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Dry Eye Disease and Ocular Surface Disorders in Endurance Athletes - A Narrative Review of Current Evidence and Prevention Strategies

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Abstract

Background. Endurance athletes (long-distance runners, cyclists, triathletes, swimmers, rowers, cross-country skiers) are repeatedly exposed to wind, ultraviolet radiation, low humidity, chlorinated water, air pollution, and autonomic stress that may compromise tear film homeostasis. Despite growing clinical interest, dry eye disease in this population remains under-recognized.

Aim. To synthesize current evidence on the prevalence, pathophysiology, clinical presentation, diagnostic findings, and prevention of dry eye disease and ocular surface disorders in endurance athletes.

Material and methods. A structured narrative review was conducted using PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar (2016–2025), with priority given to systematic reviews, meta-analyses, and the TFOS DEWS II and III reports.

Results. Endurance athletes show a markedly higher prevalence of dry eye symptoms and signs than age-matched non-athletic controls, with the evaporative subtype predominating. Convergence of environmental, mechanical, autonomic, and inflammatory stressors produces cumulative ocular surface damage that is often clinically silent. Evidence supports pre-, intra-, and post-exercise prevention strategies, including protective eyewear, eyelid hygiene, preservative-free artificial tears, environmental modification, and targeted anti-inflammatory therapy.

Conclusions. Dry eye disease in endurance athletes is multifactorial, predominantly evaporative, and largely preventable. Sport-specific, evidence-based prevention should be integrated into routine sports medicine practice, and further prospective longitudinal research is needed.

Key words: dry eye disease, ocular surface, tear film, endurance athletes, prevention, meibomian gland dysfunction, ultraviolet radiation, swimming, running, cycling.

1. Introduction

Dry eye disease (DED) is one of the most common ocular conditions encountered in clinical ophthalmology, with a global prevalence estimated between 5% and 50% depending on the diagnostic criteria, age structure of the studied population, and geographic region [1, 2]. According to the second Tear Film and Ocular Surface Society Dry Eye WorkShop (TFOS DEWS II), DED is defined as a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles [3]. The disease is increasingly recognized as a disorder of the entire lacrimal functional unit - an integrated system comprising the lacrimal glands, meibomian glands, ocular surface epithelium, goblet cells, and the sensory and autonomic nerves that connect them [4]. This systemic framing is essential for understanding how environmental and physiological stressors converge on the ocular surface in conditions of repeated exposure, such as endurance exercise.

Over the last two decades, participation in endurance sports has expanded dramatically worldwide. Mass-participation events such as city marathons, half-marathons, ultramarathons, Ironman triathlons, long-distance road and mountain-bike races, open-water swimming competitions, and competitive rowing have attracted millions of amateur and professional athletes across all age groups. World Athletics, World Triathlon, and the Union Cycliste Internationale (UCI) have documented steady annual growth in both registered competitors and recreational finishers. This demographic shift has created a large, previously understudied population that is repeatedly and often chronically exposed to environmental and physiological stressors with a documented potential to compromise the ocular surface.

The ocular surface is particularly vulnerable during sustained aerobic exercise because endurance activity imposes several converging insults on the lacrimal functional unit. Taken together, the available evidence indicates that outdoor running and cycling accelerate evaporative loss of the aqueous tear layer through high-velocity airflow over the cornea; that

ultraviolet A and UVB radiation induce squamous metaplasia of the conjunctival epithelium, reduce goblet cell density, destabilize the tear film mucin layer, and trigger chronic low-grade inflammation, with recent population-level data linking ambient UV exposure independently to meibomian gland dysfunction and DED [5]; that low ambient humidity and cold-air exposure during winter endurance sports compound evaporative stress; that chlorinated pool water alters tear film pH and osmolarity, destabilizes meibomian gland secretions, and chemically irritates the corneal and conjunctival epithelium in swimmers, with open-water athletes additionally facing osmotic and microbiological stress from salt and variable water chemistry; and that sweat contaminating the marginal tear film during prolonged exertion transiently raises tear osmolarity through its content of sodium chloride, lactate, urea, and other osmotically active solutes [6]. The autonomic and systemic dimension is equally relevant: sympathetic nervous system dominance during sustained aerobic effort reduces parasympathetically mediated lacrimal secretion and alters meibomian expression, while systemic dehydration may further compromise aqueous tear availability. These mechanisms are not independent; rather, they interact multiplicatively over the course of repeated training sessions, which helps explain why acute, subclinical perturbations can progress toward clinically significant ocular surface disease across consecutive competitive seasons.

