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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*

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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,

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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.

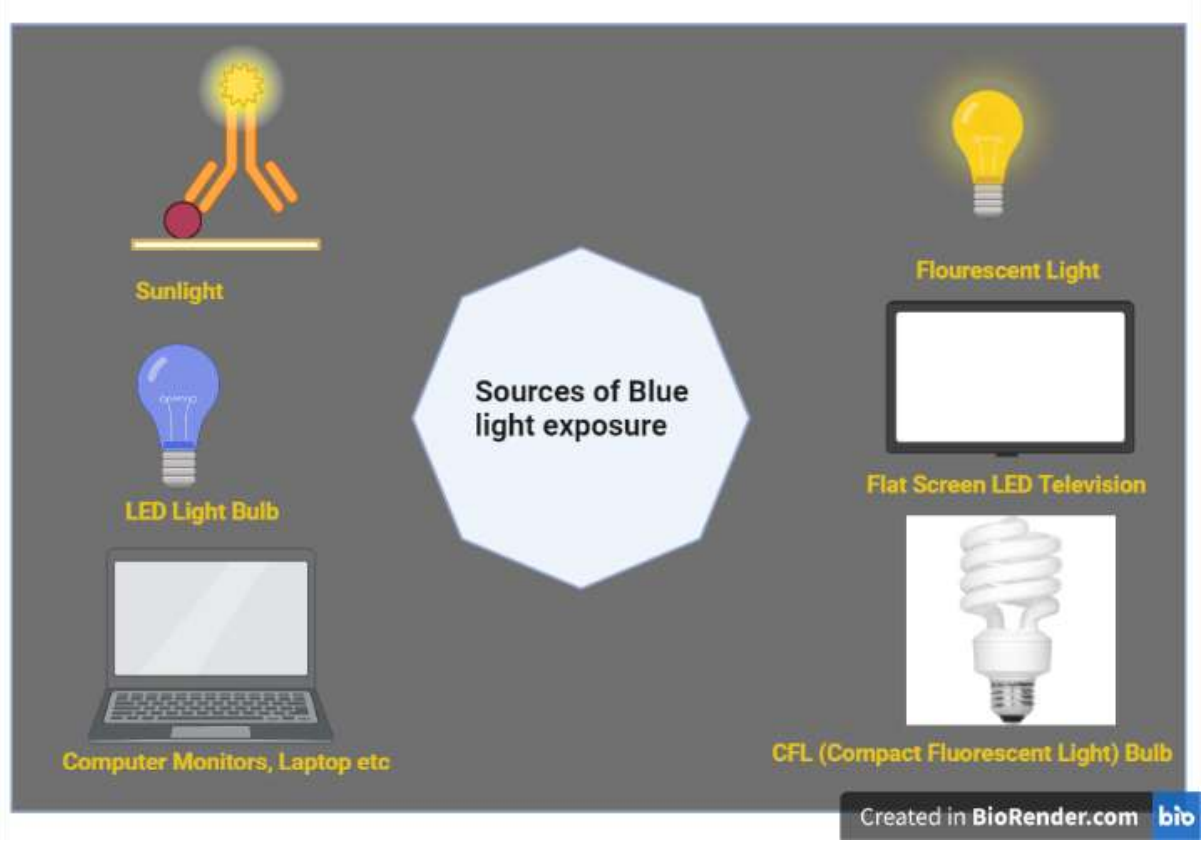


Fig 1: Sources of blue light exposure in our daily lives.

2. EFFECT OF BLUE LIGHT

2.1. Effect of Blue Light on the Ocular Surface

Histologically, the ocular surface includes-

- Tear film,

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- 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]

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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

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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

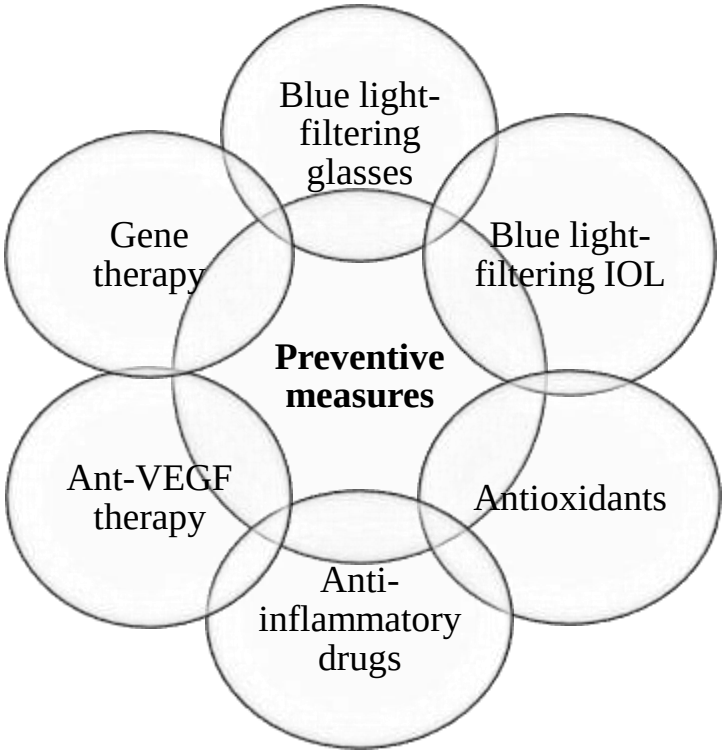
Many studies have shown that blue light can help regulate the biological clock and improve cognition, memory, and alertness. The major reason is that blue light causes the pineal gland to release melatonin, which regulates human circadian rhythm by raising or lowering cortisol expression based on time 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]

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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]

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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.

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