How much blue light should an IOL transmit?

M A Mainster, J R Sparrow

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 authoritative 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.

The retina exists in a dangerous environment. Exposure to high concentrations of light and oxygen can damage photoreceptors and retinal pigment epithelial cells. Intraretinal and extraocular UVR is potentially hazardous and not useful for vision, so it is prudent to protect the retina from it with chromophores in IOLs. Determining authoritative 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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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. 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.

Photic retinopathy has been studied extensively since it was first reported in 1966. It occurs at chorioretinal temperature elevations far too low for retinal photoagulation. Retinal photoagulation 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 and elevated blood oxygen concentration. Genetic factors, time of day, and diet 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. There is reciprocity between retinal irradiance (power/area) and exposure time, so longer exposures produce threshold phototoxicity at lower irradiances. Retinal phototoxicity is probably additive so that previous exposure increases the risk of subsequent damage.

Retinal defences against photic retinopathy decline with ageing. Environmental light exposure has been postulated to be a potential causative factor in macular degeneration for almost a century, and there are striking similarities in the retinal abnormalities caused by age related macular degeneration and repetitive acute phototoxicity. 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. Additionally, studies correlating cataract surgery with postoperative progression of macular degeneration also have produced conflicting results.

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_0 (for aphakic) in Figure 1. This UVR-blue type of retinal phototoxicity 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_0 (for blue) in Figure 1. For prolonged exposures typically longer than 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_s) in Fig 1. 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.

The photosensitisers responsible for photic retinopathy have not been determined conclusively. Rhodopsin, its photoproducts, or cytochrome-c oxidase in mitochondria may be involved. A growing body of evidence suggests that lipofuscin fluorophores—for instance, the pyridinium bisretinoid A_2E, may play significant parts in RPE phototoxicity and macular ageing, and that the oxidative products of A_2E are the agents that damage cellular molecules.

A_2E has an excitation maximum of approximately 430 nm, a property that may contribute to the susceptibility of RPE to blue light damage in vivo. Most of the lipofuscin that is amassed by RPE originates from conjugates generated by visual cycle retinoids in photoreceptor cells. This material being deposited in RPE cells subsequent to outer segment disc phagocytosis. These retinoid conjugates accumulate because they are not broken down enzymatically. Accordingly, lipofuscin levels in the RPE increase with age, and the highest levels are present in macular RPE. The role of RPE melanin as a...
photosensitiser and/or photoprotective agent in photic retinopathy remains under investigation, but the presence of melanin is not essential for RPE phototoxicity.

**IOL PROTECTION AND PERFORMANCE**

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

The advantages of UVR-only absorbing IOLs are well documented. UVR-only protective IOLs transmit more blue light than a crystalline lens, but they decrease the incidence of erythropsia and blue cone sensitivity loss in pseudophakic patients. They also decrease blood-retinal barrier disruption in pseudophakic eyes as measured by vitreous fluorophotometry, and the risk of retinal phototoxicity in experimental animals. UVR+blue absorbing IOLs increase photopic and mesopic contrast sensitivity at intermediate spatial frequencies. UVR-only protective IOLs were reported initially to decrease the risk of angiographically apparent cystoid macular oedema (CMO), 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.

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. 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, 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’) and aphakic phototoxicity (A) 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’ and A curves in Figure 1 represent total scotopic sensitivity and total aphakic UVR-blue retinal phototoxicity, respectively. If V’ and A 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 the Aβ gene mutations. (2) V’ does describe overall scotopic performance, but it represents the performance of a phakic “standard CIE observer” 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 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 standard...
may significantly overestimate the UVR-blue type phototoxicity of shorter wavelength blue light.\textsuperscript{46} Thus, international phototoxicity standards may change, and results in Table I based on $A_2$ 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 choroidal condition. Adaptive photochromic IOLs are not available. The two individual’s visual requirements and chorioretinal condition.

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As shown in Table I, 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 achieved 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 photochrome 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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