SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

IMPACT OF BLUE LIGHT EXPOSURE ON THE OCULAR STRUCTURES AND ITS PREVENTIVE MEASURES OR MANAGEMENT OPTIONS

Salal Khan[1*], Kamal Pant[2], Jamshed Ali[1], Zainul Abedin[3], Ragni Kumari[1], Sunil Kumar Gupta[1]

[1]Assistant Professor, Department of Optometry (EIAHSR), Era University, Lucknow, U.P (India).
[2]Professor & Head, Department of Optometry, UP University of Medical Sciences, Saifai, U.P (India).
[3]Assistant Professor, Department of Paramedical Sciences (IIAHSR), Integral University, Lucknow, U.P (India).

Corresponding Author: *Salal Khan, Assistant Professor, Department of Optometry (EIAHSR), Era University, Lucknow, U.P (India).ORCID I'd: 0000-0002-1269-3428*

Article DOI: https://doi.org/10.36713/epra20750
DOI No: 10.36713/epra20750

ABSTRACT

To define the risk of blue light exposure to human health, there has been rising research interest. The relatively high energy level of blue light has the potential to cause lasting photochemical damage to eye tissue. Excessive blue light exposure commonly causes the development of dry eye illness, glaucoma, and keratitis, resulting in a variety of alterations such as oxidative stress, mitochondrial apoptosis, inflammatory apoptosis, mitochondrial apoptosis, and DNA damage.

Descriptive research was conducted to examine literature about the blue light hazards that were accessible on PubMed, Google Scholar, Medline, Publon, Orcid, Healthstar, Science Open, Cochrane Library, Paperity, and other databases. Peer-reviewed articles/studies were used to determine available screening tests, preventative strategies, and the impacts of blue light. Some writers stated that excessive blue light exposure can cause a variety of eye disorders and consequences.

However, the therapeutic care of blue light hazards makes considerable use of physical shielding, chemical and pharmacological preventative measures, gene therapy, and other methods. We investigated research on putative blue light-induced signalling routes and processes in the eye, as well as studies on blue light hazard therapy possibilities.

KEYWORDS: *Blue Light Hazard, Preventive Measures, Dry Eye Disease, Blue Filtering Glasses*

1. INTRODUCTION

Blue light is a form of visible light that has a short wavelength between 380 and 500 nm. Because of its tremendous energy, it is quite penetrating. [1]

Various biochemical and physiological changes in the eyes are brought on by blue light exposure.[2] According to previous research, blue light has a high photochemical energy. Prolonged exposure to this high energy light can exacerbate nearsightedness [3-4] and age-related maculopathy, as well as other eye conditions like dry eye and cataracts.[5-9] Blue light can also impair the quality of sleep by disrupting the hormonal balance and preventing the release of melatonin. [10-13]

According to studies, the blue light spectrum between 415 and 455 nm produces the most permanent photochemical retinal damage since it may pass past the lens and directly impact the retina.[14-18] Because of its phototoxicity, high intensity blue light can cause necrosis, but moderate intensity blue light can cause non-necrotic cell death or apoptosis. [19-21] Reactive oxygen species (ROS) and oxidative stress are thought to have a role in the cause of this damage. [22-23]

According to some data, consistent exposure to blue light may have an influence on gene expression in addition to the impacts on eye structure and function. According to genetic research, extended blue light exposure can increase Bax expression while decreasing Bcl-2 and Bcl-xL expression. [24] It has been demonstrated unequivocally that N-retinylidene-N-retinylethanolamine (A2E) is a mediator of blue light damage in the retinal pigment epithelium. Blue light produces a large amount of free radicals, which can damage messenger ribonucleic acid (mRNA) and proteins under aerobic settings. [25-28]

People have recently been exposed to manufactured light sources more due to the development of advanced lighting technology (e.g., LED lights, mobile phones, computers and other devices). [29] This light belongs to the visible blue portion of the spectrum,

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

having a wavelength between 400 and 500 nm. According to research, blue light may impact circadian entrainment, lengthen perceived duration, and increase alertness [30-32], all of which are related to the stimulation of photosensitive retinal ganglion cells (ipRGCs). [33]

Currently, the majority of blue light research focuses on acute light harm caused by high levels of illumination (>1000 lux).[34-36] However, research on the influence of low-illuminance (100 lux) blue light on retinal tissue structure is relatively limited, with the bulk of testing involving exposure to a single source of blue light.[37-39] The irradiance of a point light source disperses with increasing irradiation distance. [40] The irradiance of a light source at a given distance from the vertical centre is less variable, more uniform, and the experimental data are more trustworthy for an area array light source with a large irradiance range in the same plane. [41-42]

In recent years, people have become increasingly aware of the eye pain produced by blue light. Previous research established a good foundation for future eye illness investigations and the creation of blue light-blocking filters.

However, the cellular and molecular pathways underlying blue light-induced retinal damage are not completely understood. These studies indicated that blue light has a major influence on the anatomy and function of the eyes.

1.1 Where are you exposed to blue light?

The most abundant source of blue light is sunshine. In addition, there are numerous additional sources:

- Fluorescent light
- CFL (compact fluorescent light) bulbs
- LED light
- Flat-screen LED televisions
- Computer displays, laptops, smartphones, and tablets.

