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An Updated Review of Blue Light Effects on Ocular Diseases

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Abstract: Blue light, a high-energy component of the visible spectrum (400–480 nm), has been explored for its potential involvement in various ocular conditions, including age-related macular degeneration (AMD), retinitis pigmentosa (RP), and cataract formation. This narrative review examines the potential mechanisms by which blue light exposure may influence the initiation and progression of these diseases, drawing on evidence from in vitro, in vivo, and selected clinical studies. Experimental findings suggest that shorter blue wavelengths (435–450 nm) can increase oxidative stress in retinal cells under specific exposure conditions, potentially contributing to AMD-related pathological changes. In RP models, blue light has been linked to photoreceptor vulnerability, while blue-light filtering strategies have been explored for potential visual and circadian benefits. Regarding cataracts, laboratory studies indicate that prolonged exposure to certain blue wavelengths (455–463 nm) may promote lens protein aggregation and inflammatory responses. However, outcomes appear to depend strongly on intensity and biological context. Blue-light-filtering intraocular lenses (IOLs) and optical filters have been proposed as protective interventions, although their long-term clinical benefits remain under investigation. Overall, current evidence suggests that the biological effects of blue light are influenced by wavelength, irradiance, intensity, and cell susceptibility. Further standardized exposure protocols and well-designed clinical studies are needed to clarify its role in long-term ocular health.

Keywords: blue light; AMD; RP; cataract; blue-light-blocking filters.

1. Introduction

Blue light is a component of the visible light spectrum, which ranges approximately from 400 to 700 nm and represents the portion of the electromagnetic spectrum detectable by the human eye [1]. Sunlight is the primary natural source of visible light and is essential for life on Earth. However, rapid advances in artificial lighting and digital technologies have introduced additional sources of visible light, particularly light-emitting diodes (LEDs) used in

smartphones, computers, tablets, and other electronic devices. As digital device use begins at increasingly younger ages and screen time continues to rise, concerns have emerged regarding the potential health implications of prolonged exposure to artificial light sources [2–4]. The digitalization of modern environments has transformed sectors such as business, tourism, and education [5]. underscoring the need to understand its broader biological effects better.

Recent discussions have focused on the potential ocular effects of prolonged exposure to short-wavelength (blue) light emitted from LEDs and digital screens, which has been associated with symptoms such as eye strain, dryness, blurred vision, and musculoskeletal discomfort, particularly among young and healthy individuals [6,7]. Epidemiological studies have reported increasing prevalence of visual fatigue, retinal dysfunction, and sleep disruption among individuals with prolonged digital screen exposure [2–4]. However, these symptoms are generally linked to digital eye strain and near work rather than direct structural retinal damage. Blue light, with its relatively higher photon energy, is capable of reaching the retina after passing through the eye's optical media [8–10]. While experimental studies suggest that blue light can induce oxidative stress and cellular changes under certain exposure conditions, robust population-based evidence linking routine digital screen exposure to major retinal diseases, such as age-related macular degeneration (AMD), remains limited. This highlights the need for mechanistic laboratory studies to investigate cellular-level effects and better interpret potential ocular risks.

Age-related macular degeneration (AMD), retinitis pigmentosa (RP), and cataracts are major causes of visual impairment worldwide. AMD affects the macula and leads to progressive central vision loss, with a global prevalence of 8.7% among individuals aged 50 years or older [11]. Retinitis pigmentosa (RP) comprises a group of inherited retinal dystrophies characterized by progressive photoreceptor degeneration, affecting approximately 1 in 4,000 individuals globally [12]. Cataracts, defined as opacification of the eye's natural lens, remain the leading cause of blindness worldwide, accounting for more than half of global cases [13]. These conditions significantly reduce quality of life and increase healthcare burden.

Although short-wavelength light is vital for circadian rhythm regulation and visual function, concerns remain about the biological effects of high-intensity or prolonged artificial exposure under certain conditions. This review will critically analyze current literature on the mechanisms by which blue light may alter ocular tissues, drawing on in vitro, in vivo, and clinical research, and will note existing gaps and outline ideas for future research.

2. Materials and Methods

This narrative review synthesizes current findings on “blue light effects on ocular diseases”. A structured search was conducted in Google Scholar, PubMed, Science Direct, and Scopus using Boolean operators and relevant keywords such as “blue light”, “short-wavelength light”, “phototoxicity”, “ocular diseases”, “age-related macular degeneration (AMD)”, “retinitis pigmentosa (RP)”, and “cataract”. Articles were selected based on their relevance to the effects of blue and short-wavelength light on retinal health and ocular diseases, especially AMD, RP, and cataracts (Figure 1). A total of 112 records were identified.

Studies were included if they (1) examined the biological or mechanistic effects of blue light on ocular cells, to ensure relevance to disease pathogenesis rather than general visual discomfort, (2) clearly described the light exposure parameters such as wavelength, irradiance and intensity as these variables critically determine phototoxic effects and allow meaningful interpretation, and (3) involved in vitro, in vivo, or human clinical data, to capture mechanistic,

translational, and clinical perspectives. Studies were excluded if they were review articles, book chapters, or conference proceedings to avoid duplication of secondary interpretations and ensure reliance on primary data. Non-English publications were excluded to prevent misinterpretation during data extraction. Duplicate records were removed to avoid data redundancy. Studies primarily focusing on therapeutic outcomes without addressing etiological or mechanistic aspects were excluded to maintain alignment with the review objective, which was to examine causal and pathophysiological mechanisms of blue light exposure rather than treatment efficacy. After screening and full-text assessment, 14 studies met the eligibility criteria and were included in the qualitative synthesis.

A PRISMA-style flow diagram was used to document the study identification and selection process transparently. While PRISMA guidelines are formally intended for systematic reviews and meta-analyses, the diagram was adopted in this narrative review solely to improve reporting clarity and reproducibility, not to imply a systematic review design [14].

