



## Review

# The evidentiary basis and challenges associated with studying the role of light damage on ocular health

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## ABSTRACT

The interaction between light and the eye is both essential and potentially harmful. Light regulates ocular development, supports vision, and influences systemic physiology, yet it can also induce photochemical damage. Determining whether chronic exposure to high-energy visible (HEV) light contributes to age-related ocular disease remains challenging. Randomized clinical trials, the gold standard for medical evidence, are largely infeasible for exposures that are lifelong, ubiquitous, and potentially harmful. Instead, researchers rely on a spectrum of evidence, from cellular and animal models to case studies, cohort studies, and expert consensus, each with unique advantages and limitations. This review categorizes existing evidence according to study design, evaluates its strengths and weaknesses, and considers how converging findings might inform clinical practice. We conclude that while acute light injury is well established, the long-term impact of low-level HEV exposure remains largely and necessarily inferential. Definitive evidence is difficult to obtain but improvements in exposure assessment and integration across different categories of evidence have led to a cautious but reasonable conclusion on the potential harms of long-term exposure.

## Preface

The electromagnetic spectrum spans a broad range of wavelengths, with visible light occupying a narrow band from approximately 380 nm (violet) to 780 nm (deep red). Light, however, as short as 315 nm can be reliably detected in adults under 30 years of age.<sup>1</sup> The human eye is a highly specialized organ adapted to detect and interpret light, and its anatomy and physiology are finely tuned to respond to the intensity, wavelength, and duration of light stimuli to ensure clear and accurate vision across a wide range of environments. Ocular tissues are also susceptible to damage by light based on that very diversity.<sup>2</sup> Ocular phototoxicity is typically categorized into photothermal (e.g., laser photocoagulation), photomechanical (e.g., Nd:YAG iridotomy), and photochemical mechanisms.<sup>3</sup> Photochemical damage is additive in most situations (e.g., where the Bunsen-Roscoe law applies) and arises from light-induced chemical reactions generating deleterious species (e.g., light-activated A2E leading to singlet oxygen and associated oxidative stress implicated in macular degeneration).

Studying light-induced ocular damage is obviously important in order to protect vision across the lifespan and for guiding industry in the development of mitigative technologies. Investigating the underlying mechanisms can inform safer lighting practices, display technologies, and therapeutic interventions. Studying light effects on the eye is

inherently challenging, however, due to multiple interrelated factors such as: variability in exposure durations to a wide range of wavelengths and light intensities; the interleaving of photothermal, photomechanical, and photochemical processes; latency periods before observable damage; substantial inter-individual variability; and the reliance on non-invasive assessment approaches. Additionally, translational gaps between model systems and human biology, alongside ethical and practical constraints, complicate inference. The primary aim of this narrative review is to highlight these challenges and synthesize current understanding of ocular light damage to inform prevention and management strategies.

## Introduction

Addressing the question of whether chronic exposure to high-energy visible light (380–500 nm)<sup>4,5</sup> plays a role in the pathogenesis of age-related ocular diseases presents several challenges. Age-related macular degeneration (AMD) is the most prevalent cause of blindness in many developed countries; however, only about 1–2% of those affected will, ultimately, completely lose their vision due to the disease.<sup>6</sup> Prospective longitudinal studies must monitor thousands of participants to generate a sufficient number of cases for meaningful analysis of the numerous etiological factors involved. For instance, Buitendijk et al. (2013)<sup>7</sup> tracked

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over 10,000 older adults for 11 years, resulting in only 363 cases of late AMD. This is a relatively small number given the relatively wide array of often-interacting etiological factors that must be considered; ranging from genetic predispositions to lifestyle choices, all differing across individuals and geographic regions.<sup>8</sup> The impact of each factor, when considered independently, may appear minor; however, they are frequently cumulative.<sup>9</sup> For example, an increase in disease risk by just 1–2% might be negligible when considered over a few years but could become significant over several decades due to the compounding effect of risk accumulation. While longitudinal studies provide one solution, they require extended timeframes (often exceeding many careers) and are typically very costly to conduct.

