PERSPECTIVE

How much blue light should an IOL transmit?

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Older, and even some modern, intraocular lenses (IOLs) transmit potentially hazardous ultraviolet radiation (UVR) to the retina. In addition, IOLs transmit more blue and green light to the retina for scotopic vision than the crystalline lenses they replace, light that is also potentially hazardous. The severity of UVR-blue type phototoxicity increases with decreasing wavelength, unlike the action spectrum of blue-green type retinal phototoxicity and the luminous efficiency of scotopic vision which both peak in the blue-green part of the optical spectrum around 500 nm. Theoretically, UVR+blue absorbing IOLs provide better retinal protection but worse scotopic sensitivity than UVR-only absorbing IOLs, but further study is needed to test this analysis. UVR is potentially hazardous and not useful for vision, so it is prudent to protect the retina from it with chromophores in IOLs. Determining authoritatively how much blue light an optimal IOL should block requires definitive studies to determine (1) the action spectrum of the retinal phototoxicity potentially involved in human retinal ageing, and (2) the amount of shorter wavelength blue light required for older adults to perform essential activities in dimly lit environments.

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The retina exists in a dangerous environment. Exposure to high concentrations of light and oxygen can damage photoreceptors and retinal pigment epithelial cells.[1–3] Intraretinal defences decline as tissues age.[3–9] Cells can self destruct when chemical triggers are activated.[10–15] Intraocular lenses (IOLs) increase light exposure of an ageing retina as its defences are declining.[16–18] Ageing cannot be stopped but the optical radiation that IOLs transmit can be controlled.

Ultraviolet radiation (UVR) and visible light can cause photic retinopathy, also known as retinal photoxicity or foveomacular retinitis.[19–23] Solar and operating microscope maculopathy are examples of acute retinal phototoxicity. The cornea and crystalline lens help protect the retina from photic retinopathy by preventing UVR from reaching the retina. The cornea blocks UVR with wavelengths below 300 nm.[24–28] The crystalline lens blocks UVR between 300 nm and 400 nm.[24–28] The ageing crystalline lens also blocks potentially phototoxic shorter wavelength blue light.[24 26 29]

Cataract surgery increases the amount of optical radiation that reaches the retina. Intraocular lenses can compromise ocular defences against photic retinopathy, a problem first reported in 1978.[16 17] IOLs with UVR blocking chromophores bonded to optic polymers (UVR-only absorbing IOLs) were introduced in the early 1980s, but even some modern IOLs have inadequate UVR protection.[18 30 31] IOLs that absorb blue as well as UVR radiation (UVR+blue absorbing IOLs) were introduced in the 1990s.[32]

UVR is not useful for human vision, so it makes good sense to use IOL chromophores to prevent it from reaching the retina. How much blue light to block is a more difficult decision, however, because the action spectrum of retinal phototoxicity potentially involved in macular ageing is currently unknown, scotopic vision decreases faster than photopic vision in older adults, and blue light is more important in scotopic than photopic vision.[33–39] In essence, light transmission through an IOL is a trade off between visual performance and protection against retinal phototoxicity. That balance can be quantified theoretically using standard data on scotopic visual sensitivity[40] and retinal phototoxicity.[41–44]

SCOTOPIC VISION AND AGEING

Photopic sensitivity for an eye adapted to bright luminances peaks at 555 nm in the green-yellow part of the spectrum.[40] Scotopic sensitivity for an eye adapted to dim luminances peaks at 506 nm in the blue-green part of the spectrum.[40] The Commission Internationale de l'Eclairage (CIE) standard spectral luminous efficiency functions for photopic and scotopic vision are V_λ and V'_λ, respectively.[40] They are illustrated in Figure 1, which shows that scotopic vision is much more dependent on blue light than photopic vision. The similarity between scotopic luminosity V'_λ and the absorption spectrum for rhodopsin in human rod photoreceptors (which peaks at 498 nm)[45] was a key reason for concluding that rhodopsin mediates scotopic vision.[46] Similarly, the resemblance between the absorption spectrum of rhodopsin and the action spectrum of blue-green type retinal photoxicity suggests that rhodopsin may have an important role in this type of photic retinopathy.[6 19 47 48]

Visual performance decreases with ageing on most sensory tests, even in individuals with normal high contrast visual acuity.[49–53] Ageing has little effect on the number of human foveal cone photoreceptors, but parafoveal rod photoreceptors decrease by 30% with increasing age.[38 54] The mechanisms of this loss remain under investigation, but it probably has a significant role in the declining scotopic vision of older adults.[55 56] Scotopic visual sensitivity decreases twice as fast as photopic sensitivity with increasing age, a loss that contributes to older adults' visual difficulties in dim

