Special Issue

IMI: The Role of Light in Refractive Development and Myopia: Evidence from Animal and Human Studies

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Spending time outdoors is consistently associated with delayed myopia onset in children and has been incorporated into prevention programs. While the underlying mechanisms remain under investigation, substantial evidence supports sunlight exposure as a key contributing factor. This review evaluates the evidence supporting this association. Animal studies demonstrate that light characteristics—such as intensity, chromaticity, and photoperiod—can influence refractive development, often postulated to function through modulation of the dopaminergic system. However, translating these findings to humans is challenging due to limited data. Evidence remains insufficient regarding how specific light properties—including intensity thresholds, exposure durations, spectral composition, and temporal patterns—affect human myopia. Consequently, although clinical recommendations for outdoor time (e.g., 2 hours daily) are well supported by epidemiological studies and widely endorsed, current literature does not yet support evidence-based guidelines concerning specific characteristics of light exposure. Address-

iovs.arvojournals.org | ISSN: 1552-5783

Investigative Ophthalmology & Visual Science

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Received: July 25, 2025
Accepted: October 22, 2025
Published: December 2, 2025

Citation: Ashby R, Harb EN, Ostrin LA, et al. IMI: The role of light in refractive development and myopia: Evidence from animal and human studies. *Invest Ophthalmol Vis Sci.* 2025;66(15):5. https://doi.org/10.1167/iovs.66.15.5

ing this gap requires further randomized controlled trials using standardized wearable technologies to better quantify children's visual environments and identify light-related cues relevant to myopia development. Interest in light-based therapies is growing, but most interventions remain in early stages of development/testing. Due to limited efficacy data or unresolved safety concerns, no clinical recommendations can currently be made. Interpreting light-related research—especially from across animal models—requires caution. Species-specific differences in ocular transmittance and opsin distribution/tuning complicate the translation of chromatically related findings between species and to humans. Moreover, the term "white light" can be misleading, as artificial sources vary spectrally from each other and from sunlight, resulting in varied patterns of opsin activation. Studies should therefore attempt to report not only light intensity, but also the radiant power emitted at each wavelength to enable meaningful comparisons.

Keywords: light, myopia, lux, outdoor activity, wearable technologies, visual environment

This review summarizes, as best as possible, current peer-reviewed published evidence (English manuscripts, published up to June 2025, chosen by matter experts [no formal inclusion criteria applied]) on how various characteristics of light—such as intensity, spectrum, and photoperiod—may affect refractive development via image- and non-image-forming pathways. For each light-related factor, foundational animal studies and supporting human evidence are reviewed. Key knowledge gaps are identified and future research directions proposed. Based on current evidence, clinical recommendations for light-based interventions in myopia management are offered. Finally, the need to standardize the measurement and reporting of light-related metrics is discussed and suggested guidelines to support this effort are provided.

Rationale: Time Outdoors

In recent years, increasing attention has focused on light as a regulatory cue in refractive development, driven in part by epidemiological evidence that children who spend more time outdoors, as subjectively assessed, are less likely to be or become myopic. Jones et al.[1] showed that children who spent more time playing sports/being outdoors (per parental reports) had reduced odds of having myopia, especially for children that had one or two myopic parents. Rose et al.[2] further showed that children who spent more time outdoors, as subjectively assessed, had lower myopia prevalence, even with high levels of near work. The lowest prevalence was observed in children within the highest tertile of time outdoors (>2.7 hours per day outdoors).

These foundational studies were followed by several randomized clinical trials which demonstrated that increased time outdoors could reduce the incidence of myopia.[3–6] Meta-analyses support these findings, showing a consistent reduction in myopia incidence across clinical trials.[7,8] However, variability between studies remains in the reported changes in refractive error and axial length over the trial period.[7,8] More recently, the Shanghai STORM study using light-sensing smartwatch data suggested that a reduced myopic shift was associated with light exposures of at least 15 minutes in duration at intensities above 2000 lux.[9]

Initial evidence was quickly integrated into national myopia prevention programs—beginning in Singapore (2008) through parental guidance, and in Taiwan via the Tian-Tian 120 outdoor initiative, which recommended 120 minutes of daily outdoor time in schools, but not in preschools. Follow-up analysis of Taiwan's program suggests a reversal in the long-term trend of declining visual acuity, presumed to be due to uncorrected refractive error, over the 5 years following implementation.[10] As positive outcomes have emerged, these recommendations have been strengthened. More recently, mainland China has adopted the 2-hour daily outdoor recommended target, introducing mandatory school-based programs on an exploratory basis to identify effective strategies.[11]

Large scale implementation of increased time outdoors is also happening in Taiwan. The Yilan study based on promotion of time outdoors in preschool children produced 50% reduction in the prevalence of myopia over the past 7 years – an effect that has been maintained during the coronavirus disease 2019 (COVID-19) pandemic.[12,13] Recent studies indicate that the protective effect of preschool exposures can still be detected in grades 1 and 2 of primary school.[14]

Although each of these studies has limitations—particularly those inherent in working with large populations and the challenges of standardizing or closely monitoring everyday behavior—the collective findings clearly indicate that increasing time spent outdoors in school settings slows the onset of myopia. The evidence for a role in slowing myopia progression is less definitive, although several lines of support exist. These include seasonal variations in progression rates,[15–19] the randomized controlled trial (RCT) findings of Wu and colleagues,[4] (although similar effects were not observed in other RCTs[5,9]), and the increased progression rates reported during the COVID-19 pandemic, a period marked by reduced outdoor activity.[20–23] However, at present, whereas time outdoors is recommended to delay myopia onset, clinical interventions (e.g., optical and pharmacological interventions, for a review see Ref. 24) remain more robustly validated for controlling progression.

What Could Explain the Protection of Time Outdoors?

As early as 2008, Rose et al.[2] postulated that the protective effect of being outdoors could be mediated by the brighter light experienced outdoors during the day leading to greater release of retinal dopamine (DA) and subsequent slowing of axial elongation. This hypothesis was grounded in animal studies showing that bright light increases DA release and that pharmacological activation of DA receptors inhibits axial growth (for a review see Ref. 25). This hypothesis was rapidly supported by several animal experiments

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that showed that bright light slowed or blocked the development of experimental myopia in most experiments,[26–30] with one exception,[31] and that such protection was diminished or lost in the chicken model when the DA system was pharmacologically blocked.[27,32] These findings suggest that light intensity is an effective factor in the protective effects of outdoor exposure.

It is unlikely that light-induced changes in DA release alone provide a mechanism for "encoding" refractive error. The maintenance of emmetropia requires a feedback loop that informs the eye whether its current shape produces a focused retinal image. Light intensity by itself is unlikely to serve this function. Rather, as previously postulated,[32] light-induced DA release may act as a regulatory brake, inhibiting excessive axial elongation of the eye.

Beyond light intensity, natural outdoor scenes may reintroduce other critical visual cues essential for normal ocular development and the anti-myopic effects observed—cues that are often diminished or absent during prolonged indoor exposure.[33,34] These include several dynamic differences, such as peripheral blur, pupil size, dioptric range, temporal vision, central versus peripheral vision, spectral composition, and spatial structure (for review see Refs. 25 and 35). A role for vitamin D levels has also been proposed, but is difficult to separate from time outdoors, given the known impact of UV exposures on vitamin D levels. However detailed epidemiological[36] and Mendelian randomization studies[37–39] appear to argue against this possibility.

Understanding the mechanism/s by which outdoor time protects against myopia is critical—not only for advancing scientific knowledge but also for addressing practical questions frequently asked by parents/clinicians. Currently, clear clinical evidence is lacking to answer common concerns such as: Does wearing sunglasses or a hat reduce the protective effect? Is sitting under a tree or reading outdoors still beneficial? What happens on cloudy or rainy days? Can weekend outdoor time compensate for limited exposure during the week? Does the time of day a child goes outdoors matter? Is it better to go outdoors for one long period or several short periods? Do certain activities performed outdoors provide a greater protective effect? Identifying the specific visual cues critical to this protective effect will help clarify these uncertainties and support the development of evidence-based recommendations.

Retinal Physiology of Common Animal Models of Myopia

By enabling the selective modulation of specific environmental factors, animal models have played a fundamental role in advancing our understanding of how different aspects of the visual experience influence ocular development. These models will remain essential, particularly for generating robust evidence to support clinical trials and for exploring aspects of the visual environment that cannot be practically studied in humans. Animal models are particularly critical in understanding the molecular and biochemical pathways underlying the regulation of ocular growth, and the physiological processes they drive.

Two primary methods have commonly been used to induce excessive axial elongation in animals: form-deprivation myopia (FDM) and negative lens-induced myopia (LIM). In both monocular paradigms, the eye responds to altered visual input by rapidly thinning the choroid and later increasing scleral growth.[40–61] While FDM and LIM share similarities (for review see Refs. 25 and 59), it is unclear which better reflects human myopia. Although many findings from animal studies have successfully translated to humans, there are limitations to what can be understood without direct investigation in patients, particularly regarding what aspects of our complex and dynamic visual environment are critical for growth regulation.

When comparing across animal models, it is essential to consider distinct differences in ocular physiology, including variations in ocular media, photoreceptor diversity, spectral sensitivity, interneuronal processing and circuitry, and circadian entrainment. Recognizing these differences is crucial not only for understanding why certain results may not replicate across species, but also for appreciating the limitations of directly extrapolating animal data to humans. For example, whereas ocular growth in one species (i.e. the chicken[62–64] or mouse[65]) may be significantly influenced by ultraviolet light, another species (i.e. human[62,66]) may show little or no response due to differences in lens transmittance or photoreceptor availability.

Animal models commonly used in myopia research, each of which offer unique advantages, include but are not limited to, rhesus monkeys, chickens, tree shrews, guinea pigs, and mice. The visual physiology of these species has been reviewed in detail in a previous International Myopia Institute (IMI) white paper.[25] Key relevant distinctions, such as photoreceptor subtypes, distribution, and sensitivities, cornea and lens transmittance, visual acuity, and non-image forming opsins, are summarized below and in Figure 1; the Table.

Chickens are diurnal and possess a cone-dominant retina, with a ratio of rods to double cones to single cones of 2:2:1 in most regions.[67] Rather than a fovea, the chick possess an area centralis, which is mainly rod free. Chick visual acuity is approximately 7 cycles/degree.[68–70] Chicks have 4 types of single cone photoreceptors; long, medium, short 1, short 2,[71,72] and one double cone photoreceptor (comprised of 2 "members," both of which possess a long-wavelength sensitive opsin).[70,73]

Mice are nocturnal rodents that can also be active during the day. Mice are dichromats, possessing short (can detect UV) and long wavelength sensitive cones[74] (see Fig. 1; the Table) that are regionally distributed; long wavelength cones are more dominant in the superior half of the retina, and short wavelength cones are more dominant in the inferior-nasal retina.[75,76] The mouse retina is rod-dominated (approximately 97%),[77] and has a visual streak with a visual acuity of approximately 0.3 to 0.6 cycles/degree.

