

Ocular Hazards Associated with Eclipse Viewing

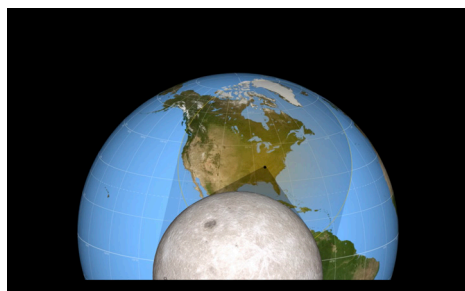
Arthur Bradley¹ and Todd Peabody²

Abstract

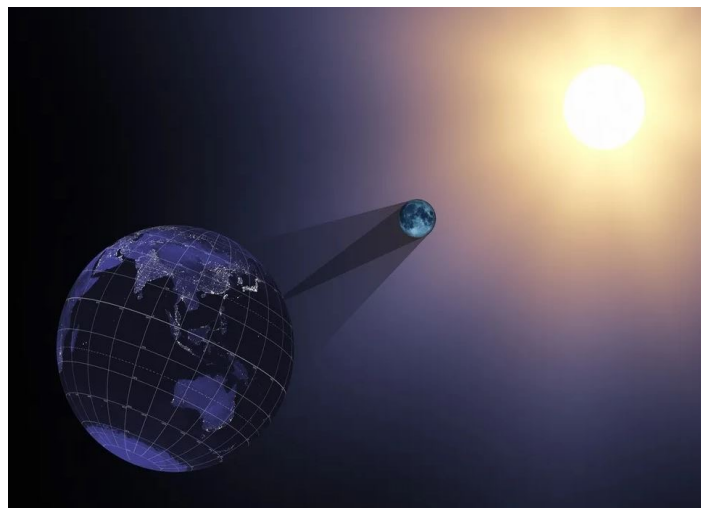
You have probably heard the warnings about direct viewing of an eclipse and the damage it can do to your eyes. What is the real threat, and how does sunlight during an eclipse pose a threat? In this article, re-printed from the 2017 Indiana University CelestFest website, hear from faculty at IU's School of Optometry about the medical information surrounding this topic.

On Monday, April 8, 2024, Indiana will experience a solar eclipse. The path of totality, or the area that will experience a full eclipse, will be only about 115 miles wide, arcing from the southwest to the northeast. Those outside the path of totality will experience a partial eclipse.

When the Moon passes between the Earth and the Sun, it casts a shadow onto the earth. Because of the large size of the Sun the shadow is only complete during umbra, the phase of the fully shaded inner region of the Sun which occurs during a total solar eclipse. This is the inner and darkest part of the Moon's shadow and the only time you can view the eclipse safely with your eyes. It lasts for only a few minutes depending on how close to the center of the path you are viewing from. The much larger shadow we refer to here is its penumbra, the outer and lightest part of the Moon's shadow. This is when only part of the sun's light is blocked. It is unsafe to look at any part of the Sun directly.



Full listing of authors and contacts can be found at the end of this article.

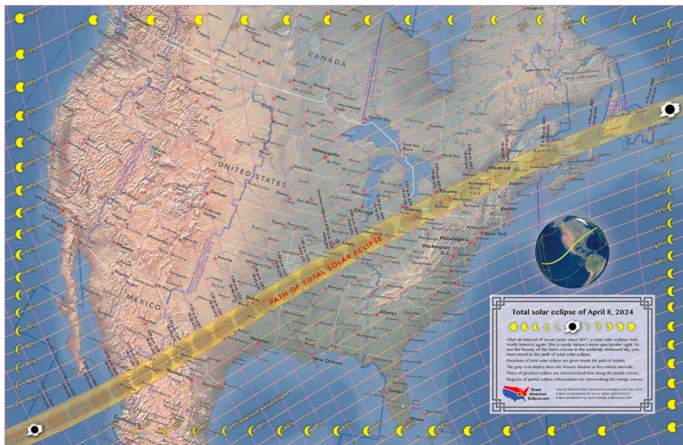


Shadow umbra, the dark inner shadow and penumbra, the lighter outer shadow. (NASA's Scientific Visualization Studio)