Despite this convergence of risk factors, dry eye disease and other ocular surface disorders remain underrecognized in endurance athletes. Athletes frequently under-report symptoms because they normalize them as part of training adaptation, attribute them to fatigue, or accept them as an unavoidable cost of performance. The few narrative and systematic reviews available to date confirm that ocular surface symptoms and signs are more prevalent in athletic populations than in age-matched non-athletic controls; but a comprehensive synthesis covering the major endurance disciplines, integrating recent meta-analytic evidence, and translating findings into sport-specific prevention strategies has not been available [7]. As a result, ocular surface disease in this population is often identified only at advanced stages, when it has begun to affect visual performance parameters critical to athletic success such as contrast sensitivity, glare tolerance, and dynamic visual acuity, and may compromise training quality, competitive safety, and recovery.

The review synthesizes current knowledge on the epidemiology of DED across the principal endurance disciplines in relation to the general population, describes the dominant pathophysiological mechanisms with attention to sport-specific risk profiles, reviews the clinical presentation and the spectrum of objective diagnostic findings reported in athletic

populations, and evaluates the evidence base for prevention and management strategies structured around the pre-exercise, intra-exercise, and post-exercise phases of training.

The aim of this narrative review is therefore to provide an integrated, clinically relevant synthesis of the current evidence on dry eye disease and ocular surface disorders in endurance athletes.

By consolidating evidence across running, cycling, triathlon, swimming, and rowing, this review is intended to serve clinicians (ophthalmologists, sports medicine physicians, optometrists, and team physicians) as well as coaches, athletic trainers, and the athletes themselves, with the ultimate goal of supporting earlier recognition of ocular surface disease in endurance athletes and promoting sport-specific, evidence-based prevention strategies that protect both ocular health and athletic performance.

2. Research materials and methods

This narrative review summarizes current evidence on dry eye disease and ocular surface disorders in endurance athletes, focusing on prevalence, underlying environmental, mechanical, autonomic, and inflammatory mechanisms, the spectrum of clinical presentation and objective diagnostic findings, and evidence-based prevention and intervention strategies. Relevant literature published in peer-reviewed journals was identified through a structured search of PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar (2016–2025), with priority given to sources published within the last five years. Priority was given to systematic reviews and meta-analyses, complemented by high-quality primary studies where higher-level evidence was unavailable, together with the TFOS DEWS II reports as the conceptual framework for definition, classification, and management. Selected publications were analyzed narratively to provide an up-to-date overview of the epidemiology, pathophysiology, clinical characteristics, and prevention of dry eye disease and ocular surface disorders in endurance athletes.

2.1. Participants

The review included studies enrolling adult amateur and professional endurance athletes (long-distance runners, marathon and ultramarathon runners, road and mountain-bike cyclists, triathletes, competitive swimmers, rowers, cross-country skiers). Pediatric populations were excluded except where data were directly relevant to adolescent athletes, as were studies focused on non-endurance sports, anterior segment trauma unrelated to surface disease,

conference abstracts without full data, and animal-only studies except where directly relevant to mechanism.

2.2. Procedure / Test protocol / Measure / Instruments

The TFOS DEWS II framework was adopted as the diagnostic and therapeutic backbone of this review. Inclusion criteria comprised original research articles, systematic reviews, meta-analyses, narrative reviews, and relevant case reports published in English or Polish between 2016 and 2025, dealing with ocular surface findings in adult endurance athletes or with controlled laboratory conditions simulating endurance-type ocular surface stress. Reference lists of key articles and recent systematic reviews were hand-searched for additional sources.

2.3. Data collection and analysis / Statistical analysis

2.3.1. Statistical Software

No quantitative meta-analysis was performed, consistent with the narrative-review design. No statistical software was used for pooled estimation.

2.3.2. AI

AI tools were used to support text analysis of the source literature and to assist in linguistic refinement of the manuscript. All AI-assisted outputs were verified and finalized by the human authors; AI tools served only as assistive instruments under expert clinical supervision.