Another challenge lies in the relative importance of risk and protective factors across different moderators, such as individual traits, generational timeframes, and geographical regions (e.g., distance from the equator). Such large variation is difficult to quantify. Chronic light exposure, for instance, is particularly challenging to measure accurately over extended periods.<sup>10</sup> In contrast, intense acute light exposure is relatively straightforward to quantify, as are its biological effects, especially on outer structures such as the skin (e.g., sunburn) or sclera (e.g., scleritis or episcleritis). In contrast, assessing the biological effects of low-level light exposure over time presents significant measurement difficulties. For example, in healthy individuals, the loss of transparency in the crystalline lens increases consistently over time, driven largely by ultraviolet light, leading to opacification that can be well described by a linear regression model.<sup>11</sup> The age-related change in lens optical density (O. D.) amounts to approximately 0.01–0.02 per year, which translates to an O.D. change of about 0.05–0.10 at the shortest wavelengths (~410 nm) over a five-year span.<sup>12,13</sup> Notably, this degree of change is nearly equivalent to the measurement error associated with assessing lens density for a single individual in a single experimental session. For example, Grewal et al. (2009)<sup>14</sup> report a measurement error of approximately  $\pm 0.05$  to  $\pm 0.10$  OD units when employing Scheimpflug imaging to evaluate lens density. Most current methodologies lack the precision required to detect the subtle changes that could result from light damage to the eye over relatively short periods, such as five years.

Even if exposure can be accurately characterized throughout an individual's lifetime, its effects are influenced by numerous between-subject factors, such as diet, ocular melanin, eyewear, behavior, crystalline lens density, and macular pigment. This variability highlights why within-subject designs are often more powerful than between-subject comparisons, since individuals effectively experience different retinal light environments even under identical external illumination. For example, crystalline lens optical density at 407 nm can vary by more than a log unit even among young individuals,<sup>15</sup> affecting the amount of high-energy light that reaches the retina. Additionally, individual differences in macular pigment (MP) represent one of the most variable endogenous spectral filters of the fovea, even in young children. At peak optical density (460 nm), for instance, children can exhibit MP levels ranging from a high of 100% transmission to a low of around 5–10%.<sup>16</sup> Such significant variation over decades can result in substantial differences in the amount of potentially harmful short-wave light reaching the sensitive outer segments of photoreceptors (photopigment itself is a potent photosensitizer), or the underlying retinal pigment epithelium (RPE). Even a nutritious meal rich in photoprotective factors before exposure to intense light may mitigate some of the harmful effects of that exposure.<sup>17</sup> The multitude of interacting covariates makes it challenging to quantify these effects over time.

Importantly, many of these sources of variability are not merely methodological noise but represent biologically meaningful modifiers of retinal light exposure. The crystalline lens and MP, for instance, continuously shape the spectral and energetic composition of light reaching photoreceptors and the retinal pigment epithelium. The crystalline lens progressively increases in optical density across the lifespan, preferentially attenuating short-wavelengths, while macular pigment selectively absorbs light centered near 460 nm. Together with ocular melanin, pupil

size, and behavioral factors such as gaze direction and blinking, these elements determine the effective retinal irradiance. This means that two observers exposed to identical light conditions may experience substantially different doses of light at the level of their retina. For example, an individual with high macular pigment optical density and a relatively dense crystalline lens may receive only a fraction of the short-wave retinal irradiance experienced by a younger observer with clearer ocular media and low macular pigment. Retinal exposure is not defined solely by the external lighting environment but by a personalized optical filtering system intrinsic to the eye itself. This biological modulation complicates attempts to estimate cumulative exposure and helps explain why epidemiological associations between ambient light and ocular outcomes are often weak or inconsistent despite well-established mechanisms of acute photochemical injury. Studies of light exposure in humans resemble studies of radiation exposure without personal dosimeters: the relevant dose is internal rather than environmental.