Guinea pigs are crepuscular rodents, meaning they are most active around dusk and dawn and are not strictly diurnal or nocturnal. They are dichromatic, with short and long wavelength sensitive cones[78,79] that, like mice, are regionally distributed; long wavelength cones are more dominant in the superior half of the retina, and short wavelength cones are more dominant in the inferior-nasal retina.[75,76] The guinea pig retina is rod dominated (83%–92%).[80] The guinea pig has a visual streak with a visual acuity of approximately 2.7 to 3 cycles/degree.[81,82]

Tree shrews are diurnal and in the Scandentia order, closely related to primates. Their retina is cone dominated, with only approximately 14% rods.[83] Tree shrews have an area centralis with a visual acuity of approximately 2.4

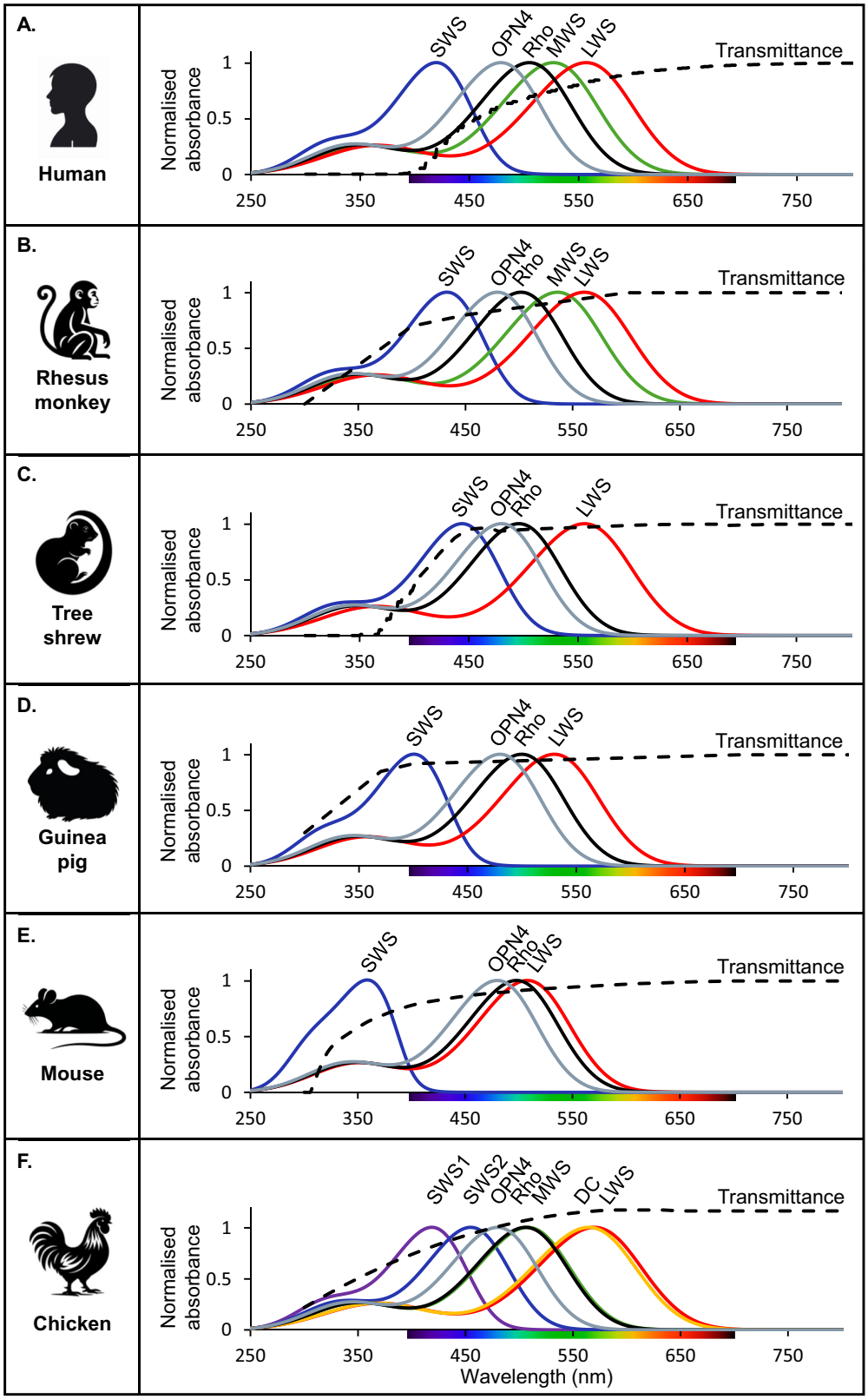

Figure 1. Normalized photopigment absorbance spectra and ocular transmittance across species. Absorbance curves were calculated as described by Govardovskii et al.[104] using λ max values for each species. Ocular media transmittance values (cornea/lens/aqueous humor/vitreous humor), as best known, are displayed as *dotted lines* for each species (**A**) human,[96,99] (**B**) rhesus monkey,[91,105] (**C**) tree shrew,[88,89,106] (**D**) guinea pig,[78,107] (**E**) mouse,[74,108] and (**F**) chicken[71,109]). For reference, the visual spectrum for humans is displayed as a *color bar* under each species panel. OPN3 and OPN5, as well as the effects of ellipsoid membranes and oil droplets are not plotted due to limits in the characterization of their absorbance spectra across all species. It is also important to note that λ max and transmittance data are currently limited for some species, and, as such, these values may be subject to revision as additional data become available. Key: SWS, short-wavelength sensitive cone opsin (*blue and violet lines*); OPN4, melanopsin (*light blue line*); Rho, rhodopsin (*black line*); MWS,

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medium-wavelength sensitive cone opsin (*green line*); DC, double cone opsin, chick only (*yellow line*); LWS, long-wavelength sensitive cone opsin (*red line*). Figure adapted from Reference 32.

Table. Peak Wavelength Sensitivities for Major Photopigments Across Species

	λ max						
Species	**SWS**	**MWS**	**LWS**	**Rho**	**OPN3**	**OPN4**	**OPN5**
Human[101,103]	420	527	557	505	^	479	380
Rhesus monkey[91–93]	433–440	536	561–565	502	^	480	^
Tree shrew[88–90]	428–444	–	556	496	^	480	^
Guinea pig[78,79]	401–429	–	530	500	^	480	^
Mouse[74]	360	–	508	498	^	480	380
Chicken[71,72]	419, 455*	508	570	506	470	480	360

SWS, short-wavelength sensitive cone opsin; MWS, medium-wavelength sensitive cone opsin; LWS, long-wavelength sensitive cone opsin; rho, rhodopsin (rod opsin); OPN3, encephalopsin; OPN4, melanopsin; OPN5, neuropsin; "-" not present, "*" λ max values for the two chicken SWS opsins (SWS1 and SWS2), "^" detected, but absorbance characteristics unknown.

cycles/degree.[83–87] Tree shrews are dichromats with short and long wavelength sensitive cones.[88–90]

The rhesus monkey, a diurnal mammal, is the closest animal model to humans with regard to retinal physiology. Their retina is rod-dominant (rod to cone ratio of roughly 20:1), with a fovea that is cone-dominant, having a visual acuity of approximately 44 cycles/degree. Rhesus monkeys are trichromatic, with short, medium, and long wavelength sensitive cones.[91–93]

For comparison, humans are diurnal with a rod-dominant retina (rod to cone ratio of roughly 20:1).[94] The human fovea, which is cone-dominant, has a visual acuity of approximately 60 cycles/degree.[95] Humans are trichromatic, with short, medium, and long wavelength cones.[96]

Each species also possesses several, non-classical, opsins involved in non-image forming processes. Among these, 3 major families have come to prominence: opsin 3 (OPN3; encephalopsin),[97] opsin 4 (OPN4; melanopsin[98–100]), and opsin 5 (OPN5; neuropsin).[101,102] With regard to spectral sensitivity, whereas OPN4 has been well-characterized across species, less is known about OPN3 and OPN5. OPN4 demonstrates consistency (i.e. deep evolutionary conservation) across species. The role of non-classical photoreceptors in ocular growth has become an area of significant interest.

As noted, research on animal models has the power to impose environmental exposures that will rarely be achieved in humans, providing clearer insights into the basic mechanisms involved in the regulation of eye growth. However, caution is required in applying these insights to human studies. For example, if a particular exposure is identified in animal studies, if it is to be considered as a risk factor for myopia, it is important to identify conditions under which humans might be exposed to this factor at a level that would produce population level changes in prevalence. For example, exposure to light during the dark cycle disrupts refractive development in animal studies but is there evidence that these sorts of exposures have a role in creating the current patterns of prevalence of myopia in human populations? In considering these possibilities, a question that could be posed is whether light exposures in East Asia and Singapore are unique in a way that could contribute to the current epidemic of myopia. Even if factors identified in animal models fail to explain the population epidemiology of myopia, they may provide insights into methods for controlling the onset and progression of myopia.

Spectral Diversity of Common Light Sources and Impact on Opsin Activation

The spectral composition of common light sources is shown in Figure 2. Natural sunlight is broadband and perceived as white light, with a spectral distribution spanning UV, visible, and infrared wavelengths.[110] The spectrum varies over the day, across the year, and by geographic location due to solar elevation and atmospheric composition. For example, short (blue) wavelengths are enriched during dawn and dusk when the angle of incidence is low, whereas long (red) wavelengths are enriched during the middle of the day when the sun sits higher in the sky.[110] This also causes seasonal deviations in the spectral composition of sunlight, with lower red enrichment during the autumn/winter months when the sun remains lower in the sky and higher amounts of atmospheric water vapor are present.[110,111] In contrast, and as illustrated in Figure 2, artificial lighting systems have, for the most part, varied but narrower spectral distribution.

Due to their distinct spectral compositions, each "white light" source noted in Figure 2 produces a unique activation pattern across classical and non-classical photoreceptors. This pattern is further shaped by two species-specific factors: (1) the eye's aperture (the ratio of pupil area to retinal area) and ocular media transmittance, and (2) photoreceptor distribution and tuning. Figure 3 illustrates how aperture and transmittance affect the number of photons reaching the retina in various model species when exposed to two different white light sources—sunlight and fluorescent light. This initial analysis shows that, at the retinal surface, both light sources generate a similar chromatic activation pattern skewed toward long wavelengths, regardless of species.

Figure 3 can be further refined by incorporating photoreceptor distribution and spectral tuning. Although this information is not available for all animal models commonly used in myopia research, Figure 4 illustrates how retinal activation patterns differ among four distinct "white light" sources when these data are included for humans and chickens. This comparison highlights that, although all four sources are classified as "white light," they elicit different patterns of photoreceptor activation—patterns that also vary across species.

These observations prompt an important question: could the chromatic and flicker characteristics of modern artificial lighting—which are distinct from natural sunlight—influence eye growth and the development of myopia? Although current evidence does not provide a definitive answer, animal work has shown that changes in the chromatic composition of white light,[112–116] or exposure to narrow-band light,[62–65,112,117–138] can influence refractive development. Given the increasing exposure to artificial lighting during daily activities—including increased exposure through digital devices—further investigation into its potential effects on visual development is warranted.

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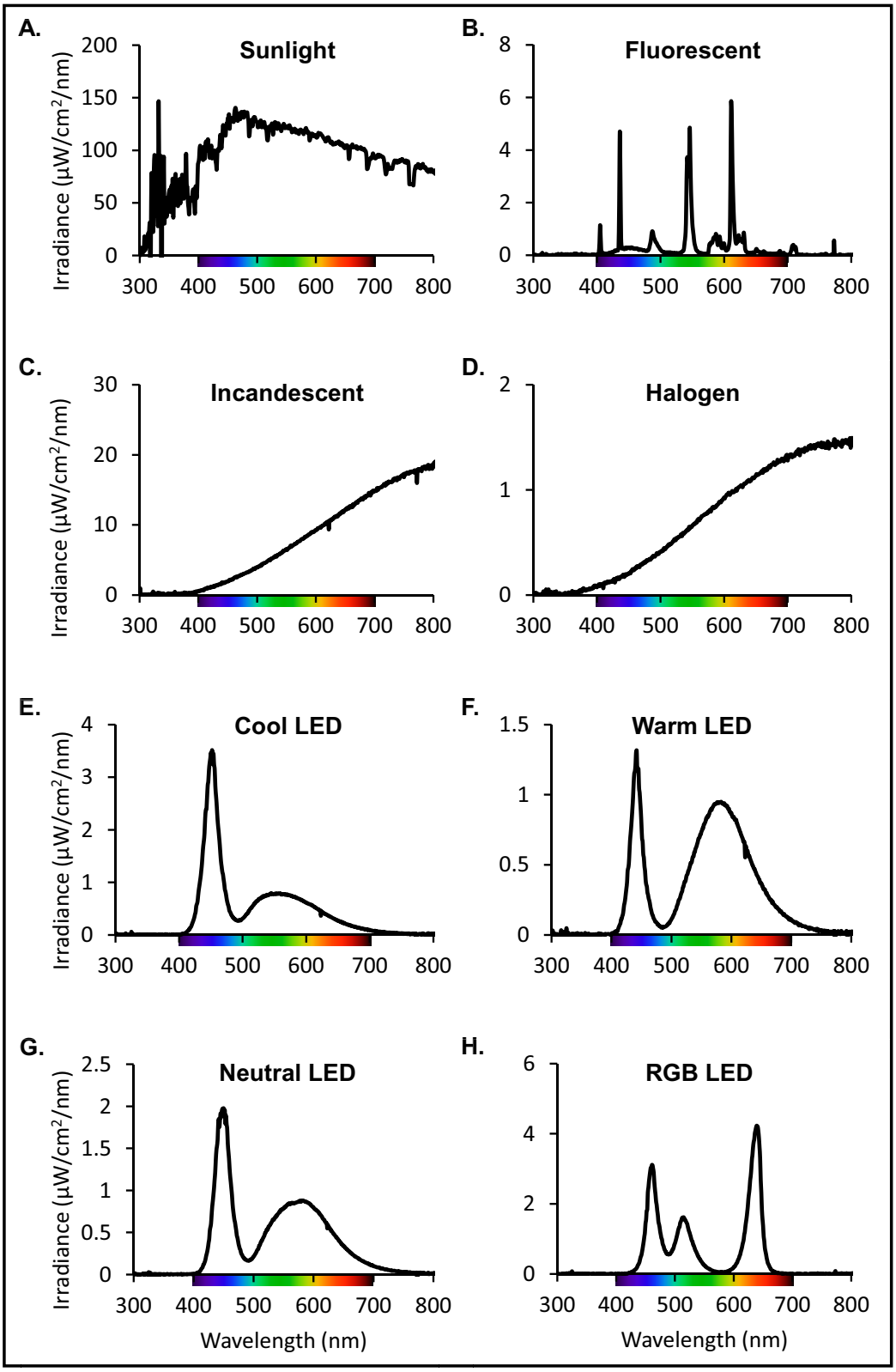

Figure 2. Spectral distribution of different "white light" sources. The irradiance at wavelengths between 300 and 800 nm is presented for (**A**) approximately 90,000 lux sunlight (as measured in the early afternoon during summer in Canberra, Australia; correlated color temperature (CCT) = approximately 6000 K), (**B**) 500 lux fluorescent light (CCT = approximately 4000), (**C**) 500 lux incandescent light (CCT = approximately 2700), (**D**) 500 lux halogen light (as used in initial studies examining the role of light in experimental myopia[26,27]; CCT = approximately 3000), (**E**) 500 lux "cool white" using light emitting diodes (LEDs; CCT = 6000–6500), (**F**) 500 lux "warm white" light using LEDs (CCT = 3000–3500), (**G**) 500 lux "neutral white" light using LEDs (CCT = approximately 4500), and (**H**) 500 lux white light generated from red-green-blue LEDs (CCT = approximately 4000). For reference, the human visual spectrum is displayed as a color bar under each panel. Figure adapted from Reference 32.

