

Solar radiation and the eye

“When Isaac was old and his eyes were so weak that he could no longer see, he called for Esau his older son ...” (Genesis 27:1; written about 1400 BC)

This is one of the oldest descriptions of a patient with vision loss that was probably caused by solar radiation. The passage indicates that Isaac’s ocular condition was age-related (he was over 100 years old at the time), so he probably had cataracts or macular degeneration, both of which are caused by overexposure to UV radiation. With respect to UV-induced ocular damage, Isaac lived in high-risk region (Canaan) and worked in a high-risk profession (nomadic farmer). Three thousand years later solar radiation continues to be a major cause of blindness worldwide. Today’s optometrists should therefore be familiar with the ocular hazards of sunlight, and know how to protect the eyes.

The solar hazard

Solar energy, including visible light is essential for all life on earth. Without light, vision is impossible, and well-focused optical images are required for normal development of the visual system. It’s somewhat ironic that solar energy is one of the primary causes of aging (which ultimately leads to death), and solar radiation damage is one of the major causes of blindness in the world.

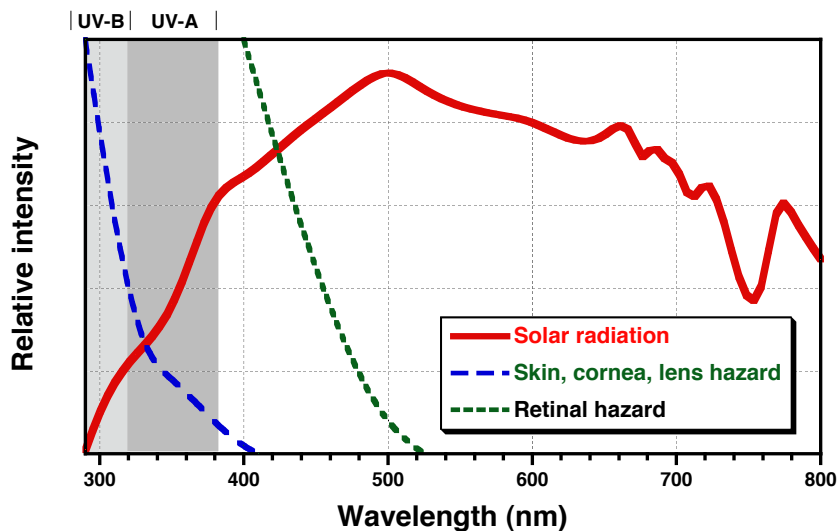


Figure 1. Relative intensity of solar radiation and relative risk spectrum for damage to the skin, cornea, lens (wide dashes) and retina (fine dashes).

Figure 1 shows the relative intensity of the different wavelengths of solar energy reaching the earth. The two dashed curves show the relative risk for damage to the skin, cornea, and lens (left dashed curve) and the retina (right dashed curve) as a function of wavelength. The risk spectra indicate the degree to which those wavelengths can damage anterior and posterior ocular tissues.

UV-C from the sun is completely absorbed in the upper atmosphere, so it is not an ocular hazard. Man-made sources of UV-C, such as arc welding, or electric lamps, however, can be very dangerous to the eye. UV-A and -B cause diseases at the front of the eye because that’s where they are most strongly absorbed. The back of the eye, however, is mostly protected from these wavelengths because they are absorbed by the cornea and lens. A small amount of UV-B is transmitted by the cornea and lens to the retina, especially in very young eyes. Visible light, of course, is transmitted all the way to the retina. Long-term exposure of the higher-energy shorter wavelengths (380-500 nm) of visible light can gradually damage the retina and cause ARMD.