2.3.3. Statistical Methods

No original statistical hypotheses were tested. The methodological quality of cited systematic reviews and meta-analyses was informally appraised against AMSTAR 2 criteria [23], and primary observational studies against the Newcastle-Ottawa Scale [24]. Heterogeneity in diagnostic thresholds and outcome measures was noted as a principal limitation.

3. Research results

3.1. Epidemiology of dry eye disease in endurance athletes

Available evidence consistently indicates that adult endurance athletes report ocular symptoms and meet diagnostic criteria for dry eye disease more frequently than age-matched non-athletic populations, although the precise magnitude of this excess varies across studies because of methodological heterogeneity in case definition, diagnostic thresholds, and sample selection [7]. Estimates of symptomatic DED in endurance athletes, derived primarily from Ocular Surface Disease Index (OSDI) and 5-item Dry Eye Questionnaire (DEQ-5) scores, cluster between 30% and 55%, compared with population-based estimates of 5–30% reported in the TFOS DEWS II Epidemiology Report, with the highest figures documented in symptomatic elite or competitive subgroups rather than in recreational athletes [1]. The TFOS DEWS III Digest [9] provides an updated synthesis of interdisciplinary research on DED published since the TFOS DEWS II reports, with particular emphasis on environmental, lifestyle, and population-level determinants relevant to endurance athletes. The TFOS Lifestyle Report on environmental conditions reinforces this picture by identifying outdoor exposure, low ambient humidity, and air pollution - all characteristic of endurance training environments - as independent population-level risk factors for DED [5]. A recent International Olympic Committee consensus paper specifically highlights ocular surface disease among the most common vision problems encountered in elite athletes across all sport disciplines [26].

Discipline-specific data suggest that the prevalence and severity of ocular surface involvement differ across endurance sports. Swimmers, particularly those training in chlorinated pools, consistently report the highest symptom burden and the most pronounced tear film instability, with available cross-sectional evidence documenting shortened tear break-up time, elevated tear osmolarity, and increased ocular surface staining in pool swimmers compared with non-swimming controls. Long-distance runners and ultramarathon runners show a similarly high prevalence of DED signs, attributed to the combined effects of high-velocity airflow, evaporative stress, and prolonged UV exposure. Competitive road cyclists, although studied less extensively, also show measurable ocular surface compromise, with tear film instability and lid wiper epitheliopathy documented after prolonged training sessions in dry or windy conditions. Triathletes occupy an intermediate position, exposed successively to the stressors of all three disciplines within a single event, which makes them a particularly informative population for studying cumulative ocular surface load. Open-water swimmers face the

additional osmotic and microbiological challenges of variable salinity and organic contamination.

The relationship between training exposure and ocular surface disease appears to be dose-dependent. Several primary studies report positive correlations between weekly training volume, years of competitive practice, or cumulative hours of outdoor exposure and the prevalence or severity of DED signs. Environmental modifiers further modulate this relationship: training in cold, dry, or high-altitude conditions accelerates evaporative loss, while training in polluted urban environments introduces particulate matter and ozone, both independently associated with ocular surface inflammation and DED [5, 12]. Sex differences mirror those observed in the general population, with female endurance athletes reporting higher DED symptom scores than male counterparts; however, the male athlete subgroup still shows prevalence rates well above those of age-matched male non-athletes, indicating that training-related factors elevate risk independently of sex hormones [7, 11]. Comparative work by Abokyi et al. [8] further demonstrates that trained athletes respond to maximal exercise with greater transient reductions in tear secretion than sedentary controls, providing a mechanistic substrate for cumulative ocular surface damage across repeated training sessions. The largest population-based cohort to date - 79,866 adults from the Dutch Lifelines study - confirmed female sex, age, and modifiable lifestyle factors as major determinants of DED prevalence in the general population, providing a quantitative reference for interpreting the elevated rates observed in endurance athletes [28].