Can the findings from studies utilizing high-intensity light exposure for short intervals be applied to predict the long-term effects of prolonged low-intensity light exposure in real life? Some researchers in the area have argued that they likely generalize.<sup>18</sup> Numerous light safety standards recommend limiting exposure to short-wave light.<sup>19</sup> This type of light can largely penetrate the crystalline lens, particularly in younger individuals, reaching wavelengths down to 315 nm.<sup>1</sup> Short-wave light passed through the cornea and crystalline lens is still sufficiently energetic to cause photochemical damage to the retina.<sup>20,21</sup> Research on the accumulation of lipofuscin (oxidized lipids) throughout life indicates a linear increase between the ages of approximately 10 and 70, with an escalation of roughly 30% every decade (although note that this is a rough approximation, the slope of the function varies significantly by age and the retinal eccentricity measured<sup>22,23</sup>). This suggests that significant "aging" of the retina-RPE begins in childhood. This relation itself, however is complex. Children possess more transparent ocular media, resulting in greater light stress but lower overall levels of photosensitizer; as photosensitizer levels rise, the media becomes increasingly opaque.<sup>24</sup> Recent laboratory findings indicate that UV and short-wave light are also necessary for proper emmetropization,<sup>25</sup> although real-world data suggest otherwise.<sup>26,27</sup> How do we distinguish between necessary light exposure and excessive exposure? This question has prompted the exploration of using less actinic red light as a tool for facilitating proper emmetropization.<sup>28</sup>

How does one integrate data that comes from so many methods, models and domains? Many real-world queries are not conducive to experimental studies (due to ethical and practical constraints), particularly those involving the "gold standard" of randomized clinical trials. Should we adopt a different evidentiary standard for preventive strategies compared to those we utilize for evaluating treatments for individuals with pre-existing conditions?

## Overview of evidentiary categories

### *Anecdotal and expert opinions*

Anecdotal evidence refers to individual observations, case reports, or unstructured patient stories that may hint at patterns or associations but are not controlled data. Expert opinions come from individuals with substantial domain experience (clinicians, researchers, regulatory scientists) and often contribute through structured consensus processes or narrative insights. In ocular research, these inputs can help identify rare events, novel phenomena, or practical considerations that formal studies might overlook. Expert opinion in the field of ocular phototoxicity is not simply speculative. A broad international consensus exists regarding the potential for acute retinal injury from sufficiently intense light exposure. This agreement is reflected in established photobiological safety standards developed by organizations such as the American National Standards Committee and the International Commission on Illumination (CIE), which define spectral weighting functions and exposure limits for

the blue-light hazard.<sup>5,19</sup> These standards, widely adopted in clinical practice, industry design, and occupational safety, indicate strong expert agreement that short-wavelength visible light can produce retinal photochemical damage when exposure exceeds defined thresholds. Thus, the existence of a blue-light hazard under acute conditions is largely untested; debate instead centers on whether chronic, low-level exposures encountered in everyday environments produce clinically meaningful long-term effects.

A review<sup>29</sup> conducted in 2023 by Hipólito et al. identified 4237 articles pertaining to blue light and its potential ocular damage. Organizations such as the International Commission on Illumination have issued position statements that define exposure limits and note that exceeding these limits can result in light-induced damage.<sup>29</sup> Many clinical practices actively address these concerns. For instance, incorporating UV chromophores into intraocular implants has become a standard practice.<sup>30</sup> Numerous brands of intraocular lenses (IOLs)<sup>30–32</sup>, spectacles<sup>33</sup>, and contact lenses<sup>34</sup> are designed to absorb high-energy visible (HEV) light. Professions utilizing light for therapeutic purposes adhere closely to safety guidelines. For example, blue light therapy is employed to break down bilirubin in infants suffering from jaundice<sup>35</sup>, and dentists use HEV light to cure dental composites.<sup>36</sup> In such instances, light-curing instruments are often equipped with filters situated near the tip (such as amber-tinted dental curing light shields) to protect users from the emitted light.

In recent decades, the general public has become increasingly skeptical of expert opinions. The growing use of the internet and social media has democratized information, often obscuring the lines between genuine expertise and misinformation. This environment has fostered echo chambers<sup>37</sup> and eroded trust in traditional authorities, such as scientists. Political polarization has further intensified this distrust often along ideological lines.<sup>38</sup> Fields of study that are inherently complex, such as light damage, are particularly vulnerable to this type of skepticism.<sup>39,40</sup>

