

Violet and blue light blocking intraocular lenses: photoprotection versus photoreception

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Br J Ophthalmol 2006;90:784–792. doi: 10.1136/bjo.2005.086553

Aim: To analyse how intraocular lens (IOL) chromophores affect retinal photoprotection and the sensitivity of scotopic vision, melanopsin photoreception, and melatonin suppression.

Methods: Transmittance spectra of IOLs, high pass spectral filters, human crystalline lenses, and sunglasses are used with spectral data for acute ultraviolet (UV)-blue photic retinopathy ("blue light hazard" phototoxicity), aphakic scotopic luminous efficiency, melanopsin sensitivity, and melatonin suppression to compute the effect of spectral filters on retinal photoprotection, scotopic sensitivity, and circadian photoentrainment.

Results: Retinal photoprotection increases and photoreception decreases as high pass filters progressively attenuate additional short wavelength light. Violet blocking IOLs reduce retinal exposure to UV (200–400 nm) radiation and violet (400–440 nm) light. Blue blocking IOLs attenuate blue (440–500 nm) and shorter wavelength optical radiation. Blue blocking IOLs theoretically provide better photoprotection but worse photoreception than conventional UV only blocking IOLs. Violet blocking IOLs offer similar UV-blue photoprotection but better scotopic and melanopsin photoreception than blue blocking IOLs. Sunglasses provide roughly 50% more UV-blue photoprotection than either violet or blue blocking IOLs.

Conclusions: Action spectra for most retinal photosensitisers increase or peak in the violet part of the spectrum. Melanopsin, melatonin suppression, and rhodopsin sensitivities are all maximal in the blue part of the spectrum. Scotopic sensitivity and circadian photoentrainment decline with ageing. UV blocking IOLs provide older adults with the best possible rhodopsin and melanopsin sensitivity. Blue and violet blocking IOLs provide less photoprotection than middle aged crystalline lenses, which do not prevent age related macular degeneration (AMD). Thus, pseudophakes should wear sunglasses in bright environments if the unproved phototoxicity-AMD hypothesis is valid.

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Accepted for publication
7 January 2006

Optical radiation includes ultraviolet (UV) radiation (200–400 nm) and visible light (400–700 nm).¹ Violet (400–440 nm) and blue (440–500 nm) light comprise the shorter wavelength part of the visible spectrum.^{2,3} The cornea prevents UV radiation shorter than 300 nm from reaching the retina.⁴ The crystalline lens blocks most UV between 300 nm and 400 nm.^{4–6} Light transmission by the crystalline lens decreases with ageing, particularly at shorter wavelengths.^{4–8} The first poly(methylmethacrylate) intraocular lenses (IOLs) transmitted UV in addition to visible light.⁹ UV does not provide useful vision but it can harm the retina in acute intense exposures.^{9–13} Most IOLs incorporated UV blocking chromophores by 1986.¹⁴

Interest in blocking visible light as well as UV is motivated by the unproved hypothesis that phototoxicity from environmental light exposure can cause or accelerate age related macular degeneration (AMD).^{11,13,15–25} This phototoxicity-AMD hypothesis is popular in part because lipofuscin accumulates with ageing in the retinal pigment epithelium (RPE), perhaps increasing the retinal phototoxicity risks of older adults.^{26–28} None the less, six of the eight major epidemiological studies found no correlation between AMD and lifelong light exposure,^{29–37} caused by (1) its absence, (2) difficulty in accurately estimating a subject's cumulative light exposure retrospectively, (3) variability in genetic susceptibility, or (4) other potentially obfuscating factors such as differences in the age at which subjects experience bright environmental light exposure.

Light can damage the retina by photomechanical, photo-thermal, or photochemical mechanisms.^{1,11,20,38} The two classic types of acute retinal photochemical injuries ("photic retinopathies" or "retinal phototoxicities") can be

distinguished by their action spectra, as shown in figure 1.^{1,3,20,39–42} An action spectrum characterises the variation in potential phototoxicity with wavelength.

The first type of phototoxicity is blue-green ("Noell-type," "class 1," or "white light") photic retinopathy. Its action spectrum is similar to aphakic scotopic sensitivity because rhodopsin mediates both processes. Thus, blue-green phototoxicity hazardousness actually decreases in the blue and violet part of the spectrum below rhodopsin's peak sensitivity around 500 nm (cf, fig 1).^{43–46} Furthermore, any spectral filter that reduces blue-green phototoxicity causes an equivalent percentage decrease in scotopic sensitivity.