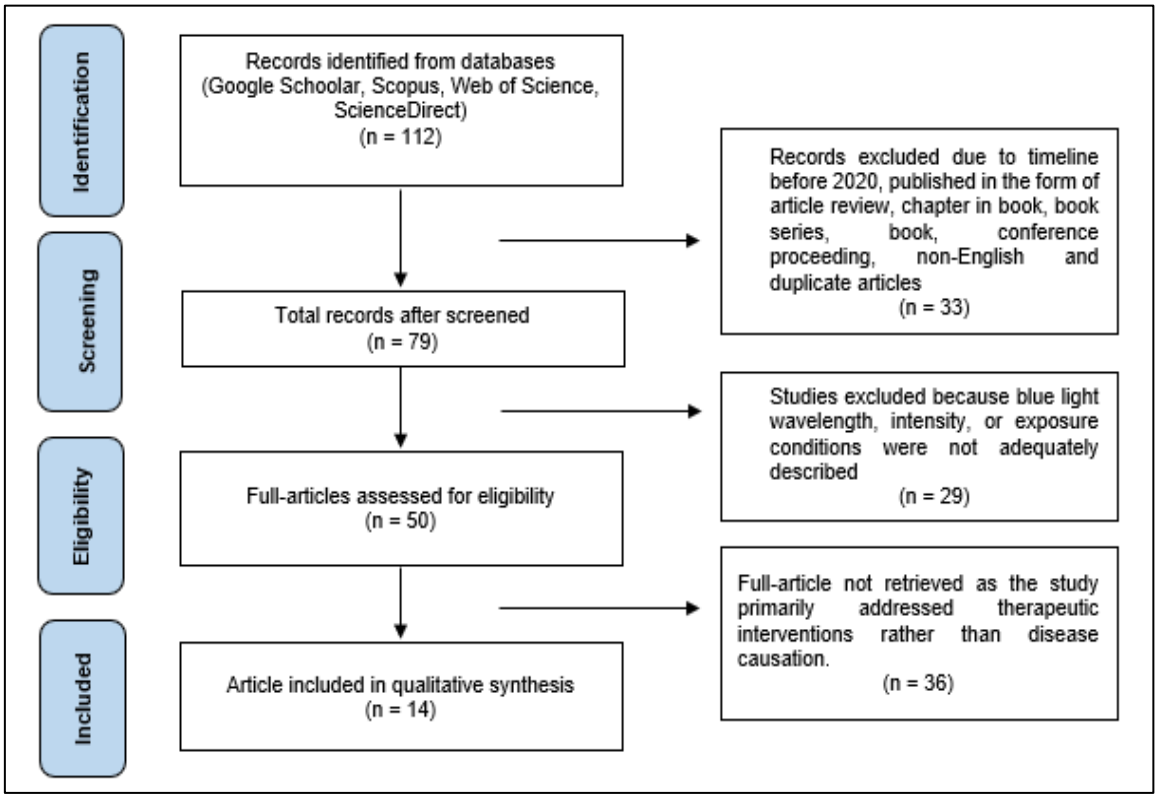

Figure 1. Preferred Reporting Items for Narrative Reviews and Meta-Analyses flow diagram.

3. Results

The results and discussion synthesize findings from experimental and clinical studies examining the effects of blue light exposure on retinal and lens health, particularly in age-related macular degeneration (AMD), retinitis pigmentosa (RP), and cataracts. Across models, blue light exposure has been associated with oxidative stress, mitochondrial alterations, and photoreceptor vulnerability. However, the magnitude and nature of these effects vary considerably depending on wavelength, irradiance, exposure duration, and biological context. Importantly, many experimental studies employ high-intensity or acute exposure paradigms that differ substantially from typical environmental or digital device exposure. Therefore, interpretation of these findings requires careful consideration of exposure conditions.

3.1. Blue light causes age-related macular degeneration (AMD).

Experimental studies constantly show that blue light disrupts retinal health, with evidence linking it to the early stages of AMD (Table 1). In aged C57 mice, blue light exposure (450 nm, 2,000 lux) led to retinal damage, mitochondrial impairment in the retinal pigment epithelium (RPE), oxidative stress, and dysfunction of photoreceptor outer segments [15]. In human ARPE19 cells, blue light (465 nm to 475 nm) induced oxidative stress and RPE dysfunction, which is a main factor in AMD [16]. Similarly, blue light exposure (445 ± 18 nm) with A2E accumulation generated reactive oxygen species (ROS), mitochondrial dysfunction, and apoptosis [17]. In porcine retinal explants, blue light exposure (435 ± 20 nm) amplified p53-mediated apoptosis [18]. However, a study from Marie *et al*. [18] demonstrates a dual effect of blue light by reduced VEGF mRNA and protein levels and also increased VEGF, which will increase oxidative stress and apoptosis in A2E-loaded cells. It signifies that while blue light may suppress VEGF, it also worsens oxidative damage, contributing to AMD progression (Figure 2).

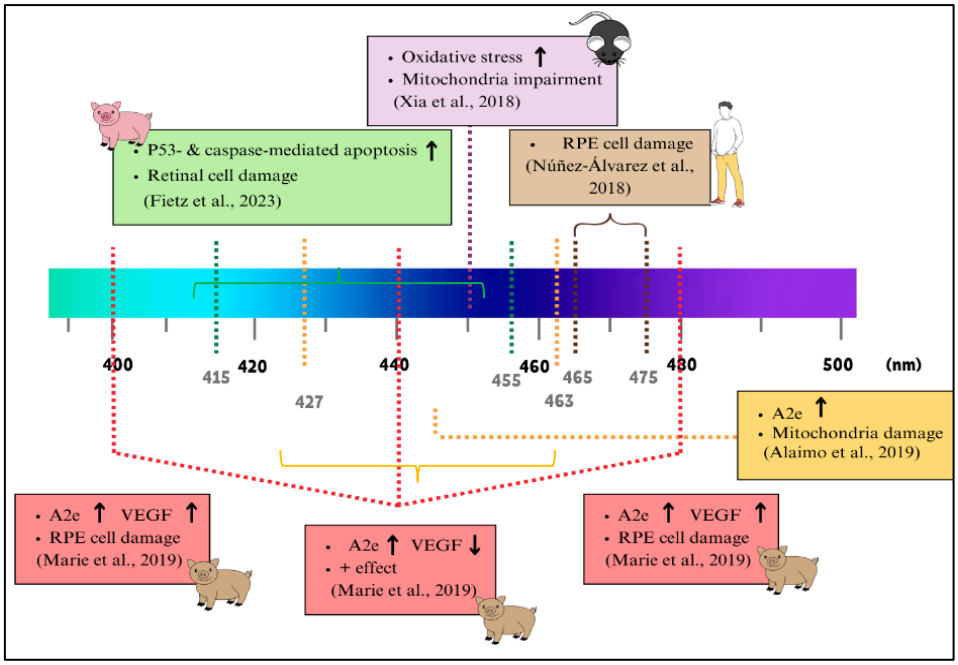

Figure 2. Schematic representation of findings on blue light caused age-related macular degeneration (AMD). Created by authors based on data from [15–19].

Table 1. Related studies on blue light cause age-related macular degeneration (AMD).

Reference	Test subject	Experimental models	Wavelength settings	Intensities (lux) or irradiance (mW/cm2) or luminance (log*cd/m2)	Findings	Remarks
[15]	Mice	Aged C57 mice	Peak: 450 nm	2,000 lux	• Mitochondrial impairment in RPE • Increasing oxidative stress in retinal tissue • Photoreceptor damage • Early-stage AMD	Oxidative stress; early AMD
[16]	Human	ARPE19 cells	Blue light: 465–475 nm Red light: 625–635 nm	Blue light: 800 lux Red light: 950 lux	Blue light: • Increasing oxidative stress in retinal tissue • Damaged RPE cells • Dry AMD Red light:	Oxidative stress; dry AMD

Reference	Test subject	Experimental models	Wavelength settings	Intensities (lux) or irradiance (mW/cm2) or luminance (log*cd/m2)	Findings	Remarks
					• blunted the toxic effect of the various mitochondrial toxins	
[17]	Human	ARPE19 cells	Wavelength: 445±18 nm	4.43 mW/cm2	• Increasing in A2E accumulation • Dysfunction of mitochondria • Increasing production of ROS, oxidative stress • Activates caspase-3; apoptosis	Oxidative stress, apoptosis
[18]	Porcine	Retinal explants	Wavelength: 435 ± 20 nm	15 mW/cm2	• Oxidative stress • Overactivation of p53 and increasing caspase-mediated apoptosis	Oxidative stress, apoptosis
[19]	Porcine	RPE cells	Wavelength: 400, 440, and 480 nm.	1.5 mW/cm²	• Increasing VEGF expression, which reduces angiogenesis (+) • Increasing VEGF generates oxidative stress, leading to apoptosis of RPE cells (-)	Dual effect

3.2. Blue light caused retinitis pigmentosa (RP).

The findings strongly emphasize the detrimental impact of blue light on retinal health, particularly in the context of RP (Table 2). Clinical study in autosomal dominant RP (adRP) patients due to NR2E3 mutations shows that blue light exposure impairs rod dysfunction in RP patients, which serves as a hallmark of progressive rod photoreceptor loss [20]. In contrast, Kawasaki *et al*. [19] suggested that cone function assessed under red light conditions often remains preserved, further highlighting the selective vulnerability of rods to blue light. Similarly, in patients with early-onset RP caused by CRB1 mutations, blue light sensitivity decreased as the disease progressed [20].