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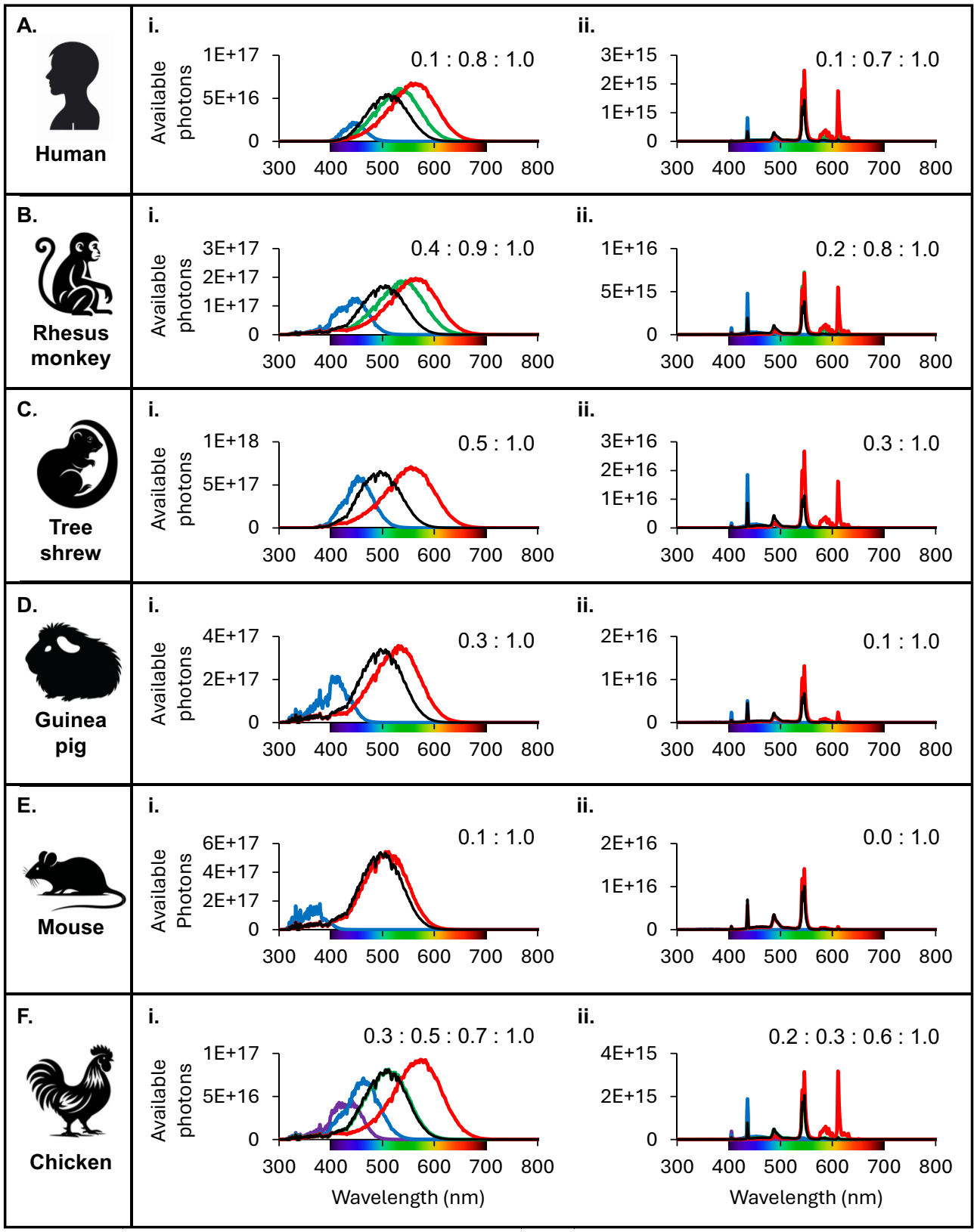

Figure 3. Differences in total photons presented to the retina across species for two different light sources. The total photons presented to each classical photoreceptor type were calculated between 300 and 800 nm for (**A**) human,[96,99,139,140] (**B**) rhesus monkey,[91,105,139,141] (**C**) tree shrew,[87–89,106] (**D**) guinea pig,[78,107,142,143] (**E**) mouse,[74,108] and (**F**) chicken.[32,71,109] Total photons available to the retina were calculated for (**i**) approximately 90,000 lux sunlight (as measured in the early afternoon during summer in Canberra, Australia; CCT = approximately 6000 K) and (**ii**) 500 lux fluorescent light (CCT = approximately 4000 K). The ratio of total photons available to each cone photoreceptor type is presented in the top right of each panel. Ratios were calculated by normalizing to the photons presented

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to the LWS cone, which is given a value of 1. Ratios are presented for tetrachromatic (SWS1:SWS2:MWS:LWS), trichromatic (SWS:MWS:LWS), and dichromatic (SWS:LWS) species. Total available photons were calculated as described by Wilby and colleagues,[73] taking into account photoreceptor spectral sensitivity (Fig. 1), ocular light transmission, and the multiplication factor of the (eye's aperture pupil area:retinal area ratio) for each species (references given for each species when first listed above). The peaks depicting total effective photons for rods (rhodopsin) and MWS/LWS cones overlap in some species and may not be visible. For reference, the human visual spectrum is displayed as a color bar under each panel. Key: Rhodopsin (*black line*), short-wavelength sensitive cone opsin (*blue and violet* [chicken only] *lines*); medium-wavelength sensitive cone opsin (*green line*); and long-wavelength sensitive cone opsin (*red line*). Figure adapted from Reference 32.

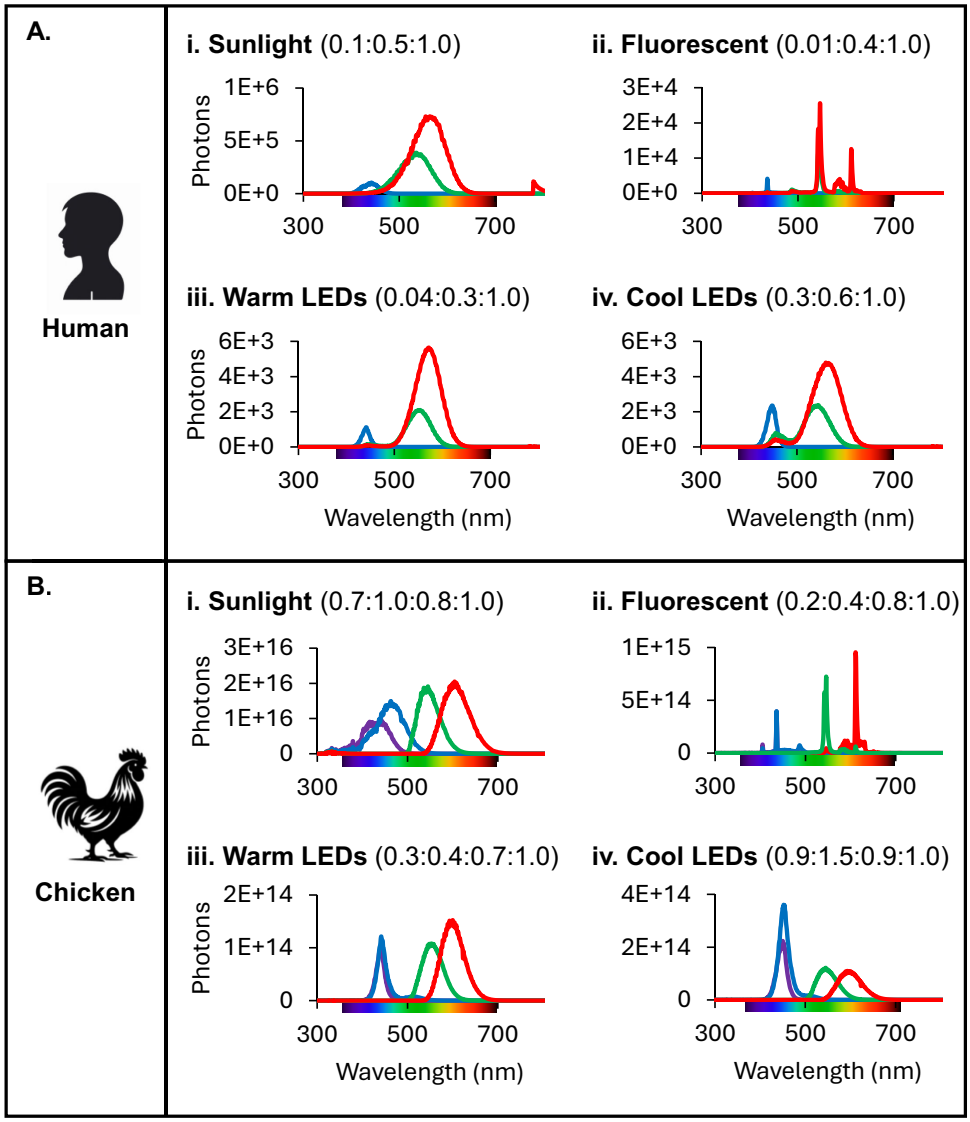

Figure 4. Differences in cone opsin activation patterns in response to four different light sources as demonstrated for humans and chicks. Total effective photon estimates were calculated for (**A**) humans and (**B**) chicks exposed to four different light sources: (**i**) approximately 90,000 lux sunlight (as measured in the early afternoon during summer in Canberra, Australia; CCT = approximately 6000 K), (**ii**) 500 lux fluorescent light (CCT = approximately 4000 K), (**iii**) 500 lux "warm white" LED light (CCT = approximately 3000–3500 K), and (**iv**) 500 lux "cool white" LED light (CCT = approximately 6000–6500 K). Total effective photon estimates were calculated between 300 and 800 nm following the protocol described by Wilby and colleagues.[73] These calculations took into account photoreceptor spectral sensitivity (Figs. 1, 3), ocular light transmission,[109] the multiplication factor of the eye's aperture (pupil area:retinal area ratio), photoreceptor cell light transmission (representative of the total photons able to reach a photopigment)[73,99] and the relative ratios[141,144] of each cone type (to take into account the differences in cone numbers). For reference, the human visual spectrum is displayed as a color bar under each panel. The ratio of total photons available to each cone photoreceptor type is presented in each panel title within the *brackets*. Ratios were calculated by normalizing to the photons presented to the LWS cone, which is given a value of 1. Key: Short-wavelength sensitive cone opsin (*blue and violet* [chicken only] *lines*), medium-wavelength sensitive cone opsin (*green line*), and long-wavelength sensitive cone opsin (*red line*). Figure adapted from Reference 32.