The second type of phototoxicity is UV-blue ("Ham-type," "class 2," or "blue light hazard") photic retinopathy.^{10,12,20,25,40,47} As shown in figure 1, its severity increases with decreasing wavelength, similar to that of lipofuscin which is one of its primary mediators.^{23,25,48} Figure 2 illustrates that macular xanthophyll protection declines rapidly in the violet part of the spectrum,^{49–52} where porphyrin and cytochrome oxidase phototoxicity peak.^{23,43,53–58} The weakly phototoxic²⁵ pyridinium bisretinoid A2E component of lipofuscin also has peak phototoxicity around 430 nm in the violet part of the spectrum.^{59,60} Separating acute photic retinopathy into the two preceding categories is useful heuristically, but it may oversimplify phototoxic interactions currently used to study retinal degeneration and cell biology.^{22,57,61–63}

There are three types of retinal photopigments: (1) cone photoreceptor photopigments that provide photopic (bright

Abbreviations: AMD, age related macular degeneration; IOL, intraocular lens; RPE, retinal pigment epithelium; UV, ultraviolet

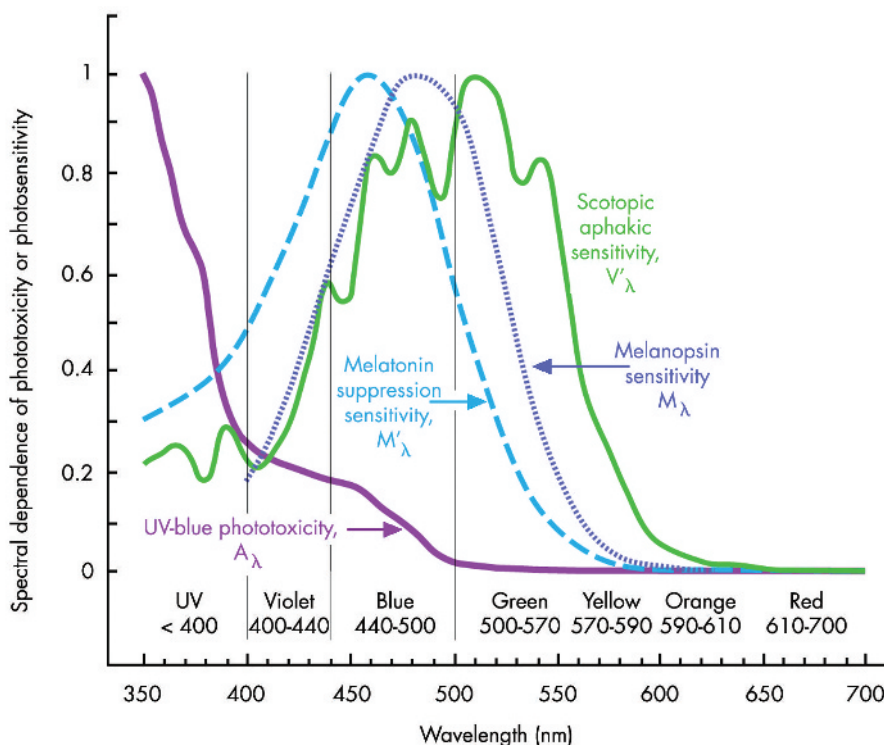


Figure 1 Acute aphakic UV-blue phototoxicity (A_{λ}),^{10 12 73} aphakic scotopic luminous efficiency (V'_{λ}),⁴⁶ melanopsin spectral sensitivity (M_{λ} , peak sensitivity, 479–483 nm),^{68-71 188} and melatonin suppression sensitivity (M'_{λ} , peak sensitivity, 459–464 nm).^{66 67 72} Original rather than smoothed aphakic scotopic luminous efficiency (V'_{λ}) data are shown.⁴⁶ The potential hazardousness of acute UV-blue type phototoxicity increases with decreasing wavelength. Acute blue-green retinal phototoxicity has an action spectrum similar to aphakic scotopic sensitivity because rhodopsin mediates both processes.^{43 44} Melatonin suppression and melanopsin sensitivity are more heavily dependent on blue light than rod (rhodopsin) mediated visual functions.

light) and mesopic (intermediate light) vision,^{64 65} (2) rhodopsin in rod photoreceptors responsible for mesopic and scotopic (dim light) vision,^{45 46} and (3) melanopsin in blue light sensitive retinal ganglion cells that modulate circadian photoentrainment, pupillary function, and possibly conscious vision.⁶⁶⁻⁶⁹ IOLs that block UV and visible light potentially reduce the risk of acute UV-blue phototoxicity.^{3 11 14 42} They also decrease the light

reaching S-cones, light sensitive retinal ganglions, and rod photoreceptors,^{3 42} which have peak spectral sensitivities around 426 nm (violet),^{64 65} 480 nm (blue),⁶⁹⁻⁷¹ and 500 nm (blue-green),^{45 46} respectively. Melanopsin containing light sensitive retinal ganglion cells control circadian photoentrainment through melatonin suppression. Melatonin suppression has a peak sensitivity in human subjects of roughly 460 nm (blue),