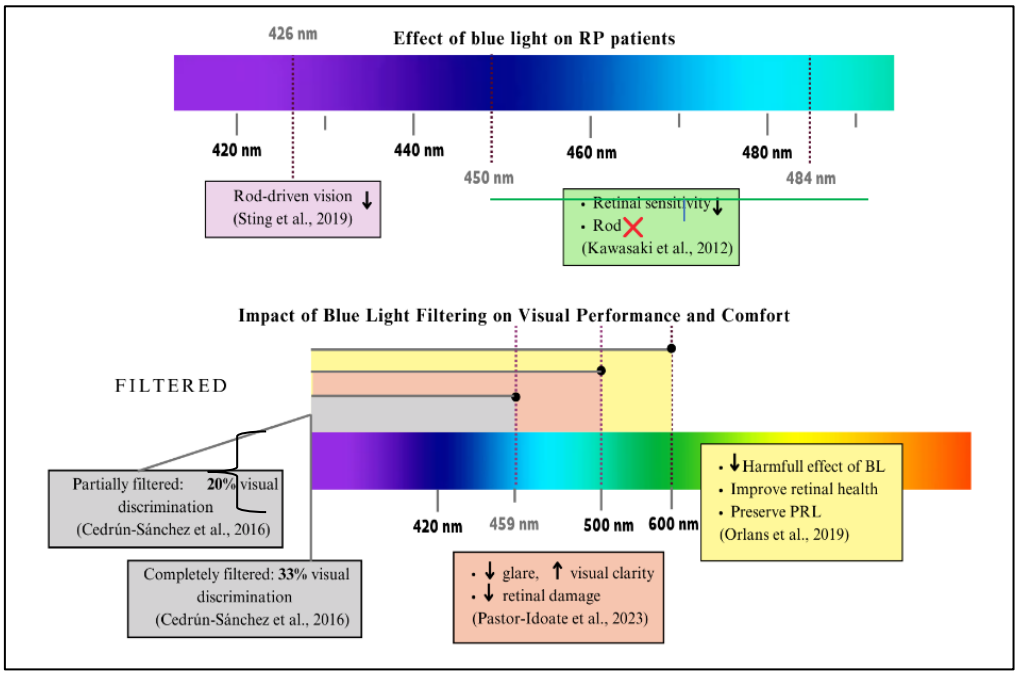

Figure 3. Schematic representation of findings on the effect of blue light and the impact of blue light filtering on RP patients. Created by authors based on data from [19–23].

Animal models of RP, such as the RhoP23H/+ mouse, reveal that exposure to unfiltered blue light accelerates photoreceptor layer thinning and retinal dysfunction [21]. Based on Figure 3, filtering out short-wavelength light (<600 nm) in these models conserved photoreceptor morphology and function, suggesting that blue light exacerbates the structural and functional decline characteristic of RP [22].

Table 2. Studies on the effects of blue light exposure on retinitis pigmentosa (RP) models.

Reference	Test subject	Experimental models	Wavelength setting	Intensities (lux) or irradiance (mW/cm2) or luminance (log*cd/m2)	Findings	Remarks
[19]	Human	RP patients	Blue light wavelength: 467 ± 17 nm Red light wavelength: 640 ± 10 nm	6.0 to 2.6 log cd/m²	Blue light:- • Decrease retinal sensitivity • hallmark of progressive rod dysfunction Red light:- no significant difference from controls, indicating preserved cone function	Rod dysfunction
[20]	Human	RP patients	Peak Wavelength: Blue light: 426 nm Red light: 606 nm	Blue and Red light = 28 lux	Blue-FST:- • Beyond 30 years: rod-driven response diminishes; decline in rod photoreceptor function. Red FST:- • Reduced sensitivity in RP patients • Exhibits no clear correlation with disease duration • Highlighting their potential as a baseline functional marker, but not as an indicator of progression.	Photoreceptor dysfunction
[21]	Mice	RhoP23H/+	Filtered <600 nm	-	1) Red-tinted cages that filtered short-wavelength light (<600 nm):- • mitigated the harmful effects of blue light exposure • preserved Photoreceptor Layer (PRL) • improved retinal health 2) Unfiltered SWL gives deteriorated retinal health and function	Filtering blue light
[22]	Human	RP patients	SWL (filtered)	-	1) Optical SWL filters can help RP patients by:- • Reducing glare and improving visual clarity. • Possibly slowing retinal damage, offering protection to the retina. 2) Unfiltered (SWL) exacerbates symptoms in RP patients	Filtering blue light
[23]	Human	RP patients	Partial filter: partially filter < 450 nm. Full Filter: Completely filter <450 nm.	-	• Partial absorption filters: Increase 20% of visual discrimination • Full absorption filters: increase 33% visual discrimination	Filtering blue light

In human RP patients, blue light-blocking filters reduced glare, enhanced visual function, and perhaps reduced retinal degeneration by regulating circadian rhythms, while a study with 57 RP patients observed that blue light-absorbing filters enhanced visual discrimination, mainly in low-light conditions, and that complete filters improved visual discriminationy 33% [22,23].

3.3. Blue light causes cataract.

Numerous studies underscore the cataractogenic effects of blue light, suggesting its involvement in lens damage and cataract formation (Table 3).

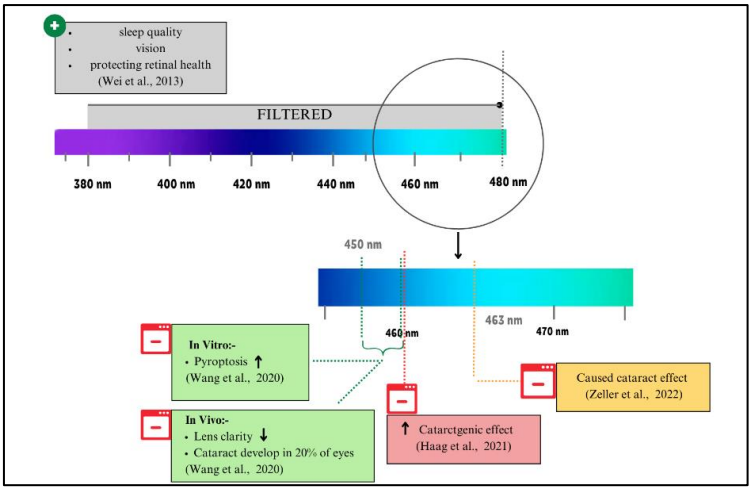

Figure 4. Schematic representation of findings on the summary of findings on blue light caused cataract. Created by authors based on data from [24–26].

Table 3. Related studies on blue light have caused cataract.