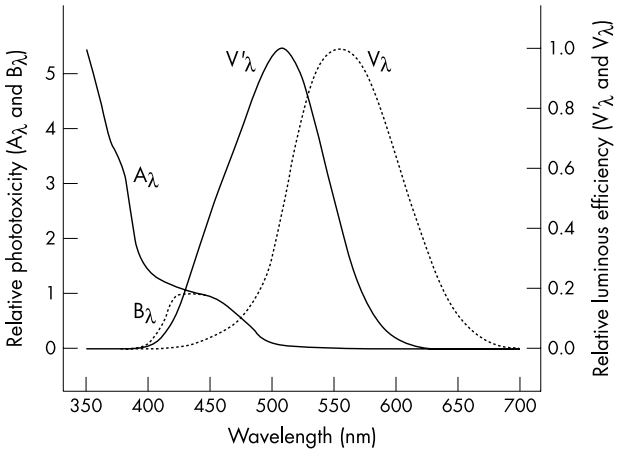

Figure 1 A_λ and B_λ describe how UVR-blue type phototoxicity varies with wavelength in an aphakic and a phakic eye, respectively.[41–43] V'_λ and V_λ are the relative spectral luminous scotopic and photopic efficiencies, respectively, of the standard CIE observer.[40] They characterise how scotopic and photopic visual sensitivity vary with wavelength in a normal phakic eye.[40 170]

environments and that occurs independent of retinal disease or ocular optical problems.[34 36 38] Rod mediated dark adaptation slows progressively with ageing because of delayed rhodopsin regeneration.[34] Scotopic contrast sensitivity at low and high spatial frequencies declines with increasing age.[35] Ageing related loss of scotopic sensitivity is worst in the blue part of the spectrum.[57] Macular degeneration worsens age related decreases in scotopic vision.[37 38 58 59]

Visual difficulties can limit the activities and reduce the quality of life of older adults.[52 60–64] Fear of events such as a hip fracture from a fall can prompt older individuals to limit their activities.[63] Impaired dark adaptation is associated with an increased risk of falling.[65] Older individuals need to be closer to road signs to read them effectively at night.[66] Vision problems may prompt older drivers to curtail their night-time driving activities.[39 52 67–69] In general, visual problems in dim environments increase with ageing, and improved scotopic vision is an appropriate goal for cataract surgery and IOL design. The bottom line is that older adults have increasing problems seeing at night or in dim environments even when they don't have crystalline lens or retinal problems.[49–53]

PHOTIC RETINOPATHY AND AGEING

Photic retinopathy has been studied as an ocular hazard and used as a technique to investigate retinal degeneration and cell biology. The oxygen rich environment of the neural retina and retinal pigment epithelium (RPE) increases their vulnerability to light damage.[48 70–74] Ocular media are the first line of defence against photic retinopathy, but the retina has its own internal defences against phototoxicity, including agents such as superoxide dismutase, catalase, glutathione peroxidase, vitamin E, vitamin C, lutein, and zeaxanthin.[6 71 75–77]

Photic retinopathy has been studied extensively since it was first reported in 1966.[19] It occurs at chorioretinal temperature elevations far too low for retinal photocoagulation.[78–80] Retinal photocoagulation is thermal damage caused by radiant heating of the retina and choroid, whereas photic retinopathy is actinic damage caused by photochemical reactions in the neural retina and/or RPE.

Phototoxicity is accelerated by higher body temperature[19 78 81] and elevated blood oxygen concentration.[1 2] Genetic factors,[82–84] time of day,[85–88] and diet[89–92] all affect the susceptibility of experimental animals to photic retinopathy. Different mechanisms cause phototoxicity in the neural retina and RPE, selective damage at each of these sites being dependent on exposure protocols and animal species.[48 93 94] There is reciprocity between retinal irradiance (power/area) and exposure time, so longer exposures produce threshold phototoxicity at lower irradiances.[48] Retinal phototoxicity is probably additive so that previous exposure increases the risk of subsequent damage.[95]

Retinal defences against photic retinopathy decline with ageing.[3–9] Environmental light exposure has been postulated to be a potential causative factor in macular degeneration for almost a century,[6 17 23 77 96–100] and there are striking similarities in the retinal abnormalities caused by age related macular degeneration and repetitive acute phototoxicity.[17 97 101 102] Unfortunately, epidemiological studies correlating macular degeneration with light exposure are problematical because individual susceptibility varies and lifelong photic exposure is difficult to determine accurately in retrospective studies. Some studies have shown a correlation between macular degeneration and lifelong light exposure, whereas others have not found them to be correlated.[103–110] Additionally, studies correlating cataract surgery with postoperative progression of macular degeneration also have produced conflicting results.[111–119]

An action spectrum characterises the relative effectiveness of different wavelengths in producing a photochemical effect. There are at least two classes of action spectra for retinal phototoxicity.

