oxidative stress on the outer retina, there is a growing need for blue-violet light protection against retinal pathologies, and in particular AMD. Photobiology safety standards for lighting products need to keep up with our rapidly evolving light exposure profile, maintaining a strong focus on conducting health risk assessments and extensive experimental studies on LED-based systems. Blue light protection norms are trailing behind those of UV light, with no current standards for cumulative toxicity limits.

Various international initiatives have highlighted concerns over potential health issues of solid-state lighting, including the ANSES 2008 task group (the French Agency for Food, Environmental and Occupational Health & Safety) and the 2014 SSL Annex (4E IEA), calling for photobiological safety assessments for all SSL devices (LED-based) using the joint CIE S009 / IEC 62471 standard. Risky light exposure profiles need to be identified and related to high-risk populations (pre-existing eye conditions, children and the elderly, etc). A new ANSES task group was formed in 2015 to address these issues head-on.

It is important to keep in mind that blue light encompasses wavelengths which perform essential functions. These key metabolic non-visual functions driven by blue-turquoise light including circadian resetting, melatonin suppression, pupil light-induced reflex, cognitive performance, mood, locomotor activity, memory, body temperature, etc; are thought to be mediated by photosensitive retinal ganglion cells containing the melanopsin photopigment with an absorption peak at 480 nm. When designing ophthalmic lenses, it is thus essential to filter only the harmful blue-violet light while ensuring beneficial blue-turquoise light reaches the retina during the day.

![Figure 9. Altered mitochondrial cellular distribution after 15h light exposure at 440 nm in 10 µM A2E-loaded RPE cells (left) compared to 630nm (right)](image)

For over half a century, a large body of in vitro and in vivo experimental evidence has progressively revealed a strong scientific rationale for blue-light induced toxicity in the outer retina. Many of these studies suffer limitations such as not evaluating the toxic risk of each blue wavelength or illuminating with very high irradiances that trigger acute light-toxicity mechanisms rather than cumulative exposure damage which should be sought when studying the pathogenic mechanisms of AMD. To go a step further from a photometry standpoint, we joined skills with the Paris Vision Institute in 2008. In research performed prior to 2013, we scanned the phototoxic risk of each 10 nm band of the blue-green spectral range, simulating physiological retinal exposure to sunlight. Since 2013, we focused our research on the comprehensive understanding of the role of blue light on each step of the RPE cell degenerative process, from the earliest stages through to cell death. We explored the photomodulation of oxidative stress and cell defense mechanisms in the outer retina with two questions in mind. First, does blue-violet light act as an inducer of reactive oxygen species? Second, does blue-violet light act as an inhibitor of antioxidant defense mechanisms?

Very interestingly, in 2013, we found that it is a narrow spectral range, blue-violet light from 415 to 455 nm, that induces the highest apoptosis of RPE cells (Arnault et al., PlosOne, 2013).

In 2015, we further confirmed this specific toxic action spectrum of light with oxidative stress biomarkers. First, we highlighted a strong accumulation of reactive oxygen species in response to blue-violet light. Second, we demonstrated that blue-violet light also acts as a strong inhibitor of antioxidant defense mechanisms. This means that blue-violet light is not only a strong stress inducer but also a defense inhibitor. This double negative effect strongly supports the hypothesis of blue-violet light as an important contributor of oxidative stress in the earliest stages of cell damage, and thus of accelerated retinal ageing, potentially leading to cell death and ultimately to faster AMD onset or progression.

As age-related oxidative changes in the outer retina are a hallmark of early AMD, the identified deleterious effect of blue-violet light at each step of the damaging cycle of RPE cells strengthens the role of blue-violet light as an initiating cause of AMD. Together, our latest photobiology data provide strong scientific evidence on the role of blue-violet light induced retinal damage, providing comprehensive evidence that the most harmful light band to RPE cells is indeed between 415 and 455 nm.
PHOTOPROTECTION: FROM CELL RESEARCH TO LENSES

A timeline of smart filtering protective lens

The last decade has seen major innovations in clear everyday lenses with the incorporation of evolving photoprotective technology. Essilor’s photoprotective research program started about 10 years ago, delivering in 2011 the first antireflective coating with low UV reflection on back-side with Crizal® UV. Thanks to the collaborative research program between the Paris Vision Institute and Essilor, the Crizal® Prevencia® coating was released in 2013, the first clear lens to integrate an antireflective coating that filtrates both UV and partially harmful blue-violet light while maintaining a maximum of the essential blue-turquoise light.

2016 brings Essilor’s latest technological advance, the Smart Blue Filter™, a novel approach using embedded blue-violet light protection, compatible with any antireflective coating. The Eye Protect System™ lens brings together the Smart Blue Filter™ and UV protection while ensuring minimal aesthetic compromise. Bringing protection to a higher level, the Smart Blue Filter™ lens has been combined with Crizal® Prevencia® coating.

Refining the test system: a new adjustable fibered white light device

Today Essilor has the edge in the ophthalmic optics industry, by testing in vitro the photoprotective potency of lens filters. The photoprotective effect of the Smart Blue Filter™ lens feature was compared between each narrow illumination band within the blue-violet range from 400-450 nm to better differentiate the different spectral profiles.

To validate the photoprotective effect in real light conditions, a polychromatic light source (as opposed to the monochromatic light source used in the in vitro model) was envisaged.

Over an 18-month period, researchers at Essilor developed an innovative adjustable fibered white light illumination device which can generate programmable and variable spectra and irradiances within the visible range. This new device offers greater flexibility than the previous blue-green light device, delivering any spectrum within the visible range, thus in addition to daylight spectra it can also mimic warm-white or cold-white LED, fluorescent, and incandescent spectra, and even quasi-monochromatic light. The photoprotective potency of the Smart Blue Filter™ lens feature was measured in terms of the reduction in apoptotic cell death with the filter versus without.

UV protection: the E-SPF® index

UV is a constant source of potential eye damage irrespective of the weather conditions. Exposure occurs directly from the sun’s rays, however more than 50% of UV radiation reaching the eye is indirect, coming from cloud scatter and reflection. Public awareness of UV eye hazards has been increasing since the widespread UV SPF campaigns to protect against skin cancer. Most clear lenses provide a high level of UV protection by absorption. Crizal Forte® UV coating offers additional UV protection on the back surface of the lens to limit UV reflection that can reach the eye.

The E-SPF® (eye-SPF) index takes into account both UV transmission through the lens and UV reflection off the back surface of the coated lens, although it does not account for light coming around the lens or for variations with facial morphology, gaze direction and glasses shape. Essilor is currently offering an E-SPF® up to 35 on clear lenses.