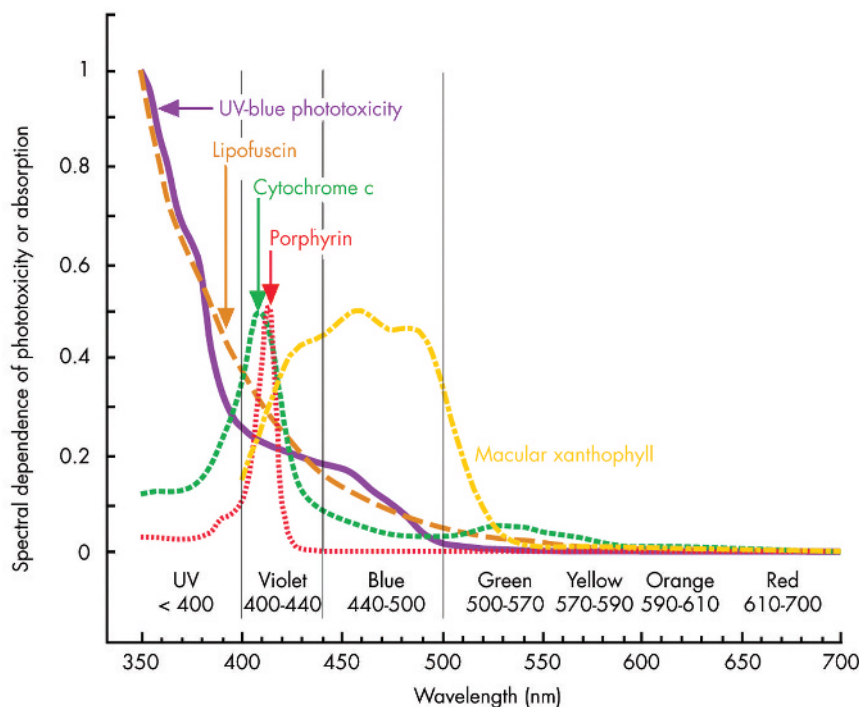


Figure 2 Acute UV-blue^{10 12 73} and lipofuscin^{23 25} phototoxicity rise rapidly in the violet part of the spectrum, where porphyrin^{23 36} and cytochrome oxidase^{23 58} phototoxicities peak and macular xanthophyll protection declines.⁷⁵ Conversely, as shown in figure 1, violet light is much less important than blue light for circadian photoentrainment and vision in dim environments.