Despite the convergence of these findings, important gaps remain. Few longitudinal studies have documented the natural history of DED in athletes across consecutive training seasons, and cross-sectional designs predominate in the available literature. Symptom under-reporting is likely substantial, because athletes normalize ocular discomfort as part of training adaptation and may decline to seek ophthalmologic evaluation until visual performance is demonstrably affected [7]. The few intervention studies available, such as the controlled trial of moderate aerobic exercise by Sun et al. [13], suggest that the relationship between exercise and the ocular surface is bidirectional and intensity-dependent, with low-to-moderate exertion transiently improving tear film parameters in symptomatic patients while high-intensity or prolonged effort in trained athletes produces the opposite effect. This duality complicates simple prevalence estimates and underscores the need for sport-specific, exposure-stratified epidemiologic studies using standardized TFOS DEWS II and III diagnostic criteria [9, 10]. Population-level meta-analyses estimate global DED prevalence at 11.59% based on Bayesian methods [27] and 34.6% in a more recent systematic review [31], with the largest population-based cohort to date

reporting a prevalence of 9.1% in 79,866 Dutch adults and confirming female sex, age, and lifestyle factors as major determinants [28, 29, 30]. Across all ages and regions, female sex has consistently emerged as one of the strongest non-modifiable risk factors for DED, as documented in the TFOS DEWS II Sex, Gender, and Hormones Report [11]. Demographic and lifestyle risk factors - including smoking, alcohol consumption, screen time, contact lens wear, and certain dietary patterns - have likewise been quantified in a cross-sectional study of dry eye subtypes [29], and a recent comprehensive review confirmed that these factors together explain a substantial proportion of population-level DED burden across the aqueous-deficient and evaporative subtypes [30].

3.2. Pathophysiology and risk factors

The pathophysiology of dry eye disease in endurance athletes is best understood within the evaporative subtype of the TFOS DEWS II classification, although aqueous-deficient components frequently coexist and may become clinically dominant in the later phases of prolonged events [4, 3]. In contrast to age-related or autoimmune DED, in which meibomian gland dysfunction or systemic inflammation predominates, athlete-associated ocular surface disease arises primarily from the convergence of environmental, mechanical, autonomic, and systemic stressors acting on an otherwise healthy ocular surface. These mechanisms are not independent but operate simultaneously during a single training session or competitive event.

Evaporative stress from high-velocity airflow. Outdoor running, cycling, and cross-country skiing expose the cornea to sustained airflow that accelerates thinning of the tear film lipid layer and increases aqueous evaporation. Recent population-level evidence indicates that ambient wind and outdoor exposure are independent risk factors for DED, with the TFOS Lifestyle Report on environmental conditions identifying low humidity and air movement as significant contributors to ocular surface decompensation [5]. This mechanism is amplified by incomplete blinking during high-concentration effort, which reduces the frequency of tear film reformation.

Ultraviolet radiation. UVA and UVB exposure induces squamous metaplasia of the conjunctival epithelium, reduces goblet cell density, destabilizes the tear film mucin layer, and triggers chronic low-grade ocular surface inflammation through the generation of reactive oxygen species and the activation of inflammatory cascades [4]. Recent epidemiological work has demonstrated that ambient UV levels are independently associated with meibomian gland dysfunction and dry eye disease at the population level, providing a direct mechanistic link

between chronic outdoor exposure and the evaporative phenotype observed in endurance athletes [5].

Chlorinated and natural water exposure. In swimmers, direct contact with chlorinated pool water alters tear film pH and osmolarity, destabilizes meibomian gland secretions through surfactant-like disruption of the lipid layer, and produces direct chemical irritation of the corneal and conjunctival epithelium. Open-water swimmers additionally face osmotic stress from variable salinity, organic contamination, and microbiological challenge, including Acanthamoeba and gram-negative bacteria, which can convert subclinical surface compromise into frank keratitis in contact lens wearers [14].

Sweat-induced osmotic stress. Sweat, which contains sodium chloride, lactate, urea, and other osmotically active solutes, frequently contaminates the marginal tear film during prolonged exertion, transiently raising tear osmolarity and lowering pH [6]. Although the osmotic effect of a single contamination episode is brief, repeated episodes across training sessions contribute to cumulative hyperosmolar stress on the ocular surface epithelium.

Low ambient humidity and cold-air exposure. Winter endurance sports (cross-country skiing, winter running, alpine skyrunning) compound evaporative loss by exposing the cornea to cold air with low absolute humidity, a combination that markedly increases tear film evaporation rate relative to temperate conditions [5].

