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PHOTOPHOBIA AS A RESULT OF OCULAR AND NEURONAL MECHANISMS: PATHOPHYSIOLOGY AND CLINICAL IMPLICATIONS

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ABSTRACT

Background: Photophobia is a common symptom across a wide range of neurological and ophthalmological disorders that might be defined as an abnormal aversion or sensitivity to light. Despite its prevalence, the clinical spectrum and underlying pathophysiological mechanisms remain incompletely understood.

Aim of the review: The present study was aimed at summarizing and comparing data from available literature regarding the current evidence on the conditions associated with photophobia and to clarify the mechanisms contributing to light sensitivity.

Methods: This narrative review was conducted through a structured literature search of the PubMed database covering publications from January 2000 to March 2025. Additional relevant articles were identified through manual screening of reference lists. Eligible articles were selected based on predefined criteria and synthesized narratively due to the heterogeneity of study designs.

Results: Photophobia was mostly associated with migraine, ocular surface disease, retinal disorders, and traumatic brain injury. Across studies, light sensitivity was linked to interactions between intrinsically photosensitive retinal ganglion cells, trigeminal nociceptive pathways, and thalamocortical processing networks. The reviewed literature also highlighted considerable variability in clinical presentation, reflecting the diverse mechanisms contributing to photophobia across different conditions.

Conclusions: Photophobia is a multifaceted symptom arising from complex interactions between retinal, trigeminal, and central neural pathways. A deeper understanding of its neurobiological mechanisms and broad clinical spectrum enables clinicians to make more accurate diagnoses and to develop targeted interventions for diverse patient populations.

Keywords: Photophobia, Light sensitivity, Migraine, Headache

INTRODUCTION

Photophobia is a frequent and clinically relevant symptom [2] that occurs in a variety of medical conditions, including primary ocular, central nervous, and mental health disorders [3]. The term is not straightforward to define [2]. Its etymology comes from the Greek photo (“light”) and phobia (“fear”), signifying a “fear of light” [3].

Light serves as the primary stimulus for photophobia, indicating the involvement of photoreceptors [4]. Although it is frequently observed in migraine, this symptom can also occur in tension-type headache and other neurological conditions, indicating a broader neurobiological basis [12].

Photophobia is increasingly recognized as a symptom involving not only ocular structures but also central nervous system pathways. Neuroimaging studies have shown that light exposure can activate pain-processing brain regions, particularly in patients with chronic ocular surface pain, supporting a central component of photophobia [21]. Although traditionally considered a secondary symptom, growing evidence indicates that photophobia is tightly linked to the neurobiological pathways that underlie migraine attacks [11].

This narrative review integrates current evidence from retinal physiology, trigeminal nociceptive pathways, and central sensory processing to provide a unified, clinically oriented overview of photophobia as a multidimensional symptom rather than a secondary complaint. By comparing photophobia across multiple conditions, this review highlights shared and distinct mechanisms that may inform differential diagnosis and targeted management strategies in clinical practice.

MECHANISMS

Ocular mechanisms

Humans have five primary types of photoreceptors: three types of cones, rods, and intrinsically photosensitive retinal ganglion cells (ipRGCs). Rods and cones, located in the outer retina, are the main photoreceptors responsible for image-forming vision due to their high temporal and spatial sensitivity. In contrast, ipRGCs, that contain the melanopsin photopigment (encoded by the OPN4 gene), play a key role in non-image-forming light responses [4].

Both melanopsin and cone luminance signals contribute to photophobic responses, with melanopsin-related pathways having a stronger influence on discomfort thresholds than cone-mediated signals [14]. Wang et al. (2022) suggest that the co-occurrence of headache and photophobia may result from the integration of light and nociceptive signals at different levels of the central nervous system. These include the visual cortex, hypothalamus, thalamus, midbrain, and retina [5].

Genetic or acquired dysfunctions of cones and rods can significantly alter light perception, leading to heightened light sensitivity in affected individuals. Achromatopsia arises from pathogenic variants in one of several cone-specific genes, most commonly in CNGA3 or CNGB3, which encode subunits of the cone cyclic nucleotide-gated channel. Mutations in these genes disrupt normal phototransduction, contributing to the functional loss of cone photoreceptors that underlie the clinical phenotype. Up to 90 % of individuals with achromatopsia carry mutations in CNGA3 or CNGB3, highlighting the pivotal role of these channels in cone function [13].