For lengthy exposures typically shorter than 12 hours in aphakic animals, retinal phototoxicity has an action spectrum that increases with decreasing wavelength, as shown by A_λ (for *a*phakic) in Figure 1.[20 41–43 47 48 120–123] This UVR-blue type of retinal photoxicity has been termed blue light, class 2 or Ham type photic retinopathy. It has also been termed ''blue light'' damage because its action spectrum peaks around 440 nm when a crystalline lens blocks UVR and shorter wavelength blue light, as shown by B_λ (for *b*lue) in Figure 1.[41–43]

For prolonged exposures typically longer that 12 hours, phototoxicity has an action spectrum that peaks in the blue-green part of the spectrum, similar to the absorption spectrum of rhodopsin or that of scotopic luminous efficiency (V'_λ in Fig 1).[19 41–43 47 48 121 124] This blue-green type of retinal phototoxicity has been referred to as white light, class 1, or Noell type photic retinopathy. Blue-green type retinal phototoxicity occurs at substantially lower retinal irradiances than UVR-blue type retinal phototoxicity, but very prolonged exposures are required to produce damage in a single irradiation.[47 48 121]

The photosensitisers responsible for photic retinopathy have not been determined conclusively. Rhodopsin, its photoproducts, or cytochrome-c oxidase in mitochondria may be involved.[19 21 93 125–128] A growing body of evidence suggests that lipofuscin fluorophores—for instance, the pyridinium bisretinoid A2E,[129] may play significant parts in RPE phototoxicity and macular ageing,[15 73 130–137] and that the photoxidative products of A2E are the agents that damage cellular molecules.[136 138 139]

A2E has an excitation maximum of approximately 430 nm,[133 140] a property that may contribute to the susceptibility of RPE to blue light damage in vivo.[20 141] Most of the lipofuscin that is amassed by RPE originates from conjugates generated by visual cycle retinoids in photoreceptor cells,[138 142–144] this material being deposited in RPE cells subsequent to outer segment disc phagocytosis.[145] These retinoid conjugates accumulate because they are not broken down enzymatically. Accordingly, lipofuscin levels in the RPE increase with age,[146–148] and the highest levels are present in macular RPE.[148–151] The role of RPE melanin as a

photosensitiser and/or photoprotective agent in photic retinopathy remains under investigation,[135 152–154] but the presence of melanin is not essential for RPE phototoxicity.[133]

IOL PROTECTION AND PERFORMANCE

IOLs were initially fabricated from poly(methylmethacrylate) (PMMA) without UVR blocking chromophores.[16] The dangers of retinal exposure to near-UVR transmitted by clear PMMA IOLs were recognised in 1978[16 17] and most IOLs had UVR absorbing chromophores by 1986.[18] Unfortunately, UVR protection in contemporary IOLs is inconsistent, and some manufacturers still produce IOLs that transmit potentially phototoxic near-UVR to the retina.[31]

The advantages of UVR-only absorbing IOLs are well documented. UVR-only protective IOLs transmit more blue light than a crystalline lens,[155] but they decrease the incidence of erythropsia[156–159] and blue cone sensitivity loss in pseudophakes.[160] They also decrease blood-retinal barrier disruption in pseudophakes as measured by vitreous fluorophotometry[161] and the risk of retinal phototoxicity in experimental animals.[162 163] UVR+blue absorbing IOLs increase photopic and mesopic contrast sensitivity at intermediate spatial frequencies.[32] UVR-only protective IOLs were reported initially to decrease the risk of angiographically apparent cystoid macular oedema (CMO),[164] but a later study found no such effect in individuals with a ultraviolet protective lens in one eye and a non-ultraviolet-absorbing lens in their other eye.[165]

Blocking UVR with IOL chromophores increases protection from photic retinopathy without decreasing visual sensitivity. It also seemed appropriate in 1986 to use IOL chromophores to decrease the amount of shorter wavelength blue light reaching the retina.[18] Now that a growing body of scientific evidence has demonstrated that ageing related decreases in scotopic sensitivity cannot be attributed solely to optical changes but may involve rod and ganglion cell loss as well as central visual pathway alterations,[34–36 38 166] how much shorter wavelength blue light should be attenuated by IOL chromophores to reduce the potential risk of retinal phototoxicity?