Global protection with the Eye Protect System™ lens

The Smart Blue Filter™ innovation was designed to distinguish harmful blue-violet light from essential blue-turquoise light, absorbing the former and transmitting the latter, using specific absorbers for blue-violet light, embedded inside the lens such that blue-vio-
let light reaching both the front and the back-surface of the lens is filtered [Figure 10].

This new embedded innovation offers the key advantage of being compatible with all antireflective coatings. Building on the existing Crizal® UV coating, Essilor combined the embedded Smart Blue Filter™ feature with E-SPF®, offering a clear lens with both UV and blue-violet protection called Eye Protect System™.

As such, the lens filters on average 20% of the blue-violet light between 400-455 nm, combined with UV protection [Figure 14]. Its blue light photoprotective effect on retinal cells (RPE) in vitro is equivalent to that offered by Crizal® Prevencia® coating giving a 25% (±5%) decrease in retinal cell death.

A new lens without aesthetic compromise

The absorber filtering partially blue-violet light attributes a natural yellow-orange colour to the lens which is not acceptable for a clear day to day lens. To counteract this, two neutralising molecules were added to the Smart Blue Filter™. To assess the accuracy of transparency, a sensory analysis** with trained judges was performed evaluating four parameters; lens colour

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Figure 10. Blue-violet light reaching the lens from the front or back surface is absorbed by the embedded filter, while not affecting other wavelengths.

Figure 11. Effect of colour neutralisation on transmittance on clear lens. Both lenses with Crizal® UV coating and transmittance were measured on prototypes 1.5-index CR39 Plano (2 mm centre thickness).
EYE PROTECT SYSTEM™ LENSES: FROM RESEARCH TO HARMFUL LIGHT FILTERING

EYE PROTECT SYSTEM™ LENSES:
FROM RESEARCH TO HARMFUL LIGHT FILTERING

(through the lens for the wearer), skin colour (through the lens for the observer), lens transparency (through the lens for the wearer and observer), and perception of a colour picture (through the lens for the wearer). All parameters consistently rated better for the neutralised lens than for the yellow-orange one.

Transmittance was almost identical between a yellow-orange lens and a neutralised lens [Figure 11]. In addition to the sensory analysis, a consumer test* was performed to assess the acceptance of this new lens without explanations on the additional benefit brought. After one month of wear, 96% were satisfied by its aesthetic. This test shows that the new embedded technology is not noticeable by wearers.

*EUROSYN Acceptance Wearers Test (N=57 lens wearers wearing previously lenses with Crizal® coating that have been equipped with Eye Protect System™ lenses with Crizal Forte® UV coating (same index and Rx) / results after 1 month) - FRANCE - 2016.

** Standard deviation based on a calculation model for all substrate

Essilor Ultimate Protection: the Eye Protect System™ lens with Crizal® Prevencia® coating

Combining the embedded Smart Blue Filter™ feature with the Crizal® Prevencia® antireflective coating gives a maximized protection; blue-violet and UV reflection off the front-surface with Crizal® Prevencia® coating, partial blue-violet and UV filtration by the Eye Protect System™ lens itself, and minimisation of UV reflection off the back-surface of the lens with Crizal® Prevencia® coating [Figure 12]. Combining these two solutions of blue-violet filtering offers a maximized protection for a clear lens, filtering on average 30% of blue-violet light (1.59 index lens) between 415 nm and 455 nm and reducing retinal cell apoptosis by 35% (±5%**), approximately 10% more than with Crizal® Prevencia® coating alone.

Figure 12 - The Eye Protect System™ lens combined with Crizal® Prevencia® coating ensures filtering of harmful UV and blue-violet light protection while ensuring transmittance of valuable blue light.
The Eye Protect System™ lens with Crizal® Preven-
cia® coating offers a transmittance profile filtering
the greatest proportion of the harmful blue-violet
light while allowing the beneficial blue-turquoise light
through [Figure 13] with comparable lens aesthetics
to Crizal® Prevencia® coating alone.

Finally, the Eye Protect System™ lens with Crizal® Pre-
vencia® coating offers a significant reduction in dis-
comfort glare compared to a standard Crizal Forte®
UV lens. A study* with nine young healthy subjects
suffering moderate or high photosensitivity showed
the highest photosensitivity threshold was with the
Eye Protect System™ lens with Crizal® Prevencia®
coating, giving a 1.5-fold improvement in discomfort
and glare compared to Crizal Forte® UV coating.

* Essilor R&D study (N=9 Discomfort glare & Blue-filtering lenses Focus on Smart Blue
Filter™ with Crizal® Prevencia® coating) - FRANCE - 2015
Essilor’s current range of Eye Protect System™ lenses*

At Essilor, we recommend three levels of protection; ESSENTIAL composed of the Eye Protect System™ lens (HC version or antireflective with E-SPF® 10), ADVANCED UV protection adding a UV optimized back-side AR coating such as Crizal® Forte® UV with E-SPF®25 or E-SPF®35 and ULTIMATE UV protection and partial blue-light filtration with the antireflective coating Crizal® Prevencia® [Figure 14].

* The commercial offer can differ depending on country.
EYE PROTECT SYSTEM™ LENSES:
FROM RESEARCH TO HARMFUL LIGHT FILTERING

KEY FACTS

PHOTORECEPTION

• Blue light encompasses both harmful blue-violet radiations (415-455 nm) which can damage the retina and beneficial blue-turquoise waves (465-495 nm) essential for normal physiological functioning during the day (rhythmic biological functions).
• The visual cycle, highly involving retinal pigment epithelium (RPE) is fundamental to vision and its progressive dysfunction may be associated with retinal pathologies.
• Blue-turquoise light needs to be transmitted by the lens, especially during the day.

PHOTOTOXICITY & NEW PHOTOBIOLOGY RESEARCH

• UV is a risk factor for diseases of the anterior part of the eye (cataracts...).
• Lipofuscin, the age pigment, accumulates with age in the outer retina, and reacts with energetic blue-violet light, which contributes to accelerated photo-ageing of the outer retina.
• The toxic action spectrum of light on the outer retina (RPE cells) is identified as blue-violet light 415–455 nm (Arnault, Barrau et al., PlosOne, 2013).
• New data confirmed:
  - The toxic action spectrum with oxidative stress biomarkers.
  - Blue-violet light induces high ROS accumulation (H₂O₂, O₂−): it is a STRESS INDUCER
  - Blue-violet light acts as a strong inhibitor of antioxidant mechanisms (glutathione, SOD, catalase): it is a DEFENSE INHIBITOR
  - Blue-violet light directly impacts mitochondria: peri-nuclear clustering, globular shape, decreased respiration rate.
• Low-irradiance blue-violet light induces apoptotic cell death.
• Cumulative (i.e long-term with moderate irradiance) damages induced by light are wavelength-dependent and relevant for eye ageing.
• Blue-violet light is an accelerator of retinal ageing: it is a risk factor for AMD.

