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A comparison of blue-light transmissions through blue-control lenses

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Background: Many people are exposed to blue light through devices such as cellular phones, tablets and computers. Such light may affect us depending on its wavelength and blue-control lenses are now frequently used, thus influencing our daily lives.

Aim: This study provides an analysis of the transmissions of blue light through 10 lenses with different blue-control coatings.

Setting: The study was conducted at the Department of Optometry, University of Johannesburg, South Africa.

Methods: Transmission curves of 10 lenses with different blue-control reflective coatings were compared. A control lens (the achromatic lens) was also included. The Cary Varian 5000 photo spectrophotometer from the Department of Physics at the University of Johannesburg was used to measure the spectral transmittances of these lenses with refractive indices ranging from 1.5 to 1.6. The geometric centre of each lens was aligned with the measuring axis of the spectrophotometer and spectral transmittance between 300 nm and 500 nm was measured.

Results: For the 10 lenses studied, the transmission of wavelengths below 460 nm varied from 48% to 69% and for wavelengths between 460 nm and 500 nm from 33% to 55%. The differences between lenses were greater than 20%. If we changed the range of transmission to between 480 nm and 500 nm, the percentage transmitted varied from approximately 71% to 83% to give about a 12% difference between all the lenses.

Conclusion: Not all lenses displayed similar transmissions of blue light and different manufacturers do not agree as to what percentage of blue light should be reflected or transmitted.

Keywords: blue light; transmission; reflective coatings; cumulative curves; normalised cumulative curves.

Introduction

Visible light is that part of the electromagnetic spectrum that ranges from approximately 380 nm to 760 nm in wavelength.[1] Blue light is the radiation that ranges from about 400 nm to 500 nm. Wavelengths ranging from approximately 400 nm to 460 nm are believed to be harmful to the human eye. Recent research has found that light of this band triggers critical physiological responses, including pupil constriction and circadian rhythm synchronisation.[2] Blue light of this wavelength cannot be absorbed by the cornea or crystalline lens and is transmitted directly to the retina. Excessive blue light of 400 nm – 460 nm may cause damage to the crystalline lens proteins and account for cataracts, accelerate the degeneration of retinal pigment cells and increase the acidification of retinal cells.[3] *In vitro* studies done on animals have shown hazardous effects of blue light in ageing eyes through the accumulation of lipofuscin, which is commonly known as 'the age pigment' within the retinal pigment epithelium (RPE). Lipofuscin is stimulated by blue light and makes the retina and the fovea more vulnerable to high-energy blue-light radiation, leading to epithelial cell death.[1,2] As a result, lipofuscin accumulation has been implicated in the pathogenesis of age-related macular degeneration (ARMD) and intense auto-fluorescence is frequently observed in regions surrounding the leading edges of geographic atrophy lesions in the retina.[2]

Other studies have shown that excessive exposure to harmful blue light induces the formation of toxic reactive oxygen species (ROS) that cause photochemical damage resulting in apoptosis of

retinal cells. This leads to the worsening of severe ocular damage such as ARMD.[1,2] Apart from ARMD, blue light is also related to uveal melanoma as wavelengths of 400 nm – 500 nm, specifically in the 425 nm – 475 nm range, can reach the posterior uveal tract with sufficient energy to cause destruction to the components of the eye, such as creating ROS in the mitochondria, resulting in cell dysfunction or cell death. Reactive oxygen species attacks many molecules within the eye. For example, ROS attacks polyunsaturated fatty acids, which form one of the major components of cell membranes. The large concentration of fatty acids in cell membranes in the retina makes the retina highly sensitive to oxidative stress. This stress may disrupt the membranous structures of the photo-receptor outer segments of the retina, causing incomplete phagocytosis and digestion of oxidised outer segments in RPE which may result in the accumulation of the lipofuscin waste product and RPE cell granules.[2] The exact mechanism by which blue light increases the proliferation of uveal melanoma cells is unknown; however, it is known that light of shorter wavelength provokes RPE cell death by ROS production. Blue-light therapy used for neonatal purposes, such as treating jaundice, increases the risk of developing dysplastic nevi in the eyes and skin, and is a further risk factor for melanoma. Therefore, it is imperative for eye protection to be worn to save the neonate from exposure to the lower-frequency blue light. Blue light is also a possible mechanism for tumourigenesis by inducing nuclear deoxyribonucleic acid (DNA) lesions due to the presence of lipofuscin. Blue light is not directly absorbed by the DNA but rather exerts its effect through a photochemical interaction with melanin. These biological effects coincide with UVA (ultraviolet A) wavelengths as they are both capable of producing ROS and causing melanocyte mutation. In addition, blue light also plays a role in neovascularisation, seen as a vital component of developing solid tumours such as uveal melanomas.[4]