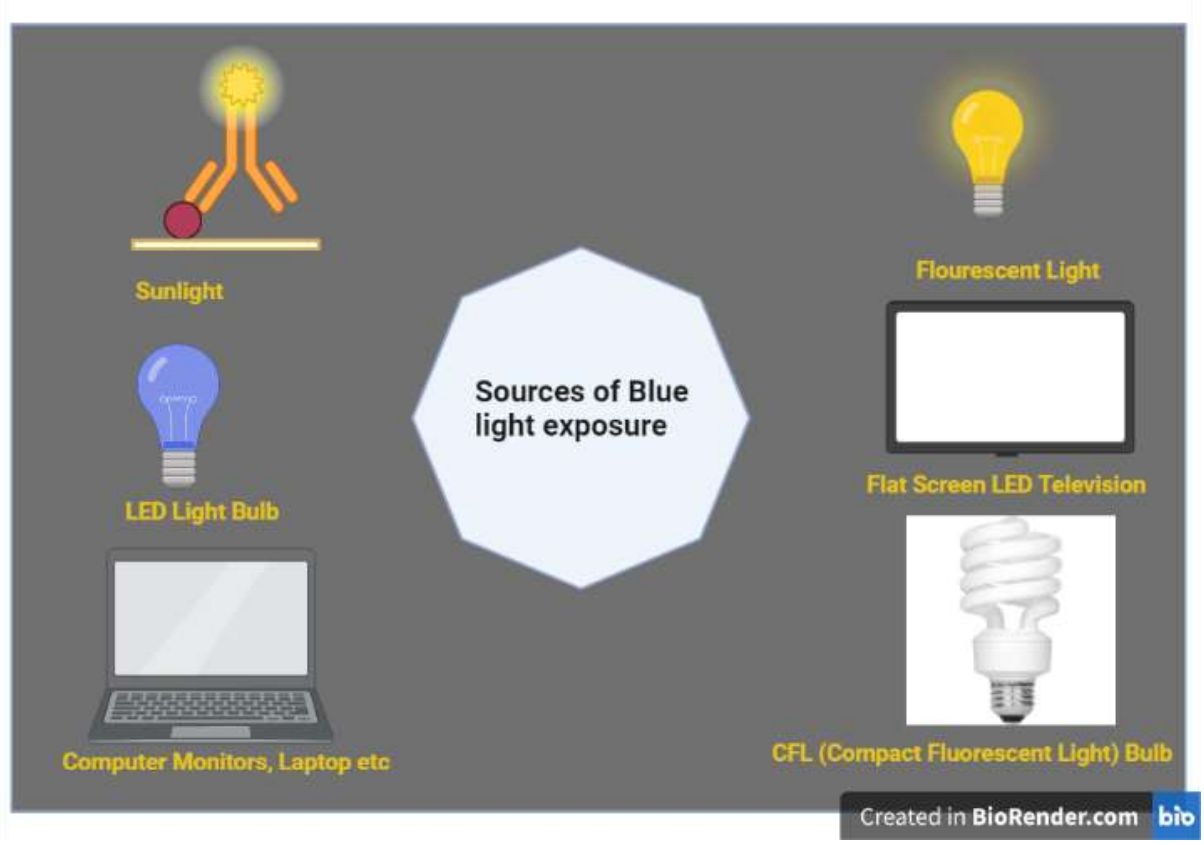


Fig 1: Sources of blue light exposure in our daily lives.

2. EFFECT OF BLUE LIGHT

2.1. Effect of Blue Light on the Ocular Surface

Histologically, the ocular surface includes-

- Tear film,

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

- Corneal epithelial tissue, and
- Conjunctival tissue.

The initial line of defence against irradiant energy is the ocular surface, which is vulnerable to light hazards and abnormalities. Numerous studies have demonstrated that oxidative stress damage, ocular surface inflammation, and cell apoptosis play a role in the process of blue light hazard on the ocular surface (Table 1).

The cornea suffers severe oxidative damage and apoptosis from prolonged exposure to blue light. In addition to raising the rate at which O2 is produced in mitochondria and impairing their ability to function [43], excessive ROS in the cornea also cause the migration of macrophages and inflammatory cytokines. The oxidation byproduct facilitates the NLRP3 inflammasome's activation. The ocular surface is the first line of defence against irradiant radiation and is susceptible to light hazards, which can injure the ocular surface and exacerbate dry eye symptoms [44–45].The cornea absorbs almost all of the radiant light below 295 nm, including all UVC and most UVB.

Aside from the well-known effects of UV light, long-term exposure to blue light with short wavelengths may affect the ocular surface through three major mechanisms: oxidative stress damage, ocular surface inflammation, and cell death [45,46]. These can either exacerbate the ocular phototoxicity of blue light in dry eye patients or contribute to the formation and pathophysiology of dry eyes [46].

Through the P38 and JNK signalling pathways, the active NLRP3 inflammasome hydrolyzes the IL-1 precursor and releases active IL-1, which further stimulates the secretion of IL-6 [47-48]. The development of dry eye illness is linked to the cornea's inflammatory response. When inflammatory substances are released, less tears and mucin are secreted, the tear film becomes more unstable, more tears evaporate, and the ocular surface becomes hyperosmotic [49-51].

Furthermore, it appears that corneal cells are more susceptible to injury than conjunctival cells. The cornea possesses an excellent antioxidative defence mechanism: blue light activates superoxide dismutase (SOD1) in corneal epithelial cells, therefore preserving the cornea. While glutathione peroxidase (GPx1) expression is reduced in conjunctival epithelial cells, SOD1 expression has not altered much, increasing the danger of blue light [48].

Additionally, the conjunctiva is the initial location of eye inflammation because it contains a variety of immune cells and actively contributes to the immunological defence of the ocular surface. Additionally, because the majority of the conjunctiva is exposed to the environment, it is more prone to injury and conjunctivitis. Numerous immune cells that can contribute to the immunological defence of the ocular surface are present in the conjunctiva.

The conjunctiva is so susceptible to inflammation. The data presented above imply that prolonged exposure to short-wavelength blue light can increase oxidative stress, increase the production of inflammatory markers in the cornea and tear film, decrease cell viability, and cause dry eye.

2.1.1 Effects of Blue Light on Cornea

The cornea is the first structure light contacts as it passes through the eye. It is placed at the front of the eyeball.
Blue light have been shown to increase the production of reactive oxygen species (ROS) in corneal epithelial cells, activate the ROS-nucleotide-binding domain, leucine-rich containing family, pyrin-domain containing-3 (NLRP3)-interleukin (IL)-1 signaling pathway, and cause inflammation of human corneal epithelial cells (HCECs) in response to hyperosmotic pressure. Some investigations have demonstrated that the survival rate, induced oxidative damage and apoptosis create further ocular inflammation and xerophthalmia. [52-53]

2.2. Effects of Blue Light on Lens

Lens opacity develops cataracts, one of the leading causes of blindness worldwide. [54] As early as the 1980s, people recognised that the lens could efficiently filter short light waves to reduce the possibility of retinal light damage while yet giving main optical power (measured in diopters). The lens contains structural proteins, enzymes, and protein metabolites that absorb short-wave light. The addition of these compounds and their derivatives to the lens's protein causes the lens to darken and become yellow.

The lens absorbs blue light far more efficiently, reducing potential retinal damage. [55] However, when the lens protects the retina, it may lose transparency or change colour, resulting in cataract formation. As everyone knows, exposure to sunshine is known to increase the risk of acquiring cataracts. According to research, blue light can induce lens epithelial cells (hLECs) to create ROS in their mitochondria, potentially leading to cataract development. [56-57]

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

2.3. Effect of Blue Light on the Retina

The retina, as a light signal sensor, plays an important role in vision development. The two main cell types involved in vision development are photoreceptors (rod and cone cells) and retinal pigment epithelium cells (RPE cells).