Photographs taken from deep space show this shadow moving across the planet at very high speed (between 1500 and 3000 km/hr). A NASA animation depicts how the Moon's shadow moves across the Earth during a total eclipse on August 21, 2017. The small darker circle of the umbra and the larger lighter circle of the penumbra can be seen. [Click here or on the image at the left to view an animation.](#)

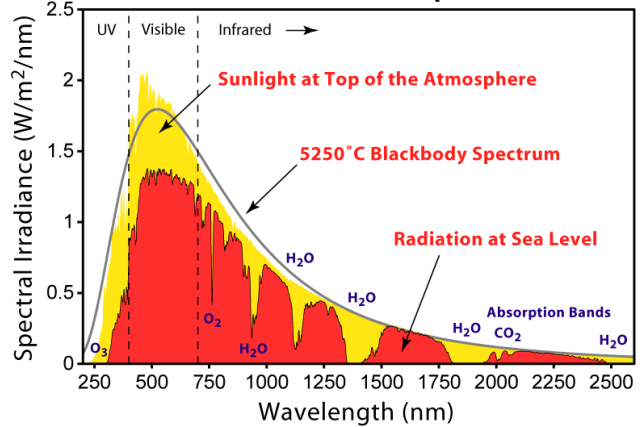
This essay summarizes the ocular dangers posed by this solar eclipse.

Those in the penumbra of the moon’s shadow, viewing a partial eclipse, will be especially susceptible to eye damage during eclipse viewing.



Michael Zeiler, GreatAmericanEclipse.com, by permission.

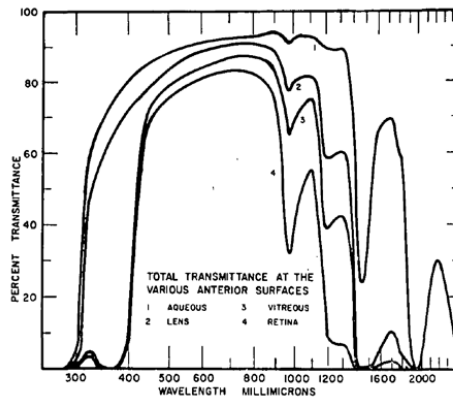
Solar Radiation Spectrum



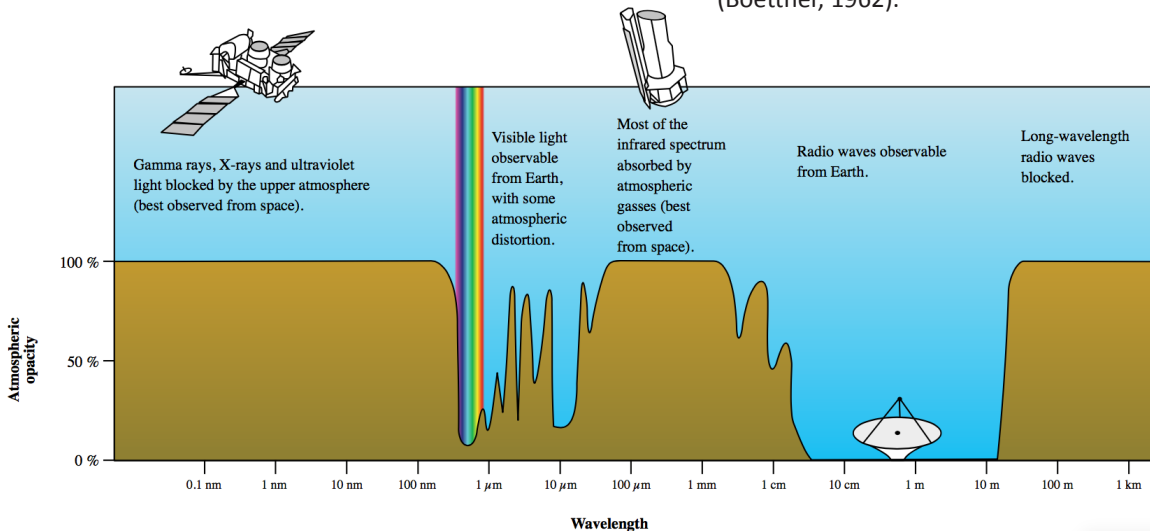
Due to availability in the solar spectrum, and the high transmission of our atmosphere, most animals on earth are visually sensitive to the 400 – 700 nm range of the electromagnetic spectrum. (Robert A Rhode, 2013)