Neuronal mechanisms

Functional MRI studies have demonstrated that light exposure in individuals with chronic ocular surface pain elicits increased activation of brain regions involved in nociceptive processing, including the trigeminal brainstem, primary somatosensory cortex, anterior mid-cingulate cortex, and the insular cortex, compared with healthy controls. These findings highlight the involvement of trigeminal nociceptive pathways in linking peripheral ocular input with central pain processing mechanisms [21].

Photophobia is not solely mediated by retinal response. Specific wavelengths of light can also activate central neural mechanisms [11]. Communication between visual and pain pathways appears to involve neuropeptides such as pituitary adenylate cyclase-activating polypeptide (PACAP) and calcitonin gene-related peptide (CGRP). Additionally, cortical spreading depression, that plays a role of an important trigger of headache, may also contribute to photophobia, by amplifying nociceptive input reaching the thalamus [5].

In certain conditions, such as viral meningitis, photophobia reflects activation of meningeal sensory pathways [19]. Neuroimaging findings indicate that photophobia associated with chronic ocular surface pain is not solely a peripheral ocular phenomenon but involves altered central pain processing. This central component may explain limited responses to purely topical therapies [21].

Typical ocular anesthesia has been shown to modulate light-induced neural activity in selected cortical regions, such as the somatosensory and mid-cingulate cortices. However, changes in symptom severity are variable, suggesting that central sensitization may contribute to persistent photophobia in this patient population [21].

Pathophysiology in migraine

Interaction between trigeminothalamic pathways and ipRGCs appears to mediate the heightened light sensitivity observed in migraine patients [12]. Although the precise neurobiological substrates of photophobia remain under investigation, the integration of sensory inputs from cranial nociceptive and visual pathways is recognized as a key element in the exaggerated light sensitivity observed in migraineurs [15].

In the study by Zele et al. (2021), subjects with migraine showed larger post-illumination pupil responses, consistent with enhanced melanopsin-driven ipRGC activity during light exposure. These physiological differences between migraineurs and headache-free individuals underscore distinct sensory processing mechanisms underlying photophobia [14]. Additionally, altered light exposure, possibly affecting circadian regulation through ipRGCs, may help explain the association between photophobia and disrupted sleep patterns in migraineurs [18].

As it was demonstrated by Wilkins et al. (2021), ipRGCs have been implicated in signalling ambient light intensity, they do not fully account for discomfort produced by non-irradiance visual stimuli in migraine. The authors propose that cortical hyper-responsivity may offer a more unified explanation for migraine photophobia across a range of visual provocations, including flickering lights and spatial patterns [20].

According to Burstein, Noseda and Fulton (2019), neural circuits responsible for light aversion are distinct from those that amplify headache in migraine or those that participate in mediating the ocular discomfort. In migraine, nociceptive signals traveling along the trigeminovascular pathway may interact with visual pathways terminating in the visual cortex, thereby modifying visual perception. Conversely, photic inputs can influence headache perception by converging on trigeminovascular nociceptive pathways that project to cortical areas involved in pain processing [11].

Ocular discomfort induced by light likely results from indirect activation of intraocular trigeminal nociceptors, which subsequently stimulate second-order neurons situated in the spinal trigeminal nucleus. General aversion to light may involve modulation of neural circuits that control affective responses and autonomic functions [1]. Moreover, In Zele et al. (2021) study, patients exhibited lower photophobia thresholds compared to controls, suggesting hypersensitivity in light-detecting neural circuits [14].

Nevertheless, neuroimaging studies indicate that photophobia in migraine is associated with region-specific alterations in thalamic activity. Using ^18F-FDG PET, Suzuki et al. (2024) identified increased glucose metabolism within the bilateral medial thalamus in migraine patients with persistent photophobia compared with those without light sensitivity, indicating a selective involvement of thalamic sensory relay regions in photophobia [17].

Clinical observations indicate that treatment with calcitonin gene-related peptide (CGRP) monoclonal antibodies, like galcanezumab, can lead to a noticeable reduction in light sensitivity during migraine attacks. Furthermore, alleviation of photophobia often parallels decreases in headache frequency and analgesic use, suggesting that effective migraine management may concurrently improve associated sensory disturbances. Retrospective studies have shown that a substantial proportion of migraine sufferers experience improvement in photophobia shortly after initiating CGRP-targeted therapy, highlighting a potential link between these neuropeptides and sensory hypersensitivity [12].

CLINICAL SPECTRUM

Ocular causes

Photophobia is commonly linked to a wide range of medical conditions, including ocular disorders—ranging from anterior segment problems like dry eye disease, iritis, and blepharitis to patologies of posterior segment such as retinal dystrophies or retinitis pigmentosa [7].