The basic function of photoreceptors is to catch light photons and convert them into observable signals. RPE cells, located between the choroid and the top layer of retinal nerve cells, are essential for the development of the eye and various elements of vision, including:

- Growth factor secretion,
- Antioxidant protection,
- Phagocytosis of cell fragments from the outer segment of the photoreceptor,
- Blood-retinal barrier maintenance, and
- Other physiological functions.

Thus, it is crucial for the development of vision to preserve photoreceptor and RPE cell normality. Numerous studies have demonstrated that prolonged exposure to blue light can seriously photochemically harm the retina.

2.3.1 Lipofuscin and blue light hazard

RPE cells' phagocytosis of severed photoreceptor outer segments is crucial for maintaining the retina's physiologically appropriate structure and function.

Undigested membranous discs and lysosomal storage bodies both contain lipofuscin. A2E and its oxidation products are the primary components of lipofuscin. [58-59] A2E exhibits the features of spontaneous fluorescence, which makes it a possible photosensitizer. Blue light (480 nm) exposure was used in research on RPE cells by Sparrow JR et al. They discovered that the exposure caused the A2E-containing RPE cells to die, whereas the non-A2E-containing cells survived.[60]

Furthermore, the blue light hazards were dependent on A2E concentration. The retina's blue light danger mechanisms involving lipofuscin include the following:[61]

(1) The inflammatory response
(2) DNA damage
(3) Damage of mitochondria
(4) Damage of lysosomes

2.3.2. Rhodopsin and Blue Light Hazard

11-cis-retinal and opsin act as chromophores in rod cells, compensating for rhodopsin. Rhodopsin is required in the retina for the development of dark vision as well as light reception. Rhodopsin is now recognized as the chromophore for light damage. Christian Grimm revealed that mice with rhodopsin have significant light hazards when exposed to blue light, but mice lacking rhodopsin are protected against blue light. [62]

Blue light, in a process known as photoreversal of bleaching, can restore activatable rhodopsin and increase its photon-absorbing capacity. Furthermore, blue light-induced rhodopsin localization from the inner and outer segments to ONL activated the transcriptional activator AP-1 in photoreceptor cells, resulting in death. [63-65]

2.3.3. Growth factors and blue light hazard

According to Melanie Marie [66], the photosensitizer A2E activates L-type calcium channels in A2E-loaded RPE cells, which increases VEGF mRNA levels while decreasing PEDF.

The balance of VEGF and PEDF in the retina is critical for maintaining both the permeability of retinal blood vessels and the formation of new ones. PEDF effectively inhibits vascular endothelial growth factor (VEGF) from increasing the proliferation of vascular endothelial cells. [67-68]

The dynamic balance between VEGF and PEDF is disrupted as a result. VEGF activity increases the toxicity of blue light on RPE cells. These data suggest that exposure to blue light causes vascular dysplasia and increased vascular permeability. A pleiotropic growth factor known as hepatocyte growth factor (HGF) can promote the proliferation and migration of a number of ocular cells.

As a result, the RPE and retinal neurons are effectively protected. Blue light exposure lowers HGF mRNA levels in the RPE and retinal neurons, potentially enhancing the toxicity of blue light. [69] As a result, blue light exposure may upset the balance of VEGF

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

and PEDF and decrease HGF production, which has been linked to the development of eye diseases such as diabetic retinopathy (PDR) and glaucoma.

2.3.4. Related genes of blue light hazard

Numerous studies have demonstrated that after exposure to blue light, many genes are differentially regulated. The primary route of blue light harm is the caspase-dependent apoptotic pathway. Caspase-3 and Caspase-9 are activated by LED light exposure. The permeabilization of the lysosomal membrane may be linked to these occurrences. [70] In order to control the mitochondrial apoptosis pathway, Bcl-2 and Bax are essential. The release of cytochrome C is prevented by the anti-apoptotic protein Bcl-2, which is found in the outer membrane of mitochondria. After receiving the apoptotic signal, pro-apoptotic Bax, which is present in the cytoplasm, is sent to the mitochondria to encourage cytochrome C. [71]

Long-term exposure to blue light significantly enhances Bax expression while decreases Bcl-2 expression, which is connected to GADD45 overexpression. Apoptosis is triggered by activating the MKK/JNK and mitochondrial apoptosis pathways, whereas GADD45 overexpression may be mediated via the PI3K/AKT or p53 pathways. [72-73] Furthermore, previous study has demonstrated that after exposure to blue light, the JNK and p38 pathways can remain engaged, phosphorylate c-jun and c-fos, and eventually trigger apoptosis. [71]

2.3.5. Other Mechanisms

RPE cells contain the unsaturated fatty acid docosahexaenoic acid (DHA). The lipid peroxidation of DHA and production of HOHA could be mediated by singlet oxygen and hydrogen peroxide. HOHA often induces cell apoptosis by damaging lysosomes and mitochondria. HOHA is also a predecessor of CEP. Interferon (IFN) and interleukin-17 (Il-17) are produced by CEP-specific T cells as a result of the immunological response to the CEP, which results in cellular inflammation. [74]

Tight connections between choroid and retinal cells form a blood-retinal barrier. The blood-retinal barrier plays a crucial role in the transfer of nutrients, water, and electrolytes. Blue light lowers barrier function by downregulating the scaffold protein zonulae occludentes-1 (zo-1) via the PKC pathway. [71] Furthermore, oxidative stress generated by LED illumination elevated the expression of unfolded protein response genes.

2.4. Effects of Blue Light on Refractive Development

Myopia can be prevented from developing and occurring through outdoor activities, according to epidemiological evidence[75], however there is no clear connection between the lower myopia rate and the quantity and intensity of nearer work. [76]

A recent research looked at the impact of screen reading on schoolchildren's visual acuity. The findings show that screen reading can cause impaired eyesight to appear and develop in children, and that there is a link between increased screen reading time and a higher prevalence of nearsightedness. [77] We discovered that outdoor activities are exposed to natural light, which is higher in short-wave blue light than other artificial light sources, unlike screen reading. According to Rucker et al.'s study [78], sunshine contains significantly more short-wavelength light than the bulk of artificial illuminants, causing the eye length to shrink through the release of retinal dopamine.