The Solar Spectrum

Acute solar damage to the retina occurs most frequently during solar eclipses. Worryingly, these sight threatening events occur in spite of an educated population, and children and young adults are most vulnerable (MacFaul, 1969). Typically, we avoid looking directly at the sun, because it is so bright, and generates a sense of discomfort. However, during an eclipse, people might look at the sun for many minutes, during which accumulating damage builds up in the retina. However, because there are no pain detectors in the retina, this damage goes un-noticed until much later when vision is impaired.



In order for us to see this spectral range, the optical components of the eye must be transparent at these wavelengths, because the photo-detecting cells are at the back of the eye (the retina). The eye’s optics are highly transparent between 450 and 800 nm (Boettner, 1962).



The Solar Spectrum. (NASA, 2018)



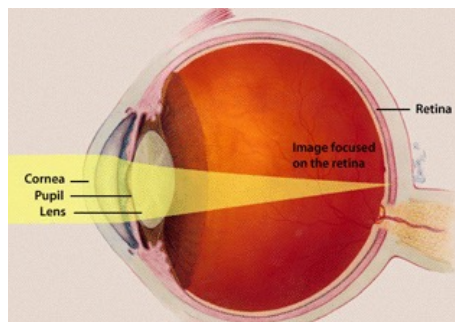
So, the spectral characteristics of the eye are nicely adapted to the realities of our solar spectrum, atmospheric transparency, and the need to create images at the back of the eye. However, although we have evolved to employ indirect solar radiation (reflected from objects in the environment) for vision, direct viewing of the sun can be damaging to the retina. For example, our laptop computer may have a luminance of 160 cd/m², but the sun has a luminance of 1.6×10^9 cd/m² at noon, or 10 million times brighter than your computer screen.

Intensity Amplification by optical systems including the eye

In addition to generating images, optical systems also amplify the intensity (flux density) of light. We are all familiar with this phenomenon from our childhood experienced using magnifying glasses to burn holes in paper by imaging the sun directly onto the paper. For example, a typical magnifying glass 5cm in diameter with a 20 cm focal length will increase the concentration of rays by a factor of about 800x between the lens and the solar image. This is typically sufficient to burn paper. Performing the same calculation for the human eye, we discover that for a large pupil (e.g. a nighttime pupil diameter of 7mm, or a dilated pupil following an eye exam) this amplification effect is >1000x, but of course, when looking at the sun, the pupil will normally shrink to about 2mm, but still the amplification is high, above 100X. Studies of rats have shown that solar and eclipse exposure when the pupil has been dilated by drugs dramatically increases the retinal damage (Thanos et al., 2001). The image of the sun on our retina has an area of about 0.017 mm², which is small enough to fit completely within the foveolar.



(BCCampus Pressbooks CC-BY 4.0)



(National Eye Institute, US Gov't Works)

Source of Retinal Damage

The above analogy of the microscope burning paper is useful, but misleading in one important way. Our retina is not a piece of dry paper in air, but biological tissue surrounded by water, blood and other heat conducting materials. Because of this, although heat is created in the pigment rich outer retina by the image of the sun, it is dissipated through heat conduction via the adjacent tissue, and when viewing the sun, the retinal temperature does not increase sufficiently to damage the retina (imagine using a magnifying lens to raise the temperature of paper in a glass of water). Passive heat conduction, and not active blood flow through the choroid is primarily responsible for heat dissipation, and solar imaging will not raise retinal temperature by the 10 degrees required to cause photocoagulation in the retina (Mainster, 1998).