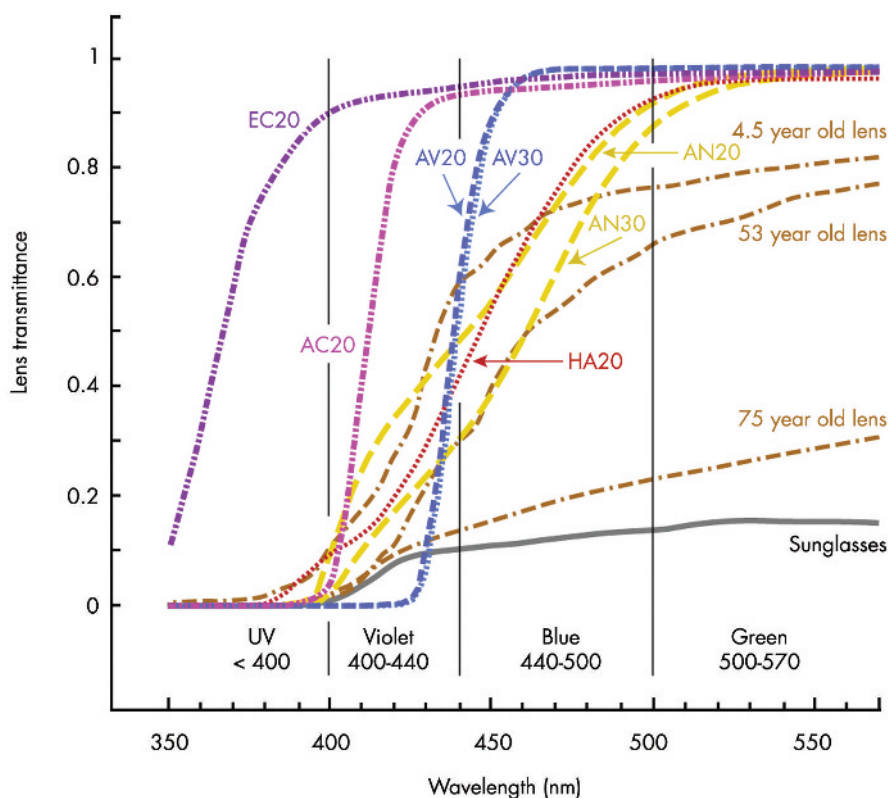


Figure 3 The spectral transmittance of UV transmitting (Eyeonics Crystalens AT-45: EC20), UV only blocking (AMO Clariflex: AC20), violet blocking (AMO OptiBlue: AV20 and AV30), and blue blocking (Alcon AcrySof SN60AT: AN20 and AN30; Hoya AF-1: HA20) IOLs. The number 20 or 30 in each IOL label is the power in dioptres of the IOL tested. Also shown are the spectral transmittance of (1) neutral grey sunglasses (Sunglasses)⁷⁴ and (2) 4.5, 53, and 75 year old crystalline lenses.⁴

approximately 20 nm shorter than the peak sensitivity measured experimentally for the photopigment melanopsin.⁶⁶⁻⁷²

The action spectra for acute experimental blue-green and UV-blue phototoxicities are well characterised, but if there is chronic light damage in humans, its action spectrum is unknown. If the phototoxicity-AMD hypothesis is valid and chronic retinal damage arising from lifelong repetitive acute phototoxic injury does have a significant role in AMD, then action spectra for the two classic types of photic retinopathy can be used to estimate the relative protection afforded by different IOL spectral filters. UV-blue phototoxicity is characterised by the international standard aphakic retinal hazard function (A_λ , fig 1),⁷³ based on Ham's studies of light damage in young primates.¹⁰⁻¹² Blue-green phototoxicity may be specified by the aphakic scotopic sensitivity function governed by rhodopsin light absorption (fig 1).⁴⁶

Retinal photoprotection, scotopic sensitivity, and circadian photoentrainment relative to a conventional UV only blocking IOL were computed for: (1) hypothetical UV + violet blocking high pass filters that have different cut-off wavelengths, (2) UV + violet and UV + violet + blue blocking IOLs, and (3) crystalline lenses of different ages,⁴ using (1) spectral data for acute UV-blue retinal phototoxicity,⁷³ aphakic scotopic luminous efficiency,⁴⁶ melanopsin sensitivity,⁶⁹ and melatonin suppression,⁶⁷ (2) transmittance spectra measured for each IOL, and (3) published data on the spectral transmittance of crystalline lenses⁴ and sunglasses.⁷⁴ The terms "violet blocking" and "blue blocking" will be used for IOLs that attenuate UV + violet and UV + violet + blue light, respectively.

MATERIALS AND METHODS

A Beckman-Coulter DU 800 UV/visible microcomputer controlled spectrophotometer (Beckman-Coulter, Fullerton, CA, USA) was used to measure the spectral transmittance of UV

transmitting (eyeonics Crystalens AT-45), UV only blocking (AMO Clariflex), violet blocking (AMO OptiBlue), and blue blocking (Alcon AcrySof SN60AT and Hoya AF-1) IOLs. Each IOL was aligned in a saline filled cuvette. Transmittance spectra were recorded from 350 nm to 700 nm. Three independent spectral transmittance measurements were performed for three IOLs of each type. Differences in the spectral transmittances of IOLs of the same type were less than 0.2% (spectral bandwidth ≤ 1.8 nm; wavelength repeatability ± 0.1 nm).

Acute aphakic UV-blue phototoxicity (A_λ),⁷³ aphakic scotopic luminous efficiency (V'_λ),⁴⁶ melanopsin spectral sensitivity (M_λ),⁶⁹ and melatonin suppression sensitivity (M'_λ)⁶⁷⁻⁷² were used to estimate the effect of each IOL, crystalline lens, or hypothetical violet blocking filter on phototoxicity or photoreception. Hypothetical high pass filters were used to study how photoprotection and photoreception are affected by blocking optical radiation below 400, 410, 420, 430, and 440 nm. Each hypothetical violet filter blocks all optical radiation below but transmits 99% of the optical radiation above the specified cut-off wavelength.

Areas under the A_λ , V'_λ , M_λ , and M'_λ curves in figure 1 represent total UV-blue phototoxicity, aphakic scotopic sensitivity, melanopsin sensitivity, and melatonin suppression sensitivity, respectively. A_λ , V'_λ , M_λ , and M'_λ were multiplied wavelength by wavelength with the transmittance of each spectral filter to determine how much the filter decreased phototoxicity, scotopic sensitivity, melanopsin photoreception, or melatonin suppression, respectively. Calculations were performed from 350–700 nm for A_λ and V'_λ using an isoquantal spectrum⁴² and from 400–600 nm for M_λ and M'_λ using daylight illumination.⁷² Results are expressed in terms of the percentage difference between the performance of a particular filter and a conventional UV only blocking filter (AMO ClariFlex).