Extensive studies has shown that outdoor activities can help reduce the onset and development of myopia [79–83]. Increased time spent indoors can cause myopia, as described following COVID-19 confinement [84-85], due to a lack of exposure to sunshine. Other issues cited were the significant increase in screen time during confinement. Increased computer use in youngsters from the birth cohort study (Generation R) was linked to myopia development [86]. Continuous smartphone usage in teens from the same cohort was related with increased refractive error, particularly in those with minimal outside exposure [87].

2.5. Effects of Blue Light on Circadian Rhythm

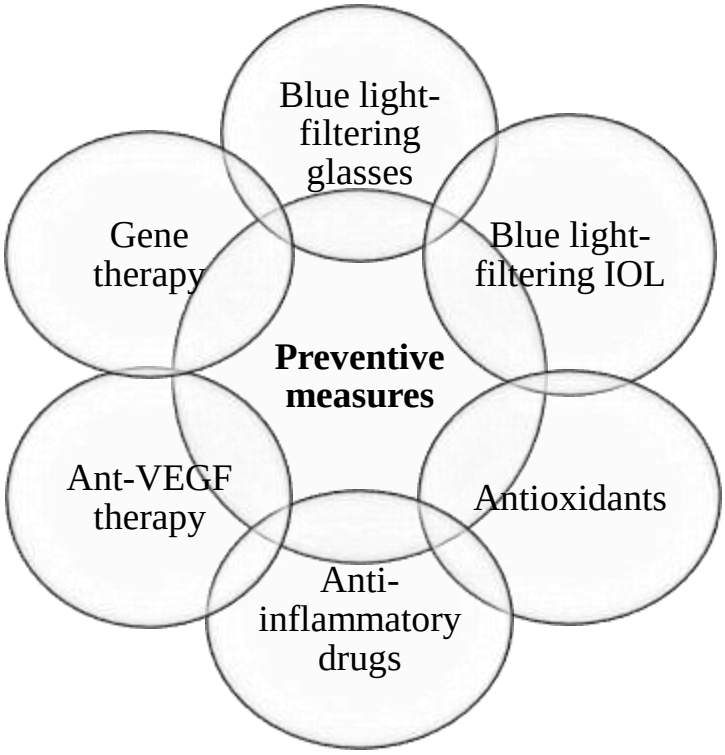
Many studies have shown that blue light can help regulate the biological clock and improve cognition, memory, and alertness. The major reason is that blue light causes the pineal gland to release melatonin, which regulates human circadian rhythm by raising or lowering cortisol expression based on time of day. [88-89] Researchers investigated how well elderly people slept after cataract surgery and determined that they slept better overall. This improvement can be due to transparent artificial crystals, which allow more blue light to enter the eye [90], confirming that blue light can regulate the circadian cycle.

However, if blue light is excessive, particularly at night when melatonin production is highest, it can not only damage the retina via the ocular surface, but it can also stimulate the brain, inhibit melatonin secretion, and increase corticosteroid production, destroying hormonal secretion and directly affecting sleep quality.[91]

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

3. PREVENTIVE MEASURES AGAINST BLUE LIGHT HAZARD

Several ways have been developed to reduce the harm that blue light causes to the eye, based on the pathophysiology of the danger. Physical protection, such as blue light glasses, eyewear shields, and software that limits blue light output, are the traditional blue



light defences. High-energy, short-wave blue light can be filtered to improve vision and avoid harm. Furthermore, several strategies are used in the therapeutic care of blue light danger, such as gene therapy, retinal transplantation, chemical and pharmacological preventive measures, among others.

Fig 2: Preventive Measures of Blue Light Exposure

3.1. Blue Light-Filtering Glasses

To relieve eye strain and weariness while avoiding phototoxic retinal damage, blue-filtering spectacle lenses with varied levels of short wavelength light protection (10% to 100%) are now being pushed to the general public. [92]

Standard eyewear normally protects UV rays with wavelengths up to 380 nm, however adding a yellow chromophore has been proven to limit the transmission of blue light. Furthermore, adding the aforementioned coating to the surface of the front and posterior lenses can selectively filter the blue-violet light spectrum (415-455 nm).[93]

In addition to their retinal protective function, blue-filtering glasses have been proposed to help relieve eye weariness symptoms during intensive computer jobs. [94] Blue light-emitting devices have been related to eye fatigue, namely computer vision syndrome (CVS) and visual display terminal (VDT) syndrome [92]

3.2. Blue Light Filtering Intraocular Lens

Cataracts are the leading cause of vision loss among the elderly. Intraocular lenses are an efficient therapy for cloudy lenses. After cataract surgery, the cloudy lens is removed, allowing more light to reach the retina; nevertheless, this light is more prone to cause blue light risks. Clinical experiments have revealed that blue light-filtering intraocular lenses preserve the retina and retinal fundus of cataract patients while also partially filtering high-energy short-wave light. [95-96]

3.3. Antioxidants

Oxidative stress is a significant contributor to the risk posed by blue light. According to research, antioxidants such as lutein, curcumin, vitamin E, and Prunella vulgaris can help minimise oxidative stress buildup. A2E is produced by free radical scavenging, MAPK pathways, and inflammatory cytokine signalling. Lutein, a common carotenoid, is the predominant pigment in the macular area of the retina. Lutein has been shown to have preventive advantages in retinal disease by raising the density of macular pigment, which can absorb damaging blue light and shield photoreceptors from damage. [97]

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

Anthocyanin pigments, a typical plant pigment, are crucial for improving visual acuity because they lower inflammation and oxidative damage, limit lipid peroxidation, and stop photoreceptor cell activity from being hampered by retinal inflammation. [98-99] Additionally, plant extract has been used as a potential medication to prevent eye problems. The plant Curcuma longa Linn's roots are where you can find the active ingredient known as curcumin. By lowering ROS levels and raising VEGF, GSH, and GSH-Px levels in RPE cells, curcumin has shown to be protective against blue light irradiation.[100]

Prunella vulgaris can prevent retinal degeneration from blue light-induced oxidative stress by inhibiting the Nrf2/HO-1 signalling pathway, NF-B, and inflammatory factors. [89] Furthermore, E2 has a strong anti-neurodegenerative therapeutic effect, owing to its anti-oxidant capabilities. [101] H2O2's cytotoxic impact is reduced, caspase-3 and Bax expression is downregulated, p-AKT and Bcl-2 expression is upregulated, and autophagy is induced.

3.4. Anti-VEGF Therapy

Blue light damage results in an imbalance of PEDF and VEGF, which is linked to diabetic retinopathy. As of now, the only clinical treatment for diabetic retinopathy is the intravitreal injection of an anti-VEGF medication. [102]

According to research, MSC-derived exosomes (MSCExos) are essential for inhibiting VEGF.[103] MSC-Exos transplantation is a novel therapeutic approach with significant benefits, including self-renewal, multidirectional differentiation, and simple sampling.

