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Ultraviolet-Related Ocular Disease: Tissue-Specific Effects, Outdoor Exposure and Prevention - A Literature Review

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ABSTRACT

Background. Ultraviolet radiation (UVR) is a well-established cause of skin damage, photoaging and cancer but its ocular effects are less consistently addressed in prevention. The cornea, conjunctiva, limbus and crystalline lens are exposed to wavelength-dependent photochemical stress, particularly during outdoor activities and sports.

Aim. To review the pathophysiology, clinical features, epidemiology and prevention of UV-related ocular disease, with emphasis on wavelength-dependent exposure, peripheral light focusing and high-risk settings such as outdoor and sports environments. Key conditions include photokeratitis, pinguecula, pterygium, cortical cataract and age-related macular degeneration.

Material and methods. A literature review of scientific publications on UV-related ocular disease was conducted, focusing on major disorders, mechanisms of damage and ocular photoprotection.

Results. UVA (315–400 nm), UVB (280–315 nm) and UVC (100–280 nm) differ in atmospheric transmission and tissue absorption. UVB is mainly absorbed by the cornea and causes acute epithelial injury, while chronic exposure contributes to conjunctival degeneration, limbal remodeling and lens opacification. Peripheral light focusing

increases irradiance at the nasal limbus and lens cortex, consistent with the distribution of pinguecula, pterygium and cortical cataract. Outdoor sports increase cumulative UV exposure.

Conclusions. UV-related ocular disease represents tissue-specific responses to a shared environmental exposure. Prevention is essential in outdoor and sports settings and requires certified UV-filtering eyewear, proper frame design and regular use. Lens tint alone is insufficient.

Keywords: ultraviolet radiation; UVA; UVB; photokeratitis; pinguecula; pterygium; cataract; age-related macular degeneration; ocular photoprotection.

1. Introduction

Ultraviolet radiation is commonly discussed in relation to the skin. Public health communication routinely links UVR with sunburn, photoaging, actinic keratoses and melanoma, whereas its ocular effects are mentioned less often. This imbalance persists even though the eye is exposed to the same outdoor radiation field and develops both acute and chronic photic injury [1,2].

From a biologic perspective, UV-related ocular disease is best interpreted as a tissue-specific response to a shared environmental exposure. The cornea, limbus, bulbar conjunctiva, crystalline lens, and, more indirectly, posterior-segment tissues differ in wavelength absorption, repair capacity and cumulative susceptibility. Acute high-dose exposure tends to produce corneal epithelial injury and photokeratitis, whereas chronic repeated exposure contributes to pinguecula, pterygium and cortical cataract over years or decades; the link with age-related macular degeneration is less direct and remains more heterogeneous [3,4].

Environmental context modifies ocular dose. Altitude, reflection from snow, water, or sand, prolonged time outdoors, wind and surface dryness increase effective exposure. These conditions are frequent in outdoor work and in sports such as skiing, sailing, surfing, cycling and endurance running [3,5].

Most UV-related ocular lesions also show a reproducible anatomic distribution. This distribution reflects exposure geometry. Peripheral light entry, interpalpebral exposure and local surface conditions help explain why chronic actinic lesions tend to arise in the nasal conjunctival-limbal region and why cortical cataract often begins in the nasal lens cortex [1].

Research Objective. The objective of this review was to analyze how solar radiation contributes to different ocular disorders through tissue-specific mechanisms, exposure geometry and environmental conditions, with particular attention to anterior-segment disease, cortical cataract, age-related macular degeneration, high-exposure sports settings and the preventive role of protective eyewear.

2. Research materials and methods

A literature review was conducted based on available scientific publications concerning ultraviolet-related ocular disease, with particular focus on photokeratitis, pinguecula, pterygium, cataract, macular degeneration, ocular photoprotection and outdoor exposure. The analysis included review articles, systematic reviews, epidemiologic studies, mechanistic studies and selected clinically informative case series published between January 2018 and April 2026.

A comprehensive literature search was performed using database such as PubMed. The search strategy was based on combinations of predefined keywords and Medical Subject Headings (MeSH) terms, including: "*ultraviolet eye exposure*", "*UVA*", "*UVB*", "*UVC*", "*photokeratitis*", "*pinguecula*", "*pterygium*", "*cataract ultraviolet*", "*macular degeneration sunlight*", "*ocular photoprotection*", "*eye-sun protection factor*", "*sunglasses ultraviolet*" and "*peripheral light focusing eye*." Boolean operators (AND, OR) were used to refine the search and improve specificity.