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Defining and Measuring Light

There are a variety of units in which the intensity, the amount of energy or light emitted by a source, can be measured and reported (watts, lumens, and candles, see definitions in Box 1). While valuable to demonstrate the power of a light source, these measures are difficult to contextualize with regard to the visual experience as the angle and distance from the light source is not accounted for. Therefore, irradiance and illuminance are more commonly reported, as both quantify the light falling on a surface, with irradiance expressed in watts per square meter (W/m^2) and illuminance in lumens per square meter (lux) or lumens per square foot (foot-candles). Irradiance and illuminance measures are not directly comparable. Illuminance measures the amount of light falling on a surface, spectrally weighted to match human visual sensitivity according to the photopic luminous efficiency function.[145–147] Measuring illuminance using a lux meter is cost effective and therefore a commonly reported parameter, especially in the myopia literature. However, lux meters do not measure radiant energy and are wavelength dependent, with lux calculated relative to 555 nm.[145–147] As a result, they cannot provide information about the spectral distribution of light. This limitation makes it challenging to compare lighting systems with differing spectral profiles. For instance, two light sources may produce similar illuminance values in lux but have vastly different spectral compositions, leading to different photoreceptor activation patterns (Fig. 5).

In contrast, irradiance measures the total radiant power incident on a surface per unit area (e.g., W/m^2), integrated across all wavelengths without accounting for human visual sensitivity.[145–147] Spectroradiometers are the primary instruments used to measure irradiance and to capture the spectral power distribution (SPD) of a light source. SPD describes how much radiant power is emitted at each wavelength, offering a detailed profile of the light's spectral composition.[145–147] This makes SPD measurements more versatile, as they enable meaningful comparisons between spectrally dissimilar lighting systems. Crucially, irradiance data allow for the calculation of the total number of effective photons reaching the retina, which as discussed above, is required to understand the pattern of opsin activation produced by a particular light source. Such information is essential for understanding the influence of chromatic cues on ocular growth regulation. Thus, radiance is a more appropriate metric than illuminance, and, where feasible, future studies on light exposure should aim to include this measure.

Definitions

Emittance: The total radiant energy emitted from a light source (or reflected from a surface) per unit area, integrated over all directions (measured in W/m^2).

- For example, the energy emitted in all directions from a light source.

Radiance: The amount of radiant energy (light) leaving a light source (or reflected from a surface) in a specific direction, per unit area per unit solid angle (measured in $W/m^2 \cdot sr$).

- For example, the energy emitted from a light source toward a table.

Luminance: The amount of light reflected or emitted from a surface per unit area in a given direction (measured in candela per meter squared).

- For example, the amount of light reflected from a table towards a specific point (e.g., an observer).

Irradiance: The total radiant power received by a surface per unit area, regardless of direction (measured in W/m^2).

- For example, the energy from a light source that reaches a table.

Illuminance: The amount of visible light (measured in terms of human visual perception) incident on a surface per unit area (measured in lux).

- For example, the amount of visible light cast on the table.

Spectral Power Distribution (SPD): The radiant power emitted by a light source at each wavelength - frequently generated from irradiance measures.

- For example, the energy from a light source, at each individual wavelength, that reaches a table.

The Role of Light Intensity in Ocular Growth Regulation

Bright light exposure was initially put forward to explain the anti-myopic effects of time spent outdoors.[2] In the following section, key findings from animal models on how light intensity influences ocular growth are summarized and their relevance to human myopia is examined. Supporting clinical evidence is reviewed and gaps in understanding are described.

Findings From Animal Models

Rearing in Low Intensity Illumination. Initial studies on light intensity were prompted by the seminal finding that disrupting diurnal or circadian entrainment—by rearing animals in constant light or darkness—can lead to abnormal eye growth (see Section 8.0 The Role of Circadian Entrainment for further information).[148–150] From this, early chick studies showed that rearing under dim light (5.5–60 lux) during the light phase led to axial elongation and a myopic shift.[151–153] This finding was later expanded on by Cohen and colleagues,[154] who demonstrated that over 90 days, chicks reared under dim light (50 lux) showed a myopic shift in refraction, whereas chicks reared under brighter light (10,000 lux) remained hyperopic. Dim light has also been shown to have a significant effect on ocular growth in guinea pigs[155] and rhesus monkeys,[156] although the direction of change is variable. Specifically, guinea pigs raised under dim light (20 lux) were found to be the least hyperopic after 6 weeks of treatment.[155] In contrast, rhesus monkeys raised under dim light (55 lux) remained more hyperopic compared with age-matched animals kept under normal light (500 lux).[156]

The effect of dim light (≤55 lux) rearing on experimental myopia has also been studied. For FDM, the

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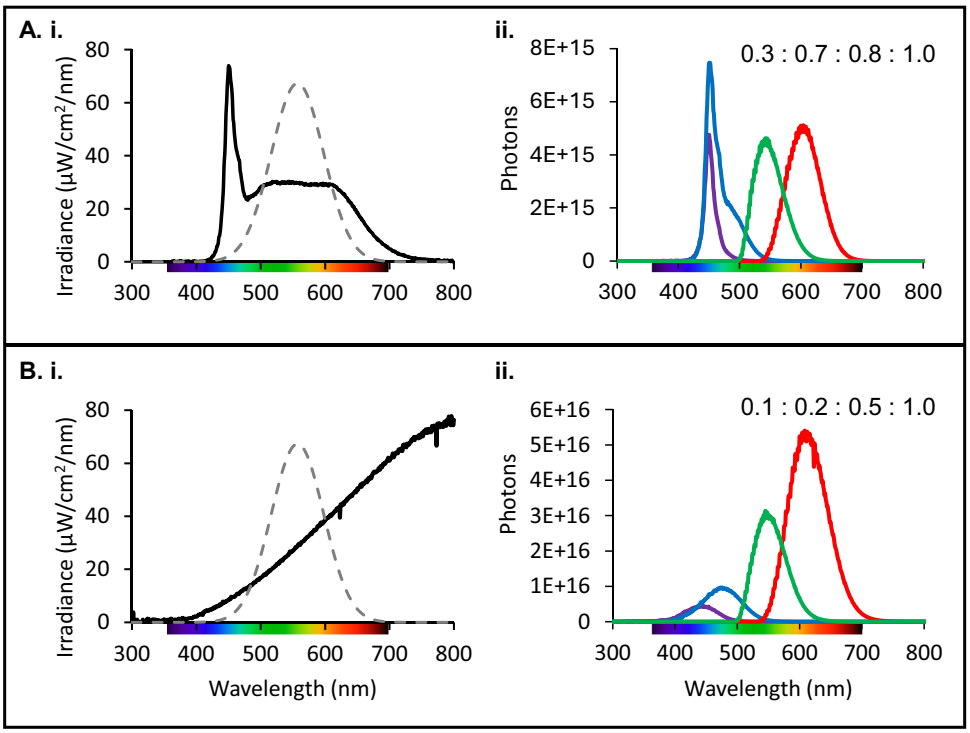

Figure 5. Differences in the spectral power distribution (SPD) of two "white light" sources with matched illuminance, and their corresponding cone opsin activation profiles in the chick retina. Presented between wavelengths of 300 and 800 nm are the SPDs of 2 light sources matched for illuminance (approximately 20,000 lux): (**A.i.**) LED "neutral white" light (CCT = approximately 4500 K) and (**B.i.**) halogen light (CCT = approximately 3000 K). In both panels, the *gray dotted line* represents the human photopic luminosity function (peaking at 555 nm), which is used to calculate illuminance in lux. Although both sources emit approximately 20,000 lux, the halogen light delivers more than twice the number of photons (LED = 1.3×10^{14} photons versus Halogen = 3.35×10^{14} photons). Due to differences in SPD, the spectral distribution of photons reaching the retina varies between sources (**A.ii.** for LED and **B.ii.** for halogen). Total effective photons for chicken cone opsins were calculated as described in Figure 4. For reference, the human visible spectrum is shown as a color bar beneath each panel. The relative photon availability for each cone type is shown in the *top right* of *each panel*, normalized to the long-wavelength sensitive (LWS) cone, which is assigned a value of 1. Key: Short-wavelength sensitive cone opsins (*blue and violet lines*), medium-wavelength sensitive cone opsin (*green line*), and long-wavelength sensitive cone opsin (*red line*). Figure adapted from Reference 32.

degree of myopia induced in chicks,[26] guinea pigs,[155] and rhesus monkeys[157] is unaffected by dim light rearing when compared with animals reared under normal light. In contrast, in mice (1.6×10^{-3} candela/m^2)[158] and monkeys (55 lux),[159] dim rearing slows (but does not stop) the rate of compensation to negative lenses.

Rearing in High Intensity Illumination. Cohen and colleagues[154] demonstrated that chicks exposed to higher illuminance levels exhibited more hyperopic refractive states during normal development. Concurrently, evidence emerged demonstrating that elevated light levels influence the development of experimental myopia (for a review see Refs. 25, 30, 160–164). Specifically, in chicks,[26,165] rhesus monkeys,[28] tree shrews,[29,30] guinea pigs,[166] and mice[167] daily exposure of 2 to 6 hours of bright artificial indoor light (2500–40,000 lux) inhibits the development of FDM and also induces a hyperopic shift in untreated eyes for the majority of species. Where studied, the anti-myopic effect was intensity-dependent, showing a strong negative logarithmic correlation ($R^2 = 0.95$, $P < 0.001$).[165] In chicks, near full protection against FDM was observed by 6 hours per day of 40,000 lux,[165] with such intensities also halting further progression in already myopic eyes.[165] These effects were not driven by UV, changes in corneal power or the physical movement (optic flow) of the animals, three early hypotheses were put forward to explain the protection observed.[26]

Bright light also influences the response to LIM in chicks,[27,32] tree shrews,[29] and guinea pigs.[168] In all three species, bright light slows the rate of lens compensation in an intensity-dependent manner[32] but does not prevent it. This inability to halt LIM suggests a mechanistic distinction from FDM, likely due to the presence of a defocus-driven growth endpoint in LIM that is absent in FDM.[32] Another key difference is observed in rhesus monkeys, where 3 hours per day of bright indoor light (25,000 lux)—sufficient to inhibit FDM—failed to alter LIM progression.[31] However, a subsequent study reported an anti-myopic effect from 3 hours of outdoor light exposure.[169] It is important to note, although, that this study compared monkeys wearing −3D lenses reared indoors to those reared outdoors under higher light levels, introducing differences not only in intensity but also in spectral composition.

Taken together, animal studies across multiple species have demonstrated that bright light exposure inhibits the excessive ocular growth associated with FDM. Except for one study, bright light also consistently reduces the rate of ocular growth during compensation to negative lenses. These findings support the hypothesis that light intensity plays a meaningful role in modulating eye growth and, thus,

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may contribute to the mechanism underlying the protective effects of time spent outdoors.

Research on the impact of light levels on growth suppression associated with lens-induced hyperopia (LIH; myopic defocus) has only been investigated in chicks.[27,170] Similar to findings with negative lenses, exposure to bright light does not inhibit compensation to positive lens wear; rather, it accelerates the rate of compensation. Specifically, bright light further slows ocular growth rates above that seen in response to myopic defocus alone. More recently, Zheng et al.[171] examined the effects of bright light on compensation to dual-focus lenses in chicks and found that it amplified the growth-inhibitory effect of a +10 diopter (D)/−10 D (50:50 ratio) lens, while concurrently suppressing the growth-stimulatory effect of a plano/−10 D (50:50 ratio) lens.

The impact of bright light on experimental myopia is also dependent on the timing, duration, and pattern of the exposure. A recent study by Biswas and colleagues[172] highlighted a duration-dependent effect when −10 D lens-wearing chicks were exposed to 15,000 lux for a period of 2, 4, or 6 hours over 8 days. The efficacy was duration-dependent, with even 2 hours of bright light exposure showing a significant reduction in myopic refraction and axial elongation.

Increasing attention has been given to whether the effectiveness of brief periods of bright light exposure—or other anti-myopic treatments—is influenced by the time of day, particularly in light of growing interest in the role of circadian rhythms in refractive error development (for review see Ref. 173). In chicks, full-day exposure to 2000 lux was reported to be more effective at inhibiting FDM than 2-hour exposures to equi-luminant light (10,000 lux) delivered in the morning, midday, or evening.[174] However, among the brief exposure groups, midday light was more effective than morning or evening exposure. A subsequent study in chicks found that both FDM and LIM (−10 D lens wear) were significantly inhibited by 3 hours of bright light (25,000–30,000 lux) administered in the evening, whereas morning exposure had no effect.[175] Although further research is needed, these initial findings suggest a complex interaction between the timing of bright light exposure and its anti-myopic efficacy.