3.5. Anti-Inflammatory Drugs

Previous research has demonstrated the crucial role that inflammation plays in the pathophysiology of blue light hazard, which raises the possibility that inflammatory factors could be the focus of treatment for eye illnesses. In CCR2-knockout animals exposed to blue light, microglial cell recruitment and activation were reduced. Through lowering ROS levels and preventing caspase3/7 activation, CCR3 inhibition in RPE cells reduced the rate of cell death.

Thus, the treatment of blue light hazard may focus on CCR2 and CCR3. Through the stimulation of MEK/ERK/CREB pathways, silibinin greatly protects RGCs from damage caused by blue light. [104]

3.5. Gene Therapy

Ciliary neurotrophic factor (CNTF) has been found to protect against retinal disorders by inhibiting cell apoptosis, activating Muller cells, and secreting neurotrophic factors. The degeneration and death of retinal photoreceptor cells caused by light can be significantly slowed by injecting CNTF into the vitreous cavity. [105]

When utilised to treat blue light-induced optic nerve damage, the development of bone marrow mesenchymal stem cells containing the CNTF gene has demonstrated high effectiveness. CNTF-BMSCs can increase the antioxidant capacity of the retina by decreasing MDA and VEGF levels, increasing SOD levels, and activating retinal autophagy. After blue light exposure, Panax notoginsenoside saponins (PNS) increased miR155 and SHIP1 expression in the retina, decreased retinal oxidative stress and inflammation, and protected against photoreceptor loss. [106]

4. METHODOLOGY

A descriptive research was conducted to examine literature about the blue light hazards that were accessible on PubMed, Google Scholar, Medline, Publon, Orcid, Healthstar, Science Open, Cochrane Library, Paperity, and other databases.

Peer-reviewed articles/studies were used to determine available screening tests, preventative strategies, and the impacts of blue light. Some writers stated that excessive blue light exposure can cause a variety of eye disorders and consequences.

5. CONCLUSION

Based on evidence indicating that blue light causes ocular diseases, the review summarises how several strategies have been used to protect against the risk of blue light, such as wearing blue light-filtering glasses to block out harmful light or modifying the emission spectrum of smartphones and visual displays to reduce blue light transmission. Injectable anti-VEGF medications and topical or oral antioxidants have also shown promise in improving eyesight. Clinical trials have also shown that blue light-filtering intraocular lenses, neurotrophic factor treatment, and gene therapy were effective.

However, these procedures cannot heal all ailments. As science advances, study into the mechanism of blue light damage will reveal possible treatment targets and continue to enhance visual quality in individuals, with further studies needed to investigate the consequences of blue light.

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

REFERENCES

1. *Lian Y, Lu W, Huang H et al. The Long-Term Effect of Blue-Light Blocking Spectacle Lenses on Adults' Contrast Perception. Front. Neurosci. 2022 Jul 15;16:898489.*
2. *Khan S, Khan S, Md Zakaria Midya et al. Comparison of prevalence data about digital eye strain (DES), pre-lockdown versus post-lockdown period in India: a systematic review study. International Journal of Research and Review. 2021; 8(5): 59-68.*
3. *Kaido M, Toda I, Oobayashi T et al. Reducing short-wavelength blue light in dry eye patients with unstable tear film improves performance on tests of visual acuity. PLoS One 2016;11(4):e0152936.*
4. *Lee HS, Cui L, Li Y et al. Influence of light emitting diode-derived blue light overexposure on mouse ocular surface. PLoS One 2016;11(8):e0161041.*
5. *Krigel A, Berdugo M, Picard E et al. Light-induced retinal damage using different light sources, protocols and rat strains reveals LED phototoxicity. Neuroscience 2016;339:296-307.*
6. *Algvere PV, Marshall J, Seregard S. Age-related maculopathy and the impact of blue light hazard. Acta Ophthalmol Scand 2006;84(1):4-15.*
7. *Zhao ZC, Zhou Y, Tan G et al. Research progress about the effect and prevention of blue light on eyes. Int J Ophthalmol 2018;11(12):1999-2003.*
8. *Niwano Y, Kanno T, Iwasawa A et al. Blue light injures corneal epithelial cells in the mitotic phase in vitro. Br J Ophthalmol 2014;98(7):990-992.*
9. *Rozanowska M, Jarvis-Evans J, Korytowski W et al. Blue light-induced reactivity of retinal age pigment. In vitro generation of oxygen-reactive species. J Biol Chem 1995;270(32): 18825-30.*
10. *Lee SI, Matsumori K, Nishimura K et al. Melatonin suppression and sleepiness in children exposed to blue-enriched white LED lighting at night. Physiol Rep 2018;6(24):e13942.*
11. *Motamedzadeh M, Golmohammadi R, Kazemi R et al. The effect of blue-enriched white light on cognitive performances and sleepiness of night-shift workers: a field study. Physiol Behav 2017;177:208-14.*
12. *van der Maren S, Moderie C, Duclos C et al. Daily profiles of light exposure and evening use of light-emitting devices in young adults complaining of a delayed sleep schedule. J Biol Rhythm 2018;33(2):192-202.*
13. *West KE, Jablonski MR, Warfield B et al. Blue light from light-emitting diodes elicits a dose-dependent suppression of melatonin in humans. J Appl Physiol 2011;110(3):619-626.*
14. *Wu JM, Seregard S, Algvere PV. Photochemical damage of the retina. Surv Ophthalmol 2006;51(5):461-81.*
15. *Tosini G, Ferguson I, Tsubota K. Effects of blue light on the circadian system and eye physiology. Mol Vis 2016;22:61-72.*
16. *Narimatsu T, Ozawa Y, Miyake S et al. Biological effects of blocking blue and other visible light on the mouse retina. Clin Exp Ophthalmol 2014;42(6):555-63.*
17. *Ratnayake K, Payton JL, Lakmal OH et al. Blue light excited retinal intercepts cellular signaling. Sci Rep 2018;8(1):10207.*
18. *Vicente-Tejedor J, Marchena M, Ramírez L et al. Removal of the blue component of light significantly decreases retinal damage after high intensity exposure. PLoS One 2018;13(3):e0194218.*
19. *Pang J, Seko Y, Tokoro T. Processes of blue light-induced damage to retinal pigment epithelial cells lacking phagosomes. Jpn J Ophthalmol 1999;43(2):103-108.*
20. *Li JY, Zhang K, Xu D et al. Mitochondrial fission is required for blue light-induced apoptosis and mitophagy in retinal neuronal R28 cells. Front Mol Neurosci 2018;11:432.*
21. *Moon J, Yun J, Yoon YD, et al. Blue light effect on retinal pigment epithelial cells by display devices. Integr Biol (Camb) 2017;9(5):436-443.*
22. *Marek V, Melik-Parsadaniantz S, Villette T et al. Blue light phototoxicity toward human corneal and conjunctival epithelial cells in basal and hyperosmolar conditions. Free Radic Biol Med 2018;126:27-40.*
23. *Yan GG, Zhang L, Feng C, et al. Blue light emitting diodes irradiation causes cell death in colorectal cancer by inducing ROS production and DNA damage. Int J Biochem Cell Biol 2018;103:81-88.*
24. *Lin CH, Wu MR, Li CH et al. Editor's highlight: periodic exposure to smartphone-mimic low-luminance blue light induces retina damage through bcl-2/BAX-dependent apoptosis. Toxicol Sci 2017;157(1):196-210.*
25. *Sparrow JR, Zhou JL, Cai BL. DNA is a target of the photodynamic effects elicited in A2E-laden RPE by blue-light illumination. Invest Ophthalmol Vis Sci 2003;44(5):2245-51.*
26. *Sparrow JR, Nakanishi K, Parish CA. The lipofuscin fluorophore A2E mediates blue light-induced damage to retinal pigmented epithelial cells. Invest Ophthalmol Vis Sci 2000;41(7):1981-89.*
27. *Sparrow JR, Cai B. Blue light-induced apoptosis of A2E-containing RPE: involvement of caspase-3 and protection by Bcl-2. Invest Ophthalmol Vis Sci 2001;42(6):1356-62.*
28. *Sparrow JR, Zhou JL, Ben-Shabat S et al. Involvement of oxidative mechanisms in blue-light-induced damage to A2E-laden RPE. Invest Ophthalmol Vis Sci 2002;43(4):1222-27.*
29. *D.H. Sliney, What is light? The visible spectrum and beyond, Eye (Lond) 30 (2) (2016) 222–29.*
30. *S.W. Lockley, E.E. Evans, F.A. Scheer et al. Short-wavelength sensitivity for the direct effects of light on alertness, vigilance, and the waking electroencephalogram in humans, Sleep. 29 (2) (2006) 161–68.*
31. *K. Dong, E.C. Goyarts, E. Pelle et al. Blue light disrupts the circadian rhythm and create damage in skin cells, Int J Cosmet Sci. 41 (6) (2019) 558–62.*
32. *C. Blume, C. Garbazza, M. Spitschan, Effects of light on human circadian rhythms, sleep and mood, Somnologie (Berl) 23 (3) (2019) 147–56.*