Studies published in English and directly addressing ultraviolet-related ocular disease were included, particularly those concerning pathophysiology, histology, clinical presentation, epidemiology, diagnosis and prevention. The review considered review papers, systematic reviews, epidemiologic studies, mechanistic studies and selected clinically relevant original articles. Duplicate records, irrelevant publications, inaccessible full texts when more reliable equivalent sources were available and papers with insufficient clinical or mechanistic detail were excluded.

2.1. AI

Artificial intelligence tools were used only for editorial support during manuscript preparation. Their role was limited to linguistic refinement, structural polishing and assistance in reducing repetition. Study selection, interpretation of findings, synthesis of mechanisms and all final scientific conclusions remained under direct human control. AI was not used to generate data or references.

3. Research results

3.1. UV awareness: skin and eye

Awareness of UV damage is greater for the skin than for the eyes. This difference is visible in routine counselling: sunscreen use, pigmented lesion surveillance and melanoma prevention are widely discussed, whereas ocular photoprotection is often reduced to glare reduction or comfort. The consequence is not the absence of exposure but weaker recognition of ocular risk [2,4].

The comparison with skin is useful only because it shows the communication gap. Ophthalmic disease has its own anatomy and optics. The key question is how the same environmental UV source produces different ocular lesions according to wavelength, exposure geometry and tissue susceptibility [1].

3.2. Spectral bands and determinants of ocular exposure

Ambient UV index is not identical to ocular UV dose. The amount of radiation reaching the eye depends on latitude, season, time of day, cloud conditions, altitude, reflective surroundings, head posture, gaze direction, brow anatomy, eyelid position and geometry of protective eyewear. Ocular exposure may therefore differ substantially from the exposure estimated from a horizontal surface or from standard weather reports [3].

Wavelength is central to tissue targeting. UVC spans 100-280 nm and is almost completely filtered by the atmosphere under natural terrestrial conditions. UVB spans 280-315 nm and reaches the eye in smaller amounts but with high biologic activity; it is absorbed predominantly by the cornea and is the main driver of acute photokeratitis. UVA spans 315-400 nm, penetrates more deeply and contributes to cumulative injury of the ocular surface and lens [1].

Outdoor sport and outdoor work magnify these determinants. High altitude reduces atmospheric filtration. Snow, water and sand increase reflected exposure. Long repeated sessions outdoors enlarge cumulative dose. These conditions are common in skiing, sailing, surfing, cycling, endurance running, farming and construction work [5,21].

Diffuse radiation should also be considered. Ocular exposure does not disappear in shade or under light cloud cover, because scattered UV still reaches the eye from broad angular directions. Brow anatomy and eyelid position reduce some superior exposure, but they do not adequately block temporal side-entry or reflected light from below [3].

3.3. Peripheral light focusing and nasal predilection

Peripheral light focusing explains the characteristic nasal distribution of several UV-related ocular lesions. UVR incident from the periphery is refracted into the eye. Because of the focusing effect of the cornea, irradiance is on average about 22-fold higher at the nasal limbus and about 8-fold higher at the nasal lens cortex than would be expected from a uniform exposure model [1]. The nasal limbus is the typical site of pinguecula and pterygium and the nasal lens cortex is the typical site of cortical cataract.

This distribution has a clear anatomic and optical basis. Peripheral temporal entry, interpalpebral exposure, tear-film stress and limbal vulnerability act together. The result is a reproducible pattern in which the nasal conjunctival-limbal region and the nasal lens cortex receive a disproportionate cumulative actinic burden [1].

Dermatology also recognizes preferentially exposed facial subunits in which chronic UV damage accumulates faster than in more sheltered areas. In the eye, however, the main explanation is optical concentration of peripheral light at specific anatomic sites [1,2].

The limbal stem cell niche is particularly relevant in this context. The limbus maintains the boundary between corneal and conjunctival phenotypes. Repeated UV-related injury at the nasal limbus may alter epithelial renewal, stromal signalling and barrier function. This helps connect optical dose concentration with the later development of fibrovascular limbal disease [11].

3.4. Tissue-specific pathophysiology and histology

3.4.1. Cornea and photokeratitis

Photokeratitis is an acute UV-induced keratoconjunctivitis centred on the corneal epithelium. The lesion is photochemical. UVB exposure induces epithelial DNA damage, reactive oxygen species generation, cytokine release, apoptosis and loss of barrier integrity. Histologically, the main findings are superficial epithelial cell injury, punctate epithelial erosions, desquamation and a transient inflammatory response that exposes subepithelial nociceptors [10].