#### *Animal and cell studies*

Due to various challenges, many studies in the light damage literature rely on laboratory data derived from acute, intense, and monochromatic exposures.<sup>41</sup> It is reasonable to question the generalizability of such findings. A common question is whether *ex vivo* cellular models can effectively characterize how actinic light affects a living eye over time and at lower intensities. For instance, one approach<sup>42,43</sup> for assessing the risks associated with exposure to blue light from digital devices involves exposing retinal pigment epithelial (RPE) cells, often loaded with a photosensitizer (e.g., N-retinylidene-N-retinylethanolamine, or A2E), to prolonged illumination with short-wave light. RPE cell senescence is frequently targeted since it is believed that the degeneration of these cells plays a critical role in the development of degenerative retinal diseases.<sup>44</sup> In a living eye, however, short-wave light does not directly illuminate photosensitizer-laden RPE cells for extended periods due to factors such as overlying retinal layers, pre-retinal filters, individual blinking, and gaze shifts. The primary aim of cellular studies is to unravel mechanisms. Are these mechanisms, however, the same *in situ*? Similar questions also arise in other fields. How similar are cancer cells cultivated in 2D monolayer cultures to tumors present in living organisms?<sup>45</sup> Tumors engage actively with their surrounding tissues, blood vessels, immune factors, and more. As a result, chemotherapy that proves effective in *ex vivo* settings often fails in actual patients; for instance, tumors may employ protective mechanisms such as hypoxia and drug efflux pumps.

Research examining the impact of light on ocular damage in humans frequently employs animal and cell culture models. Notably, it was through action spectra studies on rats<sup>46</sup> that the idea of light overexposure as potentially harmful gained traction, despite being acknowledged by clinicians Duke-Elder and MacFaul as early as the 1920s.<sup>3</sup> How well animals model humans is also a good question. Rats are nocturnal with rod-dominated retinas consisting of only about 1% cones<sup>47</sup> and a relatively short lifespan. Similar criticisms have been directed at avian

models. In contrast, primates, although more costly, may provide a more suitable model.<sup>48</sup> A study by Sykes et al. (1981)<sup>49</sup> demonstrated that acute exposure to broad-spectrum fluorescent light (approximately 25,000 lx for 12 h over a span of up to four days) inflicted structural damage to the macula and paramacular regions of monkey retinas. In comparison, control eyes that were patched during the exposure showed no structural abnormalities at any exposure level. Even monkeys, however, seem to have a lower threshold for photochemical retinal injury compared to humans. For example, a 15-minute exposure to retinal irradiance of 0.27 W/cm<sup>2</sup> from an indirect ophthalmoscope led to significant damage to photoreceptors and alterations in the retinal pigment epithelium in monkeys.<sup>3</sup>

Animal models have proven to be quite valuable in elucidating the mechanisms and effects of acute light damage (refer to Table 1 in MacFarlane et al, for studies on cataract formation).<sup>50</sup> However, their utility in assessing the significance of long-term damage resulting from less intense exposures has been more limited. For instance, Cuthbertson et al. (2009)<sup>51</sup> conducted a review of the literature on light damage, primarily focusing on animal studies, and remarked, "It would seem sensible to reduce short-wavelength light exposure using blue light-filtering IOLs, especially in cases where damage may be accelerated due to underlying pathology, such as early AMD." Sensible but the evidence is lacking. The effects of blue light-filtering IOLs on the progression of retinal diseases remain mixed.<sup>52</sup> A recent<sup>53</sup> population-based cohort study evaluated whether BLF-IOLs influence the risk of developing exudative AMD stratifying patients by their baseline status of non-exudative AMD. The investigators found no overall protective effect of BLF-IOLs against progression to exudative AMD compared with standard IOLs. It is conceivable that interventions need to occur much earlier to successfully influence degenerative diseases that likely begin at an early stage in life.

#### *Case reports and series*

Numerous clinical cases illustrate the acute effects of light damage on the eye. For instance, solar burns affecting the sclera, known as pinguecula, are prevalent among children in sunny regions such as Australia.<sup>54</sup> Patients who observe eclipses without appropriate protection frequently sustain specific retinal damage.<sup>55</sup> Liang et al. (2017)<sup>56</sup> detailed a case of a patient who experienced visual disturbances, including a central scotoma and reduced visual acuity, following accidental exposure to a high-intensity blue laser, which was (somewhat ironically) attributed to regular nightclub lighting. Ophthalmologic examination of this patient, including optical coherence tomography, revealed localized macular damage characterized by disruption of the photoreceptor and retinal pigment epithelium layers.<sup>56</sup> These reports demonstrate that brief but intense exposures can lead to significant ocular damage in humans.

