

Photophobia in Migraine: Neural Mechanisms, Epidemiological Burden, and Evidence for Wavelength-Selective Non-Pharmacological Management

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Abstract

Background: Photophobia affects 80-90% of migraine patients during ictal episodes and approximately 60% interictally, making it the most prevalent non-headache symptom of migraine. In India, with an estimated 213 million migraine cases (Global Burden of Disease 2019), approximately 170-190 million individuals experience clinically significant light sensitivity. Despite this burden, non-pharmacological management remains limited to behavioural avoidance and generic tinted eyewear that does not target the identified neural mechanisms.

Objective: To provide a comprehensive review of the neural mechanisms underlying migraine photophobia, quantify the epidemiological burden with emphasis on the Indian population, critically evaluate the evidence for FL-41 wavelength-selective filtration, analyse the dark adaptation paradox associated with indoor sunglass use, and discuss dual-band filtration approaches that address the complete mechanistic model.

Methods: Narrative review of peer-reviewed literature including mechanistic electrophysiology studies (Noseda et al. 2010, 2016), ipRGC characterisation (Berson et al. 2002; Hattar et al. 2002; Do & Yau 2010; Schmidt et al. 2011), clinical trials of FL-41 filtration (Good et al. 1991; Blackburn et al. 2009; Hoggan et al. 2016; Reyes et al. 2024), epidemiological data (GBD 2019; Stovner et al. 2022), and photophobia classification literature (Digre & Brennan 2012; Noseda & Burstein 2013).

Findings: Migraine photophobia is mediated by at least two retinal pathways: (1) a melanopsin/ipRGC-driven pathway responsive to 460-520 nm (blue-cyan), projecting directly to posterior thalamic neurons where convergence with trigeminal nociceptive input produces pain amplification; and (2) a cone-driven pathway responsive to 585-600 nm (amber) and 620+ nm (red), operating through classical retinal processing circuits independent of melanopsin. Green light (~530 nm) uniquely reduces headache intensity through a proposed inhibitory cone-mediated circuit. FL-41 lenses (480-520 nm filtration) demonstrated >50% migraine frequency reduction in paediatric populations (Good et al. 1991), 71% patient preference over alternative tints in blepharospasm (Blackburn et al. 2009), and 76% reduction in neural pathway activation in chronic photophobia (Reyes et al. 2024). Dark sunglasses paradoxically worsen photophobia through progressive dark adaptation involving photoreceptor gain increase, chronic pupil dilation, and potential central sensitisation. Dual-band filtration addressing both pathways while preserving the beneficial green band represents a theoretically optimal approach consistent with the complete mechanistic model.

Conclusion: Wavelength-selective filtration targeting identified neural pathways represents an evidence-informed non-pharmacological approach to photophobia management that avoids the dark adaptation paradox. Indian-specific epidemiological and clinical outcome data remain absent, representing a critical research gap in the population bearing one of the world's highest migraine burdens.

Keywords: photophobia, migraine, intrinsically photosensitive retinal ganglion cells, ipRGC, melanopsin, FL-41, wavelength-selective filtration, photobiological eyewear, trigeminal pathway, thalamic convergence, dark adaptation, India, non-pharmacological management

1. Introduction

Photophobia, clinically defined as an abnormal sensitivity to light producing discomfort or pain, is the most prevalent non-headache symptom of migraine and a diagnostic criterion in the International Classification of Headache Disorders (ICHD-3). The term, derived from Greek (*phos* = light, *phobos* = fear), is technically a misnomer: photophobic patients do not fear light but experience genuine pain upon light exposure. This distinction is clinically significant, as it positions

photophobia as a neurological pain phenomenon rather than a behavioural aversion.

Epidemiological estimates consistently place the prevalence of ictal photophobia (during migraine attacks) at 80-90% among migraine patients, exceeding the prevalence of nausea (~70-80%), phonophobia (~70%), and osmophobia (~40%). Interictal photophobia - light sensitivity persisting between attacks on headache-free days - affects approximately 60% of migraine patients (Digre and Brennan, 2012), suggesting chronic sensitisation of the neural pathways mediating light-evoked pain.

The Global Burden of Disease Study 2019 ranks migraine as the second leading cause of years lived with disability (YLD) worldwide, after low back pain. India bears one of the highest migraine burdens globally, with an estimated prevalence of 25-26% among adults (approximately 213 million cases annually). Applying published photophobia prevalence rates to this population yields an estimated 170-190 million Indians experiencing clinically significant migraine-related light sensitivity - a figure that exceeds the entire population of most countries.