Air pollution. Exposure to fine particulate matter (PM2.5), ozone, and nitrogen dioxide has been consistently associated with the prevalence and severity of DED through mechanisms including oxidative stress, conjunctival inflammatory cell infiltration, and goblet cell apoptosis [12]. For endurance athletes, who typically train outdoors for many hours per week, cumulative exposure to traffic-related air pollution in urban environments constitutes a chronic, low-grade ocular surface insult that is readily modifiable through training-schedule adjustment and route selection.

Autonomic nervous system dominance. Sustained aerobic effort is accompanied by progressive sympathetic activation and relative parasympathetic withdrawal. The autonomic nervous system exerts a well-documented influence on lacrimal secretion, and its dysregulation during prolonged exercise has been proposed to contribute to transient reductions in tear

secretion [4]. Comparative work in athletes and sedentary controls confirms that trained athletes show a significantly greater post-exercise reduction in tear secretion than non-athletes following maximal exertion, indicating that the ocular surface response to autonomic stress is potentiated by chronic training [8].

Systemic dehydration. Although the relationship between whole-body hydration status and tear osmolarity is debated, dehydration sufficient to reduce plasma volume has been shown to elevate tear film osmolarity and reduce aqueous tear availability, particularly when combined with the evaporative and osmotic stressors already active during exercise. Endurance events lasting several hours, in which fluid replacement is often incomplete, place athletes in a state of progressive dehydration that may exacerbate baseline ocular surface compromise.

Pharmacological and dietary cofactors. Several medications commonly used in athletic populations (isotretinoin for acne, antihistamines for allergic rhinitis, beta-blockers for performance anxiety or hypertension) are independently associated with reduced tear production or altered meibomian secretion [15]. Although the magnitude of their contribution in athletes has not been quantified, their additive effect on a pre-stressed ocular surface is plausible and clinically relevant [7].

Contact lens wear. Contact lens use is a well-established independent risk factor for DED in the general population, and it is particularly problematic in endurance athletes. Mechanical microtrauma from rapid eye movements, lens dehydration in windy conditions, and water exposure in swimmers create a compounded risk profile [14]. Contact lens-wearing endurance athletes therefore represent a clinically identifiable high-risk subgroup that warrants targeted prevention and surveillance.

Taken together, these mechanisms do not act in isolation but accumulate over repeated training sessions, and an acute perturbation clinically silent in a single exposure can evolve into measurable ocular surface disease across consecutive competitive seasons. Prevention and treatment strategies that address only one mechanism are therefore unlikely to produce sustained improvement; effective intervention requires a multifactorial approach matched to the dominant stressors in each athlete's training environment.

3.3. Clinical presentation and diagnostic findings

The clinical presentation of dry eye disease in endurance athletes typically combines classic dry eye symptoms, such as grittiness, burning, foreign body sensation, fluctuating vision, photophobia, and conjunctival redness, with sport-specific complaints that are rarely volunteered without direct questioning. These include blurred distance vision during the late phase of long runs, transient visual disturbance during high-speed cycling descents, foreign body sensation immediately after swimming that persists for several hours post-pool, and reactive tearing or irritation during winter training in cold or windy conditions [7]. Symptoms are often bilateral but may be asymmetric, reflecting dominant-side wind exposure in cyclists or unilateral splash patterns in swimmers. Importantly, available evidence indicates that endurance athletes tend to under-report symptoms because they normalize them as part of training adaptation, which means that objective clinical findings often exceed what would be predicted from symptom questionnaires alone [1].

The diagnostic approach to DED in endurance athletes should follow the standardized methodology recommended by the TFOS DEWS II report and reaffirmed in the recently published TFOS DEWS III Diagnostic Methodology Report [25], with the overarching synthesis provided by the TFOS DEWS III Executive Summary [10]. Symptom assessment is most commonly performed using the Ocular Surface Disease Index (OSDI) or the 5-item Dry Eye Questionnaire (DEQ-5), both of which are validated for DED screening and severity grading. Objective clinical signs should be interpreted using a tiered diagnostic battery, beginning with non-invasive or fluorescein-based tear break-up time (TBUT <10 seconds indicating instability), followed by corneal and conjunctival staining with fluorescein and lissamine green graded on the Oxford or van Bijsterveld scales, tear osmolarity (≥308 mOsm/L in either eye or an interocular difference >8 mOsm/L suggesting DED), and the Schirmer I test without anesthesia, which in the predominantly evaporative phenotype seen in athletes is frequently borderline rather than markedly reduced [16].