The scotopic luminous efficiency (V'_λ) and aphakic phototoxicity (A_λ) standards shown in Figure 1 can be used to examine how the optical transmittance spectrum of a crystalline or intraocular lens affects scotopic vision and the risk of photic retinopathy. The areas under the V'_λ and A_λ curves in Figure 1 represent total scotopic sensitivity and total aphakic UVR-blue retinal phototoxicity, respectively. If V'_λ and A_λ are convolved with a transmittance spectrum of a particular lens, the percentage difference between the original and convolved areas under the curve represents the percentage loss in scotopic sensitivity or gain in UVR-blue phototoxicity protection from the lens.

Calculations were performed for the five lenses shown in Figure 2, which included two UVR-only absorbing IOLs, one UVR+blue absorbing IOL, and a 53 year old and 75 year old crystalline lens. The results of this analysis are presented in Table 1. As expected, the calculations predict that increasing retinal protection with an IOL decreases its overall scotopic performance and that the UVR+blue absorbing IOL affords better retinal protection but worse scotopic performance than the conventional UVR-only absorbing IOLs. Only clinical studies can determine the potential significance of these theoretical predictions.

The results in Table 1 are subject to numerous limitations: (1) If chronic environmental light exposure does play an important part in macular ageing, it probably affects individuals quite differently depending on unrelated environmental factors such as smoking and on pigmentation and other genetic factors such as the rate at which A2E accumulates in RPE cells, which in turn may be affected by *abcr* gene mutations.[167] (2) V'_λ does describe overall scotopic performance, but it represents the performance of a phakic ''standard CIE observer''[40 168] rather than an aphakic older adult. Psychophysical studies are needed to determine: (a) how much shorter wavelength blue light is needed for older adults to perform essential scotopic tasks in dimly illuminated environments, and (b) whether the shorter wavelength blue light attenuated by a UVR+blue absorbing IOL but transmitted by a UVR-only absorbing IOL can compensate in any significant way for ageing related losses in scotopic sensitivity. (3) A_λ does characterise threshold, acute, UVR-blue type photic retinopathy in experimental animals, but it may differ significantly from the action spectrum of the repetitive or chronic retinal phototoxicity potentially involved in but not conclusively proved to have a significant role in human retinal ageing. (4) Recent threshold studies on primate retinal phototoxicity have found that some of the classic data incorporated into the international A_λ standard

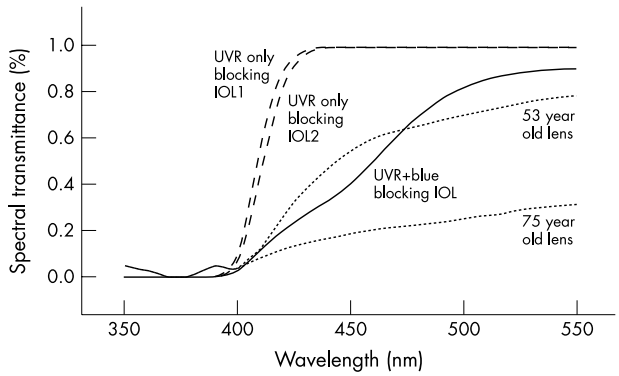

Figure 2 The percentage spectral transmittance of crystalline and intraocular lenses listed in Table 1. Spectral transmittance data on 20D UVR-only absorbing IOL 1 (Alcon AcrySof MA60BM) and 2 (Pharmacia & Upjohn 720A) 20D lenses are from Lin, *et al*.[31] UVR+blue absorbing IOL data (Alcon AcrySof Natural 20D lens) are from Mr Raphael Chan, Alcon Surgical Division, Forth Worth, TX, USA. The 53 and 75 year old crystalline lens transmittance data are from Boettner and Wolter.[24]

Table 1 Theoretical predictions of how several IOLs and human crystalline lenses decrease scotopic visual sensitivity and increase protection from blue-green type photic retinopathy

Lens	% decrease in scotopic visual sensitivity*	% increase in protection from UVR blue type phototoxicity†
75 year old crystalline lens‡	75	93
53 year old crystalline lens‡	33	86
UVR+blue absorbing IOL§	27	90
UVR-only blocking IOL, No 1¶	1.5	75
UVR-only blocking IOL, No 2**	1.6	76