Despite this scale, the non-pharmacological management of migraine photophobia in India and globally remains underdeveloped. Standard clinical approaches consist of behavioural avoidance ('avoid bright lights'), generic tinted eyewear with uncharacterised spectral properties, or dark sunglasses - the latter being counterproductive through progressive dark adaptation (Katz and Digre, University of Utah Moran Eye Center). The identification of specific neural pathways mediating photophobic pain (Noseda et al. 2010, 2016) and the availability of wavelength-selective filtration technologies provide an opportunity to develop evidence-informed management strategies targeting the identified mechanisms.

This review synthesises the mechanistic, epidemiological, and clinical evidence relevant to non-pharmacological photophobia management, with particular attention to the Indian context.

2. Clinical Classification of Photophobia in Migraine

2.1 Ictal Photophobia

Ictal photophobia refers to light sensitivity occurring during active migraine episodes. During an attack, the pain amplification is acute and dramatic: patients report that normal indoor lighting (100-500 lux) produces discomfort equivalent to direct sunlight exposure (50,000-100,000 lux). This is not subjective exaggeration - electrophysiological studies have demonstrated objectively measurable increases in visual evoked potential amplitudes and altered cortical excitability during migraine attacks (Ambrosini et al. 2003).

Ictal photophobia drives characteristic avoidance behaviours: retreating to darkened rooms, closing curtains, wearing sunglasses indoors, and avoiding screen use. While these behaviours provide acute relief by reducing retinal illuminance, they are functionally disabling and, in the case of chronic indoor sunglass use, can paradoxically worsen long-term light sensitivity (see Section 5).

2.2 Interictal Photophobia

Interictal photophobia - light sensitivity persisting between migraine attacks - represents a distinct and arguably more disabling clinical entity. Approximately 60% of migraine patients report chronic light sensitivity on headache-free days (Digre and Brennan, 2012). The persistence of photophobia between attacks suggests chronic sensitisation at one or more levels of the neural pathway: increased baseline ipRGC excitability, long-term potentiation of thalamic convergence neurons, reduced descending pain modulatory inhibition, or altered cortical excitability.

The functional impact of interictal photophobia extends across daily occupational functioning (inability to tolerate standard office fluorescent/LED lighting), driving (oncoming headlights, bright sunlight), screen-dependent work (computer and smartphone use becomes aversive), and social participation (restaurants, shopping centres, outdoor events become difficult). For patients with chronic migraine (15+ headache days per month), interictal photophobia may effectively be continuous, creating a permanent disability that current clinical frameworks inadequately address.

2.3 Grading Photophobia Severity

Grade	Clinical Description	Functional Impact	Estimated Prevalence
Mild	Discomfort in bright environments only	Minor avoidance behaviours	~20% of migraine patients
Moderate	Discomfort under standard indoor lighting	Preference for dimmed environments; screen difficulty	~40% of migraine patients
Severe	Pain under any ambient lighting; only comfortable in darkness	Significant occupational disability; indoor sunglass dependence	~20% of migraine patients

Interictal	Persistent between attacks on headache-free days	Chronic functional limitation	~60% of migraine patients
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Table 1. Proposed clinical grading of photophobia severity in migraine. Prevalence estimates are approximate and derived from pooled clinical observations; formal validation studies are needed.

3. Neural Mechanisms of Migraine Photophobia

3.1 The Third Photoreceptor: ipRGCs and Melanopsin

The mechanistic understanding of photophobia was transformed by the discovery of intrinsically photosensitive retinal ganglion cells (ipRGCs) by Berson et al. (2002) and Hattar et al. (2002), published simultaneously in *Science*. These cells constitute a third class of retinal photoreceptor, distinct from rods and cones, expressing the photopigment melanopsin (OPN4). Numbering approximately 5,000 per human eye (compared to ~120 million rods and ~6 million cones), ipRGCs serve non-image-forming visual functions including circadian photoentrainment, pupillary light reflex, and - critically for photophobia - pain pathway modulation.