PHOTOPROTECTION

• Essilor is the first ophthalmic actor to conduct in vitro tests to assess the photo-protective potency of lenses.
• The Eye Protect System™ lens protects against both harmful UV and blue-violet light.
• 3 levels of protection are available with increasing E-SPF® and blue-violet light protection from Essential, Advanced to the Ultimate level which also offers an extra filtering of blue-violet light.
References

HOW
TRANSITIONS® LENSES
FILTER HARMFUL
BLUE LIGHT

Article published in Points de Vue, International Review of Ophthalmic Optics, online publication, March 2016
Light-induced ocular damage has been investigated for decades in laboratory extensive work and several epidemiological studies. More recently, harmful effects of blue-violet light have been spotlighted by growing body of scientific research. Despite the eye’s natural defense mechanisms, it has been evidenced that cumulative exposure to blue-violet light can contribute to long-term irreversible changes in the retina. When the most critical exposure occurs in outdoor conditions, Transitions® lenses can effectively filter harmful blue-violet light and consequently provide optimal photo-protection for the patient eyes.

**Light**

**The role of light in the visual experience**

Light is essential to the development of visual function. Light is an element of life, a major environmental factor in human development. It plays a significant role in how we process sensory information, impacting our visual experience from the point of birth and throughout our lives.

Visual perception occurs when light strikes the retina of the eye. The pupil of the iris serves as the optical diaphragm of the eye affecting the path of light rays which are refracted by the cornea and the crystalline lens on their way to the retina. Numerous deprivation experiments have demonstrated that ocular growth and refraction development are regulated by visual information. Light is essential in providing this information on diurnal species by transmitting signals which are converted by the brain into visual perception. This acquisition of visual function is experienced as early as infancy and is essential to healthy development.

**KEYWORDS**

- Blue Light, photochromic lenses, light filtering, sunlight, light exposure, Retina, AMD, photo protection, Transitions® Signature™, Transitions® XTRActive®
Light plays a fundamental role in visual performance
The iris acts as a natural optical diaphragm for expanding (dilation) or retracting (constriction) its central aperture. Depending essentially on lighting conditions and age, the diameter of the pupil ranges from 2mm to 8mm. Variations in the diameter of the pupil are caused by a movement reflex that regulates the light flux incident and, subsequently, visual performance. The visual system as a whole is sensitive over a wide range of light levels from starlight to bright sunlight but, despite the regulation of the pupil aperture, it cannot operate over the entire range simultaneously. An adaptation is required to adjust the light sensitivity of the visual system to different light levels. When the adaptation is in progress, visual performance is reduced. Once the process is complete, visual capabilities depend on the new level of light.

There are two primary lighting conditions with which the visual system has to deal: daylight (photopic) and nighttime (scotopic). Between photopic and scotopic levels is a range called mesopic, which corresponds roughly to twilight. The human eye has three types of light sensitive cells (photoreceptors) in the retina – cones, rods and ganglion cells – that process sensory information (Table 1).

Cones are highly concentrated in the central area of the retina (macula) and are responsible for providing daylight sharp image resolution and color detection. Rods are largely distributed in the periphery of the retina. Having high sensitivity, they are required for scotopic vision but provide low resolution and lack of color information. The ganglion cells or ipRGCs (intrinsic photosensitive Retinal Ganglion Cells) express the melanopsin-based photopigment. These melanopsin ganglion cells are crucial for relaying light information from the retina to the brain to control circadian rhythms, pupillary light reflex, sleep and many other body functions. (Sand A. et al., 2012, Gronfier 2013).[11, 09]

The sun is the most powerful source of light
The solar spectrum
The sun emits a tremendous amount of energy in the form of wide electromagnetic radiation. From cosmic rays to radio waves (Fig. 1), the majority of solar emissions are not visible to human photoreceptors. Only a thin portion – at wavelengths (λ) between 380nm and 780nm – provides the visible light that interacts with the eye’s photoreceptors – enabling us to see the world. When visible solar radiation reaches the Earth’s surface it is scattered throughout the atmosphere, especially in the blue-violet region corresponding to the shortest wavelengths (380-460nm) of visible light and subsequently to the highest energy.

<table>
<thead>
<tr>
<th>OPERATING STATE</th>
<th>LUMINANCE RANGE</th>
<th>PHOTORECEPTOR</th>
<th>PEAK SENSITIVITY</th>
<th>CHARACTERISTICS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Photopic</td>
<td>&gt;3cd/m²</td>
<td>Cones</td>
<td>555nm</td>
<td>Fine resolution</td>
</tr>
<tr>
<td>Scotopic</td>
<td>&lt;0.001cd/m²</td>
<td>Rods</td>
<td>507nm</td>
<td>No vision color</td>
</tr>
<tr>
<td>Mesopic</td>
<td>&gt;0.001cd/m²&lt;3cd/m²</td>
<td>Cones and Rods</td>
<td>Between 555nm and 507nm</td>
<td>Reduced color</td>
</tr>
</tbody>
</table>

TAB.1 Summary of main lighting conditions (Boyce, 2001).[6]
The risks associated to UV exposure

Beyond the visible spectrum, sunlight emits ultraviolet radiation with wavelengths shorter than 380nm – commonly referred to as UV – and infrared radiations with wavelengths greater than 780nm. Ultraviolet radiation arriving on earth surface is divided into UVB (280-315nm) and UVA (315-380nm). At sea level, about 10 percent of radiation is UV, 50 percent is visible and 40 percent is infrared.

Exposure to the sun for an extended period of time produces erythema and affects skin pigmentation, causing burning or tanning. Both UVA and UVB penetrate the atmosphere freely and play a critical role in advancing more severe health conditions like premature skin aging (ex: wrinkles) and certain skin cancers (ex: carcinoma) which can affect the eyelids and facial skin. In a healthy adult, more than 99 percent of UV radiation is absorbed by the anterior part of the eye (eyelid, ocular surface, crystalline lens). Exposure to ultraviolet radiation is well established as a major cause of eyelid malignancies, photokeratitis, climatic droplet keratopathy, pterygium and cortical cataract (Yam 2014, Behar-Cohen et al. 2014). There is insufficient evidence to support the proposal that Age-related Macular Degeneration (AMD) is related to UV exposure, and it is now suggested that AMD risk is probably more closely related to exposure to visible radiation, especially blue light (Yam 2014).