Electromagnetic radiation can, however, cause damage in another way. If the small packages of energy (photons) that constitute light individually contain enough energy to dislodge electrons from the atoms that absorb them, they can cause direct molecular changes as their energy is absorbed. This type of light damage, unlike heat, cannot be dissipated, and it is much more likely to occur in tissue that is full of pigment, as is the outer retina. Short wavelength visible photons reach the retina and contain sufficient energy to create these electron changes. After viewing the sun, therefore, the pigment rich photoreceptors and pigment epithelial cells in the outer retina suffer most of the damage (Wu et al., 2006) because of the rhodopsin in the photoreceptor outer segments, and the melanin and lipofuscin in the retinal pigment epithelial cells. The chemical changes that occur generate free radicals, which in turn damage cell membranes and other cellular tissues. The high concentrations of pigment to absorb short wavelength photons in the outer retina, combined with the high levels of oxygen provided by the choroid for the normal high metabolism of photoreceptors, make the outer retina especially vulnerable to this photo-oxidative damage.

Lipid membranes are especially vulnerable to such oxidative damage, and the photoreceptor outer segments are full of lipid membranes. Also, unlike heat that dissipates, photochemical damage will simply accumulate with increased exposure. That is, the longer you look at the sun, the more retinal damage will ensue. Because

shorter wavelength photons are responsible for this photochemical damage in the retina, young eyes (and eyes with an artificial or fake lens) are more vulnerable because of higher short wavelength transmission by the lens.

Clinical Evidence of Eye Damage

Hospitals and eye clinics are often visited by patients suffering from vision loss after a solar eclipse (MacFaul, 1969; Atmaca et al., 1995; Wong et al., 2001), and in each case, the eye exam reveals that the retina has been damaged. Patients who viewed the sun for longer periods had more serious damage (Wong et al., 2001), that in many cases does not recover, causing small regions of blindness in the eye. In milder cases, there appears to be a recovery process, and the clinically observable signs of retinal damage disappear, and vision seems to recover (MacFaul, 1969; Atmaca et al., 1995). However, patients who initially present with visual acuities worse than 20/50 never recovered 20/20 acuity (Atmaca et al. 1995). Recovery is restricted to the 1 month after the lesion develops, with no additional recovery (or worsening) over future years. However, the ability to recover seems to vary significantly from one eye to another in an unpredictable way. Because the retinal lesions created by sun-viewing are small and generally located in the central fovea (Codenotti, et al. 2002)), retina close to the fovea can be healthy and thus visual acuity reductions are often small (Wong, et al. 2001). Also, visual acuity can return to 20/20 in spite of a permanent small foveal scotomas (MacFaul, 1969).

Surprisingly, these patients arrive at clinics in modern times in countries with sophisticated health systems that educate the population about the dangers of direct viewing of the sun during an eclipse. For example, after the 1999 solar eclipse in England, many patients suffered from retinal damage (Wong, et al. 2001). Most concerning are reports that children and young adults are more likely to experience damage. Especially concerning was that many patients viewing the solar eclipse in England in 1966 and Turkey in 1995 experienced retinal damage even though they had employed some protective eye-wear that presumably was thought to provide adequate protection (MacFaul, 1969; Atmaca, et al. 1995). Imperfect use of the protective devices and ineffective filtering can both lead to retinal damage when viewing an eclipse. Use of ineffective protective devices and strategies can be, therefore, doubly dangerous because they provide the illusion of safety, may cause pupil dilation, and can prevent the avoidance

behavior that normally protects us from solar damage to the retina by encouraging extended viewing.