Studies suggest that retinal changes associated with age have a significant influence over the potential for photodamage. As the eye ages, light transmission and absorption changes, primarily owing to the gradual yellowing of the crystalline lens. As a result, the ageing lens transmits less visible light with a disproportionate drop in transmission of blue light due to the yellow discolouration of the lens. Early in life, blue light represents 20% of the visible light received by the retina, dropping to approximately 14% at 50 years of age and to about 10% at 70 years. There is a gradual decrease of retinal exposure to blue light with ageing and the natural defenses and repair mechanisms at the retina become less effective; therefore, the ageing retina remains susceptible to photochemical damage from the harmful blue light even as its level of exposure drops. The potential connection between the blue-light phototoxicity and retinal diseases such as ARMD suggests that reducing exposure to the harmful blue light would be beneficial for long-term ocular health.[2]

The rods and cones photoreceptor cells in the retina are totally responsible for light sensitivity. Recent research, however, has shown that some of the ganglion cells may be performing as a third type of photoreceptor called intrinsically photosensitive retinal ganglion cells (ipRGC).[5,6] These sparsely situated cells are most sensitive to blue light. They seem to exist principally to help differentiate between day and night, therefore modulating the sleep/wake cycles, known as circadian rhythms.[7,8]

Not all blue light is destructive as blue light of wavelengths approximately 470 nm – 500 nm is essential for normal visual functions and is believed to have effects on our psychological health and increases our feelings of well-being.[1] Intrinsically photosensitive retinal ganglion cells are believed to exist along with the rods and cones. They form a photoreceptive network broadly within the inner retina, unlike the cone cells which are concentrated at the fovea. These cells are believed to contain melanopsin which is a photo pigment. The ipRGC responds to light in the chronobiological band which regulates many non-visual physiological functions in the human body including circadian entrainment, melatonin regulation, pupillary light reflex, cognitive performance, mood, locomotor activity, memory and body temperature.[2]

Blue light therefore plays an essential role in our daily lives. Filtering out the entire blue spectrum in order to reduce the blue-light hazard (see Figure 1) may interfere with the physiological functions driven by the reaction between ipRGCs and light in the chronobiological band. Other studies have shown that blocking blue light at 470 nm could disrupt the sustained phase of the pupil constriction.[2] Blue light is important because it helps the short-wavelength cone photoreceptors of the eye in colour discrimination and possibly night vision.[1] These photoreceptors have maximum sensitivity under blue stimulation.[9] Several studies have shown that the circadian cycle is primarily dependent on blue light, suggesting that its stimulation to the brain regulates the circadian cycle by inhibiting melatonin secretion.[9]

The exposure to artificial light at night not only influences sleeping patterns, but also causes weight gain, depression, cancer and heart disease.[10] The American Medical Association issued a statement claiming that bright light emitting diodes (LED) lights are causative components of chronic disease risks. The specialised cells in the retina respond to the

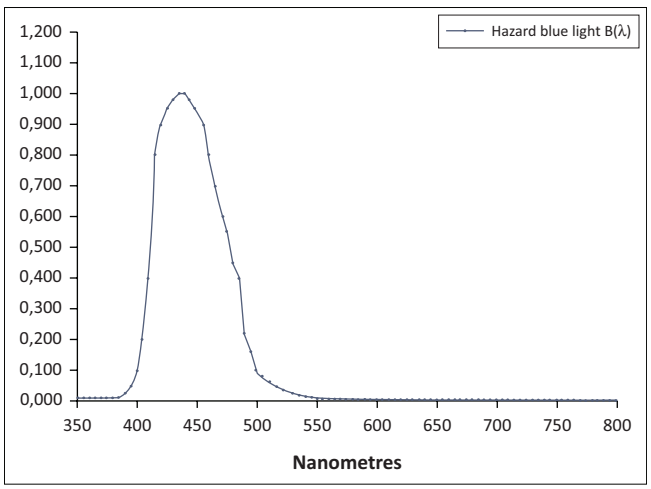

FIGURE 1: The blue-light hazard.

shorter wavelength light that affects the circadian rhythms. As these cells are stimulated, the brain is awoken, decreasing melatonin levels and driving out drowsiness, due to hormones such as cortisol and ghrelin being produced.