Blue light

The blue sky is evidence that blue light is present in direct sunlight. Since blue light is higher in energy than other wavelengths in the visible spectrum (Fig. 2), it scatters more throughout the atmosphere (Rayleigh scattering) and makes the sky appear blue. Blue light makes up 25-30 percent of daylight.

While blue light is emitted naturally by the sun, it can also be produced by numerous artificial light sources commonly found indoors. Light-emitting diodes (LEDs) are gaining an increased share of the domestic lighting market because of their high efficiency of luminance and low energy consumption. Widely found in digital screen technologies and displays, LEDs exhibit a high emission blue peak, centered at 430nm (Fig. 3).
Harmful Blue Light

The phototoxicity of blue light

As a part of visible light, blue light passes through the eye structure, reaching the retina. Due to its higher level of energy than the other wavelengths in the visible spectrum, it is potentially harmful to the retina. Depending on exposure conditions (light intensity, duration, periodicity) it may induce different types of reactions, including phototoxic damage (Rozanowska et al., 2009)[16]. Laboratory experiments showed that blue light is harmful (Sparrow et al., 2000)[14] and particularly it has been demonstrated that exposure to blue violet light with a maximum peak centered on 435+/- 20 nm can induce irreversible cell death in the retinal pigment epithelium (RPE), located in the external layer of the retina (Arnault et al., 2013). [1]

These damages contribute to the aging process of the eye and may lead to the development of pathologies such as AMD, the major cause of blindness in the elderly in developed countries. In epidemiological studies addressing long term chronic exposure to blue light, the Beaver Dam Eye study demonstrated that there is a strong correlation between outdoor activities (sunlight exposure) and early incidence of AMD changes (Cruickshanks et al., 2001, Tomany et al., 2004). [7, 15]

The different levels of blue light exposure

Amount of blue violet light is characterized by the intensity of emitted light of varied sources (Table 2). Sunlight is by far the strongest source of blue light at least 100 times greater than artificial sources (Fig. 4).

There is a significant difference in the level of blue light when facing into the sun (direct) and facing away from the sun (indirect). In actuality, no one looks directly at the sun since there is a natural aversion to sources of high glare. Humans often make adjustments by moving their head or their eyes or by relying on automatic reflexes like blinking, squinting and pupillary constriction. The eye can be subject to more serious effects due to multiple reflections of sunlight onto white surfaces. For example, the reflection of the sun at noon on sand or snow can reach 10 times more luminance than the blue sky (Behar-Cohen et al., 2011). [4]

The impact of blue-violet light exposure depends on the amount of total light reaching the retina: the retinal irradiance, which is characterized by the radiant flux (power) received by the retina per unit area. These values vary by the ocular media transmittance and – more importantly – by physical factors such as the eyelid position, which dictates the field of vision and the pupillary aperture, making ocular dosimetry far more complex than generally appreciated (Slone 2001, 2005). [12, 13] More investigations need to be done, but it seems reasonable to think that the level of retinal irradiance in the 435+/- 20 nm range is more important outdoors than indoors. Wearing appropriate glasses can be worthwhile to prevent from cumulative effects of light exposure.

Irradiance spectra of common artificial light sources (top) and direct and indirect sunlight (bottom)

The eye’s natural protections against blue light

Physiological structures around the eye, like eyelids and eyelashes, provide some protection against intense light. The iris pupil also contributes by using constriction to decrease the amount of entering light. While UV transmittance is blocked primarily by the cornea and crystalline lens in healthy adults, blue light crosses over these struc-

<table>
<thead>
<tr>
<th>SUN INDIRECT</th>
<th>PLASMA TV</th>
<th>SMARTPHONE</th>
<th>LCD MONITOR</th>
<th>CRT MONITOR</th>
<th>FLUORESCENT LIGHT OVERHEAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.71</td>
<td>0.035</td>
<td>0.007</td>
<td>0.013</td>
<td>0.025</td>
<td>0.089</td>
</tr>
</tbody>
</table>

viewing distance: (1)=6ft (2)= 1ft (3)= 2ft (4)= 2ft (5)= 6ft facing

TAB.2 | 420-440 nm integrated Irradiance values (w/m²) of common artificial light sources against solar diffused light (Transitions Optical internal measurements)

FIG. 4 | Irradiance spectra of common artificial light sources (top) and direct and indirect sunlight (bottom). (Transitions Optical internal measurements)
tures to reach the fundus of the eye (Fig. 5). The amount of blue light reaching the retina depends on the age of the eye as, during a lifetime, there is a yellowing of the crystalline lens that would typically provide some absorption in the blue violet region. The central part of the retina is covered by yellow pigments (Macula Lutea), which serve as a filter for incoming blue light because its absorbance peak in this range (Haddad et al, 2006). Due to assorted factors, macular pigment density can be variable from one individual to another and its ability to absorb light evolves during a lifetime. The children are the most exposed to harmful blue light because they have larger pupil diameter, less concentration of macular pigment and the amount of blue light reaching the retina is 65 % while it is 40 % for adults (Behar-Cohen et al., 2015).

Technical optical solutions for Blue Light long-term prevention

With the potential risks associated with outdoor conditions described and the natural protections of the human eye discussed, we now turn our attention to the technical solutions available within the eyewear industry to prevent from the long-term effects of blue-violet light. UV protection in eyewear will not be reviewed here since most high-quality lenses today offer complete protection against UV up to 380nm.

1. Coatings
Anti-reflective interferential layers may be applied to ophthalmic lenses by evaporating transparent dielectric metal oxides to the anti-scratch coating on both the convex and concave sides of the lens. The coatings essentially involve stacks created by successive deposits. Processed under vacuum on a few hundred nanometers of low index material (RI ~1.46) and high index material (RI ~ 2.2) of desired thickness (Fig. 6), they provide anti-reflective properties within the visible region of the light spectrum. It is possible to design anti-reflective stacks that offer enhanced protection in the blue-violet light region by adding a specific reflection element at the wavelength to be rejected, in this case 380-460nm. The blue-filtering reflective properties can be effective up to 20 percent while keeping superior anti-reflective properties active within the entire remaining visible range. These ophthalmic lenses display high clarity indoors and outdoors, and offer reliable indoor protection against harmful blue-violet light emitted by electronic devices and artificial lighting while providing moderate outdoor protection as well.