Title/Citation	Test subject	Experimental models	Wavelength setting	Intensities (lux) or irradiance (mW/cm2) or luminance (log*cd/m2)	Finding	Remarks
[24]	Porcine	Lens (*ex vivo* model)	UVB: 311 nm UVA: 370 nm Blue light: 460 nm	-	• All three wavelengths of blue light caused cataract development • Blue light (460 nm) revealed the strongest cataractogenic effect	Cataractogenic effect
[25]	Porcine	Lens (*ex vivo* model)	Violet: 407 nm Blue: 463 nm Red: 635 nm	70 mW/cm2	• Violet light: caused the most significant cataract formation • Blue light: caused cataract formation < violet light	Cataract formation
[26]	Human	Cataract patients	Blue light: 380 nm – 480 nm (filtered)	-	Patients with blue-light-blocking IOLs:- • Experienced significant improvements in sleep quality • Improving vision • protecting retinal health	blue-light-blocking IOL implantation

Figure 4 shows a summary of the blue light-induced cataract effect on several wavelengths. In *ex vivo* porcine lens models, exposure to UVB (311 nm), UVA (370 nm), and blue light (460 nm) leads to cataract formation, with blue light causing the greate damage [24]. However, violet light (407 nm) induced the highest level of cataract formation in porcine lenses, followed by blue light in Zeller *et al*.'s study [25]. Clinical findings also support these observations, as cataract patients who experienced surgery and received blue-light-blocking intraocular lenses (IOLs) reported substantial improvements in sleep quality [26]. These findings collectively emphasize the potential protective benefits of blue-light-blocking interventions in preventing or mitigating cataract development.

4. Discussion

4.1. Age-related macular degeneration (AMD).

Age-related macular degeneration (AMD), usually found in individuals over 50 years old, is a progressive retinal disease that disturbs the macula, leading to central vision loss. It is categorized into two forms, which are dry AMD, classified by drusen accumulation and gradual retinal pigment epithelium (RPE) breakdown, and wet AMD, which is characterized by abnormal blood vessel growth and fluid leakage, activating rapid vision distortion [27]. Risk factors include aging, genetic predispositions, lifestyle choices like smoking, poor diet, and excessive blue light exposure, as well as oxidative stress and inflammation [28]. Symptoms often comprise blurred or distorted central vision and trouble in distinguishing faces or reading, while peripheral vision remains undamaged [29]. Although incurable, treatments such as anti-vascular endothelial growth factor (VEGF) injections for wet AMD and antioxidant-rich dietary supplements for dry AMD can slow its progression [30].

Blue light (400–500 nm) has appeared as an important factor in the deterioration of retinal health, particularly in the context of age-related macular degeneration (AMD). Experimental evidence from animal models, human cell studies, and retinal explants has consistently emphasized its role in inducing oxidative stress, mitochondrial dysfunction, and structural damage in retinal cells, all of which are key contributors to AMD pathogenesis [5,15,17]. It is important to note that these findings reflect wavelength-specific responses rather than absolute irradiance, as most laboratory studies utilize higher exposure intensities than those encountered in everyday digital device use. In human ARPE19 cells, exposure to blue light (465–475 nm) caused oxidative stress, resulting in retinal pigment epithelium (RPE) dysfunction [15]. Rather than being an isolated cause, blue light may amplify pre-existing metabolic vulnerability in aging RPE cells. These findings underline the vulnerability of the RPE to phototoxic damage under chronic blue light exposure.

Studies in aged C57 mice demonstrated that blue light exposure (450 nm) caused mitochondrial impairment in the RPE and disrupted photoreceptor outer segments [5]. The accumulation of mitochondrial dysfunction not only compromises energy production but also increases oxidative stress through malfunctioning electron transport chains, creating a vicious cycle of cellular damage [16]. In addition, when blue light exposure was combined with A2E accumulation, which is a byproduct of photoreceptor metabolism, mitochondrial dysfunction was worsened, as shown by caspase-3 activation and apoptosis [16]. These results propose that the interaction between blue light and phototoxic metabolites like A2E further amplifies mitochondrial impairment, driving the progression of AMD (Figure 5). While experimental

evidence indicates that blue light can induce oxidative and mitochondrial stress in retinal cells, its role should be interpreted within the broader framework of cumulative retinal aging.

Blue light has been shown to induce apoptosis in retinal cells through both intrinsic (mitochondrial) and extrinsic (death receptor) pathways [18]. Porcine retinal explants exposed to blue light (435 ± 20 nm) exhibited p53-mediated apoptosis, highlighting its role in triggering cell death pathways [17]. Similarly, in human ARPE19 cells, blue light exposure led to apoptosis via caspase-3 activation, which is a critical effector of programmed cell death [16]. These findings validate that blue light-induced apoptosis contributes to RPE and photoreceptor loss, causing the structural and functional deterioration observed in AMD (Figure 5).

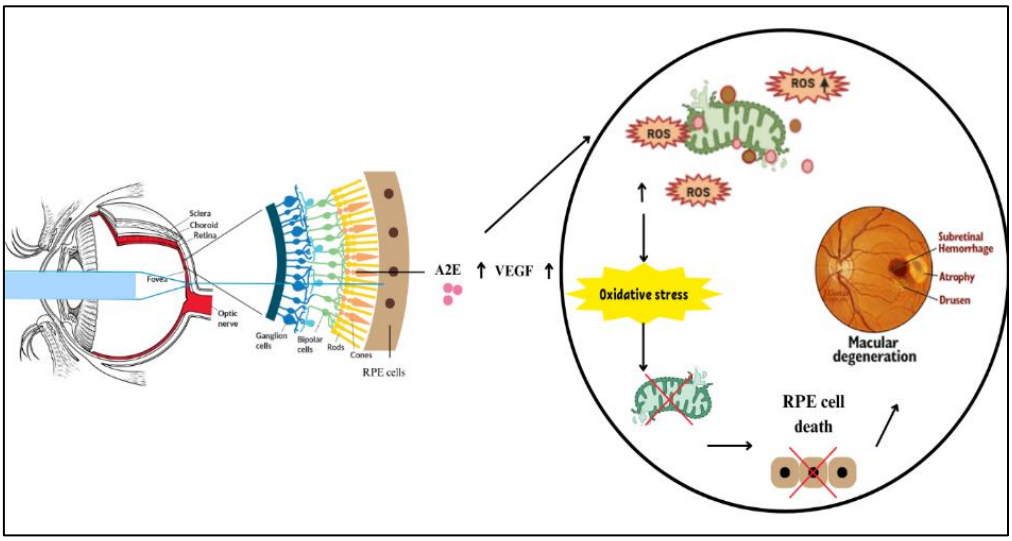

Figure 5. Schematic representation of the potential mechanism of AMD. Created by authors based on data from [5, 16–18].

Experimental findings highlight an important effect of blue light exposure on retinal health, mainly in the context of AMD. The evidence emphasizes the need for protective strategies, such as blue-light-blocking lenses, screen filters, and public health awareness campaigns, to mitigate blue light-induced damage. However, again, most evidence derives from high-intensity laboratory exposure, and robust epidemiological data linking everyday digital screen use to AMD onset remain limited.

4.1.1. Dual role of blue light.

Blue light plays a dual role in retinal health, employing both harmful and regulatory effects [18]. Blue light is pronounced in the existence of photosensitizing agents such as A2E, a byproduct of visual cycle metabolism that accumulates in the RPE [16]. High levels of A2E will increase ROS levels and cause oxidative stress. A study by Marie *et al*. [18] offers critical insights into the regulation of vascular endothelial growth factor A (VEGFa) in RPE cells, specifically under A2E accumulation and light exposure. VEGF is a signaling protein that plays a critical role in angiogenesis (the process of forming new blood vessels) and maintaining vascular health. However, excessive VEGF expression can cause pathological angiogenesis, such as in AMD, where abnormal blood vessel growth leads to vision loss (Figure 5). Marie *et al*. [18] showed that A2E accumulation significantly increases both VEGFa mRNA and intracellular protein levels in RPE cells. This increase was consistent with the previous observations, which indicate pathological AMD.