Emmetropization and ocular growth are also influenced by the temporal frequency of light exposure. Specifically, high temporal frequencies have been associated with hyperopia, whereas low temporal frequencies tend to induce myopia.[176,177] Although further research is needed, findings consistent with this concept show that intermittent exposure to bright light (15,000 lux) alternating with normal light (300 lux) in 1:1 and 7:7 minute cycles was more effective at controlling FDM in chicks than continuous bright light exposure.[178]

Evidence for a Role of Dopamine. As noted earlier, a key pathway through which light is proposed to influence ocular growth is via the retinal DA system. DA is a critical neuromodulator in the retina. It reduces horizontal and AII amacrine cell coupling,[179] thereby decreasing the amplitude of rod-driven responses during daylight hours. Additionally, DA promotes light adaptation by regulating photoreceptor retinomotor movements.[180]

DA is synthesized and released by a subset of retinal amacrine and/or interplexiform cells in a dose-dependent manner that is proportional to ambient light intensity.[32,158,181–185] Its extracellular levels exhibit significant diurnal variation, peaking during the day and declining at night.[186–190]

Extensive research in animal models has implicated DA in the development of myopia (for review see Ref. 25). Notably, the normal diurnal fluctuations in DA levels are markedly dampened during the induction of experimental myopia.[183,190,191] Conversely, pharmacological stimulation of the DA system has been shown to inhibit myopic eye growth across all species studied.[191–218]

With respect to light exposure, the anti-myopic effects of high illuminance levels appear to be mediated, at least in part, by increased DA release.[32,182,219] This is supported by findings that, in chicks, pharmacological suppression of the DA system negates the protective effects of bright light against both forms of experimental myopia.[27,32] In chicks and tree shrews, the growth modulatory effects of DA are primarily mediated through D2-like dopaminergic receptors,[191,198,199,201,203,209,210,220–223] whereas in mice and guinea pigs, a more complex interaction involving both D1-like and D2-like receptor families has been proposed.[213,214,224]

Although DA has been extensively studied in animal models of myopia, its role in human myopia development (including its hypothesized role in the protective effects associated with time spent outdoors) has not yet been definitively established due to experimental limitations. However, clinical observations suggest a potential link: in a retrospective population-based cohort study in Taiwan, children treated for attention deficit hyperactivity disorder (ADHD) with methylphenidate hydrochloride (MPH), a dopamine reuptake inhibitor, exhibited a lower incidence of myopia.[225] Additionally, a small observational study in Turkey reported a lower rate of myopia progression among ADHD children receiving MPH treatment over a period of 12 months.[226] These findings are supported by a recent animal study which reported that MPH administration inhibits the development of experimental myopia in chicks through increased extracellular accumulation of DA in the eye.[227]

Although intensity-dependent increases in DA release appear to underly the protective effects of bright light against experimental myopia, what might explain the increased effectiveness observed under different temporal (flicker) patterns of bright light noted earlier?[178] One potential explanation for these observations may be the altered modulation of retinal ON and OFF pathways. Adequate ON-pathway function has previously been proposed to play a critical role in normal ocular development, whereas ON-pathway dysfunction has been linked to dysregulated ocular growth and myopia.[177,228–233] Temporal modulation (approximately 1–4 hertz [Hz]) of the light environment to stimulate ON-pathway function has been demonstrated to significantly increase dopamine release and slow ocular growth.[177,229,234] Therefore, further stimulation of retinal ON-pathways by flickering light could in turn lead to even greater DA release than that induced by bright light alone.

However, several questions remain regarding DA's role in ocular growth regulation. Notably, extracellular DA levels are reduced in both LIM[235] and LIH,[236] despite these paradigms producing opposite growth responses. One hypothesis is that DA does not directly signal the direction of growth but instead is simply responding to any change in light and/or contrast.[32] In this model, DA may act as a permissive factor—"unlocking" the retina to enable growth modulation in response to other cues, rather than dictating the specific trajectory of refractive development.

Unanswered Questions From Animal Studies. Data from animal models have significantly advanced our understanding of the relationship between bright light exposure and myopia. Nevertheless, there remain some discrepancies between studies, particularly when comparing the impact of light exposure between FDM and LIM models (for review see Ref. 25) and how these models translate to myopia in children. These discrepancies may be due to differences in experimental protocols and non-photic environmental features the animals are raised under. These features are often under-reported in studies. Furthermore, to-date, there are uncertainties regarding the precise thresholds and parameters needed to confer protective effects in animal models using bright white light. Recent harmonization efforts, through reporting guidelines dedicated to animal and in-lab human studies using light as an intervention,[237,238] should be adopted by the vision science community to improve the reporting of experimental light characteristics. This would allow for a better comparison of findings and protocols between studies, enabling more effective meta-analyses.

Findings From Human Studies

Inferring a causal role for intensity of light in the anti-myopic effects of time outdoors from current clinical data is difficult. Exposure to sunlight could simply be a surrogate marker of being outdoors rather than being the underlying driver. However, several findings from studies using objective measures (e.g., wearables) support a role for light intensity. First, two trials have observed a reduction in myopia incidence when increasing illumination level of classrooms.[239,240] The first of these studies, conducted in northeast China, examined the impact of elevated classroom lighting on students aged 6 to 14 years.[239] In the intervention group (2 schools, $n = 178$), desk illumination was increased to approximately 550 lux, compared to approximately 100 lux in the control group (2 schools, $n = 139$). After 1 year, the intervention group showed a lower incidence of new myopia onset (4% vs. 10%, $P = 0.029$), reduced myopic refractive progression (−0.25 D vs. −0.47 D, $P = 0.001$), and shorter axial elongation in both non-myopic (0.13 mm vs. 0.18 mm, $P = 0.023$) and myopic children (0.20 mm vs. 0.27 mm, $P = 0.0001$).

In the second study, Suh and colleagues[240] tracked the effect of classroom illuminance (as measured by the proxy ratio referred to as "daylight factor") on ocular growth in first-grade students from 2 schools over a 6-month period. The authors reported that students in a low daylight factor school who had shorter eyes (axial length [AL] < 22.7 mm) at the start of the trial had a marginal increase in their axial elongation (0.04 mm longer, $P = 0.049$), as compared with their counterparts in the high daylight factor school.

Read et al.[241] found that in 10 to 15-year-old children, exposure to lower daily light intensity (459 ± 117 lux) was associated with greater axial elongation than moderate (842 ± 109 lux) or high (1455 ± 317 lux) average daily light exposure. He et al.[9] calculated that the anti-myopic effects of time outdoor was duration and light intensity dependent, such that a 9% to 30% relative reduction in myopia incidence, compared with no outdoor exposure, could be attained with significant cumulative lux/day dosing. Further evidence that the dose of daylight may be important is related to reported seasonal differences in eye growth; more axial elongation has been observed in winter compared to summer.[15–18,242–244] This is also the case for physiological eye growth, with the bulk of annual ocular elongation for 7 to 11-year-old emmetropes and low hyperopes being in winter.[245]

Higher intensity of daily light exposure has been reported to be associated with slower annual axial elongation in young adults (18–30 years of age).[246] Both emmetropic children and young adults spend more time in bright light (typically defined as >1000 lux) than those who have myopia, particularly in the summer.[241,246] However, Wu et al.[4] found no differences in bright light exposure (HOBO light sensors) during the school week. This finding may be due to concurrent public health initiatives advising all children in the region to spend more time outdoors.

Time "outdoors," typically defined by light intensity >1000 lux, may be one potential reason, in what likely is a multi-factorial problem, for the nearly 4 times greater myopia prevalence difference reported in Singaporean children (8–12-year-olds) versus Australian children (10–12-year-old). Objective assessments using wearable light sensors showed that Australian children spent on average >40 minutes more per day outdoors than their Singaporean peers.[247] Similar findings have been reported for American children aged 5 to 18 years.[248,249] There are several limitations to consider when interpreting these findings; (1) the assessment of "lighting experienced" varies with some studies using objective sensors and other subjective reports, (2) the timepoint in which some studies occur is not optimal (e.g., light experienced in school-age versus teenage years), (3) use of >1000 lux as a proxy for being outdoors has been debated[250,251] as this level of intensity can be experienced (on rare occasions) indoors when next to the window, (4) there are several other visual environmental factors that vary when outdoors versus indoors beyond bright light, (5) does wearing a myopic prescription, and in particular eyeglasses, modulate a child's behavior, such that they are more comfortable indoors and doing near work tasks, and (6) what role does coatings on glasses that reduce transmission (e.g., UV or blue blocking) play. Therefore, further work is warranted to optimize the illuminance proxy for classifying outdoor vs. indoor activity (see Supplementary Material: Wearable Technologies).

In summary, indirect evidence from human studies suggests that exposure to bright light may be associated with less myopic refraction. However, there is currently insufficient evidence to determine what constitutes a "protective" light intensity in humans, or how this may be influenced by temporal exposure patterns, seasonal variation, and geographic location. It is also unclear whether the spectral composition of the light source plays a determining role. This raises the question: can artificial light provide the same protective benefits as natural sunlight, or are additional characteristics of sunlight—such as its broader spectral composition (discussed in the following section)—also required? Importantly, current human data make it difficult to determine whether bright light directly influences refractive development or merely serves as a proxy for time spent outside, with protective effects potentially stemming from other outdoor-related factors. To this end, defining exposure to natural sunlight based on an arbitrary cutoff of >1000 lux may miscategorize a child's visual experience, as such light intensities (on rare occasions) can be achieved indoors. As discussed in the assessing the visual environment in children section of this paper (section 9), study-specific and geographically tailored thresholds may be necessary

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in future research to more accurately classify indoor versus outdoor light exposure.

Spectral Composition

An increasing body of literature suggests that the spectral composition of light can also significantly influence eye growth. This may occur through various mechanisms, including modulation of classical photoreceptors (rods and cones) and/or non-classical opsins, the evidence for which is discussed below.

Findings From Animal Studies

Animal studies have shown that chromatic cues play a role in normal refractive development. One proposed mechanism is through longitudinal chromatic aberration (LCA), whereby shorter (blue) wavelengths focus more anterior than longer (red) wavelengths due to the eye's optics.[252] LCA has been observed in all vertebrates tested. It is substantial in magnitude (up to 2 diopters in the human eye), is consistent between individuals of the same species (as it depends on the optical dispersion properties of the eye), and is relatively constant across the surface of the retina.[119]

One possible cue for emmetropization may be the detection of different image statistics (the quantitative properties that make up an image, e.g., color contrast) by the short-wavelength-sensitive and long-wavelength-sensitive cones. Such a cue can be caused by LCA, wherein any imbalance in detection across the cone types guides eye growth toward achieving a balance point.[253] LCA would seem to be an ideal cue for emmetropization: short wavelengths in better focus than long would indicate hyperopic defocus and stimulate axial elongation and long wavelengths in better focus than short would indicate myopic defocus and inhibit axial elongation. Therefore, how normal ocular development is influenced by the spectral composition of light has gained significant attention, both in animal models and humans.

If chromatic cues are important for emmetropization, then one might expect that altering the spectrum of ambient light would have strong effects on refractive development. Indeed, this is what is commonly observed (see recent review Ref. 163). Ambient narrow band light, where chromatic cues for focus are absent, often causes significant hyperopia or myopia.[62,123,124,129,130,134,138,254–261] In other words, altered chromatic cues can over-ride the cue of image sharpness.

There seems to be strong species-specific differences in the responses of emmetropization to ambient narrow band light of various wavelengths, for which there is currently no widely accepted explanation.[120] Although results are somewhat varied, short-wavelength light has been shown to be protective against experimental myopia in chicks, mice and guinea pigs.[63,112,117,118,121–127] In contrast, long-wavelength light has been found to be protective in tree shrews and primates.[128–133] Without strong chromatic cues for determining defocus, the emmetropization feedback system and the refractive state may wander unguided. In this condition, refraction might be driven by species-specific second-order phenomena that are ordinarily controlled out, and produce results that are either hard to interpret or misleading (see Ref. 133 for discussion). Despite the differing effects of narrow band light on eye growth across species and the fact that the precise details of how chromatic cues for defocus are interpreted or how they interact with other visual cues is unknown, chromatic signals seem to be important for refractive development.

Differing from other animal models tested (e.g., chick,[124,262,263] blue acara,[257] and guinea pig[123,255]), both rhesus monkeys and tree shrews[134] consistently develop significant hyperopia in response to narrow-band ambient red light.[129,130] Red light also slows the development of both FDM and LIM in these species,[135,136] with refraction changes strongly correlated with changes in vitreous chamber depth.[129,130,136] The effect was observed in tree shrews even when red light was presented for only 1 or 2 hours a day,[131] although the effect seems to require a high degree of spectral purity – that is, contamination with white light appears to greatly reduce the hyperopic effect in tree shrews.[137]

In tree shrews, amber light – relatively broad-band light only excluding wavelengths below about 500 nm, seems to have similar hyperopia-inducing effects as red light,[138] although, like with red light exposure, the effect seems sensitive to white light leakage. This effect may be due to the lack of short wavelength cone stimulation for both amber and red light. At least in tree shrews, a lack of blue light might be interpreted as a lack of blue contrast and thus inferred as myopic defocus and a signal to slow axial elongation.