Clinically, photokeratitis is usually bilateral and delayed by several hours. Patients report pain, tearing, photophobia, foreign-body sensation, blepharospasm and transient visual blur after welding exposure, germicidal lamps, tanning devices, snow fields, or intense outdoor UV events. A 2024 case series describing 8 patients

exposed to a UV display during an outdoor event reported bilateral photokeratitis after a mean exposure of 3.00 hours, with symptoms appearing after a mean of 8.88 hours and typical findings including punctate epithelial erosions and conjunctival injection/ciliary vasodilation. [9]

Management is mainly supportive and recovery is usually rapid if additional exposure is avoided [9].

The differential diagnosis includes corneal abrasion, toxic keratopathy, infectious keratitis and severe dry-eye-related epithelial disease. The history of delayed bilateral symptoms after a defined UV exposure is often the most useful clue. In uncomplicated cases, epithelial recovery occurs over one to three days, which reflects the high regenerative capacity of the corneal surface [9].

3.4.2. Conjunctiva and pinguecula

Pinguecula is a localized actinic degeneration of the bulbar conjunctiva, usually nasal within the interpalpebral fissure. Histologically, it is characterized by subepithelial elastotic degeneration, fragmented collagen, fibroblast alteration and variable epithelial acanthosis or parakeratosis. It does not invade the cornea, but it reflects chronic UV-related surface remodelling [14].

Clinically, pinguecula may remain asymptomatic or may disturb the tear film, increase blink-related friction and become inflamed. Symptoms include dryness, foreign-body sensation, redness and intermittent burning. Studies of conjunctival ultraviolet autofluorescence support the view that visible pinguecula may represent only part of the total UV-related conjunctival change [15].

On slit-lamp examination, pinguecula appears as a yellow-white, slightly elevated lesion separated from the cornea by the limbus. This distinction is useful diagnostically because pterygium crosses the limbus onto the corneal surface. In symptomatic cases, the lesion behaves as a focal ocular surface disorder [14].

The histologic distinction also explains the difference in clinical behaviour. Pinguecula remains primarily a conjunctival lesion with localized degeneration and contour change. Pterygium is an active limbal-conjunctival process with corneal extension and more substantial extracellular-matrix remodelling [14].

3.4.3. Limbus, cornea and pterygium

Pterygium is a fibrovascular proliferation arising from the limbal-conjunctival interface and extending onto the cornea. It is associated with UV-driven oxidative stress, abnormal epithelial-stromal signalling, matrix metalloproteinase activity, elastotic degeneration, neovascularization and failure of the limbal barrier. Histologic descriptions include epithelial hyperplasia or squamous metaplasia, disrupted Bowman layer, activated fibrovascular stroma, inflammatory infiltration and abnormal extracellular-matrix turnover [11].

The epidemiologic association with sunlight is consistent across outdoor and equatorial populations. Risk increases with cumulative outdoor exposure and the lesion usually arises in the nasal interpalpebral zone, where peripheral light focusing and chronic surface exposure overlap. This association is supported by the SURE RISK study, in which 9735 adults from rural India were examined; pterygium in at least one eye was found in 13.2% of participants, bilateral pterygium in 6.7%, and increasing lifetime sun exposure remained independently associated with the disease [12]. Symptoms include redness, irritation, tear-film instability, induced astigmatism, higher-order aberrations and progressive corneal encroachment [12].

Management depends on activity and extent. Conservative treatment addresses irritation and ocular surface dysfunction. Surgery is reserved for progressive corneal extension, visually significant astigmatism, recurrent inflammation or contact lens intolerance. Its main limitation is postoperative recurrence, which remains an

important consideration in treatment planning. Conjunctival autograft and limbal-conjunctival techniques are preferred over older bare-sclera approaches because they restore the ocular surface barrier more effectively and reduce fibrovascular regrowth. This postoperative behaviour supports the view of pterygium as a disorder of wound healing [11].

3.4.4. Lens and cortical cataract

Cortical cataract represents a chronic degenerative consequence of cumulative photochemical injury to the crystalline lens. In this process, ultraviolet radiation and adjacent short-wavelength visible light promote oxidation of lens crystallins, sulfhydryl modification, disruption of lens epithelial cell homeostasis and progressive failure of antioxidant defence systems, ultimately leading to loss of protein solubility and impairment of lens transparency [17,18].