Interestingly, blue light exposure (440–480 nm) could also suppress the A2E-induced increase in VEGFa protein levels. The reduction in VEGFa secretion under blue light exposure proposes that light-induced phototoxicity may interfere with VEGFa synthesis or release mechanisms [18]. This surprising outcome challenges the conventional view of VEGFa as solely protective, suggesting that under certain circumstances, such as A2E accumulation and phototoxic stress, VEGFa may contribute to cellular damage [30,31]. Besides, the increase in apoptosis in A2E-loaded RPE cells following reduction of VEGFa highlights a potential maladaptive role for VEGFa in AMD-like conditions [19]. This study underscores the complexity of VEGFa regulation in A2E-loaded RPE cells under phototoxic conditions.

Clinically, this duality suggests that blue light effects are context-dependent. While suppression of VEGF under phototoxic stress may appear protective in some settings, excessive oxidative injury may override potential benefits [18]. At present, these findings remain primarily mechanistic and should not be interpreted as direct clinical evidence that blue light exposure either promotes or suppresses AMD progression in patients.

4.1.2. Therapeutic Interventions.

Despite its detrimental effects, few studies have recognized potential interventions to mitigate blue light-induced damage. Autophagy has occurred as a promising protective mechanism, with evidence suggesting it helps to counteract oxidative stress and maintain cellular function [5]. This specifies a potential avenue for preventing or delaying retinal degeneration, particularly in aging individuals. Moreover, red light exposure has shown a protective effect against blue light-induced damage, reducing oxidative stress and promoting cellular health [15]. These findings suggest red light therapy as a non-invasive therapeutic approach for managing AMD. The exploration of these therapeutic strategies highlights the potential to balance the effects of blue light while leveraging protective mechanisms to preserve retinal health.

4.2. Retinitis pigmentosa (RP).

Retinitis pigmentosa (RP) is known as a genetic disorder triggering progressive degeneration of photoreceptor cells in the retina, causing vision loss. It is caused by genetic mutations and can be inherited through autosomal dominant, autosomal recessive, or X-linked patterns [12]. It often starts with night blindness because of rod cell damage, followed by loss of peripheral vision (tunnel vision) as cone cells are affected [32]. Advanced stages can cause complete blindness. While there is no cure, treatments like gene therapy, retinal implants, and vitamin A supplementation may slow progression [32]. Early diagnosis and genetic therapy are vital for managing the disease and exploring new treatments. Blue light exposure is a risk factor that can potentially worsen the course of RP, but it is not the cause of the disease. The primary cause of RP is genetic mutations, and blue light exposure may accelerate the retinal degeneration in genetically predisposed individuals [19,20]. However, clinical management of RP should prioritize genetic counseling and disease-modifying therapies rather than solely environmental modification.

4.2.1. Effect of blue light on RP patients.

The harmful effects of blue light on retinal health, mainly in patients with Retinitis Pigmentosa (RP), have been well-documented. Clinical studies on autosomal dominant RP

(adRP) patients with NR2E3 mutations show that blue light exposure worsens rod dysfunction [19]. This is demonstrated by significantly reduced blue light sensitivity under dark-adapted conditions, serving as a hallmark of progressive rod photoreceptor loss [19]. Similarly, in patients with early-onset RP caused by CRB1 mutations, blue light sensitivity was shown to decline with disease development, while red light responses remained stable [20]. These findings underscore the selective vulnerability of rods to blue light compared to cones. Animal models of RP, such as the RhoP23H/+ mouse, further validate these findings, indicating that unfiltered blue light accelerates photoreceptor layer thinning, mirroring the degenerative patterns detected in human RP [21]. In RP, the loss of rods caused secondary cone degeneration due to disrupted retinal homeostasis, with an increase in oxidative stress and inflammation, which may lead to vision loss [33]. Figure 6 illustrates that blue light exposure increased the ROS level in the retina. In those with RP, where photoreceptors were already damaged because of genetic mutations, this oxidative stress worsens retinal damage [33]. This deteriorates the progression of RP, causing faster loss of vision, especially in night vision and peripheral vision [32]. Nonetheless, exposure levels in laboratory settings may not reflect everyday environmental lighting. Thus, while minimizing excessive short-wavelength exposure in susceptible individuals appears reasonable, further clinical trials are needed to quantify long-term benefit.

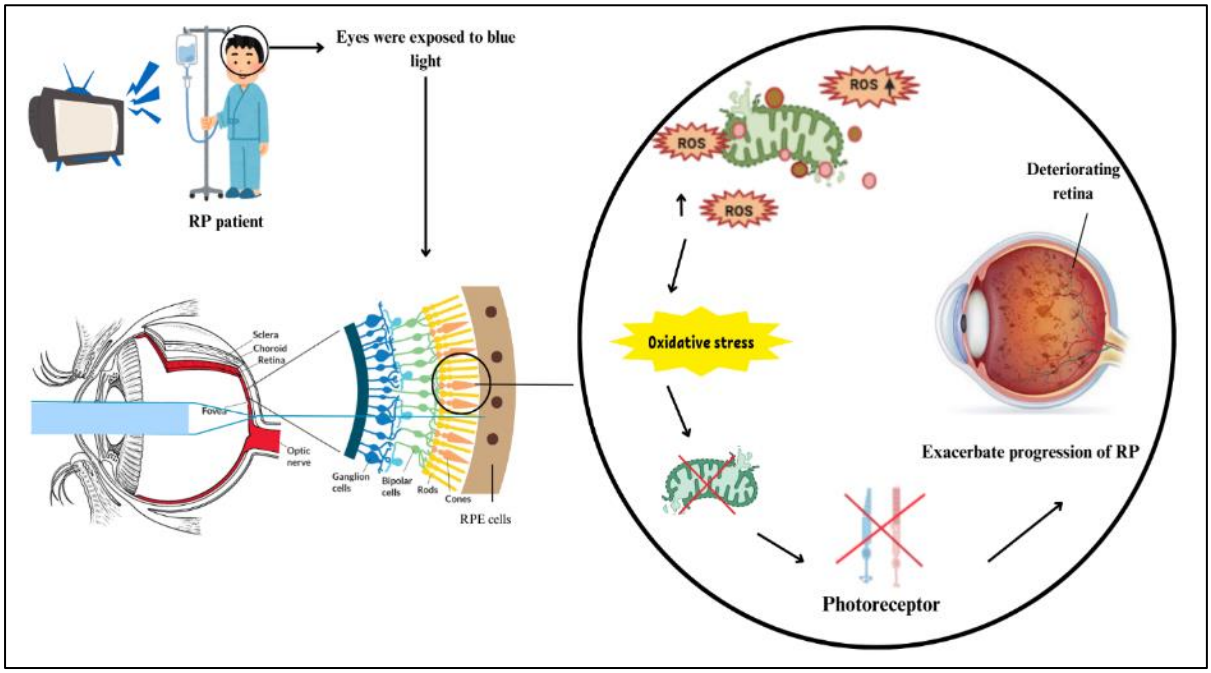

Figure 6. Schematic representation of the potential mechanism of RP. Created by authors based on data from [21,32,32]

4.2.2. Therapeutic interventions.

To counteract these harmful effects, blue light filtering has emerged as a capable intervention for RP management. Short-wavelength light-blocking filters, particularly lenses that absorb wavelengths below 450 nm or red-light filters, blocking wavelengths shorter than 600 nm, have shown significant therapeutic benefits [21]. Orlans *et al.*'s [21] study used RhoP23H/+, a mouse model of RP, and observed that red-light filters preserved photoreceptor layer thickness and cone morphology, which are often compromised under blue light exposure. Similarly, in human patients, blue-light-blocking filters significantly reduced symptoms such as glare and photophobia, improved visual clarity, and enhanced visual discrimination,

particularly in low-light conditions [22]. Notably, filters that absorbed wavelengths below 450 nm improved visual discrimination by 33% in patients with better residual vision, further emphasizing their efficacy [23]. These filters also provided an extra benefit of regulating circadian rhythms, improving sleep quality, and overall well-being.