Table 1. Electromagnetic radiation bands

Band	wavelength range (nm)	comment
UV-C	~200 to 290	blocked by atmosphere, doesn't reach earth
UV-B	290 to 320	absorbed by skin, cornea, conjunctiva, lens
UV-A	320 to 380	absorbed by the lens; small amount reaches the retina 100-500 x as abundant as UV-B
visible light	380 to about 700	transmitted to the retina 380-500 nm causes long-term cumulative retinal damage

Five major diseases that affect the eye or its adnexia are caused primarily by high-energy solar radiation. They are:

- Age-related cataracts
- Age-related macular degeneration
- Pterygium and pinguecula
- Photokeratitis
- Skin cancer

Dr. Richard Young (UCLA medical school) referred to these five diseases as the “family of sunlight-related eye diseases” in an important paper published in *Optometry and Vision Science* (February 1994). He stated that,

Each of these five ocular diseases is universally considered to represent a distinct clinical entity. ... In contrast, when attention is directed to the factors that cause these disparate ocular conditions, a remarkable similarity emerges: they all share the same primary causal factors. (Young, p. 135)

For his work in the field of UV-induced ocular damage, Dr. Young received the prestigious Charles Prentice Award from the American Academy of Optometry. The following paragraphs summarize his article.

The family of sunlight-related eye diseases

Although the five diseases mentioned above affect different parts of the eye and have different clinical manifestations, they are all the result of molecular damage caused by three things:

- solar radiation
- oxidation
- heat

Among these three causative factors, solar radiation is the most significant.

Other factors contribute to the morbidity of these diseases, such as the lack of antioxidants in the diet. Other factors create a greater risk for patients with these diseases, such as age, or living in the tropics, however the risk factors themselves do not cause the disease.

Although these different diseases have a common etiology, they have different clinical manifestations just because the different structures and physiologies of these tissues respond differently to insult. At the molecular level, the fundamental disease process is the same and is caused by three main mechanisms.

- High-energy radiation is absorbed by molecules which are directly altered.
- The radiation transforms oxygen into highly toxic free radical that destroy tissues.
- The absorbed energy increases molecular kinetic energy, which generates heat and more damage.

Radiant energy present in sunlight can degrade the native molecular structure of all parts of the eye that absorb it by its own direct effects. High-energy components of solar radiation are capable of damaging all biological molecules. The endogenous factors of oxidation and heat are also capable of damaging molecules. However, because oxygen and heat are regulated by homeostatic mechanisms within narrow limits, the amount of damage they generate is believed to be small under dark conditions. This situation changes drastically when the eye is irradiated with high-energy photons. The rate of molecular damage immediately rises due to the direct effects of the radiant energy. Some of the energy is transferred to oxygen molecules, producing the excited-state species that can set off free-radical chain reactions. Furthermore, much of the radiant energy is ultimately degraded to kinetic energy, raising the rate of thermal damage. Consequently, solar radiation damages ocular structures by its own direct effects, and exacerbates the two other causal factors, oxidation and heat. (Young, p. 135)

These mechanisms occur throughout life as a normal function of life and aging. To some degree the body is able to protect itself from the damage and repair itself. But over time the molecular damage accumulates leading to age-related degeneration. When the degeneration reaches the point that visual function is affected, it becomes pathological. Among the five diseases mentioned above skin cancer, pterygia, cataracts, ARMD are chronic diseases in which the damage accumulates for years or decades before reaching a pathological state. Corneal damage is acute, and symptoms appear within a day of UV overexposure.

UV-B and UV-A are the primary hazards, and their effects are cumulative over time.

UV-B photons (290 to 320 nm) are more damaging to biological molecules than those in the UV-A part of the spectrum (320 to 400 nm), but there is 100 to 500 times more UV-A than UV-B in sunlight. For this reason, the UV-A presents a greater hazard under natural sunlight conditions than is implied by the experimental action spectra. (Young, p. 137)

Age-related cataract

Cataract alone causes about 60% of all blindness and is therefore the leading cause of blindness worldwide. In the US, cataract surgery is the most common type of surgery, with the number of cases far exceeding the next 9 types of surgeries combined. Epidemiological studies have clearly demonstrated a higher prevalence of cataracts among people that have a greater exposure to sunlight and UV radiation.