RESULTS

Spectral transmittances measured for IOLs are shown in figure 3, along with classic human crystalline lens transmittance data.⁴ Table 1 presents the computed photoprotection and photoreception of IOLs, crystalline lenses, and hypothetical high pass violet blocking filters, relative to a conventional UV only blocking IOL. Results generally agree with previous calculations,⁴² which used (1) the international standard CIE phakic scotopic sensitivity curve⁷⁵ rather than the aphakic scotopic luminous efficiency data of Griswold and Stark,⁴⁶ and (2) blue blocking IOL spectral transmittance data for measurements made in air rather than saline. Table 1 also presents the percentage of UV, violet, blue, or green light that each filter blocks.

High pass filter calculations in table 1 show that blocking an increasing amount of violet light from 400 nm to 440 nm increases UV-blue and blue-green photoprotection but decreases scotopic, melanopsin, and melatonin suppression sensitivity.

Table 1 also shows that the UV transmitting Crystalens provides 150% less UV-blue photoprotection than a conventional UV only blocking IOL. Violet and blue blocking 20D IOLs offer approximately 40% more UV-blue photoprotection than a UV only blocking IOL. They also provide roughly 50% less UV-blue photoprotection than sunglasses and 20% less protection than a 53 year old crystalline lens.

Blue blocking IOLs offer about 20% better scotopic sensitivity and thus 20% less blue-green phototoxicity protection than a 53 year old crystalline lens. The UV transmitting Crystalens provides 10% more scotopic sensitivity than conventional UV only blocking IOLs. Blue blocking 20D IOLs offer 15% less scotopic sensitivity than a standard IOL. Violet blocking IOLs reduce scotopic sensitivity loss to 7% but provide roughly the same acute UV-blue phototoxicity protection as blue blocking IOLs.

Blue blocking IOLs provide 23% more melanopsin photoreception than a 53 year old lens but 18% less sensitivity than a conventional UV only blocking IOL. Violet blocking IOLs decrease melanopsin photoreception loss to 7% relative to a

UV only blocking IOL. Blue blocking 20D and 30D IOLs provide 27% and 38% less melatonin suppression than a UV only blocking IOL, respectively, whereas violet blocking IOLs decrease melatonin suppression by only 15%–16%.

DISCUSSION

Acute phototoxicity, IOLs, and AMD

The retina balances the production and removal of harmful reactive oxygen species in a hazardous oxidising environment that has high light levels and oxygen concentrations.^{20 21 76 77} Age related increasing accumulation of the photosensitiser lipofuscin may impair free radical control mechanisms and mechanically compromise cellular functions.^{21 23 60 78} AMD, blue-green phototoxicity, and UV-blue phototoxicity all probably involve direct or indirect oxidative damage,^{23 25 76 77} but this shared pathogenesis mechanism does not mean that phototoxicity causes AMD any more than it means that AMD causes phototoxicity. Additionally, both classic phototoxicities involve intense acute rather than lifelong normal light exposures.^{10 12 43 44} These acute exposures can injure the retina but they cannot simulate a lifetime of normal light exposure, just as scalding water can scar skin but it cannot simulate a lifetime of normal bathing.

AMD is a complex multifactorial process involving nutrition, smoking, genetics, and numerous influences other than light exposure.^{18 33 79 80} The relation between chronic light exposure and AMD is difficult to prove because of shared pathogenesis mechanisms, the size and duration of required epidemiological studies, and the difficulty of accurately estimating an individual’s cumulative light exposure retrospectively. Two large population based studies did find a weak association.^{29 36} Four other large studies did not,^{31 33 34 37} including a later study by Taylor³³ who first identified a potential association between light exposure and AMD in the Waterman study.²⁹ Additionally, two large case-control studies failed to show a correlation between AMD and environmental light exposure,^{30 32} one of which actually found that sunlight exposure was higher in the control group than in subjects with AMD.³²

Table 1 Photoprotection and photoreception relative to a UV only blocking IOL (AMO Clariflex 20D): positive and negative percentages indicate better and worse performance, respectively. Percentage of optical radiation blocked in a particular wavelength band