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

33. *P.L. Yang, S.I. Tsujimura A et al. Subjective time expansion with increased stimulation of intrinsically photosensitive retinal ganglion cells, Sci Rep. 8 (1) (2018) 11693.*
34. *Krigel, M. Berdugo, E. Picard et al., "Light-induced retinal damage using different light sources, protocols and rat strains reveals LED phototoxicity," Neuroscience, vol. 339, pp. 296– 307, 2016.*
35. *J. Vicente-Tejedor, M. Marchena, L. Ramírez et al., "Removal of the blue component of light significantly decreases retinal damage after high intensity exposure," PLoS One, vol. 13, no. 3, article e0194218, 2018.*
36. *D. Yuan, R. D. Collage, H. Huang et al., "Blue light reduces organ injury from ischemia and reperfusion," Proceedings of the National Academy of Sciences of the United States of America, vol. 113, no. 19, pp. 5239–44, 2016.*
37. *B. J. Putting, J. A. van Best, G. F. J. M. Vrensen et al. "Blue-light-induced dysfunction of the blood-retinal barrier at the pigment epithelium in albino versus pigmented rabbits," Experimental Eye Research, vol. 58, no. 1, pp. 31–40, 1994.*
38. *B. J. Putting, R. C. Zweypfenning, G. F. Vrensen et al. "Blood-retinal barrier dysfunction at the pigment epithelium induced by blue light," Investigative Ophthalmology & Visual Science, vol. 33, no. 12, pp. 3385–93, 1992.*
39. *M. Nakamura, T. Yako, Y. Kuse et al., "Exposure to excessive blue LED light damages retinal pigment epithelium and photoreceptors of pigmented mice," Experimental Eye Research, vol. 177, pp. 1–11, 2018.*
40. *S. Karaoglu, Y. Liu, T. Gevers et al. "Point light source position estimation from RGB-D images by learning surface attributes," IEEE Transactions on Image Processing, vol. 26, no. 11, pp. 5149–59, 2017.*
41. *H. M. Kim, M. S. Kim, G. J. Lee et al. "Large area fabrication of engineered microlens array with low sag height for light-field imaging," Optics Express, vol. 27, no. 4, pp. 4435–44, 2019.*
42. *Z. Zhu, M. Jiang, H. Yu et al. "Generation of blue light by sum-frequency generation of a spectrally combined broad-area diode laser array," Optics Letters, vol. 41, no. 20, pp. 4712–14, 2016.*
43. *Y. Niwano, A. Iwasawa, K. Tsubota, et al., Protective effects of blue light-blocking shades on phototoxicity in human ocular surface cells, BMJ open ophthalmology 4 (1) (2019) e000217.*
44. *Behar-Cohen F, Martinsons C, Vienot F et al. Light-emitting diodes (LED) for domestic lighting: any risks for the eye? Prog Retin Eye Res. 2011;30(4):239–57.*
45. *Scientific Committee on Health, Environmental and Emerging Risks (SCHEER). Opinion on Potential risks to human health of Light Emitting Diodes (LEDs). 5–6 June 2018: SCHEER; 2018.*
46. *Ouyang X, Yang J, Hong Z et al. Mechanisms of blue light-induced eye hazard and protective measures: a review. Biomed Pharmacother. 2020;130:110577.*
47. *V. Marek, S. Melik-Parsadaniantz, T. Villette, et al., Blue light phototoxicity toward human corneal and conjunctival epithelial cells in basal and hyperosmolar conditions, Free Radical Biology and Medicine. 126 (2018) 27–40.*
48. *T. Yamaguchi. Inflammatory Response in Dry Eye, Invest Ophthalmol Vis Sci. 59 (14) (2018) 192–99.*
49. *J.B. Lee, S.H. Kim, S.C. Lee, et al., Blue Light–Induced Oxidative Stress in Human Corneal Epithelial Cells: Protective Effects of Ethanol Extracts of Various MedicinalPlant Mixtures, Invest Ophthalmol Vis Sci. 55 (7) (2014) 4119–27.*
50. *Z.C. Zhao, Y. Zhou, G. Tan et al. Research progress about the effect and prevention of blue light on eye, International Journal of Ophthalmology. 11 (12) (2018) 1999–2003.*
51. *M. Kaido, I. Toda, T. Oobayashi, et al., Reducing Short-Wavelength Blue Light in Dry Eye Patients with Unstable Tear Film Improves Performance on Tests of Visual Acuity, PloS one 11 (4) (2016) e0152936.*
52. *Zheng QX, Ren YP, Reinach PS et al. Reactive oxygen species activated NLRP3 inflammasomes initiate inflammation in hyperosmolarity stressed human corneal epithelial cells and environment-induced dry eye patients. Exp Eye Res 2015;134: 133-40.*
53. *Lee HS, Cui L, Li Y et al. Correction: influence of light emitting diode-derived blue light overexposure on mouse ocular surface. PLoS One 2016;11(11):e0167671.*
54. *Resnikoff S, Pascolini D, Etyaale D et al. Global data on visual impairment in the year 2002. Bull World Health Organ 2004;82(11):844-51.*
55. *Xue L, Bo YU, Hong X. The establishment of a rat model for experimental retinal photic injury and observation of pathological changes in the retina. Chinese Journal of Optometry & Ophthalmology 2003;5(1):29-32.*
56. *Xie C, Li XY, Tong JP et al. Effects of white light-emitting diode (LED) light exposure with different correlated color temperatures (CCTs) on human lens epithelial cells in culture. Photochem Photobiol 2014;90(4):853-59.*
57. *Babizhayev MA. Mitochondria induce oxidative stress, generation of reactive oxygen species and redox state unbalance of the eye lens leading to human cataract formation: disruption of redox lens organization by phospholipid hydroperoxides as a common basis for cataract disease. Cell Biochem Funct 2011;29(3):183-206.*
58. *U.T. Brunk, A. Terman, Lipofuscin: mechanisms of age-related accumulation and influence on cell function, Free Radic Biol Med. 33 (5) (2002) 611–19.*
59. *J.R. Sparrow, Y. Wu, T. Nagasaki, et al., Fundus autofluorescence and the bisretinoids of retina, Photochem. Photobiol. Sci. 9 (11) (2010) 1480–89.*
60. *J.R. Sparrow, K. Nakanishi, C.A. Parish, The lipofuscin fluorophore A2E mediates blue light-induced damage to retinal pigmented epithelial cells, Invest Ophthalmol Vis Sci. 41 (7) (2000) 1981–89.*
61. *L.E. Lamb, J.D. Simon, A2E: a component of ocular lipofuscin, Photochem Photobiol 79 (2) (2004) 127–36.*
62. *C. Grimm, A. Wenzel, T. Williams, et al., Rhodopsin-mediated blue-light damage to the rat retina: effect of photoreversal of bleaching, Investigative ophthalmology & visual science 42 (2) (2001) 497–505.*
63. *C. Grimm, C.E. Reme, P.O. Rol, et al., Blue Light's Effects on Rhodopsin: Photoreversal of Bleaching in Living Rat Eye, Invest Ophthalmol Vis Sci. 41 (12) (2000) 3984–90.*

