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Seasonal Allergy and Seasonal Decrements in Athletic Performance

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Allergic diseases are among the most common chronic diseases and have been increasing worldwide over the past several decades for reasons that are still not clearly understood [1–6]. There has been considerable research elucidating the impact that allergic disease has on athletic performance. Athletes who have allergic disease can benefit from the tremendous progress that has been made in understanding the pathophysiologic basis of their disease. Accessing the host of international climatic and seasonal pollen reports available can enable athletes to be better prepared for training and performance. Additionally, athletes can benefit from an evolving repertoire of therapeutic modalities for allergic diseases that conform to current antidoping codes (www.wada-ama.org).

Pathophysiology of allergic disease

Atopic diseases such as asthma, allergic rhinitis, urticaria, and anaphylaxis are characterized by hypersensitivity to a particular allergen, resulting in secretion of specific immunoglobulin E (IgE) antibodies and acute, recurrent, or chronic inflammation. Certain individuals with an atopic predisposition synthesize IgE

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antibodies on initial exposure to allergen. IgE binding to mast cells and basophils sets the stage for the allergic response. On re-exposure, allergen cross-links IgE on cell surfaces, which causes the release of a host of inflammatory mediators. Early response mediators include granule mediators (eg, histamine, tryptase) and lipid mediators (eg, leukotrienes, prostaglandins). Cytokines such as tumor necrosis factor-alpha (TNF- α), interleukins, and chemokines (IL-8, MCP-1 and MIP-1 α) are produced minutes to hours later (Fig. 1) [7,8]. The type of allergen, the degree and length of exposure, and the atopic tendency of the individual determine the manifestation of symptoms.

Sources of allergens include the environment (eg, tree, grass and weed pollen, dust, mold), foods, drugs, and stinging insects. Aeroallergens are further subdivided into seasonal aeroallergens, like tree, grass and weed pollen, and nonseasonal aeroallergens like mold and dust [9].

Pollen counts

The concentration of pollen in the atmosphere, which correlates with allergic manifestations, is reported and disseminated at multiple centers internationally. In the United States and Canada, a useful resource is The National Allergy Bureau, which provides pollen and mold counts from approximately 75 counting stations (www.aaaai.org/nab/).

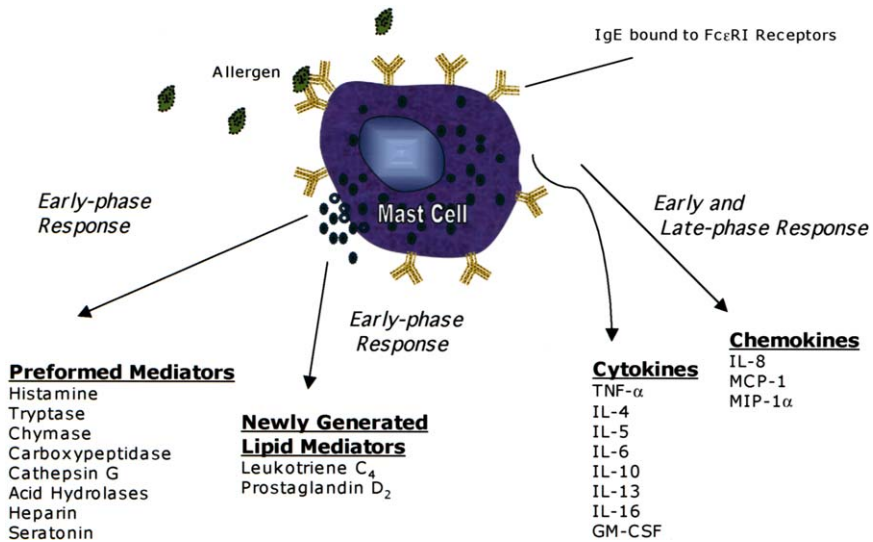


Fig. 1. Mediators of mast cells and basophils. TNF, tumor necrosis factor; IL, interleukin; GM-CSF, granulocyte-macrophage colony-stimulating factor; MCP, monocyte chemotactic protein; MIP, monocyte inflammatory protein.

Manifestations of allergy

Physical manifestation of allergy is often debilitating. In the lung there may be bronchoconstriction or asthma; in the nose, rhinitis; in the skin, urticaria; in the eyes, conjunctivitis. Systemic manifestations of allergy characterize anaphylaxis, which may be life threatening and require immediate medical attention. Anaphylaxis that occurs in conjunction with exercise, termed *exercise-induced anaphylaxis*, has been reported in certain individuals [10]. In addition to the inflammatory response, allergic disease manifestations have been associated with fatigue [11], depression [12], and decrements in cognition [13]. In fact, epidemiologic evidence suggests that seasonality of even extreme behavioral manifestation, such as suicide, may be associated with allergy [14] or with exposure to seasonal allergens, specifically tree pollen [15].

Diagnosis of allergy

In addition to the clinical manifestations of an allergic disorder, *in vivo* and laboratory-based testing for allergen sensitivity is used to strengthen the diagnosis of allergic disease. Skin-prick and intradermal testing provide a rapid and functional measure of IgE-mediated hypersensitivity in the skin with a resultant wheal and flare response that occurs within 15 minutes of allergen contact. In situations of decreased skin response from treatment with antihistamines, increased skin sensitivity from dermatographism, or logistical constraints, athletes may opt for the radioallergosorbent test (RAST), which detects specific IgE antibodies in serum with comparable sensitivity and specificity to skin-prick testing [16].