Among eye-related causes, dry eye syndrome is the most frequent and can be challenging to diagnose, making it a priority to consider in patients without headache or other neurological symptoms [2].

Dry eye disease, termed clinically as keratoconjunctivitis sicca, is a complex ocular disorder characterized by inadequate tear film insuffiiency. A variety of contributors—including aging, infrequent or incomplete blinking, systemic diseases, hormonal changes, certain medications use [9], meibomian gland abnormalities, eyelid disorders, and environmental exposures can lead to its development [10].

The condition may develop as a consequence of reduced tear production or excessive tear evaporation. Both of which impair the stability of the tear film [10] and these disturbances can leave the cornea more exposed and vulnerable, thereby heightening sensitivity to light. Dry eye disease is frequently underdiagnosed in primary care settings, photophobia linked to ocular surface disease may be mistakenly attributed to neurological causes [9].

Specific tests can help confirm or exclude dry eyes as the cause of photophobia, including evaluation of the tear film, measurement of tear film break-up time, Schirmer's test and corneal staining with fluorescin or Rose-Bengal [2].

Achromatopsia is an inherited retinal disorder characterized by a congenital loss of cone photoreceptor function, leading to severely reduced visual acuity and difficulties with color perception. This condition, also known as rod monochromatism or total color blindness, typically manifests in early childhood and affects multiple aspects of daylight vision. A hallmark symptom of achromatopsia is abnormal intolerance to light, resulting in significant photophobia for affected individuals [13].

Neurological causes

Photophobia may arise in the context of neurological conditions. These include primary and secondary headaches, traumatic brain injury, progressive supranuclear palsy, blepharospasm, meningitis, subarachnoid hemorrhage, and thalamic lesions [3].

According to Wang et al. (2022) headaches can be categorized into three main diagnostic groups in which photophobia is reported:

- Primary headaches- including tension-type headache, migraine trigeminal autonomic cephalalgias and migraine.
- Secondary headaches- such as those resulting from traumatic brain injury, non-traumatic subarachnoid hemorrhage, meningitis and ocular disorders.
- Painful cranial neuropathies- including painful optic neuritis and trigeminal neuralgia [5].

Its high prevalence of photophobia across headache disorders underscores its role as an important clinical marker [2]. According to the International Classification of Headache Disorders (ICHD), photophobia is considered a key diagnostic criterion for migraine. Photophobia is reported by up to 80% of individuals with migraine, and it may occur both during attacks and in headache-free intervals. Additionally, exposure to light can precipitate migraine episodes in approximately 30–60% of patients. Various light sources—such as natural sunlight, fluorescent lightning and flickering visual stimuli from films or television, have been identified as common triggers [2].

Migraine is a complex, genetically influenced neurological disorder characterized by recurrent episodes of moderate to severe headache, which are often unilateral and accompanied by an array of sensory disturbances, including photophobia and phonophobia Migraine without aura represents the most prevalent subtype, accounting for approximately three-quarters of diagnosed cases, and is typified by headache attacks lasting 4 to 72 hours with associated nausea and light sensitivity.

Photophobia commonly co-occurs with nausea, phonophobia, and other autonomic symptoms during ictal periods [15] and patients often seek dark environments for relief [18].

The study by Zou et al. (2024) provides evidence that photophobia in vestibular migraine is strongly linked to increased interictal photosensitivity and a greater frequency of dizziness triggered by specific visual stimuli. These associations reinforce the concept that sensory integration abnormalities, particularly involving the visual domain, play a significant role in the pathophysiology of vestibular migraine and highlight the need for further research into tailored interventions for patients with heightened visual sensitivity [11].

As it was demonstrated by Suzuki et al. (2024), persistent photophobia in migraine is linked to increased metabolic activity in the medial thalamus, supporting the involvement of central sensory processing pathways in the maintenance of chronic light sensitivity [17].

Persistent post-traumatic headache is a long-term consequence of mild traumatic brain injury, frequently accompanied by heightened sensitivity to sensory stimuli such as light and touch. In Cortez et al. (2021) study, approximately 79% of post-traumatic headache participants displayed measurable sensory hypersensitivity, with more than half experiencing both photophobia and tactile allodynia. Moreover, lower sensory thresholds for both light and touch were associated with more frequent headache episodes [16].

Case reports have described photophobia as an initial manifestation of Adie’s tonic pupil, a condition characterized by parasympathetic denervation of the iris sphincter and abnormal pupillary responses. Patients may present unilateral photophobia associated with a dilated pupil and impaired constriction to light. Adie’s tonic pupil should be considered, particularly when light sensitivity is accompanied by pupillary abnormalities, such as poor light reaction with preserved or slow near response [22].