Over time, sunlight causes molecular damage in the lens that leads to the formation of yellow pigments and accumulation of microscopic particles. The gradual yellowing seen throughout the lens gradually reduces transmission, and is what we call **nuclear sclerosis (NS)**, though it's usually not confined to the nucleus. Strictly speaking, NS is a misnomer. A more correct term would be cortical sclerosis, or cortical opacification, but most clinicians record this kind of cataract as "NS."

Scattering is mainly seen in the form of **cortical spokes**, and **posterior subcapsular cataract (PSC)** is caused by the abnormal migration of lenticular cells to the posterior lens. All of these types of cataracts are caused by UV exposure, and with time (that is, aging) the cumulative damage begins to affect vision.

The cataractous lens of the elderly eye represents an advanced state of lens deterioration. No discontinuity between senescent and cataractous changes can be detected at the molecular level in the human lens. The transition from normal aging of the lens to the presence of a cataract is signaled by the disruption of visual function. (Young, p. 126-7)

The cornea absorbs much of the UV-B incident on the eye. Therefore, although the high-energy UV-B has greater potential for damage, it probably doesn't cause as much damage to the lens as UV-A, which is 100-500 times more abundant. The lens responds to UV-A exposure by producing yellow pigments, which has a protective effect. The pigments absorb UV-A and help block transmission to the posterior eye. Antioxidant can also help protect the lens from sunlight-related damage.

There is recent evidence that insufficient dietary intake of antioxidants may be a contributing factor in cataract formation. (Antioxidants act to stop free-radical chain reactions.) When antioxidant defenses are diminished, oxidation reactions set off by UV radiation are more damaging. (Young, p. 128)

Age-related macular degeneration (ARMD)

ARMD is the leading cause of blindness among elderly Americans. Like cataracts, it may be considered an extreme of the aging process caused by exposure to sunlight. The sunlight damage is confined to the central retina and directly affects the RPE. Disruption of the RPE leads to the loss of rod and cone photoreceptors. In a young child the crystalline lens is very clear and it transmits more UV than the adult lens. This can expose the young child's retina to the damaging effects of UV. As the lens begins to yellow, UV transmission to the retina decreases. Although very little UV reaches the retina, its effects are cumulative, so over time it can lead to retinal aging, a gradual degeneration and eventually ARMD. Young states that short wavelength visible light up to 500 nm can also cause long-term damage leading to ARMD.

Q. Why do you think that ARMD is mainly confined to the outer layers of the central retina?

A. *This is where light is most clearly focused, and therefore, where solar energy is most highly concentrated. In addition, the high vascular supply provides abundant oxygen.*

In the outer layers of the retina, especially the RPE and visual cell layers, conditions for photodynamic effects are optimized by the presence of unsaturated fatty acids, high levels of oxygen, a variety of absorbing molecules and a focused beam of radiation. (Young, p. 130)

A nutritional deficiency of antioxidants compromises the retina's natural repair mechanisms, which are then unable to cope with the accumulated molecular damage.

A related condition was recorded in World War II in Southeast Asia, where malnutrition apparently reduced the efficiency of molecular defense mechanisms and thereby heightened the effect of intense sunlight. The result was the production of central retinal lesions that resembled macular degeneration insofar as they included irreversible central scotomas associated with macular pigment irregularities surrounding the fovea. Persons with low ocular pigment were most severely affected. (Young, p. 131)

Pterygium

UV exposure is generally recognized as the main cause of pterygium and pinguecula, but other things that cause conjunctival irritation and injection can also cause these diseases. The conjunctiva absorbs UV radiation much more strongly than the cornea or lens.

As with the other sunlight-related diseases, pterygium is more prevalent among people living in sunny regions, near the equator or in outdoor occupations. It is age-related in the sense that older people have longer exposures to the sun than younger people.

The likelihood of having pterygium was raised several hundred-fold among those who worked mainly on the sand, and almost 20-fold among those who worked on concrete. Spending much of the time outdoors in childhood was associated with a 20-fold increase in risk. (Young, p. 132)

Photokeratitis

This condition is known by other names: solar photoophthalmia, UV-keratitis, UV conjunctivitis, actinic keratitis, and photokeratoconjunctivitis.

