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Allergic Conjunctivitis during Outdoor Exercise: Current Management Strategies and Implications for Athletic Performance

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Abstract

Background: Allergic conjunctivitis (AC) in athletes is an inflammatory disorder of the ocular surface and a primary driver of exercise-related performance impairment, orchestrated by underlying IgE-mediated hypersensitivity and synergistic environmental triggers.

Aim of the study: This narrative review aims to synthesize current literature on the immunopathology and modern therapeutic landscape of allergic conjunctivitis (AC), with a specific focus on optimizing clinical management for athletes and physically active individuals.

Materials and Methods: A comprehensive assessment of peer-reviewed literature published between 2020 and 2026 was conducted using databases such as PubMed and Cochrane Library, prioritizing clinical trials, systematic reviews, and updated management guidelines.

Results: Modern management has transitioned toward topical dual-action agents as first-line therapy, supplemented by preservative-free formulations to protect ocular surface homeostasis.

Conclusions: Effective management of AC is essential for maintaining the quality of life and athletic performance of physically active individuals. Integrating evidence-based pharmacological strategies with non-pharmacological interventions, while avoiding the sedative effects of systemic treatments, is critical for optimizing ocular health in sporting contexts.

Keywords: allergic conjunctivitis, aeroallergens, air pollution, soft steroids, immunotherapy

1. Introduction

Allergic conjunctivitis (AC) in athletes is defined as a prevalent, allergen-induced inflammatory disease of the ocular surface, primarily characterized by a Type I IgE-mediated hypersensitivity response to normally harmless environmental triggers. These conditions typically manifest following exposure to a susceptible environment, specifically aeroallergens such as tree and grass pollens in seasonal allergic conjunctivitis (SAC) or indoor triggers like house dust mites and animal dander in perennial allergic conjunctivitis (PAC), which are frequently exacerbated by the synergistic effects of ambient air pollutants including particulate matter (PM2.5), ozone, and nitrogen dioxide (NO2)[1,2]. A central diagnostic dilemma in sports medicine is the accurate differentiation between AC and comorbid ocular surface disorders—specifically dry eye disease (DED)—as hallmark symptoms such as ocular pruritus, hyperemia, and tearing create significant clinical overlap and potential misdiagnosis between the two states [6]. While the global prevalence of ocular allergy continues to rise, affecting up to 40% of the North American population and nearly 50% of individuals in Europe, the condition remains critically underdiagnosed and undertreated, with as few as 10% of symptomatic patients seeking professional medical intervention [4, 23]. Consequently, the clinical significance of rapid recognition and performance-safe management of AC features—including intense itching, photophobia, and blurred vision—is paramount, as these symptoms can directly impair visual acuity, concentration, and safety during high-intensity outdoor training and competition.

The primary objective of this narrative review is to summarize and evaluate current evidence regarding the etiology, pathophysiology, and management strategies of AC in athletes, while highlighting essential protocols for preserving ocular health and optimizing athletic performance in challenging environmental conditions.

2. Materials and Methods

This study is a narrative literature review concerning the pathophysiology, environmental exacerbators, and management strategies of allergic conjunctivitis (AC) in athletes and physically active individuals. Publications were analyzed to provide a comprehensive synthesis of contemporary original studies, systematic reviews, and international scientific statements addressing ocular surface health, environmental risk factors during outdoor exertion, and performance-safe pharmacological interventions.

The literature search was conducted across the PubMed, Scopus, Web of Science, and ScienceDirect databases. Keywords and their combinations included: “Allergic Conjunctivitis”, “Outdoor Exercise”, “Athletes”, “Air Pollution”, “Aeroallergens”, “Topical Antihistamines”, and “Sports Performance”. In addition, the reference lists of publications considered foundational to the topic, such as international clinical guidelines, were manually examined to identify further relevant research.