Blue mirror effect of an anti-reflective coating (AR) reflectance spectra

2. Blue light absorption with dyes: yellow filters
Another way to prevent harmful blue-violet light from entering the eye is to reduce the unwanted wavelengths by absorbing them with yellow dye, a chemical compound
whose structure allows absorption in the visible part of the light spectrum of its complementary color: in this case, blue. This is why most blue-absorbing lenses appear more or less yellow depending on the level of their blue-filtering properties. A highly-efficient blue-blocking lens would appear deep yellow, while a moderately efficient blue-blocking lens would appear merely yellowish.

The advantage of the yellow dye solution is that it can reduce a significant amount of blue light, but the intense yellow color is detrimental to its cosmetic appearance and detracts from human color perception. A highly intense yellow filter, for example, will induce color distortion despite the ability of the brain to adapt chromatically.

There is a way to circumvent the yellow color of an absorbing filter that involves “color balancing” the tint by adding a small proportion of another dye. The complementary dye absorbs in another region of the visible spectrum, creating a global neutral grey filter (Fig. 7). This solution is acceptable for low yellow colors – where color balancing can be efficient – but not possible for dark yellow tones. It should be noted as well that color balancing in general is detrimental to the global photopic transmission of a lens since it causes a loss of visible transmission (or clarity).

A lens can also be surface tinted by dipping an uncoated lens substrate or a tintable coated lens in a water dye solution at an elevated temperature.

Another solution is to cast lenses with monomers that already contain yellow dyes – and its color balancing agents – in the original formulation. In this case, only light tints are achievable since darker tints would lead to a non-homogeneous appearance from center to edge due to differences in prescription lens thickness (high-minus and high-plus finished lenses).

3. Sunwear

Sunwear lenses are commonly grouped by ISO 8983-3 standards as class 3, providing 10-15% of photopic transmission (Tv), or the darker class 4 category (Tv < 8%).

In the case of prescription eyewear, sun lenses are essentially made by diffusing a mixture of dyes in a polymer substrate or in a tintable coating. For the plano sunwear business, coloring is achieved by mass mixing an injection mold of polycarbonate for instance. Polarized lenses are made by using dichroic dyes in pre-formed stretched films or encapsulated wafers. The dyes are generally a mixture of primary colors in different combinations to achieve the desired hues based on the principle of subtractive color mixing (Baillet et al., 2008). The most common hues are brown and grey.

In the fashion and high-performance sunwear business, one finds mirrored lenses manufactured on the principle of interferential light rejection stacks and/or a mix of tinting by absorption and rejection mirror technologies.

By definition and usage, sun lenses are made exclusively for outdoor purposes. The dark intensity of the lenses, both plano and Rx, allows very good protection against blue light, especially by brown lenses where the yellow dye content in the mixture is in the majority (Fig. 8).

Sun lenses in brown and grey showing that, at equal photopic transmission (15% Tv), the brown lens filters more blue light than the grey lens, as it contains more yellow dyes in its formulation.

« Depending on exposure blue light may damage the retina »
FIG. 8  |  Sun lenses in brown and grey showing that, at equal photopic transmission (15% TV), the brown lens filters more blue light than the grey lens, as it contains more yellow dyes in its formulation.

FIG. 9  |  Overlay of un-activated and activated spectra of Transitions® Signature™ grey and brown lenses [A] and Transitions® XTRActive® grey and brown lenses [B].
4. Photochromic lenses

Photochromic lenses are non-permanent tinted filters containing photochromic dyes made from molecular structures that are reversible under the action of light (Dürr et al., 1990). Their tint or color is obtained through the same principle of color-subtractive mixing as sunwear lenses.

There are, however, several notable differences in manufacturing technologies, including the cast in place (CIP) process wherein photochromic dyes are added to the monomers before polymerization, and the imbibition process, where photochromic dyes are absorbed into the surface of a lens. In these first two examples, a dedicated polymer allows the photochromic mechanism and movements to occur, and requires different polymers for each refractive index (for prescription lenses). The coating technology, meanwhile, wherein photochromic dyes are added to a coating deposited by dip – or preferentially, by spin – allows the process to be substrate independent. Photochromic lenses are highly efficient in protecting against glare, since their darkness (photopic transmission) automatically adjusts to the amount of outdoor light, whether overcast, in shadow or in bright sunlight. Because they always acclimate to various lighting levels, they help the visual system to adapt instantaneously without compromising visual performance or comfort.

The advantage of photochromic lenses like Transitions® Signature™ lenses is that they are dark outside when sunlight is bright and intense, so they offer a high level of blue light filtering much like regular sun lenses. They can be worn all the times and offer good indoor protection against artificial blue lights with no aesthetic drawbacks such as residual yellow color (Fig. 9).

As described before, color-balancing can help to limit the yellowish aspect of a given filter. For photochromic lenses, where a very low level of yellowness needs to be overcome, the smart color balancing is put to full use. Only a slight amount of dyes are used to deceive the eye (and subsequently the brain) to offset the yellowish aspect induced by chemical species providing the blue blocking properties.

A specific family of high technology products like Transitions® XTRActive™ lenses, which allow activation of the photochromic molecules behind the windshield of a vehicle, present the unique advantage of having a light tint indoor and a strong tint outdoor, leading to enhanced blue light-filtering at all times (Fig. 9 and 10) thanks to specific proprietary photochromic molecules that intrinsically absorb in the blue region of the visible spectrum.
Conclusion

Visible light reaching the retina is essential for visual perception. Despite several self-protection mechanisms, the retina in the human eye can be exposed to light levels that exceed its natural defenses and can cause long-term irreversible damage. The lifelong buildup of light-induced phototoxicity can contribute to age-related changes and retinal cell degeneration.

Preventing excess exposure and accumulation of blue-violet light indoors – and especially outdoors – during one’s life seems like common sense.

Transitions® photochromic lenses – and, in particular, Transitions® XTRActive® lenses – offer the optimum visual experience, regardless of lighting conditions, while providing an ideal protection against blue-violet light under all circumstances (Fig. 11).

• Light plays essential role in the development of visual function and visual performance
• The sun is the most powerful source of light
• Blue light is higher in energy than the other wavelengths in the visible spectrum
• Depending on exposure blue light may damage the retina
• Eyewear industry provide different solutions for blue filtering such as antireflective coatings, yellow absorbing filters, sun lenses and photochromic lenses
• Transitions® photochromic lenses offer the optimal visual experience and ideal protection against harmful blue light

KEY TAKEAWAYS

REFERENCE

3. CONSUMER TRENDS
3. CONSUMER TRENDS
THE WELL-BEING OF ‘WELL-SEEING’ - WHY ARE WOMEN AND THE OVER-50s MORE ENGAGED WITH THE HEALTH OF THEIR EYES?