Melanopsin has several properties that distinguish it from rod and cone photopigments and are directly relevant to photophobia:

- **Spectral sensitivity:** Peak absorption at ~480 nm (blue-cyan), with significant sensitivity extending from ~420 nm to ~560 nm. This places the melanopsin peak within the blue-cyan region of the visible spectrum, offset from the peak sensitivities of S-cones (420 nm), M-cones (534 nm), and L-cones (564 nm).
- **Sustained response:** Unlike rods and cones, which rapidly adapt to sustained illumination, melanopsin phototransduction produces sustained depolarisation without significant adaptation. ipRGCs function as irradiance integrators rather than change detectors, encoding absolute ambient light levels over extended time periods.
- **Phototransduction cascade:** Melanopsin uses a phospholipase C (PLC) cascade leading to TRPC channel opening and cell depolarisation. This is mechanistically distinct from the phosphodiesterase (PDE) cascade used by rods and cones, which produces hyperpolarisation. The PLC-TRPC pathway is evolutionarily ancient, sharing homology with invertebrate phototransduction (*Drosophila* rhabdomeric photoreceptors).
- **Intrinsic photosensitivity:** ipRGCs generate light responses autonomously, without synaptic input from rods or cones. However, they also receive extrinsic input from rod/cone circuits, allowing them to integrate information from all three photoreceptor classes. This dual-input architecture is relevant to the dual-pathway model of photophobia (Section 3.3).

3.2 The ipRGC-Thalamic Convergence Pathway (Nosedá 2010)

The mechanistic link between retinal light detection and migraine pain was established by Nosedá et al. (2010) in *Nature Neuroscience*. Using single-unit extracellular electrophysiology in an anaesthetised rodent model with chemical dural stimulation (to simulate trigeminovascular activation), Nosedá's group recorded from neurons in the posterior thalamus and identified a population with a remarkable convergent property: they responded to **both** retinal light input and dural nociceptive input.

The pathway can be schematised as follows:

RETINAL PATHWAY: Light (460-520 nm) --> melanopsin absorption --> ipRGC depolarisation (PLC/TRPC) --> action potential generation --> optic nerve --> posterior thalamus (LP/Po nuclei)

TRIGEMINAL PATHWAY: Trigemino-vascular activation --> dural nociceptors (C-fibres, A-delta) --> trigeminal ganglion --> trigeminal nucleus caudalis (TNC, medullary dorsal horn) --> posterior thalamus (LP/Po nuclei)

CONVERGENCE: Both pathways terminate on shared neurons in the LP/Po thalamic nuclei. When both inputs are simultaneously active (migraine headache + light exposure), the neural output exceeds the sum of either input alone (*supra-additive summation*). This amplified output is relayed to the somatosensory cortex (S1, S2) and is consciously perceived as increased headache intensity.

This convergence mechanism explains multiple clinical observations: why 80-90% of migraine patients experience photophobia during attacks (the convergence pathway is activated by concurrent trigeminovascular and retinal stimulation); why some patients report interictal photophobia (chronic sensitisation of thalamic convergence neurons lowers the activation threshold); why dark rooms provide relief (removing the retinal input); why FL-41 lenses reduce photophobic discomfort without eliminating all light (selectively reducing the melanopsin-activating wavelengths while preserving overall retinal illuminance); and why dark sunglasses paradoxically worsen long-term photophobia (dark adaptation increases retinal sensitivity, amplifying ipRGC responses when sunglasses are removed).

3.3 The Dual-Pathway Model (Nosedá 2016)

Nosedá et al.'s 2016 publication in *Brain* substantially expanded the single-pathway model. Using narrow-band light stimuli at defined wavelengths (blue ~480 nm, green ~530 nm, amber ~590 nm, red ~620 nm) presented to actively

migraining patients while recording subjective pain ratings, they identified a dual-pathway architecture:

Wavelength	Colour	Photoreceptor	Mechanism	Effect on Pain	Pathway
460-520 nm	Blue-cyan	ipRGC (melanopsin)	Direct thalamic projection via optic nerve	Exacerbation (strong)	Pathway 1 (melanopsin)
~530 nm	Green	M-cones (proposed)	Proposed inhibitory cone circuit	REDUCTION (unique)	Inhibitory
585-600 nm	Amber	L-cones	Classical retinal processing --> thalamus	Exacerbation (moderate)	Pathway 2 (cone-driven)
620+ nm	Red	L-cones (high I)	Classical retinal processing --> thalamus	Exacerbation (intensity-dependent)	Pathway 2 (cone-driven)

Table 2. Wavelength-dependent pain modulation in migraine photophobia (adapted from Nosedá et al. 2016). I = intensity.