*The percentage difference between the total areas under the V'_λ curve in Figure 1 and that curve convolved with the spectral transmittance of a particular lens.
†The percentage difference between the total areas under the A_λ curve in Figure 1 and that curve convolved with the spectral transmittance of a particular lens.
‡Data on human crystalline lens transmittance are from Boettner and Wolter.[24]
§Alcon acrylic AcrySof Natural 20D lens. IOL transmittance data are from Mr Rafael Chan, Alcon Surgical Division, Forth Worth, TX, USA.
¶Alcon acrylic AcrySof MA60BM 20D lens. IOL transmittance data are from Lin *et al*.[31]
**Pharmacia & Upjohn poly(methylmethacrylate) 720A 20D lens. IOL transmittance data are from Lin *et al*.[31]

may significantly overestimate the UVR-blue type phototoxicity of shorter wavelength blue light.[169] Thus, international phototoxicity standards may change, and results in Table 1 based on A_λ probably significantly overestimate the protection from phototoxicity provided by UVR-only and UVR+blue absorbing IOLs.

DISCUSSION

Cataract surgery removes the crystalline lens which provides optical protection against retinal phototoxicity in an ageing eye. Light absorbing chromophores in an IOL determine which optical wavelengths are transmitted to the retina, balancing retinal protection with visual performance.

An ideal IOL would adapt to changing illumination, transmitting all visible light in dim environments for optimal scotopic performance, but blocking a variable amount of visible light in bright environments depending on an individual's visual requirements and chorioretinal condition. Adaptive photochromic IOLs are not available. The two current choices are UVR-only and UVR+blue absorbing IOLs. In both cases, sunglasses and other forms of ocular protection such as a brimmed hat probably should be worn in very bright environments because of the potential risk of blue-green type retinal phototoxicity.

As shown in Table 1, UVR only blocking IOLs theoretically provide less protection from UVR-blue type phototoxicity than UVR+blue absorbing IOLs. If only the spectral region between 400–550 nm is considered, this protection is roughly a third of that of UVR+blue absorbing IOLs. Conversely, UVR-only blocking IOLs theoretically do not significantly diminish scotopic visual sensitivity. These data predict that UVR+blue absorbing IOLs diminish scotopic visual sensitivity by roughly 25%, but the practical significance of that loss is unknown. The preceding analysis addresses only UVR-blue type retinal phototoxicity, not the blue-green type retinal phototoxicity which has an action spectrum similar to the spectral sensitivity of scotopic vision or the absorption spectrum of rhodopsin. Any increase in protection against blue-green type phototoxicity that an IOL provided would be accompanied by an equivalent percentage decrease in scotopic sensitivity.

One might argue that replacing an ageing crystalline lens with a UVR-only blocking IOL increases the amount of potentially hazardous blue light reaching senescent macular RPE with its increased lipofuscin content, that decreasing blue light even in non-brilliant photopic environments could decrease background UVR-blue type phototoxic damage which might have a role in macular ageing, that shorter wavelength blue light has not been proved to be valuable for essential scotopic visual tasks of older adults after IOL implantation, and that blue light absorbing chromophores in an IOL are always there for some optical radiation protection even in individuals who fail to wear sunglasses in appropriate circumstances.

Conversely, one might argue that UVR-blue type of phototoxicity has not been proved to have a significant role in human macular ageing, that improved blue light transmission might help compensate for visual losses as a result of decreased rod photoreceptor density in ageing, that the hypothetical benefit of avoiding fractures from tripping in dim illumination is more significant than the hypothetical benefit of decreasing the risk of age related macular degeneration, and that it's easier to switch sunglasses than IOLs should future research demonstrate that shorter wavelength blue light is useful for the scotopic vision of older adults.

Neither author has an IOL, but if and when we need one, we would both make sure that it had appropriate UVR blocking chromophores. Based on current information, one of us (MAM) would choose to have a UVR-only blocking IOL that would provide maximal protection against UVR, and wear sunglasses in very bright environments, which could be removed for optimal vision in dim environments. JRS would choose a UVR+blue absorbing IOL that would provide maximal protection against UVR, afford roughly the same protection against phototoxicity and diminution of scotopic sensitivity as a 50 year old crystalline lens, and wear sunglasses in very bright environments, which could be removed for improved vision in dimmer environments. Until photochromic IOLs become available, the decision on which strategy is optimal awaits conclusive data on the role of UVR-blue type retinal phototoxicity in age related macular degeneration and the value of shorter wavelength blue light in essential scotopic activities of older adults.

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