Modern light sources and most modern windows and spectacles, filter out violet (about 400 to 450 nm) and ultraviolet (UV, less than 400 nm) light. It has been hypothesized that this lack of violet light in the modern environment could be a risk factor for myopia.[62] Supporting this, in both chicks and mice, two species that show strong transmission in the UV range and which have UV-sensitive photoreceptors, the addition of UV/violet light (360–400 nm) to fluorescent white light was observed to inhibit the development of experimental myopia when compared to animals reared in fluorescent light alone.[62,65] In chicks, the spatial resolving power of the S1 cone photoreceptors under UV light alone is sufficient for detection and compensation to optical defocus,[64] as well as the appropriate patterning of responses to FDM.[63] Of note, the effects of UV/violet light in both chicks and mice occurs only at high intensities. Data from mice suggest that the anti-myopic effects of UV light may act via a neuropsin (OPN5) mediated mechanism.[65]

However, extrapolating these findings to humans is challenging. In contrast to species such as mice, guinea pigs, or chickens (where transmission and detection of UV/violet light are evident; see Fig. 1),[264,265] young, healthy human eyes transmit only minimal UV and violet light through the lens.[66] Consequently, humans exhibit only limited sensitivity to these wavelengths (as discussed further in section 7.4. Spectral Based Approaches for Myopia Treatment).

In addition to modifying the spectral composition of ambient light, it is also possible to preserve its broadband spectrum while selectively altering the spatial frequency distribution of specific wavelengths. For example, when tree shrews viewed video displays where the blue pixels were blurred and the green and red pixels remained sharp, producing chromatically simulated myopic defocus (CSMD), axial elongation was slowed. This could overcome the effect of a myopiagenic environment, producing significant hyperopia,[266,267] further demonstrating that chromatic cues can override focus. A similar, thought transient, effect has been reported in humans by Swiatczak and Schaeffel.[268]

In summary, findings from animal models strongly suggest that chromatic cues play a critical role in refrac-

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tive development. However, their relevance to the onset and development of human myopia remains yet to be demonstrated. The precise mechanisms by which these cues are interpreted or integrated with other visual signals remains far from understood. Nevertheless, two effective strategies for modulating axial elongation using chromatic signals have been demonstrated in animal models: narrow-band red or amber light exposure, and CSMD. Finally, although humans are not typically exposed to monochromatic lighting, the potential impact of reduced or absent exposure to specific spectral regions (e.g., UV/violet light) in modern environments dominated by indoor artificial lighting warrants further investigation into its effects on ocular development.

Findings From Human Studies

Currently, most evidence regarding the role of spectral composition in refractive development is derived from animal studies. Nevertheless, some supportive data from human studies exist. For instance, clinical evidence from a 5-year cohort study found that the cumulative myopia incidence was significantly less in children with red-green color vision deficiency than without (35% vs. 57%). These children also exhibited a smaller change in spherical equivalent refraction compared with color normal participants (−1.81 D vs. −2.41 D).[269] This finding suggests a potential link between the spectrum of light perceived by the eye and refractive development. However, the observed effect is relatively weak, less pronounced in other studies,[270] and somewhat inconsistent across the literature, with methodological limitations further complicating interpretation, as highlighted in a recent review.[271]

Longitudinal Chromatic Aberration. Altering LCA in humans can be done optically by placing specially designed lenses in front of the eye to remove, double, or reverse LCA. These manipulations heavily disrupt accommodation[272–274] causing a shift in responses such that accommodation is induced when looking at a red target, showing that chromatic cues provide valuable directional signals also for accommodation.[262,275] Whereas the filtering paradigms can vary across studies, LCA can be digitally simulated on a computer screen by low-pass filtering the color channels of an RGB format display.[276–278]

Following sustained viewing (45–60 minutes) of such filtered videos, studies have found either variable changes in a cohort of myopic eyes[278] or overall axial shortening (an indirect measure of choroidal thickness change) with CSMD in emmetropic, but not myopic eyes,[276] suggesting that the myopic retina may be insensitive to chromatic cues. Transient choroidal thickness increase has been suggested to be a biomarker for long term slowing of eye growth,[279] although this theory has been debated.[279] The question as to whether CSMD could be a viable human anti-myopia therapy in children remains open. However, early findings show that exposure to simulated CSMD on a computer screen for 2 hours a day over 12 days, caused incremental choroidal thickening in a small sample of young adults.[280] Taylor and colleagues also showed that myopic eyes may have reduced sensitivity to low spatial frequency S-cone stimuli when compared to emmetropic eyes and low myopes, which could be a reason why they do not respond to the imposed blur in the blue channel.[281]

To understand the potential influence of LCA upon short term ocular responses to monochromatic lighting, Breher and colleagues[282] used a custom optical device to generate monochromatic interference fringes directly on the retina, thus eliminating the effects of the eye's LCA. In this experiment, no significant changes in axial length were found following a 20-minute period of exposure to short (450 ± 5 nm), medium (550 ± 5), and long (650 ± 5 nm) wavelength light. Given that transient choroidal changes will cause associated changes in axial length, the authors concluded that the lack of change observed in their study suggested that previously documented choroidal changes in response to monochromatic light[283,284] are likely driven by LCA induced defocus. However, the relatively short exposure time (20 minutes), may also have contributed to the lack of significant change, given that previous studies utilized a 60-minute exposure period. Whereas most research on the effects of monochromatic light on the human eye have involved short-term studies (as described in section 7.2b), some longer-term studies have been conducted (see section 7.4).

Choroidal Response to Short-Term Narrowband Light. Recent studies have examined transient choroidal thickness changes to narrowband long and short wavelength lights. Two studies showed that 60 minutes of exposure to red light (peak wavelengths 620–630 nm), through full-room illumination with LEDs, resulted in a small but significant choroidal thinning in young adults.[283,284] However, refractive status was only explicitly analyzed in one study where no difference in response was found between emmetropic and myopic participants.[283,284] On the other hand, another study showed that 10 minutes of exposure to 620 ± 10 nm induced axial shortening (i.e., choroidal thickening) in non-myopic participants, but no effects were noted in myopic participants.[285] In this study, red light exposure was produced by placing a filter in front of the eye. Across all studies, the lighting was not luminance matched, such that perceived brightness differences between conditions may have influenced outcomes.

Exposure to cyan light (peak wavelength approximately 500 nm) from light-emitting glasses for 120 minutes resulted in small but significant choroidal thickening (8–14 microns) in both young adults and children.[286] Similar results were reported for 30 minute exposures each morning for 7 days where a small (5 µm) but statistically significant increase in macular choroidal thickness, as well as an increase in the amplitude of diurnal change of choroidal thickness was reported.[287] Although this study suggests increasing light exposure in the morning results in choroidal thickening, the relative impact of the luminance and spectral content of the light upon the observed changes was not clear because only a single light exposure wavelength was tested. Two other studies used a 60-minute exposure to narrow-band short wavelength light (455–460 nm) and found either a small (6 µm) relative choroidal thickening[288] or no change in young adults.[283]

Role of Increased Device Use and Associated Blue Light in Humans. A significant source of artificial light exposure is from back lit electronic devices, including television, computers, and hand-held tablets and smartphones. These devices tend to have a high proportion of light output in the short wavelength range of the visible spectrum. Electronic device use has become a routine part of daily life, even for young children, with devices being widely available and increasingly used for educational and social purposes. Associations between screen time and myopia have been the subject of long-standing speculation and careful examination. However, current evidence regarding a causal relationship remains insufficient.[289–291] Consistent

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with this finding is the observation that mainstream adoption of devices occurred after the initial surge in myopia prevalence in regions such as East Asia. For instance, the myopia epidemic was largely established in much of East Asia by the 1990s—a period when screen use was still minimal. The internet became publicly accessible in 1993, and smartphones did not become mainstream until after 2008. Further, a recent meta-analysis examining the association between digital screen time and myopia found mixed evidence, with the more recent studies exposing a trend between hours spent by children using screens and myopia; however, results from five studies suggested that screen time was not associated with existing or incident myopia.[289] Identified gaps in knowledge to be considered for future studies encompass the use of objective measures for screen time, differentiation from other near-based activities, and accounting for other behavioral alterations with increased screen time such as reduced outdoor activities and disturbed sleep patterns.[292]

Poor sleep outcomes—which have been linked to myopia development (discussed in section 8. The Role of Circadian Entrainment)—are strongly associated with screen use immediately before bedtime.[293–296] This may be due to blue light from screens suppressing melatonin and disrupting circadian rhythms.[297] However, not all screen time is detrimental; educational content and co-viewing with parents can support learning.[296] Rather than eliminating screen use, the emphasis should be on managing timing (particularly by limiting exposure in the evening), while also promoting greater viewing distances and encouraging regular breaks.

Spectrally Sensitive Non-Image Forming Mechanisms in Ocular Development

Findings From Animal Models. Light may also affect emmetropization through non-image-forming pathways involving both classical photoreceptors and "non-classical" (or "non-canonical") photoreceptors, including OPN4-expressing intrinsically photosensitive retinal ganglion cells (ipRGCs),[100,298,299] as well as other "non-classical" opsins like OPN3 and OPN5.[300,301] Non-classical opsins respond to the spectrum, intensity, and timing of ambient light, but also influence the image forming functions of the eye,[302–304] potentially playing an important role in ocular refractive development.[305–307]

Among the non-classical opsins, OPN4 (melanopsin) is the most extensively studied. It is expressed in ipRGCs, which comprise approximately 0.2% to 2.5% of the total ganglion cell population. They exhibit widespread dendritic coverage across the retina, excluding the fovea.[308,309] The axons of ipRGCs project to brain regions involved in non-image-forming functions, such as circadian photoentrainment and the pupillary light reflex,[299,310–312] and also contribute to image-forming processes like contrast sensitivity, color perception, and pattern vision.[302,303,313] Psychophysical experiments in humans demonstrate that melanopsin photoreceptors, on their own, are sufficient to generate a visual perception in response to both spatial and temporal patterns. However, they are unable to reconstruct detailed image forms. Instead, melanopsin appears to contribute to the perception of visual information over large areas of the visual field and extended periods of time.[314]

Several direct and indirect investigations in mice and guinea pigs have provided evidence of a role for OPN4 in myopia. For example, melanopsin knockout (*Opn4*$^{-/-}$) mice demonstrate abnormal refractive development and greater susceptibility to FDM.[305] The increased myopia was associated with lower retinal dopamine and 3,4-dihydroxyphenylacetic acid (DOPAC) levels, with axial elongation partly attenuated by the administration of the dopamine precursor, L-3,4-dihydroxyphenylalanine (L-DOPA).[305] This highlights a crucial interplay between retinal DA and ipRGCs in the regulation of ocular growth and is consistent with morphological and electrophysiological evidence of synaptic drive of retinal dopaminergic amacrine cells (DACs) by ipRGCs,[315–318] although this input may not drive global retinal DA release.[319]

In guinea pigs, reduced expression of retinal melanopsin in form-deprived and lens-induced myopic eyes,[307] along with the anti-myopic effect of melanopsin antagonists on LIM,[320] further supports the involvement of melanopsin pathways in the development of experimental myopia in animal models. The ipRGC-mediated changes in ocular growth appear to primarily involve the M1 cellular subtype, with minor contributions from M2/M3 subtypes.[306] M1 ipRGCs (among others) play a crucial role in circadian entrainment by sending light information to the suprachiasmatic nucleus (SCN). As discussed later, circadian and/or diurnal rhythms have long been implicated in myopia development and may form one of the primary pathways by which light and ipRGCs influence ocular growth patterns.

There is speculation that OPN3 and OPN5 may also play a role in refractive development. OPN3 is the most widely expressed mammalian opsin, and is believed to play vital roles in cell proliferation and apoptosis, immunological processes, melanogenesis, and wound healing in humans.[301] It is expressed in the periventricular nucleus, the optic nerve, the iris–ciliary body complex,[321] the trigeminal nerve, the retinal ganglion cells, and a limited number of choroidal cells.[322,323] Recently, OPN3 knockout mice were found to exhibit refractive myopia (not typical axial myopia), characterized by reduced lens thickness, shallower aqueous chamber depth, and shorter axial length.[323] With regard to OPN5, which is expressed in the brain and retinal ganglion cells,[103,324–326] Jiang et al.[65] reported that the anti-myopic effects of violet light in mice appear to be associated with stimulation of this opsin family and their downstream pathways.