The review included English-language publications (and other languages where translation was feasible) directly related to the onset or exacerbation of AC during physical activity or exposure to outdoor environmental triggers. Inclusion criteria encompassed original clinical studies, systematic and narrative reviews, and contemporary consensus statements from major allergology and ophthalmology societies, such as ARIA, EAACI, and the Japanese Society of Ocular Allergology. Publications not directly related to ocular allergy, studies conducted outside of relevant active or young populations, and conference abstracts lacking complete data were excluded.

The analysis was qualitative in nature and consisted of a critical synthesis of data regarding the immunological phases of ocular allergy, the synergistic impact of ambient pollutants and pollen on the ocular surface, and the clinical utility of modern "soft" steroids and dual-action agents in the context of athletic participation. Due to the marked heterogeneity in exposure metrics, outcome definitions (ranging from clinical diagnoses to biomarker expression), and study designs across the literature, no quantitative meta-analysis was performed.

3. Results

3.1. Pathophysiology of Allergic Conjunctivitis

Pathophysiologically, AC is primarily a Type I IgE-mediated hypersensitivity reaction. The process initiates with a sensitization phase, where Th2 cells stimulate B cells to produce allergen-specific IgE antibodies that bind to mast cells in the conjunctival mucosa. Upon re-exposure, allergen cross-linking triggers mast cell degranulation, releasing preformed mediators like histamine and tryptase, which drive the acute early-phase response. This is followed by a late-phase reaction characterized by the recruitment of eosinophils, neutrophils, and lymphocytes, mediated by a complex cascade of cytokines such as IL-4, IL-5, IL-13, and thymic stromal lymphopoietin (TSLP) [3]. These immunological mechanisms result in chronic tissue inflammation and, in severe cases, epithelial barrier disruption and corneal damage [20]. Current clinical management faces substantial challenges, including a notable diagnostic gap; many patients are treated for ocular symptoms without formal allergy testing to identify specific triggers. Furthermore, the common use of over-the-counter (OTC) medications containing benzalkonium chloride (BAK) as a preservative poses a risk of ocular surface toxicity and can exacerbate comorbid dry eye disease (DED), which frequently overlaps with AC [1, 6]. There remains a significant unmet need for integrated, evidence-based management strategies that prioritize both symptom relief and the preservation of ocular surface homeostasis, particularly in populations with high environmental exposure.

3.2. Early-Phase Response, Late-Phase Response, and Cytokine-Mediated Inflammation

The immunopathogenesis of allergic conjunctivitis (AC) is primarily characterized as a Type I IgE-mediated hypersensitivity reaction. This immunological cascade is conventionally divided into three overlapping stages: sensitization, the early-phase response, and the late-phase response. While mild forms of AC, such as seasonal and perennial allergic conjunctivitis (SAC and PAC), are largely driven by IgE-mediated mechanisms, more chronic and severe forms like vernal keratoconjunctivitis (VKC) and atopic keratoconjunctivitis (AKC) involve complex interactions between IgE- and T-cell-mediated pathways [17, 20, 24].

3.2.1. Early and Late- Phase Response

The early-phase response initiates within seconds to minutes of allergen re-exposure on the ocular surface and typically peaks within 20 to 30 minutes. This phase is triggered when environmental allergens—such as tree or grass pollen—cross-link antigen-specific immunoglobulin E (IgE) antibodies already bound to high-affinity receptors (FcεRI) on the surface of sensitized mast cells. This cross-linking signals mast cell degranulation, leading to the rapid release of preformed mediators, most significantly histamine and tryptase, alongside newly synthesized lipid mediators including leukotrienes and prostaglandins [2].

The clinical significance of this phase is rooted in the physiological actions of these mediators: histamine stimulates conjunctival nerve endings to cause intense pruritus (itching), the hallmark of ocular allergy, and induces vasodilation and increased vascular permeability. These mechanisms manifest clinically as conjunctival hyperemia (redness), chemosis (swelling), tearing, and eyelid edema [17, 20].