These wavelengths range between approximately 380 nm and 500 nm. The blue-light hazard function peaks at about 435 nm – 440 nm. Light emitting diodes (LED) bulbs and various other devices also peak at about the same wavelengths. Therefore, a reduction of these wavelengths is the most efficient way of reducing the potential effects of blue light.

Nowadays, the human eye is potentially exposed to excessive amounts of the harmful, shorter wavelength blue light through digital devices such as tablets, computers, cellular phones, LED bulbs and all other artificial light sources emitting this type of light.[11] However, the level of blue-light exposure from computer screens and other LED-emitting devices is less than the level of blue light exposure from daily light.[1] Nevertheless, blue light from these digital devices suppresses melatonin by 23%. The use of longer wavelength light definitely has less influence than that of 'blue light' and is recommended for use at night.[10] Avoiding blue light for 2 h – 3 h before bed helps the body prepare better for improved sleep.[12] Ayaki et al. state that the reduction of blue light also lessens eye fatigue and dry eye symptoms.[13] Apart from minimising this light, the bright light that we are exposed to during the day should be maximised.[10]

Light emitting diodes (LEDs) are said to produce retinal damage especially if correlated with colour temperatures that exceed 3000 K as they generate a greater short-wavelength energy. Bullough et al. concluded that there was insufficient evidence that there is retinal damage from long-term exposure to LED light as it does not reach the threshold of a blue-light hazard.[14] Apart from the digital devices mentioned, the greatest source of blue light is sunlight. Although exposure to our devices is somewhat less than that from the sun, it is potentially damaging. While too much sun exposure can increase the risk of certain disorders such as cataracts, growths in the eye and cancer, not having enough exposure to the sun is also detrimental. When children do not have enough exposure to sunlight it affects the growth and development of the eye and can increase the risk of myopia.[12]

Potential toxicity of excessive blue light on our vision motivated optical companies to manufacture blue-blocking coatings and filters in ophthalmic lenses and intraocular lenses (IOLs). They are designed to protect our eyes from photochemical damage and alleviate the risk of retinal toxicity by blocking, or weakening, the shorter, harmful blue-light wavelengths. These lenses use filtering materials or surface coatings to reduce the transmittance of these hazardous wavelengths. The Alcon AcrySof Natural Intraocular Lens was one of the first blue-control IOLs developed.[11]

Leung et al. revealed that devices that companies are producing have blue-control coatings that offer 10% reflection of more harmful blue light with shorter wavelength and 90% transmittance of the less harmful longer wavelength blue light.[1] Vimont states that Dr Khurana suggests that the best way to protect one's eyes from eyestrain from the blue light emitted from devices is by using the '20-20-20' rule which implies taking a break every 20 min to look at an object 20 feet away for 20 seconds.[12] Vimont claims that due to the lack of evidence that a blue-control lens works to protect your eyes against blue light, the use of special eyewear is not recommended.[12]

Efforts have been made to develop prophylactic and therapeutic methods to protect retinal cells from phototoxic damage in cataract surgery. A yellow intraocular lens that blocks both UV and blue light (< 500 nm) has been introduced to reduce retinal phototoxicity in pseudophakic eyes; however, the clinical value of these lenses was debatable as they block both hazardous wavelength and those that most effectively activate the ipRGCs.[2] Blue-control coated lenses were designed primarily for protection against the harmful blue light emitted by digital devices for people who spend many hours working in front of screens. These lenses reduce the level of exposure to the harmful portion of the blue-violet spectrum while permitting the rest of the visible spectrum to enter the eye at a normal level. Accordingly, the eyes' necessary visual and non-visual functions can be maintained while exposure to hazardous wavelengths is reduced.[2]

Due to the issues relating to quality control of blue-filtering coated lenses on the market, this research project investigates the blue-light blocking effects that these lenses possess. Further investigations will allow us to determine whether the hazardous blue light is reflected while allowing the transmittance of the good blue light. This will also help us to determine if we agree with the claims and statement of Dr Khurana who recommends that one should try to limit the screen time in the 2 h – 3 h before going to bed.[12]

Methodology

Ten lenses (N = 10) with different blue-control properties were analysed and a control lens (Lens 8 in Table 1) without blue control was used as a reference. The Cary Varian 5000 photo spectrophotometer from the Department of Physics at the University of Johannesburg was used to measure the spectral transmittance of radiation 300 nm – 500 nm through

TABLE 1: Lens parameters (*n* is refractive index). Lens 8, the achromatic lens, was used as a control lens.