Meibomian gland evaluation is particularly relevant in this population. Meibography, whether by transillumination or non-contact infrared imaging, allows direct visualization of gland dropout, shortening, and distortion, while meibum expressibility scoring documents functional compromise of the lipid-secreting apparatus. Several studies in endurance athletes and recreational runners have demonstrated increased meibomian gland dropout scores compared with sedentary controls, with the effect most pronounced in swimmers and triathletes exposed to chronic chlorine or salt water [7].

Inflammatory biomarkers in tear fluid provide an additional dimension of objective assessment. Elevated tear levels of matrix metalloproteinase-9 (MMP-9), interleukin-1β, tumor necrosis factor-α, and interleukin-6 have been documented in symptomatic athletes and correlate with disease severity, supporting the concept that endurance-associated ocular surface disease is not merely evaporative but also carries an inflammatory component that may progress across training seasons if left unaddressed [4, 32, 33]. Point-of-care testing for MMP-9 is now clinically available and may be a useful adjunct in the assessment of athletes with borderline signs and disproportionately severe symptoms, with recent evidence suggesting that specific clinical phenotypes of DED can predict the presence of ocular surface inflammation as measured by InflammaDry [34]. A recent comprehensive review of tear and ocular surface disease biomarkers further documents the expanding panel of candidate molecules, including cytokines, chemokines, growth factors, and mucin-related markers, that may in the future complement established clinical tests for the diagnosis and monitoring of ocular surface inflammation in athletic and non-athletic populations [32]. An updated synthesis of biomarker research in ocular allergy and dry eye disease specifically highlights the clinical utility of matrix metalloproteinase-9 and other inflammatory mediators as objective indicators of disease activity and treatment response [33].

Contact lens-wearing endurance athletes constitute a clinically identifiable subgroup with several distinguishing features. They typically present with more pronounced ocular surface staining, more rapid tear film thinning, and more frequent reports of fluctuating vision during prolonged exercise than their non-lens-wearing counterparts, reflecting the combined effects of mechanical microtrauma, lens dehydration in windy environments, and reduced corneal oxygen availability [14]. Diagnostic thresholds developed for non-lens wearers may therefore underestimate disease severity in this subgroup, and clinical interpretation should be adjusted accordingly.

Taken together, the available evidence supports a structured diagnostic approach in endurance athletes that combines validated symptom questionnaires, a minimum objective battery (TBUT, staining, osmolarity, Schirmer I), meibography when available, and selective use of inflammatory biomarkers, with thresholds and weighting adjusted for the dominant sport-specific stressors identified in the history.

3.4. Prevention and management strategies

Available evidence supports a tiered, sport-specific prevention model organized around the pre-exercise, intra-exercise, and post-exercise phases of training. Such a model aligns with the

general management framework proposed in the TFOS DEWS II report and reaffirmed in the recently published TFOS DEWS III update, but adapts it to the specific exposure profile of endurance athletes [20, 21].

Pre-exercise measures. Routine screening of the ocular surface should be incorporated into the pre-participation medical evaluation of endurance athletes, using validated symptom questionnaires (OSDI or DEQ-5) combined with a minimum objective battery comprising non-invasive or fluorescein-based tear break-up time, ocular surface staining, and when available tear osmolarity [16]. Identification and adequate management of pre-existing DED before the competitive season reduces the cumulative load imposed by training and may prevent progression to clinically significant disease. Patient education should address the ocular impact of endurance training, the importance of protective eyewear, and the recognition of early symptoms; this is particularly relevant for novice athletes, who are least likely to attribute symptoms to ocular surface disease. Dietary supplementation with omega-3 polyunsaturated fatty acids has documented benefit in DED, particularly in the evaporative subtype; a Cochrane systematic review [17] and a more recent randomized clinical trial of re-esterified triglyceride omega-3 [18] both confirmed improvements in subjective symptoms, and a comprehensive synthesis concluded that the TFOS DEWS II Management and Therapy Report [20] and the more recent TFOS DEWS III Management and Therapy Report [21] together provide an evidence-based framework for integration of nutritional and pharmacological interventions in at-risk athletic populations. For endurance athletes, whose diet and lipid profile are often optimized for cardiovascular and metabolic performance, omega-3 supplementation may provide additional ocular benefit beyond its established cardiovascular effects.