While case studies on chronic light exposure related to ocular issues are relatively scarce, intriguing examples exist concerning dermal effects, such as unilateral dermatoheliosis. Gordon et al. (2012)<sup>57</sup> recount the case of a 69-year-old man who exhibited pronounced thickening and wrinkling of the skin on the left side of his face. A clinical examination revealed hyperkeratosis with distinct ridging, multiple open comedones, and areas of nodular elastosis. These findings were attributed to chronic ultraviolet (UV) exposure, as the patient had worked as a delivery truck driver for 28 years, leading to consistent sunlight exposure on the left side of his face through the vehicle's window. Furthermore, Weiss (2016)<sup>58</sup> noted that American truck drivers are generally more prone to developing left-sided cortical cataracts due to this asymmetrical UV exposure. While front windshields are often treated to block a significant portion of UV radiation, side windows are typically not, and they may be left open.

#### *Case-control studies*

Case-control studies are observational analytic studies that compare individuals with a specific disease or outcome (cases) to individuals

without the disease (controls) in order to look back in time to assess prior exposure to potential risk factors. For instance, Putnam et al.<sup>59</sup> investigated dentists who utilized blue lasers in their practice (cases) against a control group of participants without such exposure. The findings indicated that the dentists exhibited signs of retinal light damage. Additionally, Yang et al.<sup>60</sup> discovered that welders have higher rates of phototoxic maculopathy compared to non-welders. In certain cases, subjects can even serve as their own controls.

The first intraocular implants (IOLs) made from polymethyl methacrylate did not provide UV light protection to the retina. As a result, patients with these implants reported perceiving violet hues when exposed solely to UV light.<sup>61</sup> In a study by Werner et al. (1989)<sup>31</sup>, the researchers compared retinal sensitivity in patients with a UV-transmitting IOL in one eye and a UV-blocking IOL in the other. Over a period of five years, S-cone loss was significantly more pronounced in the UV-transmitting eye. This study aligns with the accelerated light damage observed in aphakic patients.<sup>21</sup>

#### *Cohort studies (following groups over time)*

Cohort studies are observational analytic studies that start with a defined group of individuals based on exposure status and follow them over time to observe the development of outcomes. They allow estimation of how often an outcome occurs in the exposed group compared with the unexposed group, thereby quantifying risk. Groups can be studied either prospectively or retrospectively. For instance, in the Alienor Study conducted in Bordeaux, France<sup>62</sup>, elderly participants in the highest quartile of lifetime ambient UV exposure exhibited a 53% increased risk of cataract extraction and a 59% higher risk of early age-related macular degeneration (AMD) compared to those with moderate exposure levels. This exposure was estimated based on residential history and satellite-based assessments of light ambience in specific locales. In another example, the Chesapeake Bay Watermen Study<sup>63</sup> examined 838 watermen. Results indicated that those with higher lifetime UV-B exposure faced a significantly elevated risk of developing cortical cataracts. Specifically, a doubling of lifetime UV-B exposure correlated with a 60% increase in the risk of cortical cataracts. Past UV-B exposure for these participants was assessed through detailed occupational histories, coupled with laboratory and field measurements of ocular UV-B exposure.

Prospective studies are generally regarded as more convincing than retrospective studies; however, they tend to be more costly and require longer durations to complete. Kim et al.<sup>64</sup> conducted an investigation into the relationship between long-term exposure to outdoor artificial light at night (OALAN) and the risk of developing exudative age-related macular degeneration (EAMD) in South Korea. The study included individuals aged 50 and older ( $n = 126,418$ ) and identified new cases of EAMD diagnosed between 2010 and 2011 ( $n = 4078$ ). Participants were followed prospectively until 2020, resulting in a follow-up period of up to 10 years. OALAN levels were estimated using satellite-based nighttime light imaging data, which were matched with the participants' residential districts, while controlling for factors such as age, sex, smoking status, comorbidities, income level, and residential urbanization. The findings revealed that individuals residing in areas with the highest quartile of OALAN exposure had a significantly greater risk of developing incident EAMD compared to those in the lowest quartile. This association was particularly pronounced in highly urbanized regions, where ambient nighttime light levels are typically elevated. It is worth noting that the cases analyzed in this study comprised approximately 3% of the enrolled participants and were monitored for about 10 years.