The last term, although sesquipedalian, is the most accurate, because the keratitis inevitably occurs as a part of a syndrome that also involves the conjunctiva. (Young, p. 132)

It is caused by acute overexposure to UV-B and -A, that is, sunburn of the conjunctiva. It is therefore often seen in snowy region with bright sun, especially at high altitudes. People afflicted in these regions may refer to it as **snow blindness**.

This differs from the other sunlight-induced conditions in that it's acute. As with common sunburn, there is a 6-12-hour latency between exposure and the onset of symptoms, which include a gritty foreign-body sensation, photophobia, tearing, pain and eyelid spasm. Acute symptoms last about 1 day, and then subside.

Normally damage occurs in the epithelium, but with more exposure stromal keratocytes or the endothelium may be damaged. Some research indicates that corneal damage may persist even after a symptomatic recovery.

Arc welding or exposure to some high-energy lamps can cause the same condition. During my residency I heard of a case in which an entire family came to the optometry walk-in clinic one morning with acute ocular pain, redness, tearing and diffuse punctate keratitis. The case history revealed a common cause: the entire family had been watching a softball game the night before, and was sitting in the bleachers under one of the floodlights, which apparently caused the photokeratitis.

Skin aging and skin cancer

It is well established that sunlight accelerates skin aging, and it is the primary cause of all forms of skin cancer, squamous-cell carcinoma, basal-cell carcinoma, and malignant melanoma. Up to 90% of all skin cancers are found on the head and neck.

Well before the present century the phrases 'farmer's skin' or 'sailor's skin' were part of the common parlance, referring to the dry, rough, wrinkled, leathery, sagging irregularly pigmented skin on the uncovered parts of the body in persons whose livelihood obliged them to work outdoors, where they were chronically exposed to sunlight. A comparison of the skin around the eyes of a farmer with the skin on his buttocks (both precisely the same age) is sufficient to reveal the accelerated aging produced by extended exposure of the circumorbital skin to sunlight. Thus, the ultimate cumulative effects of chronic sun exposure, by producing wrinkled, sagging skin around the eyes, can make a person look older than his chronological age. (Young, p. 133)

Sun damage affects the epidermis, causing mottling, scaling, horny growths. It also reaches the dermis, where it causes collagen damage, and destruction of elastic fibers that cause sagging and wrinkling. Every extended exposure causes permanent damage, and the effects are cumulative.

Other considerations (from Dr. Donald Pitts)

In many cases, the damaging solar radiation does not damage our eyes from above, but by reflection off of other surfaces. Therefore, when evaluating the patient's risk, you must also consider his surroundings. Table 2 compares different albedo or reflectance values for some common surfaces.

Table 2. Reflectance from different surfaces

<i>Surface</i>	<i>Albedo (% reflectance)</i>
<i>grass</i>	3-5%
<i>water</i>	3-10%
<i>dirt</i>	25%
<i>dry sand</i>	31%
<i>snow</i>	85-95%

Activities with the highest UV exposure:

1. Snow skiing is the worst
2. Next is mountain climbing
3. Beach. Note that beach umbrella can increase UV exposure for people underneath it because it collects reflected radiation and focuses it downward like a parabolic reflector.
4. Sunbathers using tanning beds

Consider the patient's environment:

- 50% of UV is transmitted to the earth's surface between the hours of 10:00 am to 2:00 pm.
- There is a 15% increase in exposure for every 1000 m increase in elevation.

Clouds absorb IR but not much UV. Therefore, you can get a sunburn on a cloudy, overcast day.

The eye is about twenty times more sensitive to UV damage than the skin.

Previous exposure to UV can render the cornea more susceptible to damage. It only takes about 0.41 times the energy to burn the cornea if it has been damaged by UV within the previous 8 hours.

All patients who are exposed to UV should protect their eyes, but the following kinds of patients are particularly at risk for UV damage.