Because there are no pain receptors in the retina, damage can happen without the person being aware. History suggests that this will happen when people employ inadequate filters in an unsuccessful attempt to protect their eyes. For example, although certified sunglasses employ powerful UV blocking filters (e.g. no more than 5% of UV wavelengths <380 nm can be transmitted by these lenses), they can meet standards by transmitting between 3%-100% of visible light ([EU standard EN 1836:2005](#)). However, to meet established safety standards for comfortable and safe solar viewing, lenses/filters must transmit no more than 0.001% (1/100,000) of the incident light at all wavelengths. This difference emphasizes that even fairly “dark” sunglasses that transmit only 10% will be transmitting 10,000 times more light than is considered safe, leading to the most crucial conclusion that **sunlasses must NEVER be used to view an eclipse!** Only specially manufactured “eclipse glasses” should be used ([AAS Eclipse Safety Report, 2017](#)). The AAS report reveals that “To date four manufacturers have certified that their eclipse glasses and hand-held solar viewers meet the ISO 12312-2 international standard for such products: Rainbow Symphony, American Paper Optics, Thousand Oaks Optical, and TSE 17.” It is important to check updated reports for each solar eclipse viewing event.

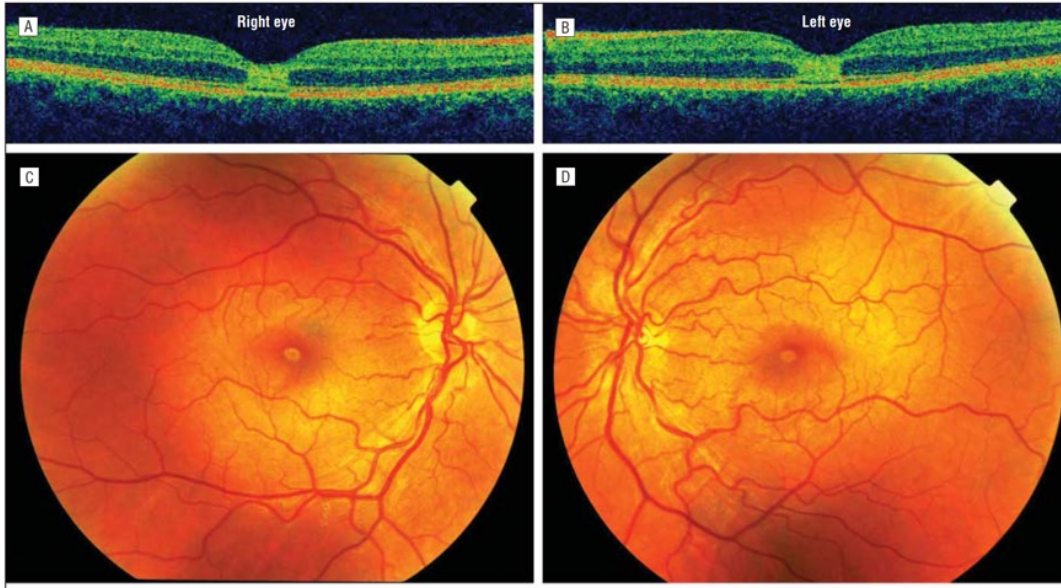
Clinical signs of Solar Retinopathy

Macular edema, loss of foveal reflex, and lamellar macular holes have all been reported in cases of solar retinitis (MacFaul, 1969).