One of the challenges identified in the Watermen study<sup>63</sup> is the difficulty in quantifying chronic light exposure. Questionnaires provide one approach, albeit a somewhat crude one. Alternative methods include estimating light exposure through geographic data, satellite

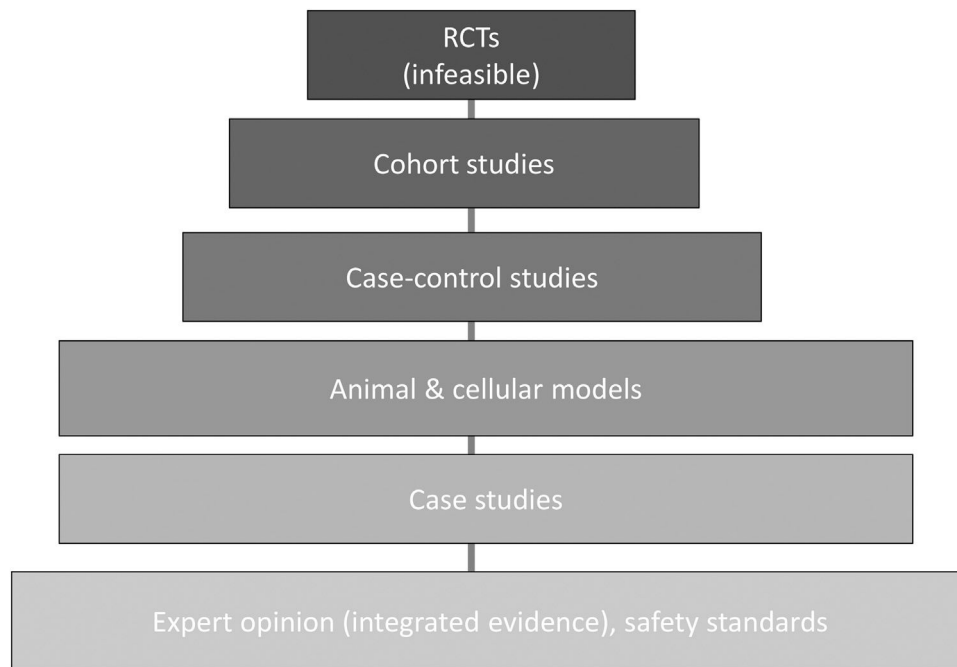
measurements, and photometric or radiometric assessments. Wearable technology presents a more promising avenue for the future.<sup>65,66</sup> Similar to how actigraphy has enhanced the measurement of physical activity, wearable devices could play a crucial role in quantifying daily or significant light exposures.<sup>66</sup> These devices, such as photosensitive elements integrated into spectacle frames or actigraphy watches, could connect to applications that offer personalized recommendations for light exposure. For instance, an app might suggest increased short-wave exposure in the morning and reduced exposure at night to help optimize circadian rhythms while avoiding overexposure.

#### *Randomized controlled trials*

In the field of medicine, double-blinded randomized controlled trials (RCTs) are widely regarded as the gold standard for making causal inferences, as affirmed by the judgments of most regulatory boards.<sup>67</sup> These experimental designs are particularly effective for many types of interventions, especially pharmaceuticals. For instance, it is relatively straightforward to identify individuals who have not previously been exposed to a specific drug, randomly assign them to either a treatment or a control group, create a suitable placebo, and monitor them for a brief period, since the effects of drugs typically manifest relatively quickly. However, this design becomes nearly impossible when investigating issues such as whether chronic overexposure to light negatively impacts ocular health; consequently, no such trials exist. For example, a control group does not exist that has never been exposed to the treatment (i.e., light). Additional questions arise about the duration of the follow-up, the specific outcomes to be measured, and the ethical considerations of exposing a treatment group to a potentially harmful intervention.

Sackett et al. (1997),<sup>68</sup> an early advocate of evidence-based medicine, asserted nearly 30 years ago that the evidence framework in medicine should "not be limited to randomized trials and meta-analyses." This notion is particularly relevant for topics that do not easily lend themselves to such studies. In the absence of RCTs, however, some have reasonably questioned the validity of the hypothesis that long-term exposure to mostly low intensity short-wave light in humans living in the natural world is truly a risk factor. Mainster et al.<sup>39</sup> contend that normal environments do not pose a blue-light hazard and that the century-old phototoxicity-AMD hypothesis remains unproven. Such statements raise an important question: what type of evidence is necessary to conclusively prove or disprove this hypothesis?

