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Protecting vision through innovation

Science behind the danger of blue light to the retina

By: Shelby Temple, BSc, MSc, PhD

Light is part of the electromagnetic radiation spectrum and, while vital for life on earth and necessary for vision, it carries energy that can be damaging, specifically to the region of the retina called the macula where light is best focussed.

There is a wealth of evidence[1,2,3,4] that it is the blue end of the visible spectrum of light that is most dangerous to the retina. Ultraviolet and infrared are almost entirely filtered out by the lens, cornea and vitreous[5] and therefore do not reach the retina. Short-wavelength, high energy visible, blue light (380 - 500 nm) has enough energy per photon (>2.5 eV) to cause damage to cells and tissues known as photochemical damage [2,3,6]. Photochemical damage typically manifests through the creation of reactive oxygen species and free radicals, which can damage proteins, cell membranes, RNA, DNA and mitochondrial DNA [7,8,9]. The accumulation of damage to DNA and mitochondrial DNA has been linked to the process of aging (free radical theory of aging) [10] and is thought to play a role in the aetiology of age-related macular degeneration[11].

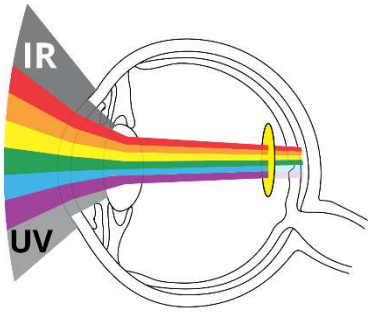

Figure 1 Schematic of the human eye showing how infrared and ultraviolet light are blocked by cornea and lens and violet-blue light is absorbed by macular pigments before it reached the retina.

Research, showing evidence of photochemical damage, has been performed on molecules [12,13,14], cells [15,16,17,18,19,20] and tissues [21,22,23] all the way to whole animals [24,25,26,27,28,29,30] including primates [31,32,33] and humans [34,35,36,37,38]. These studies consistently provide evidence that short-term high-intensity blue light causes photochemical damage, and that blue light is more dangerous than longer wavelengths. Evidence for long-term low intensity (type I) photochemical damage leading to age-related macular degermation (AMD) in humans has been obtained from epidemiological studies [3,39,40,41,42,43,44,45,46,47]. And, while there are some epidemiological studies that have failed to detect a correlation between long-term light exposure and AMD [48,49], it is probable that this is due to the multifactorial nature of aging and large number of uncontrolled variables involved in such studies.

Controlled studies of long-term photochemical damage are not possible in humans due to the length of time required for such studies (>50 years), however, research with monkeys, which have eyes that are structurally identical to our own, has shown that blue light can cause early signs of macular degeneration (lipofuscin and drusen accumulation)[33,50,51,52]. In these controlled studies, the maculae were exposed to greater amounts of blue light through a reduction in macular pigments (lutein, zeaxanthin and meso-xeazanthin), removed by eliminating carotenoids from the monkeys' diet, compared to control groups that had a normal diet. These studies have shown that in the absence of blue light protection, early signs of AMD can occur at half the age (human age equivalent of 35 instead of 65 years of age)[52]. Humans also have macular pigments that are our natural blue light and antioxidant defence system.

The French National Health and Safety Authority (ANSES) released an extensive report in April of 2019 addressing the issue of blue light, specifically in relation to LED lighting and risks of long-term photochemical damage and AMD. In that report, they recognise that the standards set on the intensity of blue light emitted from LED lights is based on Type II (short term) retinal damage and is too high given the potential long-term exposure that many people are experiencing with these commercially available light sources.

The International Organisation for Standardisation (ISO) published a technical report (ISO-TR-20772), in late 2018, as guidance for establishing unified standards against which blue light filtering lenses can be compared. This was done in recognition of the benefits they offer in relation to long-term photochemical damage to the macula by blue light. The report identified 380 - 455 nm as the wavelengths of greatest concern[36]. It represents the first step in a process that was previously undertaken for establishing the UV400 certification for sunglasses. In the ISO report, they identify the strong risk factors for accelerated aging of the macula as age, smoking, low macular pigments and genetics.

The ISO report also recognises the value of allowing the "good" blue light 455 - 500 nm to reach the eye. This is because a part of the blue light spectrum 455 - 500 nm helps entrain our circadian rhythms (sleep/wake cycle) through a pigment called melanopsin, which has a peak photosensitivity between 460 - 480 nm [4,53,54,55]. This is relevant to front of eye methods of filtering blue light reaching the eye (interocular lenses, contact lenses and glasses) because they intervene before light reaches the retina, whereas macular pigments are located in the retina itself and therefore only filter blue light in the macula thus allowing blue light to reach the rest of the retina where melanopsin can be activated.