Lens number	*n*	Manufacturer	Front surface power (D)
1	1.60	Essilor Preventia	6.00
2	1.50	Hoya Blue Control	5.50
3	1.60	Shamir Blue in mass	6.00
4	1.60	Shamir Blue Shield	5.50
5	1.50	Shamir Blue Glacier	5.00
6	1.56	Kodak in mass	6.00
7	1.56	MR Blue Coat	1.50
8	1.50	Achromatic	6.00
9	1.50	BBGR I Relief	5.00
10	1.60	Zeiss Blue Protect	3.50
11	1.56	GKB Blue Cut	3.00

the lenses. The transmissions were first measured with light entering from the front (convex) surfaces and then repeated through the back surfaces (concave) of each lens. The measurements took approximately 10 min per lens. The geometric centre of each lens was aligned with the instrument's measuring axis. The lens parameters are shown in Table 1 and all lenses were Plano in power.

Data analysis

To perform this analysis we constructed, for each transmittance curve, a new curve called the normalised cumulative integrated transmittance curve (NCITC). Before constructing this curve, we first had to construct a cumulative integrated transmittance curve (CITC) I(λ) that sums the area beneath the transmittance curve from the lowest point on the interval of interest up to the λ of interest,

$$I(\lambda) = \sum_{x \le \lambda}^{n} T(x)dx, \qquad \text{[Eqn 1]}$$

where x is the wavelength of a data point on the transmittance curve $T(x)$, with x less than or equal to λ, and dx is the increment between x and the next wavelength measured.

The total integrated transmittance (TIT) is $I(\lambda_{max})$ where λ_{max} is the largest wavelength on the interval of interest. In that way, we then integrated the amount of light transmitted by each lens on the entire interval of interest. This number is neither an intensity nor a percentage because it carries units that are different from intensity and percentage. We then used this number to compare the transmittance of one lens against any other lens. Using this number, we constructed an NCITC

$$N(\lambda) = \frac{I(\lambda)}{I(\lambda_{max})} \qquad \text{[Eqn 2]}$$

This normalisation re-scales the CITC and preserves its shape. By definition of N(λ), this curve carries no units. Therefore, each point on an NCITC corresponds to a fraction of light transmitted by a specific lens below some wavelength λ. For example, if a point on the curve N(λ) = 0.5 lies above the point on the λ on the x axis below which 50% of all transmitted light is transmitted. Similarly, the point N(λ) = 1.0 lies above a point λ on the x axis below on the interval of interest below which 100% of all wavelengths incident on the lens is transmitted. Therefore, the TIT together with the NCITC characterises the transmittance of a lens and facilitates the direct comparison of lens transmittances.

For a given lens, we construct a 50% transmission band, centred at the expectation value of E[λ]. This is the total integrated weighted transmission divided by total integrated transmission and is calculated by

$$E[\lambda] = \frac{\sum_{k=1}^{n} \lambda_k T(\lambda_k d\lambda_k)}{\sum_{k=1}^{n} T(\lambda_k d\lambda_k)} \qquad \text{[Eqn 3]}$$

The lens transmits on the interval at this average wavelength. We can then construct a 50% transmission band about this point. The band is centred at the 50% point on the normalised cumulative curve. Consider wavelengths α and β such that N(α) = 0.25 and N(β) = 0.75. Then N(β) – N(α) = 0.5. This means that 50% of the total integrated transmission through a lens occurs on the band from α to β. This band contains E[λ]. We call this the 50% transmission band and it characterises the transmission of a given lens on the interval of interest. We consider the position and width of this band among all lenses we included in this study. In particular, we consider the relative positions of E[λ] within each band and the overlap of this band with the 'bad blue light' band (400 nm – 460 nm) and the 'good blue light' band (460 nm – 500 nm) – see Figure 5.

Ethical considerations

This article followed all ethical standards for research without direct contact with human or animal subjects.