Photophobia is a recognized feature of viral meningitis and typically occurs alongside other meningeal signs, such as headache and nausea. Viral meningitis is defined as inflammation of the meninges caused by viruses and has become the most common form of meningitis due to successful reduction of bacterial cases through vaccination. The condition presents acutely with fever, headache, and neck stiffness, and may also involve sensory symptoms such as photophobia [19].

DIAGNOSTIC CONSIDERATIONS

Occurrence of this symptom requires a thorough patient history and appropriate neuro-ophthalmic and neurological examination in order to identify potential underlying causes. In cases of new-onset or sudden photophobia, clinicians should evaluate warning signs, including fever, neck rigidity, or localized neurological deficits. [2].

Considering the anatomical mechanisms involved in photophobia, along with evidence from functional neuroimaging of the occipital cortex, neurophysiological studies focusing on melanopsin-expressing retinal ganglion cells and neurochemical research examining peptide-mediated activity within the trigeminovascular system, it is possible to outline specific diagnostic criteria for isolated photophobia. These are referred to as the Santos-Ly-Porta diagnostic criteria:

- Symptom duration exceeds 3 months.
- Light sensitivity remains continuously present for more than 24 hours.
- The patient meets the diagnostic requirements for episodic migraine.
- Photophobia occurs in isolation, without any accompanying aura.
- The condition cannot be accounted for by another medical explanation, such as medication effects or psychiatric disorders [7].

The literal definition of photophobia as “fear of light” does not fully encompass the broader range of visual discomfort experienced by individuals with migraine. As it was demonstrated by Wilkins et al. (2021) emerging research suggests that simple light aversion may not be sufficient to explain migraine-related photophobia, as sufferers also show sensitivity to flicker, patterns, and colour contrasts that go beyond irradiance alone [20].

Patients often report light sensitivity not only during headache episodes but sometimes also in interictal periods, emphasizing the chronic nature of photophobia in some individuals. The intensity of photophobia can correlate with headache severity [11,13]. Recognition of photophobia as a distinct clinical feature is crucial for accurate diagnosis and effective management of headache disorders [11].

Ly-Yang et al. (2020) reported that the level at which patients begin to perceive light as uncomfortable varies widely among individuals. Those suffering from migraine or other headache disorders exhibit a lower tolerance, resulting in heightened light sensitivity. Various wavelengths can provoke discomfort, and multiple studies have shown that both short wavelengths, such as blue color and longer wavelengths, including red light, tend to be especially disturbing for migraine patients [7].

As it was demonstrated by Sharp et al. (2024) analysis of a large cohort from the American Registry for Migraine Research revealed that migraine patients reporting photophobia experienced notably poorer sleep quality compared with those without light sensitivity, independent of demographic and psychological factors. Greater severity of photophobia was linked to longer time to fall asleep, more frequent sleep disturbances, and overall reduced sleep quality, even after accounting for headache frequency, anxiety, and depression [18].

Photophobia can be associated with psychiatric disorders, including depression and anxiety, or may develop as an adverse effect of certain medications, such as barbiturates, benzodiazepines, and haloperidol [7].

CONCLUSIONS

Photophobia represents a complex and multidimensional symptom that extends far beyond its literal meaning as a “fear of light.” Current evidence demonstrates that it arises from dynamic interactions between retinal photoreceptors—particularly melanopsin-expressing ipRGCs—trigeminal nociceptive pathways, thalamic relay nuclei, cortical sensory networks, and neuropeptide-mediated signaling systems. Rather than being a purely ocular phenomenon, photophobia frequently reflects altered central sensory processing and, in many conditions, central sensitization.

n migraine, photophobia is not merely an accompanying symptom but an integral component of the disorder’s neurobiology. The bidirectional interaction between visual and trigeminovascular pathways, region-specific thalamic alterations, and cortical hyper-responsivity provide a coherent framework for understanding exaggerated light sensitivity in migraineurs. Clinical improvement of photophobia with CGRP-targeted therapies further supports its close relationship with core migraine mechanisms. At the same time, evidence from neuroimaging and neurophysiological studies indicates that melanopsin-driven pathways, cortical excitability, and sensory integration abnormalities all contribute to the variability of photophobic responses across patients.

DICLOSURE SECTION

Authors' contribution

Kamila Milewska – project development, data collection and management, data analysis and manuscript writing.

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