Overall, these studies highlight the promising role of blue light-filtering interventions in enhancing visual function and possibly slowing retinal degeneration in RP patients [21–23**]**. Given the convincing evidence from both animal models and human studies, further research is reasonable to explore the long-term effects and clinical applications of blue light filters in RP management. These findings suggest that light-filtering technologies may offer a valuable, non-invasive strategy to enhance the quality of life for individuals with retinal diseases such as RP.

4.3. Cataract.

Cataract is a common eye disorder categorized by the clouding of the eye's natural lens, leading to impaired vision. It is one of the major causes of vision loss globally, mostly in older adults [34]. The lens, positioned behind the iris, focuses light onto the retina to produce clear images. When cataracts form, the lens becomes opaque, scattering or blocking light and causing blurry or distorted vision [34]. Cataracts can appear in various forms, including nuclear cataracts that disturb the center of the lens, cortical cataracts that form at the edges and spread inward, and posterior subcapsular cataracts that develop at the back of the lens and progress rapidly [33**]**. It grows when proteins in the lens clump together, forming opaque areas that scatter or block light from reaching the retina [35]. The main causes of cataracts include aging, exposure to ultraviolet (UV) radiation, smoking, aoor diet, and certain medical conditions such as diabetes [36]. Symptoms usually include blurry or cloudy vision, increased sensitivity to glare, trouble seeing at night, and frequent changes in eyeglass prescriptions [33]. Preventative measures such as wearing UV-blocking sunglasses, maintaining a diet rich in antioxidants, avoiding smoking, and managing fundamental health conditions can delay the onset of cataracts [36].

4.3.1. Effect of blue light on cataract.

Extensive research indicates that short-wavelength visible light can adversely affect lens health, potentially contributing to cataract formation. *Ex vivo* studies using porcine lenses demonstrated that blue light (460 nm) can cause significant lens damage, including loss of transparency and structural integrity, with effects exceeding those of UVA (370 nm) and UVB (311 nm) radiation [24]. Violet light (407 nm) showed a greater propensity to induceens changes, whereas longer wavelengths, such as red light (635 nm), produced minimal or no effects [25]. Although these findings provide important mechanistic insights, caution is needed when extrapolating to humans, as in vivo lens physiology and systemic factors may influence susceptibility. Nonetheless, these data support the idea that short-wavelength visible light is a potential risk factor for lens opacity.

Cataracts form due to high levels of reactive oxygen species (ROS) and oxidative stress in the lens [37]. The imbalance of ROS level in the lens causes oxidative alterations to crystallin proteins, leading to misfolding and aggregation [37]. These changes result in aggregates of crystallin that scatter light, causing the lens to lose transparency and develop the characteristic clouding of cataracts (Figure 7). However, most studies involve irradiance levels exceeding

typical digital device exposure. These results should not be directly equated with cataract risk from routine screen use.

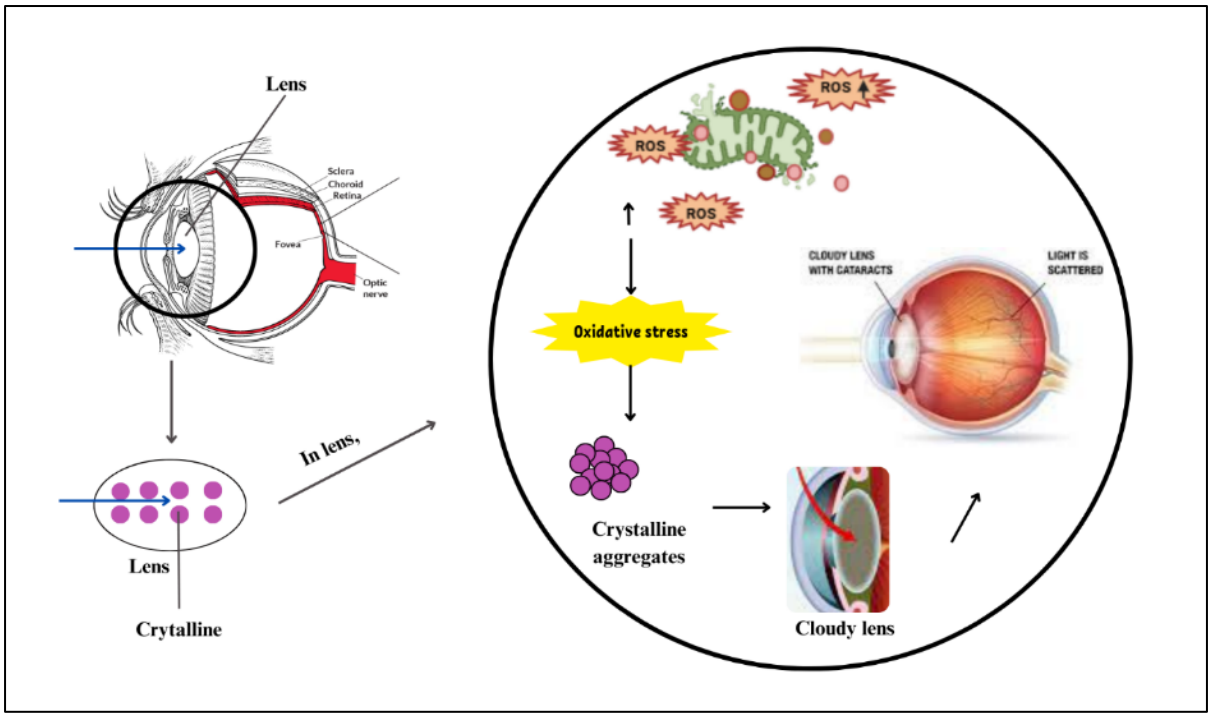

Figure 7. Schematic representation of the potential mechanism of cataract. Created by authors based on data from [24,25,37].

4.3.2. Therapeutic interventions.

Therapeutic interventions, such as blue-light-blocking intraocular lenses (IOLs), have revealed promising results in mitigating the adverse effects of blue light. In a clinical study, cataract patients who experienced surgery and received blue-light-blocking IOLs reported significant enhancements in sleep quality, as evidenced by decreases in Pittsburgh Sleep Quality Index (PSQI) scores [26]. These findings suggest that cataracts will delay blue light transmission and may disrupt melatonin regulation and circadian rhythms. By blocking blue light, these specialized IOLs not only improve vision but also restore circadian alignment, offering a dual advantage [26]. Together, these studies underscore the need for protective strategies against blue light exposure to prevent cataract development and improve overall ocular and systemic health.

Collectively, these findings deliver strong evidence for the cataractogenic effects of blue and violet light, which may contribute to the growing concern over the long-term impact of artificial light exposure on ocular health [24,25]. The use of blue-light-blocking lenses and intraocular implants seems to be a promising approach for protecting the eyes, slowing cataract progression, and improving related health outcomes such as sleep quality [26]. Further research is needed to understand the mechanisms behind blue light-induced cataracts and to explore the effectiveness of protective strategies in clinical settings.