Results

According to the results obtained, transmittance values from the front and back surface of all the lenses were approximately equal; therefore, only transmittance for light entering from the front surfaces was considered. Figure 2a, b and c show the transmittance curves for all lenses (N = 11) tested. These curves show the light transmitted for each wavelength independently. The plots show the transmittance of the incident radiation transmitted on a range of wavelengths (300 nm – 500 nm). Figure 3a, b and c show the cumulative plots (from Equation 1) for all lenses, indicating the area of the light transmitted beneath the transmission curves for each lens in Figure 2 from the lowest point on the interval up to 500 nm. This number is neither an intensity nor a percentage because it carries units that are different from intensity and percentage. We use this number to compare the area transmittance of one lens against any other lens. Table 2 shows the area as a number for each lens from the lowest on the transmission curve to 500 nm. The measurements have been categorised from the lowest wavelength to 460 nm and then the total area under the transmittance curve. Wavelengths from where each interval starts up to 460 nm represent the harmful blue light and wavelengths from 460 nm to 500 nm represent the good blue light. Figure 4a, b and c show the normalised cumulative plots (from Equation 2) showing the transmittance of light beneath the transmittance curves in Figure 2 over a range of wavelengths for all 11 lenses. From Equation 3, we can see a 50% transmittance band across two wave bands of light that is transmitted over a range of wavelengths. This is shown in Figure 5.

Consider, for example, an arbitrary curve in Figure 5 for wavelengths α and β such that N(α) = 0.25 and N(β) = 0.75. Then N(β)–N(α) = 0.5. This means that 50% of the total integrated transmittance through a lens occurs on the band from α to β. This band contains E[λ] (see Figure 5).

Discussion

The achromatic lens was used as a control lens as it does not have any blue-control properties, but only an

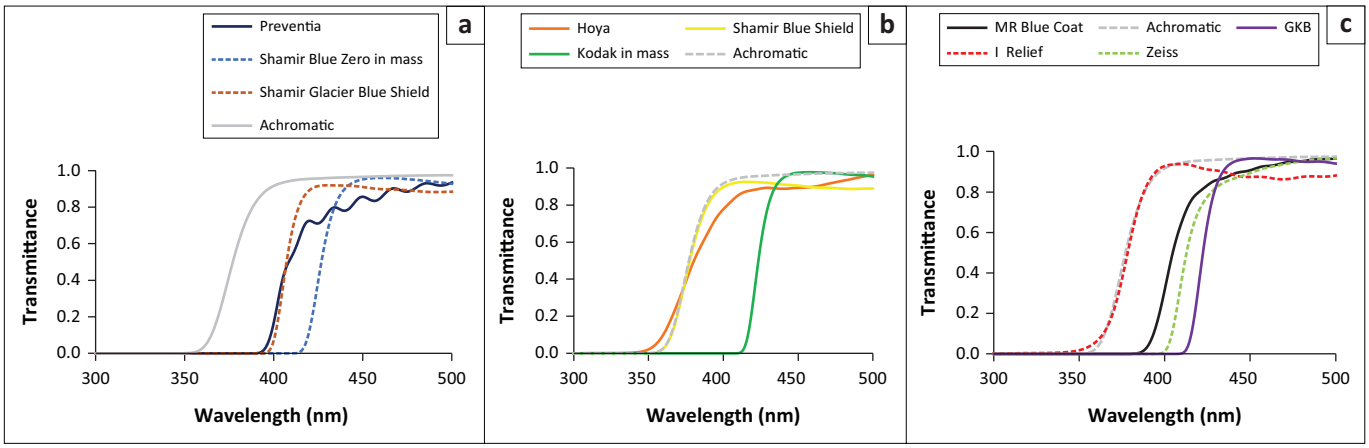

FIGURE 2: Transmittance curves for the 11 lenses (see Table 1) tested. The lenses are identified by their name and colours illustrated in parts a, b and c. The control lens is the achromatic or grey profile or curve. More harmful blue light ranges from 400 nm to 460 nm.

