Photophobia

Emeritus Prof Peter Heilig University of Vienna **February 2023**

Unusual light sensitivity is rampant: a photophobia, irritating and distracting, road safety risk: "A potentially debilitating symptom."

Search engines disappoint. Among other things, they provide the "Photo-Oculodynia Syndrome" (ICD 11: “not found“), reminiscent of the stone louse Petrophaga Lorioti and inform interested 'users' about regional sympatholysis or botulinum toxin 'treatments' up to 'beta blockers, calcium channel Blockers, Anticonvulsants, and CGRP Inhibitors'. After all, "avoiding intense light" is also mentioned - an almost prophetic final sentence, providing a small correction is made:-

“Preventing intense light“.

Too bright bluish-white light triggers physiological protective processes, such as reflexive narrowing of the lid fissures and pupil contractions, particularly avoidance of overdosed, dazzling light, even in newborn, still blind mice without cone and rod function. Intrinsically photosensitive retinal ganglion cells (ipRGC) perform this warning, even in blind patients with intact ipRGC system. The evolutionarily developed *"Avoiding Intense Light"* is ignored or completely avoided - more and more - under the influence of short-wave-dominated monitors in smartphones, tablets and PCs - from childhood on.

Even small children are often 'sedated' with 'funny' moving images from smartphones and 'child-friendly' tablets, even while they are eating. Not entirely unexpectedly, ADHD is cited in this context. The high integral brightness of these monitors causes conditioning and epigenetic imprinting - à la longue. Retinal light exposure does not decrease during adolescence - on the contrary. An 'impairment of light sensitivity' (ICD 11) develops insidiously; children - with their crystal-clear media and adolescent force themselves to stare at brilliant bluish bright screens for unlimited periods of time - until old age.

The common ailment 'dry eye' is also involved, chronic headaches and subsequent dysphoria, comorbid with blunt brain trauma. Pathophysiological processes triggered by melanopsin intrinsically photosensitive retinal ganglion cells (ipRGC) reach the posterior thalamic nuclei via trigeminothalamic pathways. Concentric cortical depolarizations can induce release of neuropeptides, such as the inflammatory mediator calcitonin gene-related peptide (CGRP), with accompanying exubewrant vascular responses in the dura and meninges.

In all cases, the trigger is bright light. ipRGCs play the inducing or even dominating role in these processes. Blue- not yellow light can cause inflammation of the trigeminal ganglia, with secondary sympathetic and parasympathetic involvement as overshootiong reaction

to short-wave dominated light. CGRP activates protein kinases including transcription factors, ultimately inflammatory cascades (interleukins and cytokines). CGRP administration causes photophobia, in animal experiments and in human clinical studies: Photophobia is often accompanied by headaches, occasionally by corneal symptoms. CGRP levels of patients with migraine are significantly increased.

Trigeminal dysfunction that goes beyond the expected dry eye symptoms provokes pain sensations. Neuronal and glial depolarizations (spreading depolarization) are the pathophysiological substrates of migraine aura. Blunt traumatic brain injuries (TBI) in sports or traffic accidents can cause disability or even worse consequences. Post-traumatic headaches (PTH) can flare up over months or years. Diffuse axonal lesions (DAI), inflammation and disturbed healing processes up to the breakdown of the blood-brain barrier can cause chronic trigeminal hypersensitivity. The symptom of photophobia runs like a red thread through the dysfunction of neuronal networks.

Photophobia can be further aggravated by emotional reactions, and depressive moods can also accompany existing symptoms. The Dry Eye Outpatient Clinic has always paid special attention to emotional factors and did not dismiss dysphoria as meaningless or harmless.

An overwhelming body of evidence convicts the blue light as the main culprit. The logical conclusion from this must be: Blue light, which cannot make any significant contribution to central vision, has to be prevented – first and foremost in traffic scenarios.

CONCLUSION: "Preventing intense light": indoors, outdoors, in traffic and: dark background for smartphones, tablets and PC monitors.

Photophobia - ICD 11: "Impairment of light sensitivity" Achromatopsia, aniridia, Adie's P., erosio corneae, conjunctivitis, iritis etc. are not listed here in full..

'Photo-Oculodynia Syndrome' - ICD 11: *“not found“* - unrelated to reality. Therapy experiments of this kind have no authorization in Evidence Based Medicine.

Lit.:

Diel RJ et al (2021) Photophobia: shared pathophysiology underlying dry eye disease, migraine and traumatic brain injury leading to central neuroplasticity of the trigeminothalamic pathway. Br J Ophthalmol;105(6):751-760.

Burstein R et al (2019) The Neurobiology of photophobia J Neuroophthalmology 38,1, 94-102

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Belliveau MJ et al (2012) Relief of refractory photo-oculodynia with botulinum toxin. J Neuroophthalmol;32(3):293.

Ghanizadeh A. (2011) Sensory processing problems in children with ADHD, a systematic review. Psychiatry Investig; 8(2):89-94.

https://www.visioncenter.org/conditions/photophobia/

Gender: beyond.

Interest: no conflict