	UV-blue photoprotection	Aphakic scotopic sensitivity*	Melanopsin sensitivity	Melatonin suppression	UV blocked†	Violet blocked†	Blue blocked†	Green blocked†
Eyeonics Crystalens AT-45	-150%	+10%	+4%	+14%	39%	7%	3%	2%
AMO Clariflex 20D	-	-	-	-	100%	34%	5%	3%
Violet 400 nm‡	-33%	+6%	+6%	+11	95%	1%	1%	1%
Violet 410 nm‡	-12%	+4%	+5%	+7	100%	19%	1%	1%
Violet 420 nm‡	+6%	+2%	+3%	+1	100%	44%	1%	1%
Violet 430 nm‡	+23%	-1%	0%	-5%	100%	69%	1%	1%
Violet 440 nm‡	+39%	-5%	-5%	-13%	100%	94%	1%	1%
AMO OptiBlue 20D	+40%	-6%	-7%	-15%	100%	90%	6%	1%
AMO OptiBlue 30D	+42%	-6%	-7%	-16%	100%	92%	6%	1%
Hoya AF-1	+43%	-15%	-18%	-27%	98%	78%	27%	4%
Alcon AcrySof Natural 20D	+40%	-14%	-18%	-27%	99%	67%	27%	3%
Alcon AcrySof Natural 30D	+57%	-21%	-27%	-38%	100%	83%	40%	5%
4.5 year old¶	+35%	-24%	-26%	-30%	97%	69%	30%	21%
53 year old¶	+61%	-37%	-41%	-48%	100%	86%	48%	28%
75 year old¶	+82%	-76%	-78%	-80%	99%	91%	81%	73%
Sunglasses§	+89%	-86%	-86%	-87%	100%	93%	88%	85%

*The percentage loss in aphakic scotopic sensitivity is the same as the percentage gain in blue-green phototoxicity protection (rhodopsin mediates both processes).
 †The percentage of optical radiation in a particular wavelength range that is blocked: UV (350–400 nm), violet (400–440 nm), blue (440–500 nm), and green (500–570 nm).
 ‡Hypothetical violet blocking high pass filters that block all optical radiation below but transmit 99% of radiation above the specified cut-off wavelength.
 ¶Human crystalline lens spectral transmittance data from Boettner and Wolter.⁴
 §Sunglasses spectral transmittance data from Marmor.⁷⁴

The risk of severe AMD has been reported to increase after cataract surgery,^{81–83} but this correlation is confounded by the possibility that cataract surgery may have been performed for decreased vision caused by AMD.^{82–84} Indeed, the AREDS study found no correlation between cataract surgery and AMD after specifically monitoring subjects for their AMD status before cataract surgery.^{84–85} If a correlation between AMD and cataract surgery does exist, it may be the result of the trauma and inflammation of operating microscope procedures and illumination on aged susceptible maculas.^{81–83}

Despite 25 years of use, the evidence documenting the clinical advantage of UV only blocking versus UV transmitting IOLs remains limited. UV only blocking IOLs have been reported to reduce pseudophakic erythroptosis, the transient reddish discoloration of vision that can occur after exposure to a bright outdoor environment.^{86–87} Short wave cone sensitivity was found to be lower in the UV transmitting IOL eye of seven bilateral pseudophakes who had a UV only blocking IOL in their other eye (no retinal abnormalities were observed).⁸⁸ Vitreous fluorophotometry demonstrated less blood-retinal barrier disruption in eyes with UV only or blue blocking IOLs than in those with UV transmitting IOLs.⁸⁹ Early studies suggested that UV only blocking IOLs were associated with a lower risk of postoperative cystoid macular oedema than UV transmitting IOLs.⁹⁰ Later studies failed to confirm that association.⁹¹ No significant difference in the incidence of exudative AMD was found in pseudophakic eyes with or without UV protection.⁹² IOL chromophores have been shown to decrease acute retinal phototoxic damage from intense violet light in cell culture and experimental animal studies.^{93–94}

Table 1 summarises theoretical pseudophakic photoprotection. It shows that violet and blue blocking 20D IOLs provide similar acute UV-blue photoprotection. Photoprotection varies with dioptric power for blue blocking AcrySof Natural IOLs but not violet blocking AMO OptiBlue IOLs. Blue-green photoprotection and scotopic sensitivity are inversely proportional, so violet blocking IOLs offer 8–9% less blue-green photoprotection than 20D blue blocking IOLs, although no IOL provides significant blue-green phototoxicity protection.

Table 1 also shows that sunglasses provide roughly 50% more photoprotection than 20D violet or blue blocking IOLs. Sunglasses have the additional advantage of removability for optimal vision in dim environments. Visible light blocking IOLs provide roughly 20% less UV-blue or blue-green phototoxicity protection than a 53 year old crystalline lens.⁴ Most AMD occurs in people over 60 years of age,⁹⁵ so 53 year old crystalline lenses do not prevent it. Thus, if acute UV-blue phototoxicity (the “blue light hazard”) is a significant risk factor for AMD, then the Boettner and Wolter data⁴ used to design blue blocking IOLs⁹⁶ show that they do not reduce an older adult’s risks, and pseudophakes regardless of IOL type should wear sunglasses in bright environments.