The identification of green light (~530 nm) as the sole wavelength associated with pain reduction is remarkable and clinically significant. The mechanism is not fully characterised but may involve M-cone-mediated activation of inhibitory interneurons in the retina or thalamus that counteract the excitatory signals from ipRGC and L-cone pathways. Regardless of mechanism, the finding has direct implications for lens design: the green band (520-560 nm) should be preserved, not filtered, in any optical intervention for photophobia.

The dual-pathway model creates a design specification that conventional single-band FL-41 cannot fully address. FL-41's filtration band (480-520 nm) targets Pathway 1 (melanopsin/ipRGC) effectively but provides no attenuation of Pathway 2 (cone-driven, 585-600 nm). The theoretically optimal photophobia management lens would simultaneously attenuate both pain-exacerbating bands while preserving the green band - a dual-band filtration requirement that exceeds the capability of conventional single-dye tinting.

4. Epidemiological Burden

4.1 Global Migraine Burden

The GBD 2019 study (published in *The Lancet*) estimates that approximately 1.1 billion individuals worldwide are affected by migraine, with a lifetime prevalence of 15-18%. The World Health Organization classifies severe migraine episodes in the same disability category (Grade IV) as dementia, quadriplegia, and active psychosis - reflecting the profound functional impairment during attacks.

Stovner et al. (2022) in their updated global headache prevalence review reported that migraine prevalence has remained stable over the past two decades despite advances in pharmacological management, suggesting that prevention and non-pharmacological approaches remain critical unmet needs in the global headache management landscape.

4.2 The Indian Burden

Parameter	Estimate	Source	Notes
Adult migraine prevalence	25-26%	GBD 2019	Among highest globally
Estimated migraine cases	~213 million annually	GBD 2019, calculated	Based on adult population
Ictal photophobia	80-90%	Digre & Brennan 2012	Applied from global data
Interictal photophobia	~60%	Digre & Brennan 2012	Applied from global data
Estimated photophobic population	170-190 million	Calculated	India-specific data absent
Gender ratio (F:M)	3:1	Stovner et al. 2022	Consistent with global
Sleep disorder comorbidity	93 million	Indian epidemiological studies	Significant migraine overlap
Domestic FL-41 availability pre-2023	Zero manufacturers	Market analysis	Reliance on imports
Imported FL-41 cost	Rs. 8,000-15,000+	Market survey	No Rx; no local support

Table 3. Migraine and photophobia burden in India. Note: India-specific photophobia prevalence data are absent; estimates apply global rates to Indian migraine prevalence. Population-based Indian studies are needed.

4.3 The Clinical Management Gap

The management gap for photophobia in India is substantial. A survey of available options reveals three categories of suboptimal interventions: (a) generic blue light glasses (Rs. 500-2,000) with broad-spectrum AR coatings targeting

400-450 nm, missing the melanopsin peak at 480 nm and providing no published spectral data; (b) imported FL-41 glasses (Rs. 8,000-15,000+) from American brands (TheraSpecs, Avulux) with no prescription availability, no local clinical support, and pricing beyond the reach of most Indian patients; and (c) dark sunglasses, which paradoxically worsen photophobia through dark adaptation (Section 5). No Indian optometrist or neurologist had access to domestically manufactured, prescription-compatible, wavelength-selective photophobia management eyewear prior to 2023.

5. The Dark Adaptation Paradox

The paradoxical worsening of photophobia by dark sunglasses represents one of the most clinically significant yet underappreciated aspects of photophobia management. Research by Katz and Digre at the University of Utah Moran Eye Center has documented this phenomenon through multiple clinical observations and the established physiology of light adaptation.