Prevention has become a focus of health strategies in many countries, especially those with growing ageing populations. Changes are also happening in the attitudes and perceptions of individuals. Two consumer groups in particular emerge as being more engaged in the health of their eyes: women and those over the age of 50.

Philippe Zagouri is the founder of Zed Marketing ‘Research’ and has a background in social sciences (Philosophy, Political Sciences). He has developed a true understanding of consumers’ needs and behaviours based on intuition, rigour and through the use of non-standardised methodologies. For the last five years he has been strongly involved in fuelling Essilor with insights from wearers and ECPs worldwide.

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Joëlle Green is a Research Manager at Zed Marketing Research, with a focus on international qualitative research. She has a background in social sciences (Durham University) and six years’ experience of working in the field of global healthcare marketing, in both advertising and market research agencies.

KEYWORDS
Prevention, protection, eye health, vision health, sun protection, seniors, women, UV, blue, light
Over the period 2012-2014, Zed Marketing conducted multiple qualitative research projects with Eye Care Professionals (ECPs: i.e. opticians, ophthalmologists and optometrists) and consumers (glasses wearers) in France, Spain, Germany, Switzerland, China, the U.S. and Canada. From these, Zed Marketing was able to gain an understanding of consumer awareness and ECP management of preventive eye care to feed into impactful and relevant communication recommendations.

We live in a visual society, but the eye is not number one

“Good vision is a part of good health, being safe and avoiding danger” (U.S., 27, female).1

The eye is a highly complex organ that allows us to see and understand the world around us. Vision has always been important and is becoming increasingly so in today’s society, where even our mobile telephones have become more visual than audio.2 Consequently, the loss of sight is the number one health fear in the U.S., higher even than HIV/AIDS or cancer.3 Despite this importance placed on being able to see, the eye is often overlooked as a part of the body that cannot be protected or get ill in the same way as other organs.

“Honestly, the health of the eye is something I just don’t think about, it’s not as important as my heart or my circulation” (France, 51, male).4

When things go wrong with our sight under the age of 40 it is thought to be due to ‘bad genes’. Everyone is then aware that from the age of 45 or so, our eyesight starts to worsen. Presbyopia is accepted as inevitable and there is often little thought or question as to what could potentially be done to prevent, slow down the onset or the rate of deterioration.

Challenging perceptions of how the environment affects our sight

In more developed countries, consumers are only just beginning to consider additional exogenous factors and the impact it can have on the health of their eyes.

Ultra violet (UV) is the main offender, and is widely known already due to its noxious effect on skin.5 However, the dangers associated with UV and its exact impact on eyes are often not clear and are underestimated. As a consequence, prevention against UV is currently often limited to wearing sunglasses on sunny days. The idea that UV can also affect the eyes on a day-to-day basis is often ignored.

Asia leads the way against Europe in terms of awareness and understanding about UV issues and related eye damage (a subject matter which has been widely reported about in Asian media).

“UV protection is very simple, therefore no excessive description is needed. This is because everybody knows the damage that UV does to eyes” (Optometrist, Taiwan).5

Globally, a dependence on technology and an increase in the number of hours spent looking at screens have raised concerns about ‘visual fatigue’ and the short term symptoms. Glasses-wearers are also beginning to notice that use of certain electronic devices for extended periods may be negatively affecting their visual performance in the long term.6

“Before we used to read only books, now we are using all sorts of vision that never existed before” (Canada, 30, female).7

Consumers in Asia are generally more anxious about ‘waves’ (radiations) in general, living in countries that rely heavily on technology and that have seen a drastic increase in the number of myopic children.8

“To prevent an issue from happening is the best way to look at health; I do whatever I can to implement preventive measures.”
Coupled with bad pollution in cities, this health-threatening environment has encouraged people to find strategies to try and avoid blue light (BL) damage with many anti-BL products available on the market.6

“It’s all those screens: too much TV, computer and playing on iPad” (China, parent of a myopic child).9

Increased awareness about UV, BL, and the rise in the use of technology have started making people question the fragility of the eye and its health, before its unavoidable ageing. Nonetheless, other than regular visits to an eye doctor, there would appear to be a general lack of awareness as to what should or can be done to keep the eye ‘healthy’.

“I see my doctor and use eye drops for dry eyes. What else is there left to do?” (France, 46, female).4

**ECPs: currently control and correct**

Even if it may contribute to ‘healthier’ eyes, the act of going to see an ECP is often more reactionary than proactive. People currently do not think of going to see an eye doctor unless they require corrective glasses/contact lenses or have a problem with their eyes. For this reason, all eye care professionals are perceived by people as helping to correct vision and solve eye issues, rather than offering any preventive benefits.

“The earlier preventive measures are taken, the more beneficial and impactful the effect can be.”

Two consumers groups in particular emerge as being more engaged in the health of their eyes: women and those over the age of 50.

**Over 50: prepared to help prevent**

With age, many people start to suffer from their first health issues, with certain diseases known to become more common. 50 is often used as a milestone for someone to take stock of their health and, where necessary, consider some healthy changes.

This belief is further underpinned by the ECPs themselves (especially opticians and optometrists), as even though in Europe and USA they are fully versed in UV damage, some are not always able to pinpoint the exact damage that can occur.10 As a result, talking about the more abstract long-term benefits of protecting against UVs to their customers can prove to be challenging. At the time of our studies (2012, early 2013), only ECPs in Asia felt comfortable talking about BL to consumers to warn them of the dangers.5

**Focus on prevention**

Prevention has become a focus of health strategies in many countries, especially those with growing ageing populations. Changes are also happening in the attitudes and perceptions of individuals, all encouraging signs that eye health may soon be taken more seriously.

“Two consumers groups in particular emerge as being more engaged in the health of their eyes: women and those over the age of 50.”

“Prevention has become a focus of health strategies in many countries, especially those with growing ageing populations.”

“Changes are also happening in the attitudes and perceptions of individuals, all encouraging signs that eye health may soon be taken more seriously.”
Over 50: more aware of what can go wrong

The over-50s have a better general awareness of eye-related diseases (especially Age-Related Macular Degeneration (AMD), glaucoma and cataracts).

“AMD is like Alzheimer’s of the eye, you lose your vision little by little, and it must be awful!” (France, 55, male). This is thanks firstly to national disease awareness campaigns (which are sometimes directed specifically at this age group) inviting them to take self-diagnosis tests or to visit a doctor; and secondly, people over 50 have more experience of peers who have already been directly affected. AMD is also becoming a much talked about topic, its incidence rate increasing with ageing populations.