This review has several limitations that should be considered. First, most of the evidence comes from high-intensity laboratory exposures using *ex vivo* models and animal studies, which may not fully reflect the effects of everyday blue light exposure from digital devices and other environmental sources on humans. Second, robust epidemiological studies linking typical human blue light exposure to ocular diseases such as AMD, RP, and cataract

are limited, restricting our ability to extrapolate experimental findings to population-level risk directly. Finally, most studies focus on short-term or acute exposures, leaving long-term consequences under physiologically relevant conditions largely unexplored. These limitations underscore the need for controlled human studies to clarify safe exposure levels and further validate the mechanistic insights identified in laboratory and animal research.

5. Conclusions

Blue light exposure, specifically at wavelengths between 400 nm and 480 nm, contributes to oxidative stress, mitochondrial dysfunction, and apoptosis, accelerating the progression of ocular diseases such as AMD, RP, and cataracts. Preventive measures, such as blue-light-blocking filters, intraocular lenses, and lifestyle modifications, have proven promising results in protecting retinal and lens health. As digital device use increases, raising awareness and normalizing protective strategies are crucial to reduce the risk of vision loss and promote long-term ocular health. While significant progress has been made in understanding the mechanisms of blue light-induced damage, particularly its role in oxidative stress, apoptosis, and retinal degeneration, significant gaps still exist. The current research lacks long-term, population-based studies assessing the efficacy of blue-light-blocking techniques in the real world. Future research should focus on longitudinal and interventional studies to evaluate the protective efficacy of blue-light filters, screen-time management, and pharmaceutical treatments. Clarifying these issues will be critical for producing evidence-based guidelines to reduce blue light-related ocular hazards.

Author Contributions

Conceptualization, F.I.S.A. and S.A.R.; methodology, F.I.S.A. and R.D.; validation, F.I.S.A., R.D., S.A.R., I.Z.H. and N.F.N.M.I.; data curation, F.I.S.A., R.D. and S.A.R.; writing—original draft preparation, F.I.S.A., R.D. and S.A.R.; writing—review and editing, F.I.S.A., R.D., S.A.R., I.Z.H. and N.F.N.M.I.; visualization, F.I.S.A.; supervision, R.D. and S.A.R. All authors discussed the results and commented on the manuscript. All authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest

The authors declare no conflict of interest.

References

1. Austin, E.; Geisler, A.N.; Nguyen, J.; Kohli, I.; Hamzavi, I.; Lim, H.W.; Jagdeo, J. Visible light. Part I: Properties and cutaneous effects of visible light. *J. Am. Acad. Dermatol.* **2021**, *84*, 1219-1231, https://doi.org/10.1016/j.jaad.2021.02.048.
2. Buxton, O. M.; Chang, A.-M.; Spilsbury, J. C.; Bos, T.; Emsellem, H.; Knutson, K. L. Sleep in the Modern Family: Protective Family Routines for Child and Adolescent Sleep. *Sleep Health* **2015**, *1*, 15, https://doi.org/10.1016/j.sleh.2014.12.002.
3. Palaiologou, C. Under Five and Digital Technologies: Implications for Early Years Pedagogy. *Eur. Early Child. Educ. Res. J.* **2014**, *24*, 5-24, https://doi.org/10.1080/1350293x.2014.929876.
4. Viner, R.M.; Gireesh, A.; Stiglic, N.; Hudson, L.D.; Goddings, A.-L.; Ward, J.L.; Nicholls, D.E. Roles of cyberbullying, sleep, and physical activity in mediating the effects of social media use on mental health and wellbeing among young people in England: a secondary analysis of longitudinal data. *Lancet Child Adolesc. Health* **2019**, *3*, 685-696. https://doi.org/10.1016/s2352-4642(19)30186-5.
5. Xia, H.; Hu, Q.; Li, L.; Tang, X.; Zou, J.; Huang, L.; Li, X. Protective Effects of Autophagy against Blue Light-Induced Retinal Degeneration in Aged Mice. *Sci. China Life Sci.* **2019**, *62*, 244–256, https://doi.org/10.1007/s11427-018-9357-y.
6. Cougnard-Gregoire, A.; Merle, B.M.; Aslam, T.; Seddon, J.M.; Aknin, I.; Klaver, C.C.; Garhöfer, G.; Layana, A.G.; Minnella, A.M.; Silva, R. Blue light exposure: ocular hazards and prevention—a narrative review. *Ophthalmol. Ther.* **2023**, *12*, 755-788, https://doi.org/10.1007/s40123-023-00675-3.
7. Chen, A.-H.; Rosli, S.A.; Basri, R.; Hoe, C.Y.W. Investigation of Screen Time Inclination and the Accompanying Visual and Musculoskeletal Discomfort in Young Smartphone Users. *Trends Sci.* **2022**, *19*, 1753, https://doi.org/10.48048/tis.2022.1753.
8. Maayah, M.F.; Nawasreh, Z.H.; Gaowgzeh, R.A.M.; Neamatallah, Z.; Alfawaz, S.S.; Alabasi, U.M. Neck Pain Associated with Smartphone Usage among University Students. *PLoS ONE* **2023**, *18*, e0285451, https://doi.org/10.1371/journal.pone.0285451.
9. Clark, S.J.; Bishop, P.N. The Eye as a Complement Dysregulation Hotspot. *Semin. Immunopathol.* **2017**, *40*, 65-74, https://doi.org/10.1007/s00281-017-0649-6.
10. Wong, N.A.; Bahmani, H. A Review of the Current State of Research on Artificial Blue Light Safety as It Applies to Digital Devices. *Heliyon* **2022**, *8*, e10282, https://doi.org/10.1016/j.heliyon.2022.e10282.
11. Apte, R.S. Age-Related Macular Degeneration. *N. Engl. J. Med.* **2021**, *385*, 539, https://doi.org/10.1056/nejmcp2102061.
12. Verbakel, S.K.; van Huet, R.A.; Boon, C.J.; den Hollander, A.I.; Collin, R.W.; Klaver, C.C.; Hoyng, C.B.; Roepman, R.; Klevering, B.J. Non-syndromic retinitis pigmentosa. *Prog. Retin. Eye Res.* **2018**, *66*, 157-186, https://doi.org/10.1016/j.preteyeres.2018.03.005.
13. Song, P.; Wang, H.; Theodoratou, E.; Chan, K.Y.; Rudan, I. The National and Subnational Prevalence of Cataract and Cataract Blindness in China: A Systematic Review and Meta-Analysis. *J. Glob. Health* **2018**, *8*, https://doi.org/10.7189/jogh.08.010804.
14. Liberati, A.; Altman, D.G.; Tetzlaff, J.; Mulrow, C.; Gøtzsche, P.C.; Ioannidis, J.P.A.; Clarke, M.; Devereaux, P.J.; Kleijnen, J.; Moher, D. The PRISMA Statement for Reporting Systematic Reviews and Meta-Analyses of Studies That Evaluate Healthcare Interventions: Explanation and Elaboration. *BMJ* **2009**, *339*, https://doi.org/10.1136/bmj.b2700.