There is a great deal of misinformation about the dangers of blue light and many sources confuse blue light with light exclusively from electronic devices. As such governing bodies in optometry in the UK (e.g., GOC and AOP) are making statements that are misleading e.g. "*There is no evidence that visible blue light causes eye disease in humans*", which is simply not true (as above). Knowledge about the danger of blue light is hindered by claims that there is a lack of "strong" or "high-quality" evidence to support the contention that blue light causes retinal damage, because people uneducated in the field think that this means that there is "no evidence" that blue light is dangerous (but see above).

It is important to recognise that the lack of evidence does not mean that the statement is untrue, but rather that the type of evidence that these organizations are looking for (e.g., randomized controlled studies in humans) is not yet, and may never be, available. The same argument was made by the tobacco industry for many years until the combination of different types of evidence (exactly like we have now for blue light) were brought together in the US Surgeon General's report in 1964[56]. The question, then, is: did smoking cause cancer before 1964? Of course, it did! Sadly, if governing bodies had responded more quickly to the evidence that had been accumulating for over 30 years, millions of lives could have been saved. We should not risk millions of people losing their vision prematurely because we ignored the evidence we have already.

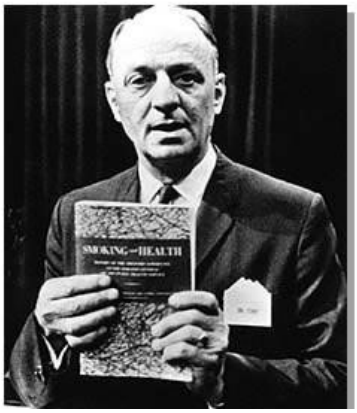

Figure 2 US surgeon general report was not supported by randomized controlled studies in humans, and no such study has ever been done to link smoking and cancer.

For blue light, the type of study expected by some of the governing bodies will never exist because of the nature of age-related macular degeneration (AMD), which is caused by a lifelong accumulation of oxidative photochemical damage due to free radicals (reactive oxygen species). As such a controlled study in humans to prove the efficacy of blue light filtering lenses would require greater than 50 years and control measures that would not be acceptable for a human study. However, there is a great deal of evidence to support the link between blue light and AMD (see citations).

In the absence of a long-term controlled study in humans showing a causative link between blue light and retinal disease, it would be advisable, based on the evidence that does exist, for eye care practitioners to recommend that patients take precautionary action by limiting their exposure (both time and intensity) to short-wavelength violet-blue light. This advice can include limiting time in direct sunlight*, wearing hats*, sunglasses*, ophthalmic lenses that reduce the proportion of blue light, as well as improving diet by consuming more dark and brightly coloured fruits and vegetables* or supplements that contain macular pigments. **These are actions recommended by the Macular Society to help prevent long-term retinal damage.*

I would urge anyone interested in this topic to learn more by reading excellent reviews by Boulton et al. 2001, Tosini et al. 2016 and Young 1988, and looking at Figure 2 in Fletcher et al. 2008 who showed that the odds ratio of getting AMD for people with low macular pigments (our natural protection against blue light) combined low vitamin C and E was nearly as high (2.6-3.7) as the odds ratio for smokers (4.0). This is relevant because only 17% of the UK population smoke, but 25-30% of UK population have low macular pigment levels. There are many other good papers below, and this is just the tip of the iceberg with respect to research in this area.

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As a visual neuroscientist working in fantastic laboratories and research stations in Canada, USA, Mexico, Brazil, Fiji, Australia, Finland, Ireland and the UK, Dr Temple has spent 20 years investigating the molecular and neural basis of vision as well as the impact of light on vision and visual behaviours. His research into sensitivity to polarized light in octopus, cuttlefish and later humans, led to the development of a new clinically relevant method of assessing the density of macular pigments in the human retina. His translation of academic research into a commercial venture saw him receive the prestigious Innovator of the Year award in 2017 awarded from the UK Biotechnology and Biological Sciences Research Council.

Dr Temple is actively involved in developing and delivering webinars and lectures on the dangers of high energy visible (violet-blue) light and free radicals, as well as the importance of macular pigments in protecting the retina from photochemical damage leading to AMD and how to assess macular pigments in the eye.