As the acute symptoms of the early phase begin to subside, the late-phase response emerges, typically occurring between 4 and 12 hours after the initial allergen contact. This phase is defined by the massive recruitment and infiltration of inflammatory cells—including eosinophils, neutrophils, basophils, macrophages, and Th2 lymphocytes—into the conjunctival mucosa. This cellular migration is directed by a gradient of chemokines, such as eotaxin, and the upregulation of adhesion molecules like VCAM-1 on the vascular endothelium.

The pathophysiology of the late phase is responsible for the persistence of symptoms and is a primary driver of chronic tissue damage. In chronic ocular allergic diseases, the repeated cycles of cellular infiltration and mediator release lead to significant tissue remodeling, epithelial barrier disruption, and sight-threatening corneal complications, such as shield ulcers or scarring [2, 20, 21].

3.2.2. Cytokine-Mediated Inflammation

Cytokine-mediated inflammation provides the underlying framework for the transition from acute symptoms to chronic disease. Cytokines derived from Th2 and Th9 lymphocytes play a central and versatile role in orchestrating this inflammatory cascade [9, 25].

Essential for the sensitization phase, IL-4 induces B-cell class switching to produce allergen-specific IgE. Pathophysiologically, it stimulates conjunctival fibroblasts to proliferate and produce excess collagen, which manifests clinically as papillae on the palpebral conjunctiva [22].

The principal regulator of eosinophil maturation, differentiation, and chemotaxis to the eye. Once activated at the ocular surface, eosinophils release toxic proteins (e.g., eosinophil peroxidase) that directly damage the corneal and conjunctival epithelium. Closely related to IL-4, IL-13 is a major driver of goblet cell hyperplasia and mucin hypersecretion, contributing to the ropy, mucoid discharge characteristic of severe AC. Interleukin-31 (IL-31) and TSLP: IL-31, known as the "itch cytokine," mediates chronic pruritus by binding to receptors on sensory neurons [12]. Thymic stromal lymphopoietin (TSLP) functions as an "alarmin," amplifying the Th2 inflammatory response and activating dendritic cells to perpetuate the cycle of allergic inflammation. Together, these phases and their molecular mediators establish AC as a complex, self-perpetuating inflammatory disorder where the early-phase triggers acute distress and the late-phase/cytokine cascades drive chronic morbidity [9, 25].

3.3. Environmental Factors Affecting Athletes with Allergic Conjunctivitis

3.3.1. Air Pollution and Ocular Allergy

Ambient air pollution is recognized as a central contributor to the clinical burden of allergic eye diseases. Multiple pollutants—including particulate matter (PM2.5 and PM10), nitrogen oxides (NOx, NO2), sulfur dioxide (SO2), carbon monoxide (CO), and ozone (O3)—have been consistently associated with increased AC incidence and symptom severity across diverse geographical contexts. These pollutants exacerbate ocular allergy through several synergistic immunological and physical mechanisms. Pollutants, particularly fine particulate matter (PM2.5), disrupt the conjunctival and corneal epithelial barrier [20]. This breach facilitates the easier penetration of allergens into the subepithelial space, where they interact with sensitized mast cells and dendritic cells to initiate the allergic cascade. Pollutants can increase the retention time of aeroallergens on the ocular surface, leading to amplified immune activation and sustained inflammatory responses. Environmental toxins often act as adjuvants that potentiate allergic responses and they can modify pollen allergenicity by enhancing protein release and modifying epitopes. [11] At the cellular level, PM exposure activates pro-inflammatory signaling cascades, such as the nuclear factor kappa B (NF-κB) and thymic stromal lymphopoietin (TSLP) pathways, which are critical mediators of the Th2-driven inflammatory response. Particulate matter exposure induces oxidative stress, DNA damage, and mitochondrial dysfunction in ocular surface cells. This toxicological impact can trigger conjunctival inflammation even when pollen counts are low or outside of typical allergen seasons. Clinically, traffic-related pollutants like NO2 and PM10 have been specifically linked to more severe phenotypes, such as vernal keratoconjunctivitis (VKC) and atopic keratoconjunctivitis (AKC), which pose a significant risk to the visual health and long-term performance of athletes.