Over 50: more aware of what is going wrong

For many, the onset of presbyopia is the first time in their lives that will require them to wear corrective glasses. As they become aware of something happening to their eye-sight, they are, by association, more concerned about the overall health of their eyes.

Women: avoid ageing at all costs

Women are more readily open to the idea of prevention as they have already integrated it for another organ: their skin. They are aware of and are often taking specific measures to slow down and prevent premature signs of ageing on their skin. The negative impact of UV on skin in this respect is well known, so the need to also protect eyes against UV light damage is more intrinsic for women. Inclusion of an AR or anti-UV coating can be an important criterion of lens choice for women.

“Mentioning that the lenses help to prevent eye diseases is not convincing enough. The customers cannot feel the benefit directly.” (Taiwan, optometrist).

Women: the importance of being seen (by an ECP)

Women visit health care professionals more often than men and are more likely to seek out preventive care. This may be due to women being more likely to report poorer health or because, as women are often responsible for their family’s health, it is in their best interest to stay healthy. This means that preventive health care measures are generally more top of mind.

“My grandma has AMD, I want to be sure that I have my eyes checked as often as possible” (Canada, 44, female).

Men, on the other hand, seem to have lower awareness about health problems and are more reluctant to go to the doctor.

ECPs: moving towards prevent and protect

ECPs, too, are thinking more preventively, as they now have better options that they can propose to consumers. Whilst prevention has always been considered more within the remit of ophthalmologists, opticians are also keen to get involved.

“The optician proposed the [anti-reflective] treatment because I work on the computer a lot and spend considerable time outside as well.” (U.S., 46, male)
MARKET WATCH

“Good vision is a part of good health, being safe and avoiding danger.”

“Glasses only correct vision, I’m not aware of how they could work as prevention” (France, 58, female).4

Looking to the future
As positive as it may be that certain groups of society and ECPs are increasingly concerned about preventive eye health, some key questions remain. Firstly, how to engage men, non-corrective eye-glasses wearers and most importantly younger generations on eye health matters that are not necessarily top of mind? The earlier preventive measures are taken, the more beneficial and impactful the effect can be. This is going to be significant in Asia, with their ever-growing number of children with myopia. Secondly, how to further educate on risks associated with cumulative exposure to harmful light, a non-tangible subject for many consumers, as it is not something that can necessarily be seen? Europe and the U.S. should perhaps look to Asia for guidance and examples on how best to communicate and raise awareness about UV and BL. These questions may become even more important in the global context of today’s screen-society – young people currently spend on average six hours 50 minutes per day looking at a screen17 – and this looks set only to increase.

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KEY TAKEAWAYS

• Despite the increasing importance of vision in today’s society, the eye is not thought about in the same way as other organs and little is done preventively.

• Consumers (especially in Asia) are beginning to understand that there may be exogenous factors that can have an impact on the eye’s health such as UV and visual fatigue from screen overuse.

• There is a general lack of awareness of what can be done to keep the eye healthy; ECPs (eye care professionals) are currently perceived as being only a measure to correct eye issues rather than prevent anything.

• However, there is a move towards thinking about ‘prevention’ at both governmental and individual level for all things health, especially amongst women and those over 50 years old.

• Over-50s are more engaged in their eye health as suffering from presbyopia and potentially other health issues (or at least aware of them) makes them more open to the notion of prevention.

• Women are more engaged in their eye health as already preventing ageing of another organ, skin, and are more aware of health issues so more likely to see an ECP than men.
PROTECTION OF EYE HEALTH: WHAT PRACTICES THROUGHOUT THE WORLD AND WHAT LOCAL SPECIFICITIES?

In 2014, the protection of eye health is a widespread practice worldwide. Yet there are behaviours specific to each country. A major international survey of 7,000 people conducted on four continents – Europe (France, Germany), North America (United States), South America (Brazil) and Asia (China, Japan, India) – revealed the similarities and differences of the various practices.

While eyes have always been seen as a precious asset, they are difficult to preserve throughout a lifetime. With the spectacular advances in ophthalmology in recent decades, it has become easier to improve and maintain one’s eyesight. In fact, far more information about eye health and safety is available than ever before. And in many countries, access to eye care specialists has greatly improved. Yet there are still areas for improvement worldwide because the question remains: at a time when we are bombarded with information, have we all become accustomed to protecting our eyes? What are the most widespread practices in today’s world? And are they evenly spread across all segments of the population?

To answer these questions, Ipsos conducted a major international survey on the following four continents in 2014: Europe (France, Germany), North America (United States), South America (Brazil) and Asia (China, Japan, India). In each country, a sample of 1,000 people representative of the national population was surveyed (urban populations in China, India and Brazil). Overall, 7,000 interviews were conducted. In each country, the same indicators were measured, enabling researchers to compare perceptions and habits across different countries.

In the end, it appears that protecting eye health is a widespread practice worldwide, but that public education on the issue differs from one country to another. The research supports the need for different communication and targeting strategies to continue improving the public’s well-being and eye health.
On a global scale, two-thirds of individuals take preventive measures to protect their eye health.

Interviewed about what they do to protect their eyesight, 68% of respondents said they take at least one preventive measure (see Fig. 1). Notable fact: this figure is comparable across countries. In countries such as China and Brazil, the health habits of the urban middle classes are increasingly dovetailing with those of populations in developed countries. There is only one noteworthy exception: in Japan, only 36% of respondents report taking one preventive measure.

The two pillars of prevention: sun protection and visiting a vision care specialist

How do people take care of their eyes? Two preventive measures particularly stand out worldwide. The wearing of sunglasses is the first measure. This is a well-established habit among 32% of respondents. Protecting the eyes from sun exposure is viewed as a very important health habit. Populations in France and the United States are most likely to be concerned about this issue. In these two countries, nearly one in two individuals reports wearing sunglasses to protect their eyes – 45% in France and 47% in the United States.

“The ubiquitous presence of screens is encouraging the most exposed individuals to protect their eyes”

The second most widespread preventive measure worldwide is visiting a vision care specialist. From this perspective, 30% of respondents believe there is nothing better than regular eye checks. It should be noted that people living in Western countries are much more likely to regularly visit a vision specialist: 48% in France, 41% in the United States and 31% in Germany. In Asia, regular check-ups are much less common (11% in China and 7% in Japan).

Respondents reported other practices as well, but they are much less widespread and their intensity varies greatly from one country to another. It should be noted, however, that the ubiquitous presence of screens is encouraging the most exposed individuals to protect their eyes. This reflects the fact that computers and tablets are now an integral part of the workplace for a large number of people, with the proportion of those using lenses for protection from touch screens numbering on average one in ten individuals among the surveyed population.