Intra-exercise measures. The use of wraparound protective sports eyewear with UV-A/UV-B filtration and side shields is the single most evidence-supported intervention for athletes training outdoors. Wraparound designs reduce airflow over the cornea, block particulate matter and allergen exposure, and filter ultraviolet radiation, addressing three of the principal mechanisms of endurance-associated DED simultaneously [5]. Photochromic or tinted lenses are useful for cyclists and runners exposed to variable light conditions. For swimmers, well-fitted goggles that minimize water exchange, combined with regular rinsing and disinfection, reduce direct chemical exposure of the ocular surface; the additional benefit of preventing contact lens-related microbial keratitis is well documented [14]. Strategic fluid and electrolyte intake during prolonged events mitigates systemic dehydration, which independently elevates tear film osmolarity and reduces aqueous tear availability. Topical decongestants or

vasoconstrictors applied immediately before exertion should be avoided, as they may transiently reduce tear film stability and mask early symptoms of evaporative stress.

Post-exercise measures. Eyelid hygiene and warm compress therapy maintain meibomian gland patency and address the evaporative component of DED directly. A recent evidence-based review confirmed that warm compress therapy improves tear film parameters, meibomian gland function, and dry eye symptoms, with outcomes critically dependent on compress type, temperature, and duration [19]. For endurance athletes, a brief post-training routine combining eyelid warming, gentle massage, and lid margin cleansing is feasible and sustainable. The use of preservative-free artificial tears, ideally with lipid-based formulations in the predominantly evaporative phenotype, accelerates recovery of tear film homeostasis after exposure to environmental stressors. Lid margin cleaning after outdoor sessions removes sweat, dust, and allergen residue that would otherwise contribute to chronic low-grade inflammation. For athletes with established DED who wish to continue training, targeted anti-inflammatory therapy with topical cyclosporine A or lifitegrast may be considered; the TFOS DEWS II and III Management and Therapy Reports summarize the efficacy and safety of these agents in DED, including in patients without Sjögren syndrome, and support their use as long-term anti-inflammatory strategies [20, 21].

Environmental and behavioral modifications. Endurance athletes should be educated to monitor ambient humidity and air quality, particularly PM2.5 and ozone concentrations, and to adjust training schedules to avoid peak pollution hours, since exposure to these pollutants is independently associated with the prevalence and severity of DED [12]. Indoor alternatives, such as treadmill running or stationary cycling, may be substituted on days of high pollution or extreme cold. Training routes can be planned to minimize traffic-related exposure, and pacing strategies can incorporate brief ocular rest intervals during prolonged sessions. These adjustments are low-cost, scalable, and supported by population-level evidence linking environmental exposure to ocular surface disease. The American Academy of Ophthalmology Preferred Practice Pattern for dry eye syndrome provides an additional evidence-based reference for the overall management approach [22].

Taken together, the available evidence supports a structured, multifactorial prevention strategy that integrates pre-exercise screening, sport-specific protective measures during exercise, and a sustained post-exercise maintenance routine, with environmental modification and patient education as cross-cutting elements. The relative weight of each component should be individualized to the dominant stressors in each athlete's training environment, and clinical

reassessment at regular intervals is recommended to detect progression and to adjust the prevention plan accordingly.

4.Discussion

The principal finding of this narrative review is that endurance athletes constitute a population at meaningfully elevated risk of dry eye disease and ocular surface disorders, with the evaporative subtype predominating and environmental and mechanical factors dominating the etiologic profile. This conclusion is supported by consistent evidence from cross-sectional studies and by population-level data linking environmental stressors to DED independent of athletic status [5, 12]. The International Olympic Committee has recently endorsed a similar conclusion in its consensus statement on sports-related ophthalmology, which identifies ocular surface disease as one of the most prevalent vision problems in elite athletes [26].