3.3.2. Aeroallergens and Outdoor Exercise

Outdoor exercise necessitates prolonged exposure to various aeroallergens, which are the primary triggers for seasonal allergic conjunctivitis (SAC). SAC, which accounts for approximately 90% of all ocular allergy cases, is typically induced by tree, grass, and weed pollens. For athletes, the burden of these allergens often peaks during prime competitive spring and summer seasons. Symptoms such as intense pruritus (itching), photophobia, and blurred vision not only cause discomfort but can directly impair athletic concentration, safety, and overall performance. Field and occupational studies provide clear evidence of strong exposure-response relationships; for instance, high-level exposure to orchard or hazelnut pollens has been shown to induce acute ocular surface disease and measurable systemic inflammation [11].

3.3.3. Climatic and Meteorological Influences

Meteorological conditions modulate the concentration and dispersal of both pollutants and allergens, thereby influencing an athlete's risk profile. Temperature and wind speed are among the strongest predictors of AC outpatient visits. High ambient temperatures often intensify the pro-inflammatory effects of pollutants, with risks peaking during warm seasons. Wind modulates the concentration of airborne particles and allergens, often exacerbating symptoms during outdoor training.

Furthermore, solar radiation and humidity have been linked to exacerbations of severe forms like VKC, particularly in semi-arid and arid regions. On a broader scale, climate change is increasingly recognized as a driver of ocular allergy, as it reshapes aerobiology through longer pollen seasons, increased allergenicity of specific species, and altered geographical distribution of allergens, further complicating the environmental management of athletes with sensitive eyes [11, 19].

3.4 Modern Pharmacological Management

3.4.1. Topical Antihistamines and Mast Cell Stabilizers

The pharmacological management of seasonal and perennial allergic conjunctivitis (SAC and PAC) primarily centers on the utilize of topical dual-action agents, which are now established as the first-line therapy. These multimodal medications combine selective H1-receptor antagonism with mast cell stabilization, providing a significant clinical advantage over single-acting formulations. By blocking the early-phase histamine-induced response, they offer rapid relief from hallmark symptoms such as pruritus (itching) and hyperemia (redness), while their stabilizing effect inhibits the late-phase release of inflammatory mediators like leukotrienes, prostaglandins, and cytokines [9, 17, 25].

Olopatadine hydrochloride remains a widely utilized dual-action agent, available in concentrations of 0.1%, 0.2%, and 0.77%. Clinical trials indicate that the highest concentration (0.77%) provides superior efficacy and a 24-hour duration of action, making it highly effective for managing ocular symptoms with a once-daily dosing regimen. This once-daily dosing is particularly beneficial for athletes and physically active individuals, as it ensures sustained symptom control throughout long training sessions or competitive events while minimizing the "washout" effect caused by heavy tearing or physical exertion. While ketotifen fumarate (0.025%) also demonstrates high efficacy and safety, patient preference often favors olopatadine due to a superior comfort profile upon instillation, as ketotifen has been associated with a higher incidence of transient stinging [13].

Technological advancements have introduced alcaftadine (0.25%), which represents a modern class of multi-action agents. Unlike traditional dual-action drugs, alcaftadine exerts inverse agonistic activity at H1, H2, and H4 receptors. The inhibition of the H4 receptor is a critical technical development, as it directly targets Th2-lymphocyte-driven allergic responses and significantly inhibits eosinophil recruitment. [15] Furthermore, alcaftadine provides a unique protective effect on the ocular epithelial barrier by preventing the degradation of tight junction proteins (such as zonula occludens-1 and E-cadherin) that typically occurs during the allergic inflammatory cascade. Recent meta-analytical data confirm that alcaftadine is remarkably effective in providing sustained relief of ocular symptoms over a 14-day period, showing statistical superiority to olopatadine in the reduction of ocular itching and conjunctival hyperemia [14, 17].

The modern therapeutic landscape also includes bepotastine besilate (1.5%), which is distinguished by its high H1-receptor affinity and rapid onset of action—often within 15 minutes of instillation. Bepotastine has been shown to provide lasting relief of pruritus for up to 8 hours following an allergen challenge and possesses additional anti-inflammatory properties, such as the inhibition of IL-5-induced eosinophil migration [15] .