- all aphakes – rare today, but occasionally you will see an aphakic patient.
- pseudophakes – should have UV protection in their IOLs.
- cataract patients – have already been damaged by UV
- those with pterygia, pinguecula – common among our patients
- people taking photosensitizing drugs – tetracyclines, naproxen, sulfonoureas, chlorothiazides, others
- people who spend much time in the sun (skiers, sun bathers, fishermen, users of sun lamps, children who play outdoors)

Also note that today's population has greater UV exposure than previous generations.

- More leisure time spent outdoors
- Style - short sleeve shirts, short pants more popular
- People don't wear hats as much
- Greater UV exposure due to less ozone

Certain vocations have a high exposure to either natural or manmade sources of UV.

- Welding – manmade sources
- electronics – manmade sources

- graphic arts – manmade sources
- some researchers – manmade sources
- watermen - sunlight
- outdoor jobs - sunlight

Uncovered fluorescent tubes or halogen lamps can also expose office workers to UV radiation.

At the end of his article, Young stated, “ ... the risk of all these serious eye diseases can be diminished simultaneously by the simple, practical, safe and inexpensive means of using eyewear that absorbs the high-energy photons present in solar radiation.” (Young, p. 138)

This leads us to the topic of protection against the solar hazard.

Protecting the eye from solar radiation

What can you do to protect these patients? The regimen for ocular protection should include the following:

- hat with at least a 4 inch brim
- avoid being outside between 10 am and 2 pm; doing so reduces exposure 50% and increases your safety factor by a factor of 4.
- wear sunglasses – the more the ocular coverage, the better. The best protection is provided by closely fitting wrap-around designs.

Relative protective effect of ophthalmic materials:

- Clear glass transmits about 16% of incident UV radiation. This is better than nothing, but still inadequate. Other materials can do better.
- Coated CR-39 lenses effectively block UV-B and some UV-A up to about 360 nm.
- Plastic lenses treated with a clear UV-400 very effectively block UV-B and UV-A up to 390 nm. UV coatings are very stable over time.
- Polycarbonate lenses block UV very well. They're better than CR-39.

Table 3: Relative UV exposure with different protection regiments:

<i>Protection regimen</i>	<i>Relative UV dose</i>
<i>indoors</i>	4
<i>when outdoors, wear hat + sunglasses</i>	8
<i>when outdoors, wear sunglasses only</i>	17
<i>when outdoors, wear hat only</i>	47
<i>no protection when outdoors</i>	72

Optimum sunglasses should:

- Reduce illumination to a comfortable level
- Generally, a good transmittance for sunglasses is 15%. Less than 8% can reduce VA.
- Darker than 10% should be reserved for special purpose sunglasses, such as for mountain climbing.
- Keep in mind that elderly patients have smaller pupils that transmit less light to the retina. You should therefore increase the sunglass transmittance 1% for every year of age over 55.
- Does not distort color perception, so normally neutral gray is best. This is important so people will still be able to recognize the colors of traffic lights correctly. Note, however, that brown lenses can help protanopes since it allows more transmission in the red end of the spectrum (boosts red for those who are red-weak).
- The so called “Blu-blocker” lenses are not recommended since they distort color perception.

Over exposure to UV during the day can interfere with dark adaptation later than night. Pilots who plan to fly at night must wear sunglasses during the day. The same goes for people who drive at night.

See figures in Borish Chapter 25 for transmission curves of different ophthalmic materials with different tints and coatings.

Light reflected off of roads or other surfaces becomes plane polarized in the 180-degree meridian. The only lens that can effectively cut this glare are polarized lenses. They are recommended for drivers or fishermen. For some occupations, however, polarized lenses may be harmful; for example, for military pilots.

Photochromic lenses are sensitive to UV, which causes them to darken. Heat and IR radiation clears them. The new Transitions plastic photochromics do a good job of cutting UV. Some contact lenses contain UV absorbers, but keep in mind that they will provide no protection for the conjunctiva (pinguecula, pterygium, etc.)

Every patient should have at least three pairs of glasses: a clear Rx with UV coat, computer glasses, and outdoor sunglasses that provide good UV and sun protection.