When the available evidence is examined discipline by discipline, a coherent pattern emerges. Swimmers and triathletes consistently show the highest symptom burden and the most pronounced tear film instability, attributable to the combined effects of chlorinated water, osmotic stress, and prolonged tear film disruption during the swim segment. Long-distance runners and cyclists rank next, with evaporative stress, UV exposure, and autonomic factors as dominant mechanisms. Rowers and cross-country skiers have been studied less extensively, but the available data suggest that cold-air exposure and low ambient humidity produce a clinical picture resembling that observed in outdoor runners. Across all disciplines, the relationship between aerobic exercise and ocular surface status appears to be bidirectional and intensity-dependent: moderate, submaximal exercise may transiently improve tear film parameters in symptomatic individuals [13], whereas high-intensity or prolonged exertion in trained athletes produces transient depressive effects that, when repeated, accumulate into chronic surface damage [8]. This duality has not always been explicitly addressed in the source literature, and it complicates the interpretation of cross-sectional prevalence estimates.

Several features of the current evidence base warrant consideration when interpreting the findings summarized above. Most available studies are cross-sectional, with relatively few longitudinal datasets tracking athletes across complete training seasons, and only a subset has applied the full TFOS DEWS II or III diagnostic battery. Diagnostic methodology is heterogeneous, environmental exposure metrics (training volume, intensity, ambient conditions) are reported inconsistently, and sample sizes in many primary studies are modest, with control groups not always age- and sex-matched. The methodological quality of cited systematic reviews, appraised against AMSTAR 2 criteria, was generally moderate [23], while

the quality of cited observational studies, appraised against the Newcastle-Ottawa Scale, was variable [24]. At the population level, recent meta-analyses converge on a global DED prevalence between approximately 11% and 35%, depending on diagnostic criteria and symptomatic versus clinical-sign definition, with female sex, age, and modifiable lifestyle factors emerging as the most consistent determinants across regions [27, 28, 29, 30, 31]. Importantly, these considerations define clear priorities for the next generation of studies rather than undermining the central clinical message of this review; the convergence of evidence across multiple disciplines, multiple study designs, and multiple research groups provides a coherent basis for the practical recommendations advanced below, even as individual studies remain open to refinement.

Despite these limitations, several clinically actionable conclusions emerge. First, the convergence of environmental stressors (wind, UV, low humidity, chlorine, air pollution) with physiological stressors (sympathetic dominance, dehydration, inflammatory rebound) creates a cumulative risk profile that justifies proactive screening rather than reactive treatment. Second, sport-specific protective strategies, including wraparound eyewear for cyclists and runners, properly fitted goggles for swimmers, lipid-based preservative-free artificial tears for the evaporative phenotype, and structured eyelid hygiene after training, have a credible mechanistic foundation and, where studied, supportive clinical evidence [19, 20, 21]. Third, athlete education and behavioral modification remain underused but high-yield interventions, particularly when integrated into existing sports medicine and coaching workflows.

Future research should focus on prospective longitudinal studies using standardized TFOS DEWS II and III diagnostic criteria [25, 10], sport-specific risk stratification models that incorporate environmental exposure metrics, randomized trials of preventive interventions (eyewear design, omega-3 supplementation, artificial tear formulations) in athletic populations, development of sport-adapted screening tools with validated cut-offs for early disease detection, and quantitative evaluation of the impact of ocular surface disease on visual performance and competitive outcomes, an area that remains underexplored despite its obvious relevance to athletic success.

5. Conclusions

Dry eye disease and ocular surface disorders are highly prevalent and clinically meaningful conditions in endurance athletes, with the evaporative subtype predominating and environmental factors (wind, ultraviolet radiation, low humidity, chlorinated water, and air

pollution) playing a central etiologic role alongside physiological contributors (sympathetic activation, dehydration, and inflammatory rebound).

The pathogenesis is multifactorial and cumulative, with repeated training sessions producing subclinical surface perturbation that may progress to clinically significant disease across consecutive competitive seasons.

The evidence supports a structured, sport-specific prevention strategy that combines pre-exercise screening, environmental control, protective eyewear, eyelid hygiene, and preservative-free artificial tears, with targeted anti-inflammatory therapy reserved for established disease.

Integration of ocular surface assessment into routine pre-participation and in-season sports medicine evaluations is recommended to enable earlier recognition and intervention.

Further prospective, longitudinal, and interventional research is needed to refine screening thresholds, validate preventive protocols, and quantify the performance impact of untreated ocular surface disease in endurance athletes.

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Supplementary Materials

Not applicable.

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