At a minimum, it would necessitate a large-scale, prospective, multi-center longitudinal cohort study, ideally integrated with mechanistic animal and cellular research. Such a study would likely need to enroll over 100,000 diverse, healthy individuals aged 18–40 at baseline (younger subjects would be preferable, but this would raise ethical concerns and extend the duration of the study). To achieve optimal results, subjects would ideally be equipped with personal dosimeters to continuously monitor cumulative exposure to UV-A, UV-B, and blue-violet visible light over the course of the study. Comprehensive ophthalmic evaluations, which could include assessments such as fundus photography, optical coherence tomography, psychophysics (e.g., isolated S-cone sensitivity), lens and macular pigment optical density, and retinal electrophysiology would be beneficial. These assessments would be optimally conducted biennially, to identify both subclinical and clinical ocular pathologies. Primary outcomes could focus on the incidence of age-related macular degeneration, cataracts, pterygium, photokeratitis, and retinal pigment epithelium atrophy, while secondary outcomes might evaluate functional visual impairments and ocular surface disease. Notably, a study duration of 40 to 50 years would likely be necessary. In the absence of such direct data (with costs estimated well over a billion US dollars), researchers may rely on a convergence of findings from multiple fields, from laboratory studies to clinical practice and fieldwork, to build a comprehensive understanding.



**Fig. 1.** Evidentiary pyramid depicting the hierarchy of study designs contributing to our current understanding of light-induced ocular damage. Randomized controlled trials sit at the apex but are rarely feasible for questions involving lifelong, ubiquitous exposure. Cohort and case-control studies provide population-level associations, while case reports, animal models, cellular preparations, and expert consensus offer mechanistic and observational guidance. Clinical inference requires integrating evidence across all these tiers.

## Conclusion

The effects of light stress on the eye depend primarily on wavelength, timing, and cumulative dose. Just as proper auditory input is essential for optimal auditory development, excessive sound exposure can damage hair cells within the cochlea. Similarly, light exposure can have beneficial, detrimental, or neutral effects depending on specific circumstances. While acute intense exposures are evidently harmful, the long-term effects of low-level cumulative light exposure are much more difficult to quantify and study. This difficulty arises from variability in individual susceptibility, environmental factors, and protective behaviors.

Evidence must be based on a convergence of data across study types (summarized in Fig. 1) as opposed to definitive experimental trials. As Sackett,<sup>68</sup> and others<sup>69</sup> have pointed out, clinical decision-making often necessitates relying on the best available evidence, even when it lacks ideal experimental rigor. Until comprehensive longitudinal, multi-center studies integrating human, animal, and mechanistic research can be conducted, it is advisable for clinicians and public health officials to adopt a precautionary approach, carefully balancing the necessary benefits of light exposure for both ocular and systemic health against the potential risks of cumulative harm. Thoughtfully designed wearables, enhanced exposure assessment tools, and collaborative epidemiological networks could help bridge these long-standing gaps, allowing us to better understand the intricate relationship between light and ocular aging in the coming decades. For instance, smart contact lenses are currently in development to monitor actinic light exposure over time, although they may also shield the eye, potentially complicating the assessment of exposure effects.<sup>70</sup>

Importantly, these challenges also suggest that progress may depend not only on improved exposure measurement but on reconsidering how outcomes are defined. It may be beneficial to shift the focus of light damage studies away from an endpoint based solely on ocular disease. Health encompasses more than just the absence of illness. One advantage of vision science is the ability to quantify function with precision. Careful psychophysics can detect even subtle visual changes,<sup>71</sup> while direct optical methods, such as adaptive optics, can assess anatomical

changes.<sup>72</sup> Additionally, physiological methods, such as multifocal ERG, can evaluate changes before clinical symptoms manifest.<sup>73</sup> One of the primary goals of preventive medicine is to optimize function. Emphasizing health-span rather than simply lifespan and disease marks a significant reorientation within the health sciences.

## Author contributions

BRH conceived the topic, conducted the literature search and synthesis, drafted the initial manuscript, and prepared all figures and tables. JB also contributed to the conception of the topic, to the literature review, assisted with interpretation of key studies, and provided critical revisions to the manuscript. Both authors reviewed and approved the final version of the manuscript.

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