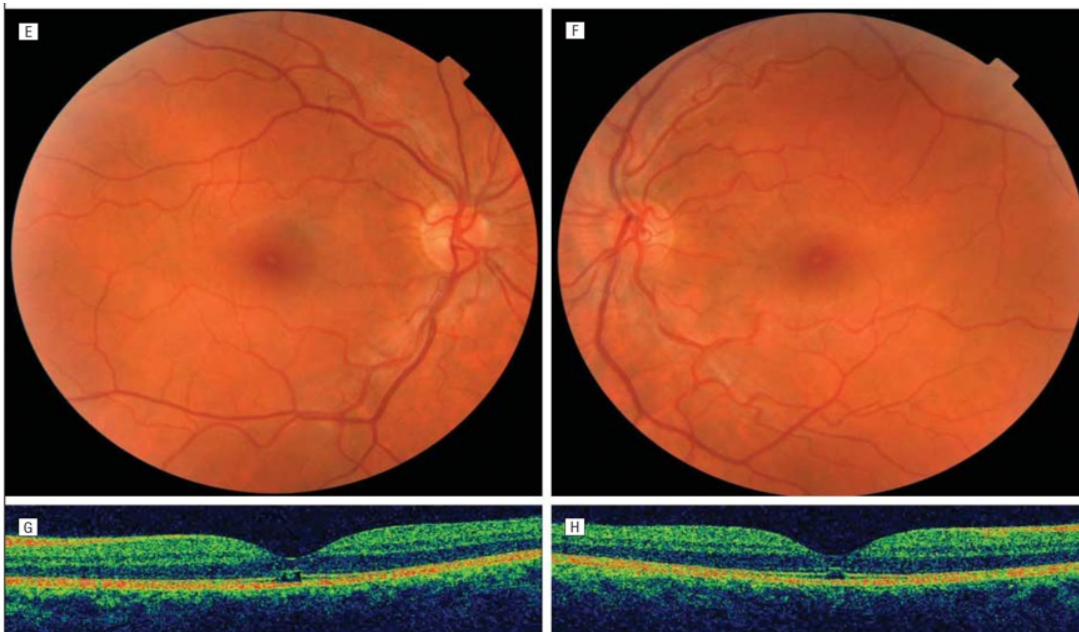
Case Study (Levy et al., 2012):

This case shows how the fundus and OCT (optical coherence tomography) images change over the first month following the acute exposure of the fovea to the sun's image, VA (visual acuity) 20/60 and 20/40 for Right and Left eyes.



These images show the foveal hyper-reflectivity of the outer retina. Note the apparent complete absence of cone cell nuclei (outer nuclear layer) and the cone outer-segments.

At a 3 month follow-up, the outer retina has locally reduced reflectivity, and VA had improved to 20/30.



Case study (Baisakhiya, Chaudhry et al. 2012):

A 21 year old male presented to the eye outpatient department (OPD) with complaints of blurred vision, central scotoma and metamorphopsia since one day after watching the solar eclipse through the radiographic film. The duration of exposure was 2 to 3 minutes. On examination the visual acuity was 6/60 in both the eyes. The Amsler grid revealed a central scotoma and metamorphopsia. The patient was treated with oral prednisolone tablets in dose of 1mg/kg of body weight for 1 week, which was then gradually reduced over a period of four weeks. The patient recovered during the first one month, with reduced metamorphopsia and recovered visual acuity with no residual ophthalmoscopically detectable damage to the macula.