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

64. *M. Nakamura, Y. Kuse, K. Tsuruma, et al., The Involvement of the Oxidative Stress in Murine Blue LED Light-Induced Retinal Damage Model, Biological & pharmaceutical bulletin 40 (8) (2017) 1219–11225.*
65. *E.K. Ozkaya, G. Anderson, B. Dhillon, et al., Blue-light induced breakdown of barrier function on human retinal epithelial cells is mediated by PKC over-activation and oxidative stress, Experimental Eye Research. 189 (2019) 107817.*
66. *M. Melanie, G. Pauline, P. Delphine, et al., Blue-violet light decreases VEGF production in an in vitro model of AMD, PloS one 14 (10) (2019) e0223839.*
67. *J.S. Penn, A. Madan, R.B. Caldwell, et al., Vascular endothelial growth factor in eye disease, Prog Retin Eye Res. 27 (4) (2008) 331–71.*
68. *H. Takahashi, M. Shibuya. The vascular endothelial growth factor (VEGF)/VEGF receptor system and its role under physiological and pathological conditions, Clinical Science. 109 (3) (2005) 227–41.*
69. *R. Rosenthal, H. Heimann, H. Agostini, et al., Ca2+channels in retinal pigment epithelial cells regulate vascular endothelial growth factor secretion rates in health and disease, Molecular Vision. 13 (2007) 443–56.*
70. *R. Chu, X. Zheng, D. Chen, et al., Blue Light Irradiation Inhibits the Production of HGF by Human Retinal Pigment Epithelium Cells In Vitro, Photochemistry and Photobiology. 82 (5) (2006) 1247–50.*
71. *J. Zhuang, Y. Liu, Q. Yuan, et al., Blue Light-Induced Apoptosis of A2E-Containing RPE: Involvement of Caspase-3 and Protection by Bcl-2, Investigative ophthalmology & visual science 42 (6) (2001) 1356–62.*
72. *C. Huang, P. Zhang, W. Wang, et al., Long-term blue light exposure induces RGC-5 cell death in vitro: involvement of mitochondria-dependent apoptosis, oxidative stress, and MAPK signaling pathways, Apoptosis 19 (6) (2014) 922–32.*
73. *M.L. Gao, W.L. Deng, N. Huang, et al., Upregulation of GADD45a in light-damaged retinal pigment epithelial cells, Cell Death Discovery 2 (2016) 16013.*
74. *C.H. Lin, M.R. Wu, W.J. Huang, et al., Low-Luminance Blue Light-Enhanced Phototoxicity in A2E-Laden RPE Cell Cultures and Rats, International journal of molecular sciences 20 (7) (2019) 1799.*
75. *Y.S. Cheng, M. Linetsky, X. Gu, et al., Light-induced generation and toxicity of docosahexaenoate-derived oxidation products in retinal pigmented epithelial cells, Experimental eye research 181 (2019) 325–45.*
76. *Rose KA, Morgan IG, Ip J et al. Outdoor activity reduces the prevalence of myopia in children. Ophthalmology 2008; 115(8):1279-85.*
77. *Rose KA, Morgan IG, Smith W et al. Myopia, lifestyle, and schooling in students of Chinese ethnicity in Singapore and Sydney. Arch Ophthalmol 2008;126(4):527-30.*
78. *Czepita D, Mojsa A, Ustianowska M et al. Reading, writing, working on a computer or watching television, and myopia. Klin Oczna 2010;112(10-12):293-95.*
79. *Morgan IG, French AN, Ashby RS et al. The epidemics of myopia: aetiology and prevention. Prog Retin Eye Res. 2018;62:134–149*
80. *He M, Xiang F, Zeng Y et al. Effect of time spent outdoors at school on the development of myopia among children in China: a randomized clinical trial. JAMA. 2015;314(11):1142–48.*
81. *Wu PC, Chen CT, Chang LC et al. Increased time outdoors is followed by reversal of the long-term trend to reduced visual acuity in Taiwan primary school students. Ophthalmology. 2020;127(11):1462–69.*
82. *Jin JX, Hua WJ, Jiang X et al. Effect of outdoor activity on myopia onset and progression in school-aged children in northeast China: the Sujiatun Eye Care Study. BMC Ophthalmol. 2015;15:73.*
83. *French AN, Morgan IG, Mitchell P et al. Risk factors for incident myopia in Australian schoolchildren: the Sydney adolescent vascular and eye study. Ophthalmology. 2013;120(10):2100–08.*
84. *Wang J, Li Y, Musch DC, Wei N et al. Progression of myopia in school-aged children after COVID-19 home confinement. JAMA Ophthalmol. 2021;139(3):293–300.*
85. *Hu Y, Zhao F, Ding X, Zhang S et al. Rates of myopia development in young Chinese schoolchildren during the outbreak of COVID-19. JAMA Ophthalmol. 2021;139(10):1115–21.*