Findings From Human Studies. Whereas ipRGC signaling has been linked to myopia in animal models, evidence in humans is limited, with several studies observing no association between refractive error and melanopsin driven pupil responses in patients.[327–331] However, one study noted that more hyperopic/less myopic adults exhibited greater pupillary adaptation (more constriction and slower dilation) to repeated blue light stimulation.[332]

Several pieces of non-direct evidence do support a role for ipRGCs in human ocular development. For example, it has been postulated that ipRGC dysfunction may underlie pathological eye growth in MYP-26 patients (MIM: 301010), a disease resulting from a mutation in the X-linked *ARR3* gene encoding the cone arrestin, but which also causes malfunction of the melanopsin system.[333]

Spectral-Based Approaches for Myopia Treatment

The potential use of chromatic cues in myopia treatment has been reviewed in detail in a concurrent IMI white paper.[24] A brief overview of several treatment options currently under

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trial (which are discussed in the context of the animal and human data outlined earlier) is provided below.

There are several approaches to using chromatic light as a therapy for myopia control. One strategy is to alter or restrict the spectral content of ambient light. Alternatively, a broad-spectrum light environment can be maintained while altering the details of image contrast as a function of spatial frequency and wavelength to simulate "protective" myopic defocus. A number of these approaches are summarized below. For certain treatments, such as red-light therapy, effectiveness may depend less on the specific wavelength or chromatic properties of the light, and more on secondary factors such as the thermal energy delivered to ocular tissues.

Ambient amber light. As noted, amber light inhibits ocular growth in tree shrews.[138] However, this anti-myopic effect was shown to be attenuated by broadband light leakage. Thus, if this result is translatable to human myopia, conventional amber (blue-blocking) spectacles would likely not be effective for myopia control, but amber tinted contact lenses or goggles that block out peripheral light might be. Amber filters pass a broader region of the visible spectrum than red filters and may provide a more acceptable visual experience. Amber contact lenses are currently used in sport vision (and other) applications,[334] with a clinical trial of amber contact lenses for myopia control currently underway (Trial ID: NCT06598423).

Repeated "Low Level" red light (RLRL) therapy. repurposed from amblyopia treatment protocols in China and supported by evidence from animal models—including studies in rhesus monkeys and tree shrews demonstrating red light's inhibitory effects on experimental myopia[129,130]—RLRL therapy emerged as a promising noninvasive intervention for myopia control (for a comprehensive clinical overview, see Ref. 24). The original and most extensively studied of the desktop RLRL devices deliver brief exposures of long-wavelength (approximately 650 nm) laser red light to the fovea, administered for 3 minutes twice daily, with sessions spaced at least 4 hours apart.[335–341] Clinical trials conducted in China using this system have reported significant reductions in myopia progression (mean 0.57 D/year) and axial elongation (mean 0.28 mm/year) compared with single vision spectacles (see reviews Refs. 342 and 343). Notably, some studies have documented axial length regression, and others have shown efficacy in delaying or preventing myopia onset.

Despite these promising outcomes, only a limited number of trials meet the IMI's recommended standards for study duration and washout periods.[344] Concerns persist regarding the long-term safety profile and the incomplete understanding of the underlying mechanisms. For instance, a study by Xiong and colleagues[345] of 120 children undergoing RLRL therapy observed a significant choroidal thickening (mean change 15 µm) after 1 month, which persisted at 12 months. These longer-term changes contrast with the short-term thinning observed in studies of red-light exposure in young adults.[283,284] This discrepancy may reflect age-dependent physiological differences, divergent short- versus long-term effects of RLRL light exposure, or potentially indicate a photo-induced inflammatory response to the specific light sources used in these studies.

The therapeutic mechanism in children likely diverges from that observed in animal models exposed to ambient red illumination. Whereas animal studies suggest that monochromatic light influences ocular growth via spectrally sensitive visual signaling pathways—from the retina through the choroid to the sclera,[163] RLRL therapy may act through direct photo-biomodulation of metabolic processes in the retina,[346] choroid,[345] and/or sclera,[347] or by alleviating scleral hypoxia through enhanced choroidal perfusion.

Importantly, the designation "low-level" may be misleading, as laser intensities used in RLRL devices often approach or, in some cases, exceed safety thresholds defined for Class 1 lasers under ANSI Z136.1-2014 standards.[348,349] Although several clinical trials have reported on the shorter-term changes associated with RLRL, there remains a significant gap in our understanding of its long-term effects—particularly those extending beyond 2 years—at the cellular and sub-cellular levels in human ocular tissues.[350] Recent reports of retinal damage following red laser therapy in humans has been reported, including reduced cone density, using optical coherence tomography (OCT), autofluorescence, and/or ERG.[351,352] Therefore, potential changes in human ocular tissue must be routinely investigated in future trials of RLRL therapy, using OCT and high-contrast visual acuity.

Cyan light. Two hours of exposure to cyan light (507 nm) has been reported to result in a significant transient reduction in axial length due to thickening of the choroid compared to broadband white light in young adults and children[286]; however, longer-term effects of cyan light on ocular growth and myopia progression are unknown. As described earlier, a 1-week period of brief morning exposure to cyan light resulted in a small but statistically significant increase in choroidal thickness in young adults,[287] although these choroidal changes likely resulted from changes in choroidal circadian rhythms. Therefore, much research is still needed to evaluate the potential utility and efficacy of cyan light for myopia control.

Ultraviolet light. As discussed earlier, a lack of exposure to UV light (due to increased time spent indoors in modern society) has been hypothesized to be a risk factor for the development of myopia.[62] In order to protect the retina from the phototoxic effects of short wavelength light, the ocular media filters out these wavelengths so strongly that it is "nearly invisible" and its effects on the retinal image are likely masked out by the rest of the visible spectrum under normal conditions.[66] The type of UV radiation that the human eye is exposed to when spending time outdoors is mainly UV-A (315–400 nm), as most of UV-B (280–315 nm) and all of UV-C (100–280 nm) is being blocked by the earth's atmosphere.[353] This in part is the rationale behind the experiments by Torii et al.[62,354,355] and Jiang et al. (i.e., increasing exposure to UV light may help inhibit the development of myopia).[65] In retrospective studies on adults and children, there are indications that UV-light transmitting contact and spectacle lenses may result in less axial length growth.[62,354,356] However, in two subsequent RCTs,[355,357] where eyeglasses that transmitted 360 to 400 nm light were worn by children aged 6 to 12 years with varied dosing, results were inconclusive. To what degree radiation in this wavelength range also plays a role in human myopia is therefore still an open question.

Although Torii et al.[355] reported no short-term adverse effects over 24 weeks, it is important to note that exposure to UV-radiation in the 370 to 400 nm range can cause damage to both the skin and the crystalline lens,[358–361] as well as affecting scleral collagen.[362] Further work is required to understand the role of 360 to 400 nm radiation in human emmetropization, requiring development of wearable light

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sensors that include measures of radiation in these ranges. Regardless, this area of investigation requires balancing the benefits and potential risks of short wavelength light as a treatment of myopia.[359–361,363]

Blue light. As outlined earlier, findings from animal models—primarily in mice—have implicated non-classical opsins in refractive development and myopia, with particular emphasis on ipRGCs that express melanopsin (OPN4).[100,298,299] As illustrated in Figure 1; the Table, OPN4 exhibits a peak spectral sensitivity approximately 479 to 480 nm (corresponding to the blue region of the spectrum)—a property that is highly conserved across species, including humans. Melanopsin is distributed throughout ipRGCs, including their axonal processes within the optic nerve head (ONH). Based on this, ongoing trials are investigating whether selective activation of ipRGCs via targeted blue light exposure to the ONH may exert an anti-myopic effect (for a comprehensive clinical overview, see Ref. 24).

The Role of Circadian Entrainment

One of the key pathways through which light may influence ocular growth and myopia is its role in circadian entrainment. As the primary cue for aligning the body's internal clock with the 24-hour day, light plays a central role in regulating circadian rhythms. Both animal and human studies provide direct and indirect evidence linking circadian entrainment to normal ocular development and the onset of myopia. Much of this evidence has recently been reviewed in another IMI white paper[279] and is only summarized in brief here.

Findings From Animal Studies

In the 1960s, initial studies in chicks revealed that disruption to circadian entrainment by rearing animals in constant light or darkness disrupted emmetropization,[148,149,364] causing excessive vitreous elongation and corneal flattening, leading to hyperopia—even in FDM and LIM models.[365–368] A minimum of 4 hours of darkness daily was needed to prevent these effects.[369]

In mammals, constant darkness induces hyperopia in mice[370] and rhesus monkeys,[371] but induces myopia in tree shrews.[372] In contrast to chicks, constant light has little effect on normal refractive development in those mammals studied,[370,373,374] possibly due to differences in their circadian systems relative to birds.[375,376] However, constant light has been observed to enhance FDM in mice.[370] Further, artificially lengthening the circadian period in mice to a 13:13 light–dark cycle induces greater myopia than a standard 24-hour cycle.[377] Collectively, these studies underscore the critical role of circadian entrainment in refractive development, further supported by time-of-day-dependent effects of anti-myopia treatments in animal models discussed earlier (see section 6.1b).

A key question remains: how does circadian entrainment influence normal and abnormal refractive development? In all species studied, the axial growth of the eye oscillates in a circadian pattern—increasing during the day and decreasing at night—with a peak (acrophase) in the afternoon.[378–382] Choroidal thickness also fluctuates diurnally, in approximate anti-phase to axial length.[379,380,383–387] Optical defocus disrupts these natural rhythms, altering growth.[378–380,382] In chicks, form-deprivation and negative lenses shift axial length rhythms into exact anti-phase with choroidal rhythms, promoting growth both day and night.[379,388] In contrast, positive lenses synchronize these rhythms, slowing eye growth.[389,390] In support of the physiological evidence, experimental myopia has been consistently reported to be associated with changes in gene pathways linked with circadian entrainment.[215,391–400]

The precise biochemical mechanisms by which circadian entrainment influences ocular growth remain unclear. One proposed pathway involves modulation of ocular dopamine levels. As discussed earlier, dopamine—which is heavily implicated in myopia and believed to mediate the protective effects of bright light—plays a central role in regulating and entraining the retinal clock. As noted, its synthesis, storage, and release follow a light-driven diurnal pattern that is altered during the development of experimental myopia.[186–191,401,402] Other neurotransmitters, such as gamma-aminobutyric acid (GABA) and melatonin, also contribute to retinal circadian regulation and may influence ocular growth.[222,403–405] Additionally, a non-retinal mechanism may be involved, wherein circadian rhythms in scleral extracellular matrix (e.g., proteoglycan) synthesis—potentially driven directly or indirectly by intraocular pressure rhythms—affect eye growth.[388,406]

Findings From Human Studies

Similar to animals, defocus-induced phase shifts in axial length and choroidal thickness rhythms have also been observed in humans.[407,408] Further, brief exposures to myopic or hyperopic defocus at different times of day produce distinct – albeit transient – effects on ocular growth.[409] Specifically, myopic defocus leads to a significantly greater reduction in axial length when applied in the evening compared to the morning, whereas hyperopic defocus causes a significant axial elongation when applied in the morning, but not in the evening.[409] Similar findings have been reported in chicks,[389,390] suggesting that the eye may be more responsive to "go" signals (promoting growth) early in the light cycle, and more sensitive to "stop" signals (inhibiting growth) later in the day. Whether timing myopigenic activities (e.g., intensive reading) or administering myopia interventions at specific times of day can more effectively combat myopia remains an important and unresolved question.

Light and circadian entrainment are key regulators of sleep–wake patterns,[410] prompting speculation about a potential link between altered sleep behavior and myopia.[173] Light exposure directly influences sleep by inhibiting melatonin secretion from the pineal gland to indicate "daytime." A study by Abbott et al.[328] found no significant difference in morning salivary melatonin levels between myopic and non-myopic individuals, although myopes did report poorer sleep quality. Conversely, Kearney et al.[411] found that young myopic adults had significantly higher serum melatonin levels than their non-myopic counterparts, however, it is important to note that measurements were only made at a single time point – a methodology which is considered flawed.[412]

Melatonin rises sharply in the evening—a phenomenon known as dim light melatonin onset (DLMO)—to prepare the body for sleep.[413] Myopic children exhibit a significant phase delay in DLMO compared with emmetropes.[414] However, while some studies report delayed sleep onset and shorter sleep duration in myopic children,[414–416] others find

no association or conflicting results.[417–419] A large review (n = 49,277 across 18 studies) concluded that the relationship between sleep and myopia remains inconclusive.[420,421] Thus, further studies using objective measures of light exposure and sleep in young children are needed to determine whether altered sleep behavior is observed in myopes, and whether any such alterations contribute to the development of myopia or is a consequence of it.