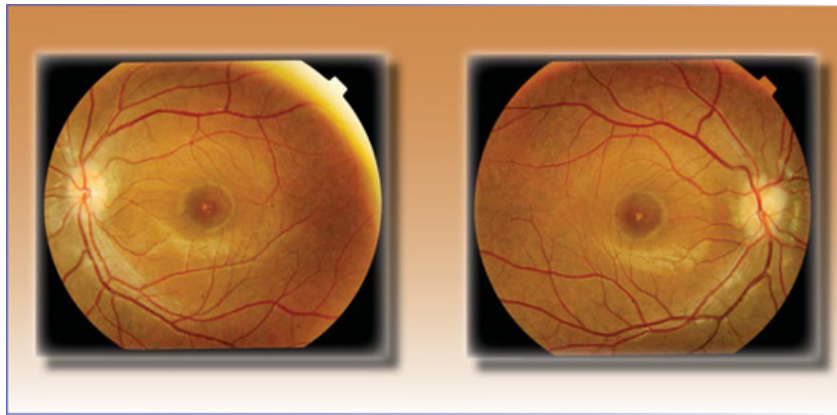


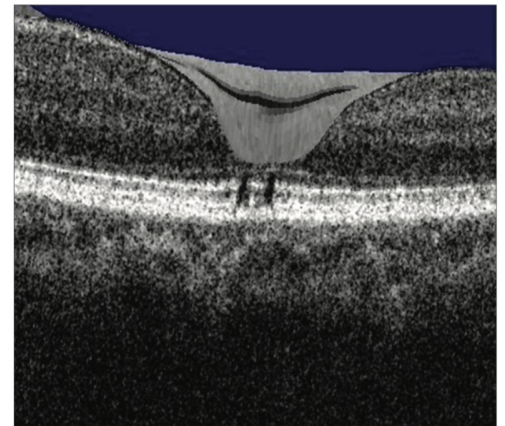
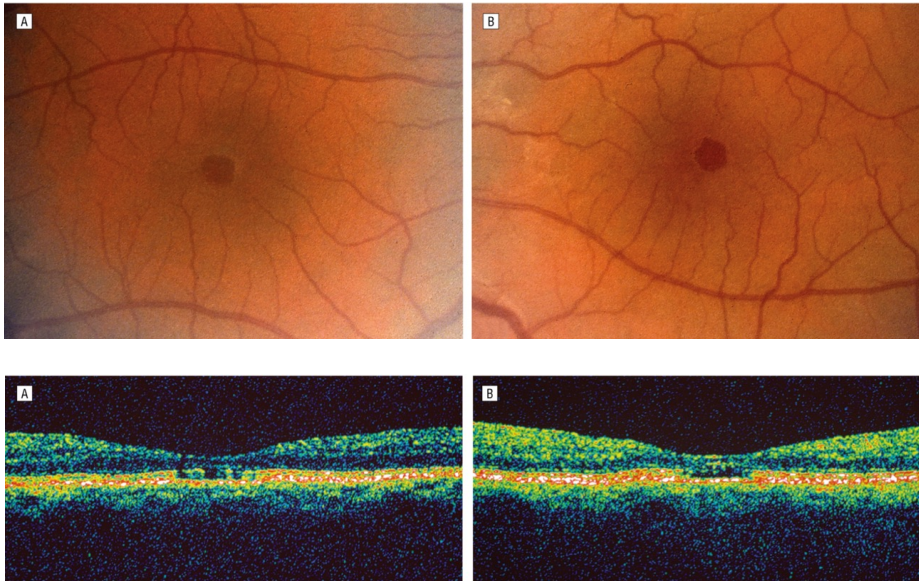
Figure 1: Fundus picture of left eye showing typical yellow white foveolar lesion on presentation.

Figure 2: Fundus picture of right eye showing typical yellow white foveolar lesion on presentation.

(Baisakhiya, et al. 2012)

Case Study (Steinkamp, et al. 2003):

Example of right and left eyes from man who repeatedly stared at sun.



A different case of repeated sun-gazing causing outer retinal (inner and outer segments of foveal cones) damage visualized with 3D OCT (Sheth, et al. 2013)

Notice hyporeflexivity concentrated in outer photoreceptor and RPE layers.

McFaul (1969) lists a series of case studies and treats with systemic steroids for most serious cases of macular edema following eclipse-gazing.

Pathophysiology of Solar Retinitis

A study of an enucleated eye from a patient with a malignant melanoma of the choroid provides some important insights into the retinal damage produced in human eyes by looking directly at the sun (Hope-Ross, et al. 1993). A patient volunteered to expose the retina to solar damage (10 minutes of looking directly at the sun) prior to enucleation. The fluorescein angiogram was normal in appearance with a very subtle but visible lesion in central fundus image.

Post enucleation histology showed dramatic changes in photoreceptors.

Fragmented discs in cone outer segments:

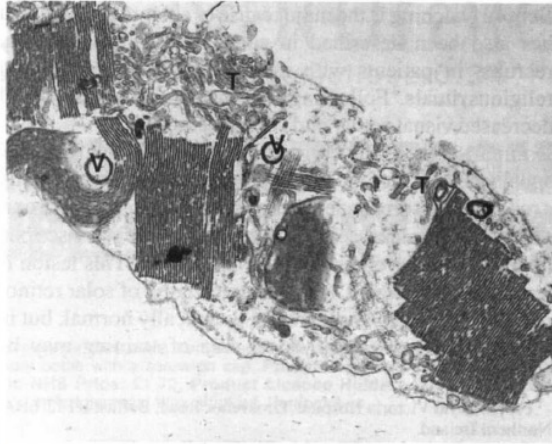


Fig. 4 (b). Cone outer segment at higher magnification shows fragmentation of the disc membranes with vesicular (V) and tubular (T) profiles. $\times 24\ 450$.