86. *Enthoven CA, Tideman JWL, Polling JR et al. The impact of computer use on myopia development in childhood: the Generation R study. Prev Med. 2020;132:105988.*
87. *Enthoven CA, Polling JR, Verzijden et al. Smartphone use associated with refractive error in teenagers: the myopia app study. Ophthalmology. 2021;128(12):1681–88.*
88. *Rucker F, Britton S, Spatcher M et al. Blue light protects against temporal frequency sensitive refractive changes. Invest Ophthalmol Vis Sci 2015;56(10):6121-31.*
89. *Münch M, Nowozin C, Regente J et al. Blue-enriched morning light as a countermeasure to light at the wrong time: effects on cognition, sleepiness, sleep, and circadian phase. Neuropsychobiology 2016;74(4):207-18.*
90. *Gabel V, Reichert CF, Maire M et al. Differential impact in young and older individuals of blue-enriched white light on circadian physiology and alertness during sustained wakefulness. Sci Rep 2017;7(1):7620.*
91. *Ayaki M, Muramatsu M, Negishi K et al. Improvements in sleep quality and gait speed after cataract surgery. Rejuvenation Res 2013;16(1):35-42.*
92. *Palavets T, Rosenfield M. Blue-blocking filters and digital eyestrain. Optom Vis Sci. 2019;96(1):48–54.*
93. *Boulton M, Rozanowska M, Rozanowski B. Retinal photodamage. J Photochem Photobiol B. 2001;64(2–3):144-61.*
94. *Ide T, Toda I, Miki E, Tsubota K. Effect of blue light-reducing eye glasses on critical flicker frequency. Asia Pac J Ophthalmol (Phila). 2015;4(2):80–85.*
95. *Leibovitch, T. Lai, N. Porter et al. Visual outcomes with the yellow intraocular lens, Acta ophthalmologica Scandinavica. 84 (1) (2006) 95–99.*

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 4 | April 2025 - Peer Reviewed Journal

96. R. Hayashi, S. Hayashi, K. Arai, et al., Evaluating the Biostability of Yellow and Clear Intraocular Lenses with a System Simulating Natural Intraocular Environment, Translational vision science & technology 5 (6) (2016) 11.
97. M. Sasaki, K. Yuki, T. Kurihara, et al., Biological role of lutein in the light-induced retinal degeneration, The Journal of nutritional biochemistry 23 (5) (2012) 423-29.
98. H.E. Khoo, A. Azlan, S.T. Tang, et al., Anthocyanidins and anthocyanins: colored pigments as food, pharmaceutical ingredients, and the potential health benefits, Food & Nutrition Research. 61 (1) (2017) 1361779.
99. Y. Nomi, K. Iwasaki-Kurashige, H. Matsumoto; Therapeutic Effects of Anthocyanins for Vision and Eye Health, Molecules. Mocular. 24 (18) (2019) e3311.
100. T. Tsuda; Curcumin as a functional food-derived factor: degradation products, metabolites, bioactivity, and future perspectives, Food Funct. 9 (2) (2018) 705-14.
101. J. Kim, K. Cho, S.Y. Choung; Protective effect of Prunella vulgaris var. L extract against blue light induced damages in ARPE-19 cells and mouse retina, Free Radical Biology and Medicine. (2019) S0819-5849(19)31257-2.
102. Q. Wei, X. Liang, Y. Peng, et al. 17β-estradiol ameliorates oxidative stress and blue light-emitting diode-induced retinal degeneration by decreasing apoptosis and enhancing autophagy, Drug Des Devel Ther. 12 (2018) 2715-30.
103. M. Cachafeiro, A.P. Bemelmans, M. Samardzija, et al. Hyperactivation of retina by light in mice leads to photoreceptor cell death mediated by VEGF and retinal pigment epithelium permeability, Cell death & disease 4 (8) (2013) e781.
104. G.H. He, W. Zhang, Y.X. Ma, et al. Mesenchymal stem cells-derived exosomes ameliorate blue light stimulation in retinal pigment epithelium cells and retinal laser injury by VEGF-dependent mechanism, International journal of ophthalmology 11 (4) (2018) 559-66.
105. Y. Shen, H.X. Zhao, Z.G. Wang, et al. Silibinin declines blue light-induced apoptosis and inflammation through MEK/ ERK/CREB of retinal ganglion cells, Artificial Cells Nanomed Biotechnology. 47 (1) (2019) 4059-65.
106. M. Ghasemi, E. Alizadeh, K. Saei Arezoumand, et al., Ciliary neurotrophic factor (CNTF) delivery to retina: an overview of current research advancements, Artificial Cells Nanomed Biotechnology. 46 (8) (2018) 1694-07.