Assessing the Visual Environment of Children

Beyond light, animal and human research has identified a wide array of visual environmental factors, including near work behaviors, contrast, and spatial frequency content, that may play a role in the regulation of eye growth and the development of myopia (for review see Refs. 25 and 35). It also appears that the temporal dynamics of each of these factors may be important, as well as interactions of signals across the near peripheral retina. The difficulty in drawing conclusion from the existing epidemiological literature may be due to the subjective nature in which the visual environmental has traditionally been quantified. Marcos[422] has recently argued for the concept of the "optical and visual diet," and proposed some significant methodologies required to elucidate aspects of light stimulation that may be crucial to the development of myopia. Wearable technologies capable of objectively quantifying such environmental factors and exposures have the potential to enhance our understanding of how they affect human eye growth and contribute to myopia development.

Wearable light sensors have been used in different studies, to quantify various aspects of ambient light exposure, including white light (broadband) illuminance,[423–426] and sensors to quantify the spectral content of ambient light exposure.[427–430] More complex visual environmental factors, such as the contrast and spatial frequency content of the environment, can be determined through analyses of images captured from wearable cameras, such as those used by head mounted mobile eye tracking devices.[431] Additional technical specifications, capabilities, and the minimal required characteristics of wearable technologies, along with relevant reporting guidelines, are detailed in Supplementary Material S1.

Factors to Consider With Regard to Wearable Devices

Studies of light exposure and myopia assessed with wearable sensors have used a range of approaches and metrics to describe personal light exposure.[241,247,251,423,424,426,427,432] A first challenge may therefore be to agree upon a limited set of parameters that will be useful and meaningful with available technologies while waiting for further development and miniaturization. Is it necessary to measure anything else but intensity? If not, what part of the solar spectrum is more important to sample; should UV-A and near infrared also be quantified? Must the full range of ambient illumination be quantified, from scotopic to high photopic illuminance? Is an observation period of 7 days sufficient? It is possible there is a need to understand not only intensity, spectral composition, and duration, but also the time of day of light exposure, as well as prior light history. Is there also a need to collect (simultaneously) non-irradiance information, including near work, eye movements, as well as contrast and spatial information about the environment? Importantly, to provide reliable objective measures of the visual environment, wearable devices must have the dynamic measurement range to capture the typical exposures encountered in the environment.

Given the potential for both dim and bright light exposure to impact upon eye growth,[433] light sensors should ideally provide measures of illuminance from scotopic through to bright photopic conditions, thus covering the range of irradiances that a human would experience during a normal day, independent of time of year. Further, a wearable light sensor should, if possible, measure spectral data covering the full range from UV-A to near infrared, 320 to 1100 nm. This would allow quantification of light in terms of both non-visual and visual responses.[147]

The frequency and duration of sampling is also important to consider. Studies aiming to quantify habitual environmental exposures need to sample frequently enough to account for relatively rapid changes in environment given the dynamic nature of human behaviors, and to record for sufficient duration to account for day-to-day variability in exposures. Few have explored this issue systematically, but Ulaganathan et al.[434] examined the effects of varying measurement sampling frequency and duration on ambient light exposure measures from a wrist-worn light sensor. The authors concluded that sampling frequency of at least 2 minutes and duration of at least 1 week was required. Studies of sleep behaviors using wearable devices also suggest that approximately 1 week of objective data recordings are needed to quantify sleep patterns reliably.[435,436] For comprehensive measures of light exposure, recordings should extend across waking hours, however, for studies examining circadian rhythms/sleep parameters, 24-hour recordings[414] are required. Given that environmental exposures are likely to vary across different times of the year,[18] capturing data over longer periods of time across different seasons, academic periods and weekdays versus weekends may more reliably capture habitual environmental exposures.

Wearable devices also come with additional challenges of form factor, body position, and adherence. Each form factor (e.g., pendant or spectacle mounted) and body position (e.g., wrist versus head mounted) has their challenges if the goal is to estimate how much light reaches the retina, and current wearable devices are less accurate at measuring higher illuminance levels.[425,437] The body position of the light logger will somewhat bias the measurement and affect both the accuracy in terms of the description of light reaching the eye as well as our ability to make comparisons across studies using different devices.[438] Presumably, light sensors mounted on the head or on spectacles better estimate the light entering the eye. Studies have shown that light measures captured from different body positions are correlated, but with significant differences.[439–441] Moreover, contradictory findings with regard to whether eye or head level measures are higher than those obtained from identical devices worn on the chest or wrist exist.[439–441] Device weight and form factor likely also affect adherence[442] and could even modulate their indoor and/or outdoor activities, which introduces fatal biases.

Many studies have used a threshold of 1000 lux to differentiate between indoors and outdoors,[241,251,423,424,426,443] but is it appropriate to use such a threshold even if its validity has been controversial?[250,251] Is just assessing the cumu-

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lative exposure of light important?[9] Or rather, is it important to find a more geographic-specific threshold, that can allow for more precise temporal analysis of outdoor activity, as has been suggested as important from animal studies? Any clinical recommendations regarding light exposure may depend not only on geographical location and variation in daylight irradiance levels (e.g., sunny versus cloudy day), but also on the individuals sensitivity to light.[444–446] In addition, there may be individual variations related to what degree the eyebrow and eyelids protect/cover the eye, pupil size and pre-receptoral filtering of light.[447] Perhaps there is a need to acquire more comprehensive knowledge about the physical environment beyond illuminance and spectral composition. For example, gaining a deeper understanding of the spatial frequency and contrast characteristics of visual environments may clarify how these factors affect the spectral quality of light and, consequently, how they may affect ocular growth. The myopia community needs to develop an understanding of, and subsequently agree upon, how to categorize the physical environment. Is it sufficient to classify the visual environment solely by the spatial frequency content?[33] Or shall we categorize visual environments for example as being natural or designed, as suggested by a recent National Academies of Sciences Engineering and Medicine Consensus Report; "Myopia: Causes, Prevention, and Treatment of an Increasingly Common Disease"?[448]

Although most visual environmental factors considered important for myopia development can be tracked across the variety of different wearable devices that are currently in use, no single device can accurately capture all parameters of interest across the full dynamic range of the visual system. Future developments therefore may involve combining multiple measurements and sensors to expand the measurements possible. This may include multiple light sensors to expand the dynamic range in terms of illuminance and spectral components, in combination with range finding or image-based systems. A common issue with all wearable devices is the potential for the act of wearing the device to alter habitual behaviors. Further miniaturization of components and devices may allow for less obtrusive devices and reduce such effects. As the field moves forward, it is imperative that standardization exist for reliable comparisons across studies and preferably such work would benefit from building on the work done by and in collaboration with the more diverse scientific community (for example frameworks see Refs. 438 and 449).

Summary and Evidence Based Clinical Recommendations

Time spent outdoors has consistently been associated with a delayed onset of myopia in children and has therefore been incorporated into official prevention programs in several countries. While research continues to refine our understanding of the underlying mechanisms driving the protection effects associated with being outdoors, substantial evidence already supports natural light exposure characteristics as a key contributing factor. This review examined the evidence that underpins this association.

Animal studies have demonstrated a strong link between light characteristics—such as intensity, spectral composition, and photoperiod—with refractive development and myopia. The influence of many of these light parameters on refractive development is often proposed to function through modulation of the retinal dopaminergic system. However, as reviewed, several other biological pathways (either interacting with or independent of dopamine) have also been proposed as potentially significant contributors.

Despite promising findings from animal models, translational evidence to human studies remains limited, leaving the role of light in myopia onset and progression—and its contribution to the protective effects of outdoor time—unresolved. Consequently, while several clinical recommendations regarding outdoor time—such as a minimum of 2 hours per day—can be made, the current literature does not yet support evidence-based guidelines concerning specific characteristics of light exposure. To address this gap, collaborative RCTs in children are needed to directly examine the effects of specific lighting characteristics on refractive development and to clarify their contribution to the anti-myopic benefits of time outdoors. These studies must be carefully designed to control for the numerous confounding visual and environmental factors that differ between indoor and outdoor settings.

As outlined in the companion IMI White Paper on Interventions, several light-based therapies—predominantly involving chromatic manipulation—are currently under clinical investigation, although most remain in the early stages of development. Although some of these technologies appear promising, due to limited efficacy data and, in some cases, unresolved safety concerns, no clinical recommendations can be made at this time.

This review outlines several key considerations for future research investigating the role of light in refractive development, across both animal models and human studies:

1. Species-specific differences—including ocular transmittance and the distribution and spectral tuning of opsins—must be acknowledged when interpreting animal data and assessing its relevance to human myopia.
2. The term "white light" is often misleading. Common light sources (e.g., fluorescent, LED, halogen, etc.) have distinct spectral compositions that result in different patterns of opsin activation, which can further vary across species. Therefore, it is essential that studies report the SPD of their light source, along with radiance metrics, as this provides more meaningful information than illuminance (lux) alone.
3. Wearable technologies can help facilitate the objective quantification of the dynamic visual environments experienced by children. In doing so, these devices can help identify which characteristics of light, along with broader visual cues, are most relevant to refractive development. Central to this effort is the development of standardized protocols for data collection and reporting—an area currently undergoing active advancement. A major limitation of current animal and human studies is the tendency to examine individual aspects of light or visual cues in isolation. In natural environments, these factors interact in complex and dynamic ways. Understanding these interactions is critical to fully elucidating the mechanisms underlying refractive development.
4. The impact of artificial lighting sources—such as LEDs used commonly in homes, schools, workplaces, and electronic devices—on refractive development remains poorly understood. Often, such sources exhibit a narrower spectral distribution (with unique

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flicker characteristics) that is skewed toward either short-wavelength (cool white) or long-wavelength (warm white) light. As exposure to artificial lighting becomes increasingly prevalent in daily life, understanding its potential influence on visual development represents an important and timely area of research.

These considerations must also be placed within the context of broader, unresolved questions surrounding the etiology, progression, and prevention of myopia.

- Why is myopia not self-limiting? Specifically, why does the inhibitory component of the emmetropization feedback loop appear to fail once myopia begins to develop in children?
- Why does myopia develop in the absence of consistent retinal defocus? This raises questions about the adequacy of current animal models of visual regulation in refractive development.
- As recently reviewed,[450] is the myopic retina/choroid in a fundamentally different state compared to a hyperopic or emmetropic eye?[268,276,280,281,285,450–467] If so, do functional deficits precede the onset of myopia, or do they emerge concurrently? If functional differences arise at myopia onset, do interventions need to be tailored differently for prevention versus progression?

In closing, more rigorous, objective and collaborative clinical research is required to identify the specific visual cues critical to myopia development as well as the protective effect of time outdoors. Results from such trials are essential for developing evidence-based recommendations and equipping clinicians with informed responses to the growing number of behavior-related inquiries from concerned parents and caregivers.

Acknowledgments

Supported by the International Myopia Institute. The publication and dissemination costs of the International Myopia Institute reports were supported by donations from the Brien Holden Vision Institute, Carl Zeiss Vision, CooperVision, EssilorLuxottica, Hoya, Thea, Alcon, and Oculus.

Disclosure: **R. Ashby**, (P); **E.N. Harb**, CooperVision (F), Meta LLC (F); **L.A. Ostrin**, Zeiss (C), Alcon (C), Essilor Luxotica (C), Meta LLC (F); **D.I. Flitcroft**, CooperVision (C), EssilorLuxottica (C), Johnson & Johnson Vision (C), Vyluma (C), Thea (C), Dopavision (C), Ocumetra (O), Myopia control monitoring tools and devices (P); **F. Schaeffel**, None; **C. Karouta**, None; **T. Gawne**, (P), Alcon (F), Meta LLC (F); **R. Chakraborty**, Essilor Luxotica (F); **K. Thomson**, None; **S. Read**, Alcon (F), CooperVision (F), Dopavision (F), Meta LLC (F); **R.C. Baraas**, None; **R.P. Najjar**, None; **K. Rose**, None; **J.R. Phillips**, (P, R); **P.-C. Wu**, None; **X. He**, None; **X.N. Liu**, ZEISS Vision Care (E); **W. Lan**, None; **B. Swiatczak**, Roche (F), Hoffmann-La Roche (E); **H. Torii**, (P); **A. Chia**, EssilorLuxottica (C), Hoya (C), Johnson & Johnson (C, F), Xingqi Pharmaceuticals (C), Santen Pharmaceutical (C, F); **I. Morgan**, Eyerising International (F), Essilor Luxottica (F)

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