Scotopic sensitivity and IOLs

The human retina contains approximately five million cone and 90 million rod photoreceptors.^{97–98} Rod and cone photoreceptors are responsible primarily for scotopic and photopic vision, respectively. They both provide mesopic vision.⁷⁵ Rod photoreceptors influence cone mediated visual functions even at photopic luminances.^{99–104} Rod photoreceptor populations and sensitivity decrease with ageing, diminishing scotopic sensitivity and other rod mediated visual functions.^{105–108} Pupil diameter also decreases with ageing,^{72–109–111} further reducing available light.

Rod photoreceptor mediated vision is important in modern society. Cone photoreceptors provide information on headlight illuminated roads during night driving, but rod

photoreceptors process the remaining visual field.^{112–113} When you arise at night and lighting is too dim to appreciate colour, you are using rod mediated vision. Aarnisalo demonstrated that filtering blue, in addition to violet, light can reduce scotopic sensitivity.¹¹⁴ Blue light provides 7% of photopic sensitivity and 35% of aphakic scotopic sensitivity. In comparison, violet light provides only 1% of photopic and 10% of aphakic scotopic sensitivity.

Table 1 shows that a UV transmitting Crystalens provides 10% better theoretical scotopic sensitivity than a UV only blocking IOL. Blue blocking 20D and 30D IOLs provide 14% and 21% less scotopic sensitivity than a UV only blocking IOL, respectively, in contrast with the 6% difference with violet blocking IOLs.

Is a 14–21% loss of scotopic sensitivity significant? This loss is difficult to measure clinically and is small in comparison with the broad range of visual sensitivity.^{115–116} None the less, (1) it is a loss, (2) standard static perimetric tests are poor surrogates for night vision tasks such as ambulation and driving, (3) scotopic vision loss is worse in people with AMD and diabetic retinopathy,^{117–121} (4) decreased night vision is well known to be a significant problem for older adults, prompting many to curtail night-time driving and other activities,^{122–127} and (5) impaired dark adaptation increases the risk of falling in older adults.¹²⁸ Forty per cent of people over 65 years of age fall each year,¹²⁹ increasing their risk of debilitating injury, long term hospitalisation, and death.¹³⁰ Additionally, a study by Jackson showed that AcrySof Natural IOL pseudophakes have decreased scotopic vision at violet and blue wavelengths,¹³¹ a type of vision loss correlated with night driving difficulties.¹³²

Circadian rhythmicity and IOLs

Spectral filters also affect circadian rhythmicity. The importance of the predominantly blue light sensitive retinal photopigment melanopsin was not widely recognised until 2002, well after the design of current blue blocking IOLs.^{66–67–133–136} Melanopsin is contained in photosensitive retinal ganglion cells.^{66–67–133–136} These ganglion cells control pineal secretion and suppression of melatonin using signals sent through the retinohypothalamic tract to the master biological clock in the suprachiasmatic nucleus.^{66–69–134–135–137} Blue light is critical in controlling circadian photoentrainment, pupillary response, and the broad range of beneficial systemic effects of endogenous melatonin.^{69–71–134–136–138–143}

Melatonin is a small, lipophilic indoleamine neurohormone that has a pivotal role in circadian rhythmicity. In response to twilight or darkness, the pineal gland secretes melatonin, core body temperatures fall, and sleep ensues.^{134–135–144} In response to bright light, melatonin secretion is suppressed, core body temperatures rise, and there is improved cognition.^{145–148}

Endogenous melatonin is an important factor in systemic homeostasis. It is a potent free radical scavenger¹⁴⁹ that modulates other antioxidants such as superoxide dismutase, catalase, and glutathione peroxidase.^{150–151} It may help protect the RPE against the oxidative stress implicated in AMD.^{152–154} It has numerous anti-cancer effects¹⁵⁵ and limits tumour cell proliferation by inhibiting telomerase.^{156–157} It has anti-inflammatory actions.^{158–159} Stimulation of electron transport and ATP production in the inner mitochondrial membranes by melatonin may affect ageing.¹⁶⁰

Disorganisation of circadian rhythmicity is more common in older adults and people with insomnia,¹⁶¹ depression,^{162–163} coronary artery disease,¹⁶⁴ acute myocardial infarction,¹⁶⁵ bronchial asthma,¹⁶⁶ many cancer types,^{167–169} Alzheimer’s disease,^{170–172} and dementia.¹⁷³ Numerous clinical studies have shown the risks of disturbed circadian photoentrainment^{174–175} and the benefits of optimal rhythmicity.^{176–177}