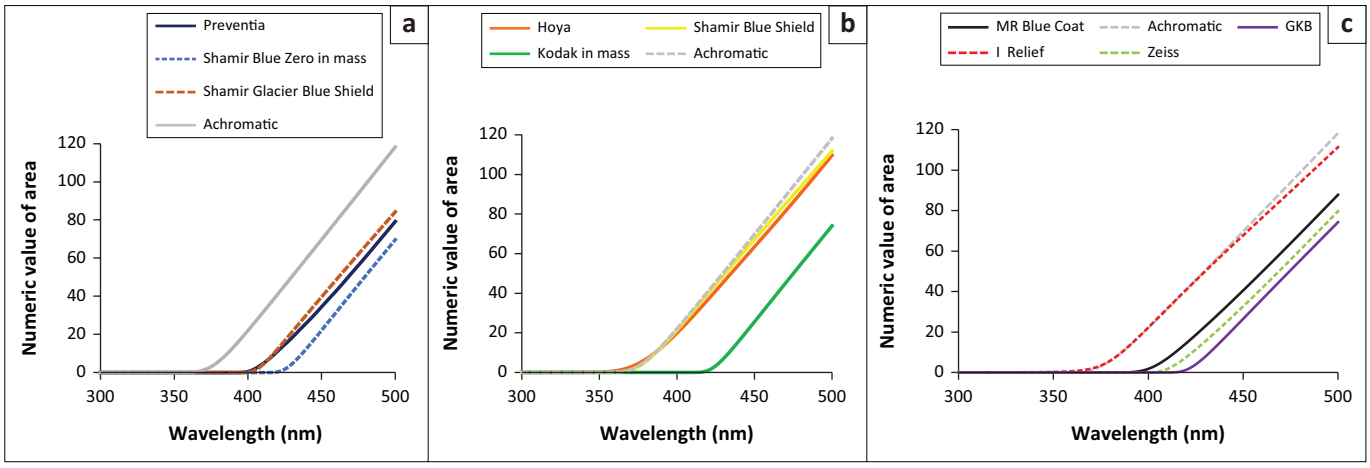

FIGURE 3: Cumulative plots a, b and c for all lenses ($N = 11$) indicated by a number showing the area of the light transmitted beneath the transmittance curves in Figure 3. The achromatic lens (in grey) is the control lens.

TABLE 2: Cumulative plots showing the area of the light transmitted beneath the transmittance curve in Figure 3 indicated by a number.

Type of lens	Wavelength ≤ 460 nm	Total area up to 500 nm
Achromatic (control)	80 (370–460)	118
Shamir Blue shield	74 (400–460)	112
I Relief	55.1 (360–460)	111
Hoya	53.3 (360–460)	110
MR Blue Coat	48 (390–460)	88
Shamir Glacier	49 (400–460)	84
Zeiss Blue Protect	41.7 (400–460)	80
Preventia	43 (395–460)	79
Kodak in mass	35 (414–460)	74
GKB	36 (415–460)	74
Shamir Blue in mass	32 (415–460)	70

Note: The numbers in brackets in the second column categorises the area from least transmittance up to 460 nm and the total area up to 500 nm in column 3.

antireflection coating. When looking at Figure 3 we observe that wavelengths less than 360 nm are not being transmitted by the control lens nor any of the other blue-coated lenses. Hoya, I Relief and achromatic lenses begin their transmissions of light only from wavelengths of about 360 nm – 370 nm. For the other lenses transmissions begin between wavelengths of 390 nm and 415 nm. Blue light ranges from approximately 400 nm to 500 nm with the more harmful being between 400 nm and 460 nm.

Table 2 represents the area of the light transmitted under each curve in Figure 3 and is represented by a number for wavelengths ≤ 460 nm (shorter) to where the transmission starts as well as for wavelengths between 460 nm and 500 nm (longer). The achromatic control lens has the largest area beneath its transmission curve (118) followed by the Shamir Blue Shield (112), i-Relief (111) and then the Hoya Blue Control (110) which all have values greater than 100. Shamir Blue in mass (70) has the smallest area followed by the Kodak and GKB (74). This is followed by Prevencia (79), Zeiss Blue Protect (80), Shamir Glacier (84) and then the MR Blue Coat (88). It can be seen that the lenses showing the smaller areas also have a smaller range. The first number in the brackets in Table 2 represents the shortest wavelength whereby transmission begins.

For wavelengths ≤ 460 nm, from Table 2 we see that the control lens (achromatic) has the largest area for wavelengths ≤ 460 nm (80) followed by Shamir Blue Shield (74). The Shamir Blue in mass has the smallest area (32) followed by the Kodak in mass (35) and then the GKB (36). The rest are then followed by Zeiss Blue Protect (42), Preventia (43), MR Blue Coat (48), Blue Glacier (49), Hoya (53) and then I Relief (55).