A major recent innovation is the approval of topical bilastine (0.6%), a preservative-free solution co-formulated with hyaluronic acid (HA). This formulation is unique for its ability to provide once-daily symptom control while simultaneously hydrating the ocular surface and promoting epithelial wound healing. The inclusion of HA increases the ocular bioavailability of the active drug through its viscosity-enhancing properties. Given that the common preservative benzalkonium chloride (BAK)—found in approximately 70% of over-the-counter ophthalmic drops—is known to cause corneal epithelial cell damage, disrupt the tear film, and trigger pro-inflammatory pathways (such as NF-κB), the transition toward preservative-free multi-dose formulations is essential for maintaining ocular surface homeostasis [1].

3.4.2. Alcaftadine in sports

For active patients, such as athletes and physically active individuals, alcaftadine (0.25%) offers several specific clinical and technical advantages that support both symptom management and the maintenance of athletic performance. Alcaftadine takes effect within a few minutes of instillation, which is essential for athletes who need immediate relief before training or competition.One of its primary advantages for active individuals is its long duration of action (up to 24 hours), which allows for once-daily administration. This reduces the need for frequent dosing during long events or training sessions and minimizes the risk of a "washout" effect from heavy tearing or physical exertion. Meta-analytical data indicate that alcaftadine is remarkably effective in providing sustained relief, showing statistical superiority to olopatadine specifically in the reduction of ocular pruritus (itching) and conjunctival hyperemia [8, 13].

Unlike many other dual-action agents, alcaftadine exerts inverse agonistic activity at H1, H2, and H4 receptors. The inhibition of the H4 receptor is particularly beneficial for active patients as it directly targets Th2-lymphocyte-driven allergic responses. Moreover, it significantly inhibits eosinophil recruitment to the ocular surface, thereby reducing the intensity of the late-phase inflammatory response. Athletes are frequently exposed to environmental stressors like wind, dust, and high pollen counts that can compromise the eyes. Alcaftadine provides a unique protective effect on the ocular epithelial barrier by preventing the degradation of tight junction proteins (such as zonula occludens-1 and E-cadherin) that occurs during allergic inflammation. [14] This helps maintain ocular surface homeostasis and prevents the deeper penetration of allergens into the subepithelial space. Due to the fact that it is a topical agent with minimal systemic absorption, alcaftadine avoids the central nervous system side effects associated with first-generation oral antihistamines, such as sedation, drowsiness, and impaired coordination.

This ensures that the athlete's concentration and safety remain uncompromised. In clinical observations, the use of topical dual-acting agents like alcaftadine has been associated with an increase in comfort for contact lens wear. This is vital for active patients who rely on contact lenses for visual acuity during sports [10]. In summary, alcaftadine's combination of rapid action, once-daily convenience, H4-receptor antagonism, and epithelial barrier protection makes it a highly effective therapeutic choice for maintaining ocular health and performance in active populations [13].

3.4.3. Preservative-Free Lubricants

Ophthalmic lubricants, or artificial tears, serve as essential adjunctive therapy by diluting and flushing allergens and inflammatory mediators from the tear film. However, approximately 70% of over-the-counter eye drops contain benzalkonium chloride (BAK), a detergent-like preservative that disrupts the tear film and induces corneal epithelial cell damage through pro-inflammatory pathways. In physically active populations and patients with sensitive eyes, the use of preservative-free (PF) lubricants is strongly recommended to maintain ocular surface homeostasis and avoid cumulative toxicity. Modern PF formulations often incorporate viscosity-enhancing agents such as hyaluronic acid (HA), which provides superior hydration, promotes epithelial wound healing, and increases the bioavailability of co-administered active drugs [1, 2, 16].