At the tissue level, these biochemical alterations translate into structural remodelling of the lens cortex. Characteristic changes include cortical spoke formation, hydration clefts and progressive disorganization of lens fibers, with early lesions typically developing in the inferior-nasal cortex. This topographic pattern is not random; rather, it is consistent with exposure geometry and with the peripheral light focusing model, according to which incident peripheral radiation is concentrated preferentially within the nasal lens region [17].

Epidemiologic data support the association between long-term solar exposure and cortical lens opacity more consistently than for several other cataract subtypes. The lens is particularly vulnerable to cumulative damage because of its minimal protein turnover and limited regenerative capacity. As a result, cortical cataract should be interpreted as a gradual exposure-related phenotype in which repeated oxidative and photochemical stress exceeds the capacity of lenticular repair and maintenance mechanisms over time [20].

Clinical expression usually begins with peripheral spoke-like opacities that may initially remain asymptomatic or cause only mild glare and reduced contrast sensitivity. With progression toward the visual axis, however, cortical changes become increasingly relevant functionally, contributing to disability glare, deteriorating visual quality and reduced performance under bright-light conditions [18,20].

3.4.5. Retina and age-related macular degeneration

The relationship between solar radiation and age-related macular degeneration (AMD) is less direct than in anterior-segment disease because most ultraviolet wavelengths are absorbed by the cornea and crystalline lens before reaching the retina. For this reason, the posterior-segment pathway is more appropriately discussed in terms of cumulative light exposure, particularly high-energy short-wavelength visible light, including blue light, rather than direct retinal ultraviolet injury alone [6,19].

From a mechanistic perspective, chronic exposure to blue light may contribute to oxidative stress within the retinal pigment epithelium and outer retina by increasing reactive oxygen species generation, promoting mitochondrial dysfunction and enhancing photochemical damage in tissues with high metabolic demand. This process is of particular interest in the macula, where lifelong light exposure, oxygen consumption and the presence of photosensitizing chromophores may favor cumulative cellular injury [19].

Blue light has also been implicated in lipofuscin-related phototoxicity, especially through excitation of bisretinoid compounds within retinal pigment epithelial cells. Such mechanisms may amplify oxidative damage, impair phagocytic and lysosomal function and promote degeneration of the retinal pigment epithelium-photoreceptor

complex. Nevertheless, AMD should not be classified as a UV-induced lesion in the same sense as photokeratitis, pinguecula, or pterygium. Instead, light exposure should be regarded as a potential modifying factor within a multifactorial disease model dominated by age, genetic susceptibility, smoking and cardiometabolic risk [6,19].

Epidemiologic evidence linking sunlight exposure with AMD remains heterogeneous, but it is not negligible. Recent epidemiologic evidence also supports a possible association between environmental light-related exposure and AMD; in a nationwide Chinese cohort including 19,707 participants without AMD at baseline, 3774 incident AMD cases occurred during follow-up, and higher UV radiation exposure was associated with increased incident AMD risk (HR 1.32, 95% CI 1.23–1.41) in the two-exposure model [19].

3.5. Epidemiology and high-exposure settings

The epidemiology of UV-related ocular disease follows exposure pattern and timescale. Photokeratitis clusters in discrete high-dose events. Pinguecula and pterygium increase with age and cumulative outdoor exposure, especially in populations living at low latitude or working in reflective environments [12]. Cortical cataract is linked to cumulative lifetime solar burden [20]. Evidence for age-related macular degeneration is less uniform, but available studies suggest a possible association with long-term sunlight exposure within a broader multifactorial risk model [6,19].

Outdoor work and outdoor sport create distinct high-exposure profiles rather than one uniform category of risk. Snow sports combine altitude with strong surface reflectance, water sports increase reflected and obliquely incident radiation, beach settings add sand reflection together with intense midday exposure, and endurance outdoor sports are characterized mainly by prolonged cumulative exposure [5]. This interpretation is supported by dosimetry data from outdoor competition: in a study of high school rowers in Aotearoa/New Zealand, the median personal UVR exposure during a single race was 1.15 SED over a mean race time of 46 minutes, and 69.6% of race-times exceeded the recommended daily threshold of 1 SED. These observations reinforce the view that ocular and periocular risk in sport depends not only on ambient sunlight, but also on exposure duration, reflective surroundings and the specific conditions of the activity performed [3,22].

Exposure history should therefore be specific. Clinically relevant questions include the setting of exposure, reflective surfaces, time spent outdoors, season, wind and dryness, regularity of eyewear use and whether the frame provides side coverage. These details are often more informative than a simple yes-or-no answer to the question of sunglasses use [3].