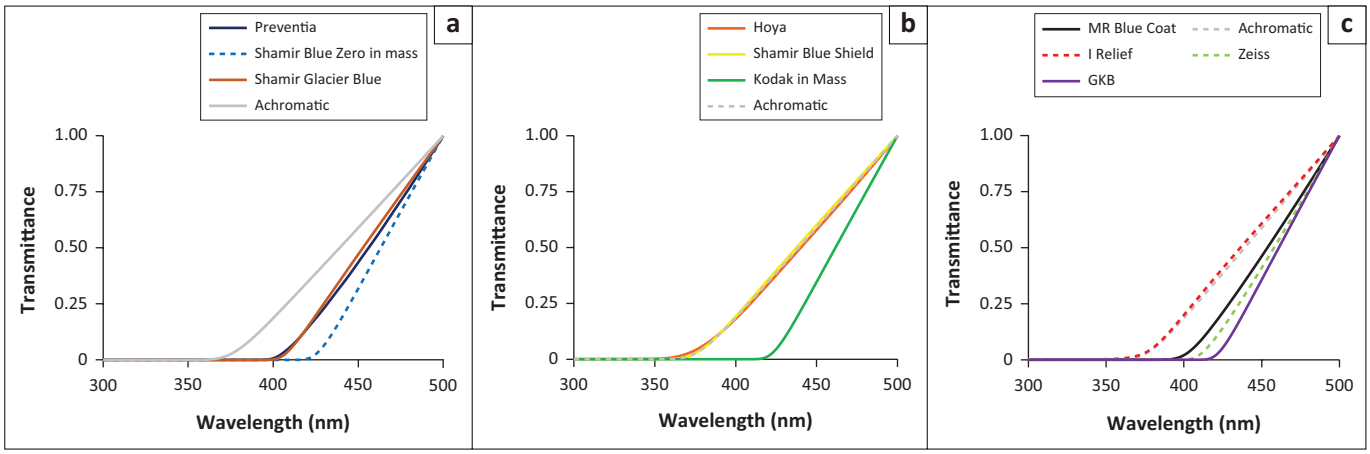

FIGURE 4: Normalised cumulative plots showing the light transmittance beneath the transmissions curve in Figure 3a, b and c over a range of wavelengths for all 11 lenses. The control lens is the achromatic lens showing the area of the light transmitted.

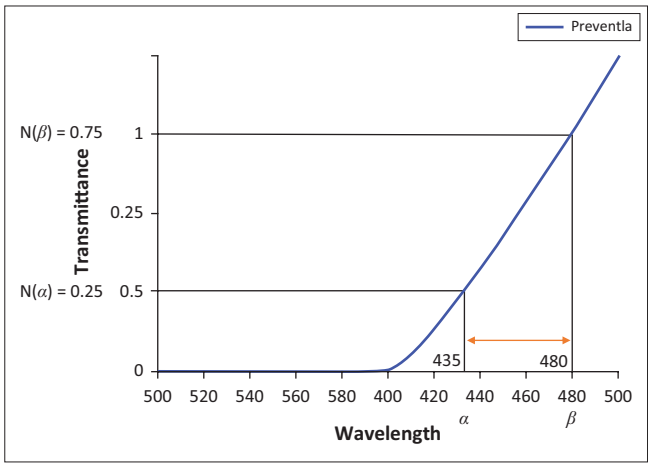

FIGURE 5: Consider wavelengths α and β such that N(α) = 0.25 and N(β) = 0.75. Then this means that 50% of the total integrated transmission through a lens occurs on the band from α to β.

TABLE 3: Normalised cumulative values showing radiation transmitted under the transmittance curves as a factor for each lens in Figure 3.

Lens type	Wavelength ≤ 460 nm	Wavelength 460 nm – 500 nm
I Relief	0.69 (360–460)	0.31
Achromatic (control)	0.67 (370–460)	0.33
Shamir Blue Shield	0.67 (400–460)	0.33
Hoya	0.66 (360–460)	0.34
Shamir Glacier	0.58 (400–460)	0.42
MR Blue Coat	0.57 (390–460)	0.43
Preventia	0.54 (395–460)	0.46
Zeiss Blue Protect	0.52 (400–460)	0.48
GKB	0.48 (415–460)	0.52
Kodak in mass	0.48 (415–460)	0.52
Shamir Blue Zero in mass	0.46 (415–460)	0.54

Note: The numbers in brackets in the second column categorise the range as a factor from least transmittance up to 460 nm and from 360 nm to 500 nm in column 3.