3.4.4. Soft Corticosteroids

For patients with moderate-to-severe symptoms or acute exacerbations refractory to first-line dual-action agents, topical corticosteroids provide potent anti-inflammatory relief by inhibiting cytokine production and mast cell proliferation. To minimize the classic risks of steroid therapy—specifically elevated intraocular pressure (IOP) and cataract formation—clinicians prioritize "soft" or ester-based steroids. Loteprednol etabonate (0.2% and 0.5%) is the preferred agent as it is rapidly inactivated in the anterior chamber, resulting in significantly lower rates of IOP elevation compared to traditional ketone-based steroids like prednisolone. Fluorometholone (FML) is another common alternative used for short-term control of moderate inflammation. Regardless of the formulation, steroid use should be limited to short bursts (one to two weeks) and must remain under the strict supervision of an eye care specialist to monitor for complications [2, 9].

3.4.5. Biologic Therapies

In severe or refractory clinical phenotypes, such as vernal keratoconjunctivitis (VKC) or atopic keratoconjunctivitis (AKC), biologic therapies are emerging as vital off-label treatment options. Omalizumab, a humanized monoclonal antibody that binds to unbound IgE, has demonstrated significant efficacy in reducing both nasal and ocular symptoms in patients with poorly controlled allergic rhinoconjunctivitis [26]. Another promising biologic is dupilumab, an IL-4 receptor alpha (IL-4Rα) antagonist that inhibits both IL-4 and IL-13 signaling [3]. While primarily approved for atopic dermatitis, dupilumab is being explored for its potential to modulate the chronic T-cell-mediated pathways driving severe ocular allergy. It is important to note, however, that conjunctivitis has been reported as a paradoxical side effect in a subset of patients receiving dupilumab for skin conditions, requiring careful clinical monitoring.

The long-term use of potent ketone-based topical steroids (such as prednisolone acetate, prednisolone phosphate, and dexamethasone) for managing severe ocular allergies carries a significant risk of serious ocular adverse effects. Because of these risks, they are generally reserved for severe cases and require close, regular supervision by an eye care specialist [13].

The most critical concern with prolonged ketone-based steroid therapy is a significant rise in intraocular pressure, which can lead to steroid-induced glaucoma. Research indicates that after 4–6 weeks of topical steroid treatment, approximately 5% of the population may experience a high rise in IOP (>16 mmHg), while 30% may see a moderate rise (6–15 mmHg). Unlike "soft" ester-based steroids (like loteprednol), which are rapidly inactivated in the eye, ketone-based steroids persist longer in ocular tissues, leading to a much higher likelihood of pressure-related complications.

Long-term or repeated treatment with these potent steroids is a known cause of cataracts, specifically posterior subcapsular cataracts. This risk is significant enough that clinicians are advised to perform baseline and periodic pupillary dilation to screen for early signs of lens opacity during treatment.

Topical steroids suppress the local immune response, which can lead to an increased risk of developing secondary bacterial, viral, or fungal infections. Moreover, use of these steroids can worsen pre-existing corneal viral infections, such as herpes simplex keratitis [18].

Steroids can interfere with protein synthesis and the inflammatory repair process, leading to delayed corneal wound healing. In severe cases of chronic inflammation (like AKC), this can increase the risk of persistent epithelial defects or even corneal thinning and perforation.

Additionaly, though sometimes less common, complications include a condition affecting the retina that has been linked to prolonged steroid therapy. Potential for ptosis (drooping eyelid), mydriasis (dilated pupil), and thinning of the eyelid skin. To mitigate these long-term risks, modern clinical management prioritizes the use of "soft" ester-based steroids (like loteprednol etabonate 0.2% or 0.5%) for moderate symptoms, as they are metabolized more efficiently and have a significantly lower correlation with cataract development and IOP elevation [1].