5.1 Mechanism of Dark Adaptation-Induced Sensitisation

Dark sunglasses reduce retinal illuminance by 80-90%, compared to 30-50% for FL-41 lenses. When worn continuously indoors, this chronic reduction in retinal illumination triggers a cascade of compensatory adaptations:

- **Photoreceptor gain increase:** Both ipRGCs and classical photoreceptors (rods and cones) upregulate their sensitivity in response to sustained low-light conditions. For ipRGCs, this may involve increased melanopsin expression density or altered intracellular signalling gain. For rods, the well-characterised process of dark adaptation involves regeneration of rhodopsin from retinal and opsin.
- **Pupil dilation:** Chronic reduction in ambient light input drives tonic pupil dilation through reduced parasympathetic drive to the Edinger-Westphal nucleus. The dilated pupil admits more light per unit time when the dark glasses are removed, amplifying the retinal illuminance change.
- **Central sensitisation:** Prolonged periods of reduced visual input may contribute to increased excitability of thalamic convergence neurons (Nosedá pathway), effectively lowering the threshold at which light-driven retinal signals produce pain amplification. This mechanism remains hypothetical but is consistent with the known plasticity of thalamic circuits.

The combined effect is a progressive spiral: each period of dark sunglass use leaves the visual and pain-processing systems more sensitive to subsequent light exposure, driving increased sunglass dependence and further sensitisation. Clinically, this presents as patients who initially required Category 2 lenses progressing to Category 3 and then Category 4 sunglasses for indoor use over months to years.

5.2 Why FL-41 Avoids This Paradox

FL-41 lenses operate through a fundamentally different optical strategy. Rather than broadly reducing total retinal illuminance (as dark sunglasses do), FL-41 selectively attenuates a narrow spectral band (480-520 nm) while maintaining moderate-to-high transmission across the remainder of the visible spectrum. The overall transmission reduction is 30-50%, which is insufficient to trigger the dark adaptation cascade. The retina receives enough total light to maintain normal adaptation levels while receiving substantially less light in the specific wavelength band that maximally activates melanopsin and drives the ipRGC-thalamic pain amplification pathway.

6. Evidence for Wavelength-Selective Filtration

6.1 FL-41: Published Clinical Evidence

Study	Year	Journal	Design	Population	N	Key Finding
Good et al.	1991	Cephalalgia	Controlled trial	Children with migraine	20	>50% reduction in migraine frequency vs blue-tinted controls
Blackburn et al.	2009	Ophthalmology	Comparative	Blepharospasm	30	71% preferred FL-41 over 6 alternative tint types
Hoggan et al.	2016	J Clin Neurosci	Technical validation	Migraine + photophobia	N/A	Validated thin-film notch filter coatings matching FL-41 profile
Reyes et al.	2024	Am J Ophthalmol	Clinical study	Chronic ocular pain	N/A	76% showed reduced neural pathway activation with FL-41

Table 4. Published clinical evidence for FL-41 tinted lenses in photophobia-related conditions.

The FL-41 evidence base, while supportive, has notable limitations: small sample sizes (Good et al. N=20), limited adult data (the landmark study was paediatric), absence of large-scale randomised controlled trials, and no India-specific studies. The Blackburn (2009) study was conducted in blepharospasm (which shares some neural mechanisms with migraine photophobia but is a distinct condition). The Reyes (2024) study in chronic ocular pain extends the evidence to a broader photophobic population.

6.2 Dual-Band Filtration: Addressing the Complete Mechanistic Model

Conventional FL-41 filtration targets Pathway 1 (melanopsin/ipRGC-mediated, 480-520 nm) effectively but provides no attenuation of Pathway 2 (cone-driven, 585-600 nm). Given the dual-pathway model established by Noseda (2016), a lens addressing both pathways simultaneously while preserving the beneficial green band (~530 nm) would more completely align with the identified mechanisms.

NeuroCalm FLX+ (Indian Patent IN 202521094370, all 10 claims granted by the Indian Patent Office) was developed to implement this dual-band approach:

- **Band 1 (460-490 nm):** Targeting the peak melanopsin activation zone within Pathway 1. This band encompasses the wavelengths with the highest melanopsin molar extinction coefficient, responsible for the strongest ipRGC-mediated thalamic input.
- **Band 2 (585-600 nm):** Targeting the amber wavelength range identified by Noseda et al. (2016) as exacerbating migraine pain through cone-driven Pathway 2, a mechanism entirely independent of melanopsin.
- **Preserved band (520-560 nm):** Maintaining high transmission in the narrow-band green range associated with reduced headache intensity in the Noseda (2016) data. This preservation is an active design constraint requiring that the two attenuation bands do not bleed into the intermediate green region.

The engineering challenge of dual-band filtration lies in simultaneously attenuating two non-contiguous spectral regions while maintaining a transmission window between them. This cannot be achieved with conventional single-dye tinting (which produces broad, unimodal absorption profiles) and requires precision optical engineering of the lens material or coating architecture.