Table 3 represents the transmittance of radiation at wavelengths ≤ 460 nm to where the transmittance starts as well as for wavelengths between 460 nm and 500 nm for each lens type. From Table 3 we see that the control lens (achromatic), Hoya and I Relief transmittance is similar for the shorter and longer wavelengths (0.67, 0.66, 0.69 and 0.33, 0.34 and 0.31 respectively) when compared to the other lens types. These lenses also transmit more UV light as they start transmitting from approximately 360 nm – 370 nm. Shamir Blue Zero in mass, GKB and the Kodak in mass transmit the lowest for the shorter wavelengths (0.46, 0.48 and 0.48 respectively) and the highest for the longer wavelengths (0.54, 0.52 and 0.52 respectively). Preventia (0.54), Glacier Blue (0.58), Zeiss Blue Protect (0.52) and MR Blue Coat (0.57) transmit more for the shorter wavelengths than the in mass lenses, that is, Shamir Blue Zero, GKB and Kodak but transmit less for the longer wavelengths, 0.46, 0.42 and 0.48 respectively. When comparing the achromatic to the Blue Shield, I Relief and Hoya, they all transmit approximately the same amount for the shorter and longer wavelengths (0.67–0.33) respectively. However, only the Blue Shield cuts out all of the UV radiation.

Research done by Tosini et al. stated that exposure to blue light in the range of approximately 400 nm – 470 nm causes damage to photoreceptors and RPE cells.[15] Kuse et al. reported that LED devices emitting light at 456 nm and 553 nm impose more damage to retinal cells, while blue light in the range of 470 nm – 490 nm is essential for physiological functions.[16] Ironically, these wavelengths overlap; however, certain LEDs do emit these wavelengths.

Studies done in London by Lawrenson et al. concluded that there is no significant difference in the improvement of macular health with intervention of blue-blocking control spectacles.[11] They further picked up that these blue-light blocking lenses affect visual performance like colour vision and contrast sensitivity. They also found that there appears to be no significant benefits of these blue-blocking lenses in improving visual performance and protecting macular health from macular degeneration when compared to normal uncoated spectacle lenses. One study, however, reported a small improvement in sleep quality in people with self-reported insomnia after wearing high compared to low blue-blocking lenses. A study involving normal participants found no observed difference in sleep quality. However, some patients wearing blue-coated lenses reported that they provide better anti-glare performance and improve their vision for computer and mobile digital screens. In conclusion, they suggest that blue-coated lenses should serve as a supplementary option for protecting the eye from potentially harmful blue light but do not guarantee a 100% protective effect.[1]

One possible limitation to our study was that we could not assess the effects of these blue-control lenses on participants in order to determine which coating proved most effective. The higher the percentage of transmission for wavelengths shorter than 460 nm, the greater the transmittance of blue light through the coating. This implies that there is more risk of exposure to the hazardous effects of blue light on the human eye, and potentially more risk of destruction of the retinal pigmented epithelium (RPE) which can further cause visual impairment such as ARMD. Laboratory studies have shown that reducing blue-light transmission of wavelengths of 430 nm through a blue-light filter by 50% could reduce approximately 80% of photochemical damage to the retina.[17] ARMD, however, is a multi-factorial eye disease. It has risk factors that include age, smoking, nutritional status, exposure to sunlight and genetic background and cannot be theoretically based on blue-light transmission alone.[18] There appears to be conflicting opinions and results among researchers; therefore, further research needs to be done on this topic.

Conclusion

The transmittance of radiation for wavelengths ≤ 460 nm varied from 46% to 69% and between 460 nm and 500 nm from 31% to 54% among the 11 lenses. The differences appear to be greater than 20%. If we changed the range to between 480 nm and 500 nm, the transmittance varied between approximately 71% and 83%, giving about a 12% difference between all lenses. It appears that the companies differ slightly in opinion as to what percentage of blue light should be transmitted.

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Competing interests

The author has declared that no competing interests exist.

Authors' contributions

I declare that I am the sole author of this research article.

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Data availability statement

Data sharing is not applicable to this article.

Disclaimer

The views and opinions expressed in this article are those of the author and do not necessarily reflect the official policy or position of any affiliated agency of the author.

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