(Hope-Ross, et al, 1993)

Swollen Inner segments and swollen mitochondria of photoreceptors

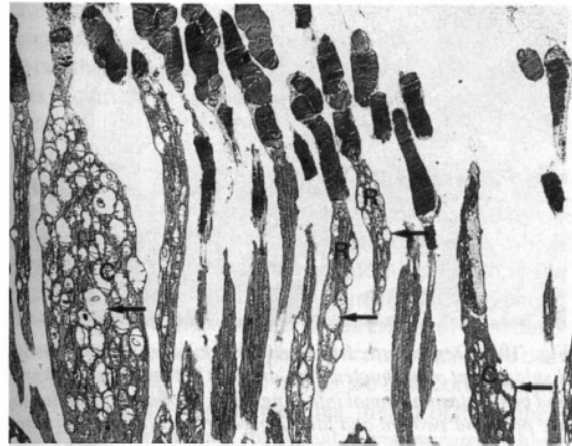


Fig. 6 (a). Inner segments of rods (R) and cones (C) in the parafovea show swollen mitochondria (arrows). $\times 4000$.

These authors also noted many abnormalities in the foveal and parafoveal retinal epithelial cells, including large amounts of lipofuscin, and loss of apical microvilli.

Animal immunolabelling studies showed wound healing responses in the neuroglial cells (Müller cells) during the week after solar exposure of mice retinas (Thanos, et al. 2001).

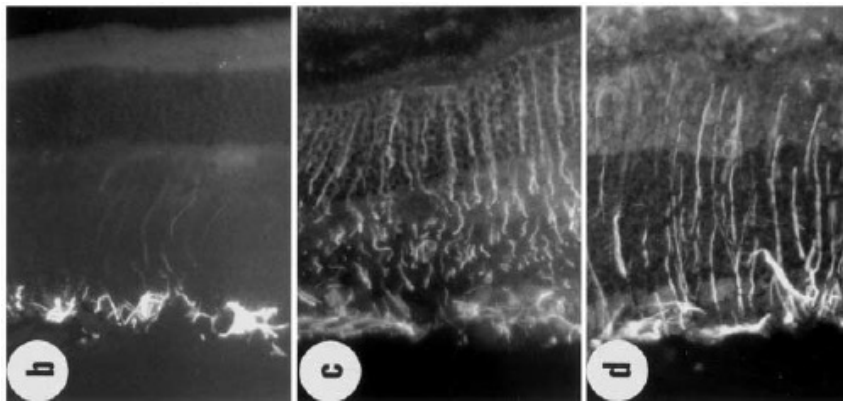


Fig. 4a–h Up-regulation of glial and vascular markers in the eclipse-exposed retina .b Control retina with glial fibrillary acidic protein (GFAP) expression in the astrocytes of the GCL. c, d GFAP is up-regulated in the Müller cells at 1 day post-exposure (c) and is more pronounced 6 days post-exposure (d).

Conclusions

The very optical characteristics of the eye that enable vision, make the retina susceptible to damage from direct viewing of the sun. Typically, humans do not view the sun directly, but a notable exception occurs during a solar eclipse. Attempting to protect the eye with inadequate filtering can exacerbate damage because of the false sense of safety promoting prolonged foveal viewing, and the fact that retinal damage is produced without any accompanying pain. Milder lesions do show clinical signs of recovery, and upregulation of Muller cell repair mechanisms may be responsible for recovery of damage caused by photo-oxidation in the outer retina. Many cases of foveal damage following eclipse viewing never recover fully, but there is some evidence to suggest that control of inflammation with corticosteroids may be an effective treatment strategy for retinal damage produced during eclipse viewing.

Sources of Information on Solar Eclipse Safety

[AAS Solar Eclipse Safety Guidelines](#)

[AAO Eclipse Eye Safety](#)

[NASA's Eclipse Safety Page](#)

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