From a public health perspective these disorders differ in how easily they are recognized. Photokeratitis is memorable because it is acutely painful. Pinguecula, pterygium and cortical cataract accumulate more slowly and are often normalized as age or irritation. Age-related macular degeneration is even less likely to be linked by patients to lifelong light exposure because its relation to sunlight is indirect and multifactorial [6,19]. This difference in symptom tempo and causal clarity partly explains inconsistent preventive behaviour despite repeated outdoor exposure [3].

3.6. Eye protection and prevention

Effective ocular photoprotection has two components. The first is spectral filtering: the lens must adequately block ultraviolet radiation, ideally through certified UVA and UVB filtration up to 400 nm. The second is geometric protection: the frame and lens shape must reduce peripheral, oblique and reflected radiation that would otherwise bypass the lens and still reach the ocular surface [1].

Lens tint does not indicate UV protection. Dark lenses reduce visible brightness and may improve comfort, but darkness alone does not mean that the lens blocks UVA or UVB. Polarization is also not a UV label; it mainly reduces reflected visible glare from horizontal surfaces such as water, snow, or roads. A clear prescription lens may therefore protect the eye better than a dark non-certified sunglass lens [7].

E-SPF and PPF describe different aspects of protection. Eye-sun protection factor (E-SPF) combines UV transmission through the lens with UV reflected from the back surface toward the eye, so it expresses the intrinsic protective performance of the lens more realistically than transmission alone. This is useful because some products that appear similar in visible light may differ in UV protection [1].

Predictive protection factors (PPFs), by contrast, model how much protection a sunglass design provides under a defined exposure geometry, head position and environment. They therefore reflect real-world performance, not only lens chemistry. A product with strong intrinsic UV filtration may still perform poorly if the frame is small, flat, distant from the face or poorly protective against temporal side-entry and reflected light from below [7].

The 3D exposure modelling study by Backes et al. illustrates the practical importance of eyewear geometry. In this model, the uncovered cornea received the highest predicted daily UVR dose, whereas goggles provided almost complete protection across the evaluated ocular and periorbital regions. Medium-sized sunglasses performed less favourably at lateral and periorbital sites and although large-sized sunglasses were highly effective under some conditions, their protection remained dependent on diffuse UVR exposure. This point is especially relevant in snow, water, beach and high-altitude environments, where diffuse and reflected radiation contribute substantially to total ocular dose [7].

Preventive counselling should start early because ocular damage is cumulative. This recommendation is particularly relevant in children and adolescents, in whom the crystalline lens is substantially more transparent than in later life. Age-related yellowing of the lens progressively reduces transmission of shorter wavelengths, whereas younger eyes permit greater passage of ultraviolet and short-wavelength visible light into deeper ocular structures. Outdoor workers and athletes then accumulate repeated exposure over many years. Regular use of appropriately fitted UV-protective eyewear should begin before symptoms appear [1].

Practical recommendations should be adapted to setting. Snow and water environments require larger frames with close facial fit or goggles because reflected light reaches the eye from below and from oblique temporal angles. For road cycling and running, stable fit, lens coverage and tolerance during motion are as relevant as nominal UV filtering. For occupational use, compatibility with corrective lenses and helmet or face-shield systems also matters [5,21].

4. Conclusions and Future Perspectives

The reviewed evidence indicates that the ocular effects of solar radiation are determined by the interaction between spectral properties of light, tissue-specific susceptibility and exposure geometry, which together shape both lesion topography and clinical expression. This framework explains why the strongest and most consistent associations concern the anterior segment and the crystalline lens, whereas posterior-segment involvement, particularly in age-related macular degeneration, remains biologically plausible but methodologically more difficult to define. An important implication is that ocular photoprotection should be evaluated not only in terms of ultraviolet transmission through the lens, but also in relation to the capacity of eyewear to modify peripheral and reflected light exposure under real conditions of use. Future research should therefore focus on standardized ocular exposure

assessment, integration of environmental and optical determinants into tissue-specific risk models, and prospective studies linking quantified exposure profiles with defined clinical endpoints. Particular priority should be given to distinguishing ultraviolet-mediated effects from short-wavelength visible light pathways and to determining whether specific eyewear parameters, such as certified UV filtration, frame geometry and peripheral coverage, are associated with measurable reduction in long-term ocular morbidity.

Disclosure

The authors declare no conflict of interest regarding the publication of this paper.

Supplementary Materials

Not applicable.

Author Contributions

Aleksandra Borowska – conceptualization, methodology, formal analysis, writing – review and editing, supervision

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Conflicts of Interest

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