3.5. Allergen-Specific Immunotherapy

3.5.1. Subcutaneous Immunotherapy (SCIT)

Subcutaneous immunotherapy (SCIT) is a well-established method, utilized since 1911, involving the regular injection of standardized allergenic extracts. To achieve sustained clinical remission, SCIT typically requires a long-term commitment of 3 to 5 years. The treatment protocol involves a gradual dose buildup phase over several months, followed by maintenance injections administered at 2- to 4-week intervals. Standardized extracts available for treatment include cat dander, grass pollen, house dust mites, and short ragweed. Clinical evidence strongly supports its efficacy, with systematic reviews indicating significant improvement in conjunctival symptoms for allergies triggered by grass mix, timothy grass, cat, *Parietaria*, and *Alternaria*. Due to a small but critical risk of severe systemic reactions, including anaphylaxis, SCIT must always be administered under strict medical supervision, and patients are required to remain in the clinic for observation following each dose [5, 7].

3.5.2. Sublingual Immunotherapy (SLIT)

Sublingual immunotherapy (SLIT) is a newer alternative where allergens are delivered via dissolvable tablets or extract solutions placed under the tongue. In North America and Europe, FDA/EMA-approved tablets are available for allergies induced by grass pollen, ragweed, and house dust mites. Meta-analytical data demonstrate that SLIT is significantly effective in reducing total ocular symptom scores, including redness, itching, and tearing, particularly in patients with pollen-induced AC. A primary technical advantage of SLIT is its safety profile; it is much less likely than SCIT to cause severe systemic reactions, allowing maintenance doses to be self-administered at home. However, local adverse effects are common, including pruritus of the mouth and ears, mouth edema, and throat irritation [5].

3.5.3. Long-Term Benefits for Athletes

For athletes and physically active individuals, AIT offers unique clinical advantages that extend beyond symptom control. By inducing long-term remission, AIT significantly reduces the overall medication burden, allowing athletes to avoid the use of systemic treatments. This is particularly vital for avoiding first-generation oral antihistamines, which are contraindicated in sports due to their performance-impairing sedative effects and potential to cause ocular surface dryness through anticholinergic action [8]. Furthermore, AIT can prevent the progression of allergic rhinoconjunctivitis to asthma, a critical factor for maintaining respiratory health and aerobic capacity during high-intensity exertion [26]. From a logistical perspective, SLIT may be the preferred immunotherapy route for competitive athletes, as the ability to self-administer treatment at home or during travel is more compatible with demanding training and competition schedules than the frequent clinical visits required for SCIT [7].

4. Discussion

The evidence synthesized in this review confirms that allergic conjunctivitis (AC) is a burgeoning global health challenge, with prevalence reaching up to 40% in North America and 50% in parts of Europe. Pathophysiologically, it is defined as a Type I IgE-mediated hypersensitivity reaction driven by a Th2-cytokine cascade (notably IL-4, IL-5, and IL-13), which orchestrates both the rapid early-phase mast cell degranulation and the persistent late-phase cellular infiltration. Diagnosis remains primarily clinical, relying on the hallmark symptom of ocular pruritus, though definitive identification of triggers via skin prick testing or serum specific IgE is essential for targeted management. Treatment has evolved into a sophisticated stepwise approach, escalating from non-pharmacological barriers and lubricants to topical dual-action agents, and reservedly, to corticosteroids and immunomodulators for refractory phenotypes.

Recent pharmacological progress has significantly refined the clinical management of AC. Topical dual-action agents (e.g., olopatadine, alcaftadine, and ketotifen) remain the established first-line therapy because they provide a multimodal mechanism: immediate H1-receptor antagonism to address acute itching and mast cell stabilization to prevent the release of late-phase pro-inflammatory mediators [13, 16, 20].

A critical technical development is the transition toward preservative-free (PF) formulations. Traditional eye drops containing benzalkonium chloride (BAK) are known to induce corneal epithelial cell damage and disrupt tear film stability, which can exacerbate symptoms and lead to comorbid dry eye disease. The approval of innovative PF solutions, such as bilastine 0.6% co-formulated with hyaluronic acid (HA), represents a dual-benefit approach: providing rapid antihistaminic relief while simultaneously hydrating the ocular surface and promoting epithelial wound healing [16].