7. Proposed Non-Pharmacological Management Framework

Clinical Scenario	Primary Intervention	Environmental	Rationale
Ictal photophobia (during attacks)	FL-41 or dual-band (NeuroCalm FLX+)	Reduced (not eliminated) ambient lighting; warm screen settings	Targets ipRGC and cone pathways without dark adaptation
Interictal photophobia (between attacks)	FL-41 or dual-band for daily indoor wear	Replace high-CCT lighting with 2700-3000K; brief outdoor exposure	Maintains light tolerance while reducing triggers
Evening circadian protection	Amber lenses (Circadian560)	Dim evening lighting; consistent sleep schedule	Protects melatonin; improved sleep reduces migraine frequency
Severe sunglass-dependent patients	Gradual transition from dark lenses to FL-41	Progressive increase in ambient light tolerance	Reverses dark adaptation cycle over weeks

Table 5. Proposed framework for non-pharmacological photophobia management. All optical interventions are comfort tools, not medical devices. Pharmacological migraine management should continue in consultation with neurologist.

8. Limitations and Research Gaps

- India-specific photophobia prevalence data are entirely absent; all estimates are extrapolated from global studies conducted primarily in Western populations. Population-based Indian studies are critically needed.
- No published Indian randomised controlled trials evaluating FL-41 or dual-band filtration in migraine photophobia. Such trials would need to account for India-specific factors including ambient lighting conditions, screen usage patterns, and genetic diversity.
- The cone-driven Pathway 2 (585-600 nm) is less completely characterised than the ipRGC pathway. The specific retinal circuits, thalamic targets, and pharmacology remain under investigation.
- The green light pain-reduction mechanism (~530 nm) is not fully understood. Whether the effect is mediated by M-cone inhibitory circuits, ipRGC subtypes with distinct spectral tuning, or thalamic mechanisms remains to be determined.
- Long-term clinical outcomes of wavelength-selective filtration in photophobia require prospective longitudinal studies. The existing evidence is limited to short-term assessments.

- Cost-effectiveness analyses comparing photobiological eyewear to pharmacological prophylaxis (e.g., topiramate, propranolol, CGRP monoclonal antibodies) in photophobia-predominant migraine are absent from the literature.
- The interaction between wavelength-selective filtration and pharmacological migraine treatments has not been systematically studied. Combination effects (additive, synergistic, or neutral) are unknown.

9. Conclusion

Migraine photophobia is a highly prevalent, mechanistically characterised, and functionally disabling condition affecting an estimated 170-190 million individuals in India alone. The neural basis involves at least two retinal pathways - melanopsin/ipRGC-mediated (460-520 nm) and cone-driven (585-600 nm) - converging at the posterior thalamus with trigeminal nociceptive signals to produce supra-additive pain amplification. A single wavelength band, green light (~530 nm), uniquely reduces headache intensity through a mechanism that remains under investigation.

Wavelength-selective filtration, particularly FL-41 (480-520 nm) and dual-band approaches targeting both identified pathways (460-490 nm + 585-600 nm) while preserving the beneficial green band (520-560 nm), represents an evidence-informed non-pharmacological management strategy. This approach specifically targets the identified neural mechanisms, avoids the dark adaptation paradox associated with conventional sunglasses, and is compatible with concurrent pharmacological management.

India-specific clinical trials, epidemiological studies, and cost-effectiveness analyses are urgently needed to validate these approaches in the population bearing one of the world's highest migraine burdens. The absence of Indian data in the photophobia management literature represents a significant gap that future research must address.

Author Affiliations and Conflict of Interest Disclosure

Suraj Dubey is the Founder and CEO of Sleepaxa Private Limited, the named inventor on Indian Patent IN 202521094370 (NeuroCalm FLX+, all 10 claims granted) and Patent Application IN 202521120977 (Circadian560). Monica Choudhary is Director of MCVI and Founding Member of Sleepaxa's Clinical Advisory Board (2% equity). Both authors have commercial interests in photobiological eyewear technologies discussed in this review. This disclosure is made in accordance with scientific transparency standards. This review presents published evidence objectively; no therapeutic or medical claims are made for any specific commercial product. Sleepaxa products are classified as comfort eyewear, not medical devices.

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