Lastly, certain measures are specific to certain cultures. For example, in Asia (mainly India and China), a significant percentage of the population reports regularly eating certain foods that supposedly have a positive effect on eyesight (47% in India and 41% in China). This type of prevention measure is much less common in Western countries, where food is more likely to be associated with health benefits unrelated to vision health.

Seniors and women are most concerned about protecting their eyesight

The survey confirms that certain segments are more concerned about protecting their eyes than the rest of the population. Not surprisingly, the older you get, the more likely you are to take steps to preserve your eyesight (see Fig. 2). Seventy-three percent (73%) of people over 50 report taking preventive measures compared to 66% of those under 35. Visiting an eye care professional is the factor that...
most differentiates the older population. While all generations have adopted the habit of wearing sunglasses, the practice of regularly visiting a vision specialist increases with age. Forty-one percent (41%) of people over 50 report doing so compared to only 25% of those under 35. Another segment being proactive about caring for their eyes is women. For one thing, more women do something to protect their eyesight: 70% versus 65% of men (see Fig. 3). Secondly, they are significantly more proactive than men when it comes to most preventive measures. Far more women than men, for example, report wearing sunglasses when they are outside (37% vs. 28%). Women are also more likely to regularly see a vision specialist (33% vs. 27%). Similarly, they are more likely than men to report taking into account the ability of certain foods to protect their eyesight (26% vs. 22%). Last but not least, more women than men lubricate their eyes (17% vs. 12%).

In short, women are currently more aware of and act accordingly. This is a tuned-in population, which means that women are also seeking information on long-term protection.

**Eye care professionals: an intermediary role among healthcare providers**

A more detailed breakdown of the figures concerning visits to eye care professionals shows that 37% of respondents reported seeing an ophthalmologist (optometrist in English-speaking countries) at least once a year and 29%, an optician. These figures are significant: on average, one-third of the population has contact with a vision specialist at least once a year.

Yet when these figures are compared with those involving other specialists, it becomes clear that visits to other professionals are far more common. (Fig 4) This especially holds true when it comes to general practitioners, whom 63% of respondents report seeing at least once a year. And respondents report visiting dentists far more often than they do ophthalmologists. Fifty-nine percent report visiting an ophthalmologist at least once a year.

**FIG. 2 | Differences between age groups.**

<table>
<thead>
<tr>
<th>% DO AT LEAST ONE ACTION FOR THEIR EYES</th>
<th>TOTAL</th>
<th>16-34 Y.o.</th>
<th>35-49 Y.o.</th>
<th>50-64 Y.o.</th>
</tr>
</thead>
<tbody>
<tr>
<td>You regularly wear sunglasses when you are outside</td>
<td>68</td>
<td>66</td>
<td>65</td>
<td>73</td>
</tr>
<tr>
<td>You wear glasses that enable you to protect your eyes</td>
<td>35</td>
<td>32</td>
<td>32</td>
<td>37</td>
</tr>
<tr>
<td>You regularly eat certain foods because you know they are good for your eyes (specific fruits and vegetables, fish)</td>
<td>24</td>
<td>22</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>You wear glasses that are specifically recommended for watching or working with display screens</td>
<td>19</td>
<td>19</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>You regularly rinse your eyes (with artificial tears, physiological saline solution, a lubricant)</td>
<td>15</td>
<td>12</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>You wear glasses that enable you to protect your eyes for specific activities (DTW, welding, sports, laboratory work)</td>
<td>12</td>
<td>16</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>You take dietary supplements specifically for your eyes</td>
<td>7</td>
<td>6</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>You take specific treatments for a diagnosed eye disease (glaucoma, others)</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

**FIG. 3 | Differences between men and women.**

<table>
<thead>
<tr>
<th>AT LEAST ONCE</th>
<th>TOTAL</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>You regularly wear sunglasses when you are outside</td>
<td>68</td>
<td>65</td>
<td>70</td>
</tr>
<tr>
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<td>35</td>
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<td>22</td>
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</tr>
<tr>
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<td>19</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>You regularly rinse your eyes (with artificial tears, physiological saline solution, a lubricant)</td>
<td>15</td>
<td>12</td>
<td>17</td>
</tr>
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<td>7</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>You take specific treatments for a diagnosed eye disease (glaucoma, others)</td>
<td>3</td>
<td>4</td>
<td>3</td>
</tr>
</tbody>
</table>

Overall, eye care professionals rank behind dentists and, for women, gynaecologists. On the other hand, they rank ahead of dermatologists, osteopaths, cardiologists and nutritionists. Vision specialists play an important albeit intermediary role. In all of the survey’s countries, patients are much less likely to visit an eye care professional than a general practitioner, which is to be expected, but also far less likely than a dentist even though vision is a precious asset, as demonstrated by numerous surveys. For example, a survey conducted by Ipsos in 2013 among young people aged
15-30 showed that the eyes, after the teeth, were the part of the body considered most important to take excellent care of as early as possible. This opinion was consistent across Europe, the United States and China. Europe ranks number one for the frequency of visits to general practitioners, while the number of visits to eye care professionals is relatively uniform among the other regions covered by the survey. Americans are most likely to get regular eye check-ups and to see an ophthalmologist than any other nationality. In emerging countries, the urban and online population has greater access to vision professionals.

Lastly, while seniors are more likely than young people to visit specialists, a significant percentage of young people get regular checkups. This fact can undoubtedly be explained, in part, by the role played by parents and schools.

Women and seniors: populations more concerned about protecting their eyesight

In conclusion, it seems that while populations in most of the countries surveyed are aware of measures to protect their eyesight, certain segments are more committed. In particular, women and seniors are more likely to see vision specialists and on a more regular basis. As a result, they are priority targets for any prevention campaign. These results should also encourage eye care professionals to also target less committed populations, i.e. young people and men. This is a public health issue that requires more targeted information.

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“The two pillars of prevention worldwide: sun protection and visiting a vision care specialist

- 32% of people worldwide wear sunglasses (45% in France, 47% in the USA)
- 30% of people see a specialist (48% in France)
- 41% in the US, 31% in Germany, 11% in China, 7% in Japan.

On average, one-third of the population has contact with a specialist at least once a year.
- 37% of respondents see a doctor-prescriber at least once a year, and 29% an optician.

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"Protecting eye health is a widespread practice worldwide"

FIG. 4

The healthcare professionals they have visited.

MARKET WATCH

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KEY TAKEAWAYS

General practitioner
Dentist
Chemist
Gynecologist
Eye doctor
Optician
Dermatologist
Physiotherapist/Osteopath
Cardiologist
Dietician/Nutritionist

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