Furthermore, the clinical role of "soft" ester-based corticosteroids like loteprednol etabonate is paramount. These agents are designed to be rapidly inactivated in the anterior chamber, offering the potent anti-inflammatory benefits of traditional steroids with a significantly reduced risk of elevated intraocular pressure (IOP) and cataract formation. For long-term disease modification, allergen-specific immunotherapy (AIT) remains the only curative option, effectively downregulating the Th2 response and inducing sustained clinical remission [13, 20].

For the physically active, the clinical manifestations of AC—specifically intense pruritus, photophobia, and blurred vision—are not merely nuisances but direct impediments to athletic performance and safety. During outdoor exertion, athletes experience prolonged exposure to aeroallergens, which can impair concentration and reaction times during high-stakes competition [16].

The choice of pharmacological management in this population must prioritize the avoidance of sedative effects. Unlike first-generation oral antihistamines that cross the blood-brain barrier and impair psychomotor coordination, topical dual-action agents target ocular tissues directly with minimal systemic absorption [8]. Additionally, the once-daily dosing potential of agents like alcaftadine 0.25% or olopatadine 0.77% is highly advantageous for athletes, as it minimizes the risk of treatment "washout" during heavy perspiration or tearing and simplifies adherence during demanding training schedules. Individualized treatment plans that account for seasonal variations and local pollen monitoring are essential to ensure that prophylactic care is initiated prior to peak exposure periods [13].

Environmental factors act as powerful exacerbators of ocular allergy, particularly for outdoor athletes. There is a documented synergy between ambient air pollutants (e.g., PM2.5, ozone, and NO2) and aeroallergens. Pollutants act as adjuvants that not only increase the allergenicity of pollen but also disrupt the conjunctival epithelial barrier, allowing allergens to penetrate deeper into the subepithelial space and trigger more severe inflammatory responses. Furthermore, climate change is reshaping the aerobiological landscape, leading to longer pollen seasons and the geographical spread of highly allergenic species, which disproportionately affects athletes who spend significant time training in outdoor environments [27].

The findings of this review align closely with the ARIA-EAACI guidelines and the 2024 American Academy of Ophthalmology Preferred Practice Pattern, which advocate for a stepwise approach beginning with dual-action topical agents. There is universal agreement on the need to minimize BAK exposure and the requirement for specialist supervision when using corticosteroids. However, controversies remain regarding the optimal timing for initiating AIT in pediatric populations and the routine off-label use of biologic therapies like omalizumab or dupilumab for severe, refractory AC outside of clinical trials.

The future of AC management lies in precision medicine. The identification of specific tear biomarkers (e.g., eotaxin-1, periostin, and TSLP) may soon allow for biomarker-guided therapy, where treatment is tailored to the patient's specific inflammatory endotype. Emerging digital health technologies and AI-driven pollen forecasting could provide athletes with real-time risk assessments to modulate their training intensity. Furthermore, biosensing technologies using two-dimensional nanomaterials offer the potential for rapid, clinic-based diagnostic testing of ocular biomarkers.

A significant research gap exists regarding athletes and physically active populations. Most current evidence is extrapolated from general population studies or murine models. There is a critical lack of large-scale randomized controlled trials (RCTs) investigating how high-intensity exercise modulates ocular surface inflammation or evaluating the performance-related safety of newer biologic agents in competitive sports environments [11].

5. Conclusions

Effective, evidence-based management of allergic conjunctivitis is fundamental to maintaining ocular health, quality of life, and optimal athletic performance. By utilizing modern topical dual-action agents—preferably in preservative-free and once-daily formulations—clinicians can provide athletes with rapid symptom relief without the risk of performance-impairing sedation. As environmental stressors continue to exacerbate the ocular allergic burden, integrating personalized precision medicine and environmental monitoring into clinical practice will be essential for the modern athlete.

Recent advances in precision medicine suggest that biologic therapies targeting type-2 inflammation, together with biomarker-based monitoring and digital health technologies, may significantly improve individualized management of allergic conjunctivitis. Further research is required to evaluate their effectiveness in athletic populations exposed to high environmental allergen burdens.

Disclosure

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