Age related pupillary miosis and crystalline lens yellowing limit the blue light reaching melanopsin for circadian rhythmicity, reducing older adults' effective retinal light exposure to one tenth that of younger people.⁷² This reduction is probably responsible for the decreased blue light melatonin suppression observed in older adults.¹⁷⁸ Additionally, elderly lifestyles may average half the total daily luminance of young adults.¹⁷⁹ All these factors conspire to weaken the photo-entrainment of older adults' circadian clock. None the less, bright environmental light exposure can restore melatonin levels in older insomniacs to 21 year old control levels, with resolution of their insomnia.¹⁷⁹ Furthermore, insomnia and depression have been shown to decrease after cataract surgery.^{180 181}

Blue light is responsible for 35% of scotopic sensitivity and 53% of melanopsin photoreception. Blue blocking 20D IOLs provide 18% less melanopsin photoreception than a conventional UV only blocking IOL. Mishima found that older adults with insomnia averaged 19% less total daily environmental illuminance than age matched control subjects.¹⁷⁹ Blue blocking IOLs provide 27–38% less melatonin suppression than a UV only blocking IOL, whereas the reduction is 15–16% for a violet blocking IOL.

Colour vision

Scotopic vision depends on a single photoreceptor, but three types of cone photoreceptors mediate normal photopic sensitivity.^{2 182} Normal cone photoreceptor reception and subsequent neural processing provide remarkable constancy of perceived colour, despite illumination changes.^{2 182–184} For example, a red apple appears red both in incandescent and outdoor illumination and the sky appears blue when viewed through different sunglass tints.

The spectral sensitivity of photopic vision is similar in aphakic and phakic patients, despite crystalline lens blockage of shorter wavelength light.¹⁸⁵ Additionally, colour appearance returns largely to normal within a few months of implantation of a UV only blocking IOL, despite the IOL's increased transmittance of shorter wavelength light.¹⁸⁶ Colour disparity problems that required explanation of a blue blocking AcrySof Natural IOL have been reported, however, in an individual with a UV only blocking IOL in their other eye.¹⁸⁷ In general, most individuals readily adjust to vision with visible light blocking IOLs.

CONCLUSION

There is no conclusive clinical or experimental proof that (1) normal light exposure causes AMD, (2) pseudophakes are at increased risk for AMD, or (3) repetitive acute phototoxicity causes AMD. None the less, if IOLs can increase retinal protection without significantly compromising photoreception, people and society should benefit. The phototoxicity-AMD hypothesis remains attractive because RPE lipofuscin concentration increases with ageing, perhaps compromising cellular function and increasing an older adults' risk of photic retinopathy.

UV, violet, and blue light are responsible for 67%, 18%, and 14% of acute UV-blue phototoxicity, respectively, in the spectral region from 350–700 nm where optical radiation can potentially reach the retina of a pseudophakic eye. Lipofuscin phototoxicity increases rapidly and porphyrin, cytochrome oxidase and A2E phototoxicity all peak in the violet part of the spectrum. UV is potentially hazardous and provides no useful vision so it is logical to block it with IOL chromophores. Violet light causes an additional 18% of UV-blue phototoxicity but provides only 10% of aphakic scotopic sensitivity. Thus, if the phototoxicity-AMD hypothesis is valid and UV blue phototoxicity (the "blue-light hazard") does have a significant role in macular ageing, violet blocking IOLs

protect the retina from most potentially phototoxic violet light while transmitting light in the blue part of the spectrum where rhodopsin, melanopsin, and melatonin suppression sensitivities all are maximal. Blue blocking IOLs have spectral transmittances similar to adult crystalline lenses.

Cataract surgery is an older adult's once in a lifetime opportunity to have improved circadian rhythmicity and vision in dim environments. UV only blocking IOLs have provided patients with their best possible scotopic vision and melanopsin photoreception for over a quarter of a century. Visible light blocking IOLs should endeavour to continue this tradition, particularly since the phototoxicity-AMD hypothesis remains unproved and blue light mediated melatonin may actually help protect the RPE from the oxidative stresses probably involved in AMD.

ACKNOWLEDGEMENTS

This research was supported in part by the Kansas Lions Sight Foundation, Inc, Manhattan, KS, USA. The author acknowledges the valuable assistance of Patricia L Turner, MD, Michael D Lowery, PhD, and Alan J Lang, PhD.

Competing interests: MAM serves as a consultant for Advanced Medical Optics, Inc, which manufactures intraocular lenses.

Presented in part at the 2005 Annual Meeting of the American Society of Cataract and Refractive Surgery in Washington DC and the 2005 Annual Meeting of the European Society of Cataract and Refractive Surgery in Lisbon, Portugal.

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