15. Núñez Álvarez, C.; Suárez Barrio, C.; Aguado, S.D.O.; Osborne, N.N. Blue Light Negatively Affects the Survival of ARPE19 Cells through an Action on Their Mitochondria and Blunted by Red Light. *Acta Ophthalmol.* **2018**, *97*, e103-e115, https://doi.org/10.1111/aos.13812.
16. Alaimo, A.; Liñares, G.G.; Bujjamer, J.M.; Gorojod, R.M.; Alcon, S.P.; Martínez, J.H.; Baldessari, A.; Grecco, H.E.; Kotler, M.L. Toxicity of blue led light and A2E is associated to mitochondrial dynamics impairment in ARPE-19 cells: implications for age-related macular degeneration. *Arch. Toxicol.* **2019**, *93*, 1401-1415, https://doi.org/10.1007/s00204-019-02409-6.
17. Fietz, F.C.; Hurst, J.; Schnichels, S. Blue Light Damage and P53: Unravelling the Role of P53 in Oxidative-Stress-Induced Retinal Apoptosis. *Antioxidants* **2023**, *12*, 2072, https://doi.org/10.3390/antiox12122072.
18. Marie, M.; Gondouin, P.; Pagan, D.; Barrau, C.; Villette, T.; Sahel, J.; Picaud, S. Blue-violet light decreases VEGFa production in an in vitro model of AMD. *PLoS One* **2019**, *14*, e0223839, https://doi.org/10.1371/journal.pone.0223839.
19. Kawasaki, S.V.C.; Kardon, R.; Leon, L.; Hamel, C. Characterization of Pupil Responses to Blue and Red Light Stimuli in Autosomal Dominant Retinitis Pigmentosa Due ToNR2E3Mutation. *Invest. Ophthalmol. Vis. Sci.* **2012**, *53*, 5562, https://doi.org/10.1167/iovs.12-10230.
20. Stingl, K.T.; Kuehlewein, L.; Weisschuh, N.; Biskup, S.; Cremers, F.P.; Khan, M.I.; Kelbsch, C.; Peters, T.; Ueffing, M.; Wilhelm, B. Chromatic full-field stimulus threshold and pupillography as functional markers for late-stage, early-onset retinitis pigmentosa caused by CRB1 mutations. *Transl. Vis. Sci. Technol.* **2019**, *8*, 45, https://doi.org/10.1167/tvst.8.6.45.
21. Orlans, H.O.; Merrill, J.; Barnard, A.R.; Issa, P.C.; Peirson, S.N.; MacLaren, R.E. Filtration of Short-Wavelength Light Provides Therapeutic Benefit in Retinitis Pigmentosa Caused by a Common Rhodopsin Mutation. *Invest. Ophthalmol. Vis. Sci.* **2019**, *60*, 2733, https://doi.org/10.1167/iovs.19-26964.
22. Pastor-Idoate, S.; Mateos-Olivares, M.; Sobas, E.M.; Marcos, M.; Toribio, A.; Pastor, J.C.; Martín, R.U. Short-Wavelength Light–Blocking Filters and Oral Melatonin Administration in Patients With Retinitis Pigmentosa: Protocol for a Randomized Controlled Trial. *JMIR Research Protocols* **2023**, *12*, e49196, https://doi.org/10.2196/49196.
23. Cedrún-Sánchez, J.E.; Chamorro, E.; Bonnin-Arias, C.; Aguirre-Vilacoro, V.; Castro, J.J.; Sánchez-Ramos, C. Visual Discrimination Increase by Yellow Filters in Retinitis Pigmentosa. *Optom. Vis. Sci.* **2016**, *93*, 1537, https://doi.org/10.1097/opx.0000000000000924.
24. Haag, R.; Sieber, N.; Heßling, M. Cataract Development by Exposure to Ultraviolet and Blue Visible Light in Porcine Lenses. *Medicina* **2021**, *57*, 535, https://doi.org/10.3390/medicina57060535.
25. Zeller, K.; Mühleisen, S.; Shanmugarajah, P.; Fehler, N.; Haag, R.; Hessling, M. Influence of Visible Violet, Blue and Red Light on the Development of Cataract in Porcine Lenses. *Medicina* **2022**, *58*, 721, https://doi.org/10.3390/medicina58060721.
26. Wei, X.; She, C.; Chen, D.; Yan, F.; Zeng, J.; Zeng, L.; Wang, L. Blue-light-blocking intraocular lens implantation improves the sleep quality of cataract patients. *J. Clin. Sleep Med.* **2013**, *9*, 741-745, https://doi.org/10.5664/jcsm.2908.
27. Marchesi, N.; Capierri, M.; Pascale, A.; Barbieri, A. Different Therapeutic Approaches for Dry and Wet AMD. *Int. J. Mol. Sci.* **2024**, *25*, 13053, https://doi.org/10.3390/ijms252313053.
28. Heesterbeek, T.J.; Lorés Motta, L.; Hoyng, C.B.; Lechanteur, Y.T.E.; Hollander, A.I. D. Risk Factors for Progression of Age?Related Macular Degeneration. *Ophthalmic Physiol. Opt.* **2020**, *40*, 140-170, https://doi.org/10.1111/opo.12675.
29. Stahl, A. The Diagnosis and Treatment of Age-Related Macular Degeneration. *Dtsch Arztebl International* **2020**, *117*, 513-519, https://doi.org/10.3238/arztebl.2020.0513.
30. Froger, N.; Matonti, F.; Roubeix, C.; Forster, V.; Ivkovic, I.; Brunel, N.; Baudouin, C.; Sahel, J.-A.; Picaud, S. VEGF is an autocrine/paracrine neuroprotective factor for injured retinal ganglion neurons. *Sci. Rep.* **2020**, *10*, 12409, https://doi.org/10.1038/s41598-020-68488-z.
31. Saint-Geniez, M.; Maharaj, A.S.; Walshe, T.E.; Tucker, B.A.; Sekiyama, E.; Kurihara, T.; Darland, D.C.; Young, M.J.; D'Amore, P.A. Endogenous VEGF is required for visual function: evidence for a survival role on Müller cells and photoreceptors. *PloS one* **2008**, *3*, e3554, https://doi.org/10.1371/journal.pone.0003554.
32. Mateos-Olivares, M.; Pastor-Idoate, S.; Martín-Vallejo, J.; García-Vazquez, C.; Pastor, J.C.; Usategui-Martín, R.; Sobas, E.M. Stress and sleep deprivation-related biomarkers in saliva in patients with retinitis pigmentosa. *PloS one* **2024**, *19*, e0304261, https://doi.org/10.1371/journal.pone.0304261.
33. Liu, W.; Liu, S.; Li, P.; Yao, K. Retinitis Pigmentosa: Progress in Molecular Pathology and Biotherapeutical Strategies. *Int. J. Mol. Sci.* **2022**, *23*, 4883, https://doi.org/10.3390/ijms23094883.

34. Lam, D.; Rao, S.K.; Ratra, V.; Liu, Y.; Mitchell, P.; King, J.; Tassignon, M.-J.; Jonas, J.; Pang, C.P.; Chang, D.F. Cataract. *Nat. Rev. Dis. Primers* **2015**, *1*, 15014, https://doi.org/10.1038/nrdp.2015.14.
35. Nizami, A.A.; Gurnani, B.; Gulani, A.C.; Redmond, S.B. Cataract (nursing). In StatPearls [Internet]; StatPearls Publishing: **2024**.
36. Wear sunglasses, stop smoking and other ways to prevent cataracts. Available online: https://health.osu.edu/health/eye-health/cataract-prevention (accessed on 11th December **2024**).
37. Babizhayev, M.A. 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*, 183, https://doi.org/10.1002 /cbf.1737.

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