Seasonal effects of allergy on athletes

Environmental exposures

Athletes are particularly vulnerable to the effects of the environment, as many athletic activities—whether they are for leisure, training, or competition—occur outdoors. When athletes train and compete, often for long durations of time, they make intimate contact with the outdoor elements that are comprised of a host of topographical, seasonal, and climatic components. Exposure to the various elements is magnified by increased ventilation (up to 200 L/min during exercise), which intensifies the contact between the respiratory system and environment, and by inhalation of poor quality and cold air, which often affects those who train year-round in temperate climates [17,18]. During the winter months, cold air inhalation during exercise exacerbates exercise-induced asthma in people

with asthma [19]. Track and field athletes have extensive exposure to various seasonal pollen allergens during the spring (trees), summer (grass), and early fall (ragweed) [20]. Susceptible athletes may develop conjunctival inflammation, rhinitis, or bronchial inflammation as a consequence of these environmental conditions.

Allergies and Olympic athletes

Based on a 15-year database of aeroallergen records, an in-depth study reported the predicted aeroallergen counts that would be observed in association with the 2004 Olympic games in Athens, Greece. The study predicted that peak pollen concentrations would be observed during training in March and May, and high levels of goosefoot, mugwort, *Alternaria*, and *Cladisporum* spores would be observed in August and September during the Olympic games [21], thus highlighting the need for preparation, testing, and therapy.

An earlier survey of allergic disease in elite athletes reported that 20% of the 1984 Australian Olympic team had allergic disorders based on clinical evaluation (not including skin testing or RAST) [22]. Of 214 Australian Olympic athletes studied during the 2000 Sydney Olympics, which took place during the period of high tree pollen (over 5000 grains per $C_{\mu}M$), a high prevalence of perennial and seasonal allergic disease was reported. Within that group, 56% reported a history of allergic rhinoconjunctivitis, 41% were symptomatic with a positive skin test to a potential allergen, 29% were diagnosed with seasonal allergic rhinoconjunctivitis based on history and skin testing, and 21% suffered from asthma [23]. Before the 2004 Olympics in Athens, Greece, information regarding circulating aeroallergens in neighboring cities was published to attempt to minimize the allergy symptoms and help athletes achieve peak performance. Understandably, the incidence of adverse responses to seasonal allergens is common amongst athletes. Although athletes with allergic diseases have successfully won many Olympic medals, the detrimental contribution of pollutants, allergens, or other environmental factors has occasionally produced severe exacerbations of allergic conditions, warranting medical attention and causing suboptimal performance.

Seasonal exposures to infectious agents may also induce seasonal decrements in athletic performance. Viral infections reviewed in Nelson et al [24], such as influenza, reovirus, and respiratory syncytial virus, manifest a seasonal peak in winter and early, cause significant morbidity, may trigger bronchial hyper-reactivity, and may result in secondary bacterial infections such as sinus or bronchial infections (Table 1).

Several studies have characterized the relationship between viral infection, which is primarily a T-helper type 1 (Th1) response, and enhancement of allergic disease, which is a T-helper type 2 (Th2) response. Viral infections like influenza A may trigger allergic asthma by interfering with tolerance to aeroallergens [25], inducing a concomitant Th1 response [26], and causing recruitment of Th2 cells

Table 1
Seasonality of infectious diseases

Infection	Peak prevalence
Malaria	Winter–early spring
Leishmaniasis	Winter–early spring
Influenza	Winter–early spring
Human reovirus	Winter
Coronavirus	Winter–early spring
Respiratory syncytial virus	Winter–early spring, summer

Adapted from Nelson RJ, Demas GE, Klein SL, et al. Seasonal patterns of stress, immune function, and disease. 1st edition. Cambridge, MA: Cambridge University Press; 2002.

into the lung [27]. In some people, seasonal allergy and mood vulnerability to inflammation may interact, and people with allergies may experience more post-flu mood worsening than those without allergies [28].

Allergic rhinitis

Allergic rhinitis in athletes

Often the initial contact of pollen and other airborne components is with nasal mucosal and eyes. Studies have shown that allergic rhinoconjunctivitis is under-recognized and certainly undertreated in elite athletes [18]. Helenius et al [29] reported the results of a survey of 49 athletes competing in summer events. The diagnosis of allergic rhinoconjunctivitis was more common among athletes than in a control group of nonathletes ($P = .037$). Helbling et al [30] surveyed 2961 Swiss athletes who participated in 68 sports. Of the 79% who responded to the questionnaire, 16.8% indicated they suffered from hay fever and 59% reported that they needed medication during the pollen season. Individuals who have allergic rhinitis also often have increased bronchial hyperresponsiveness [31,32].

Pathophysiology and presentation

Allergic rhinitis (AR) is characterized by nasal mucosa edema as a result of IgE-mediated release of early- and late-phase mediators and Th2 cytokines, which promotes the infiltration of mucosa with inflammatory cells such as eosinophils, neutrophils, basophils, T cells, and macrophages [2,33,34]. AR is characterized by nasal congestion, rhinorrhea, sneezing, itching of the nose, or postnasal drainage and is often associated with allergic conjunctivitis manifested by ocular itch, tearing, redness, and sometimes swelling and photophobia. These and other signs and symptoms of AR are listed in **Box 1**. Allergic, irritant,

Box 1. Signs and symptoms of allergic rhinitis

Itching of the nose, ears, palate, throat
Sneezing spells
Clear and watery rhinorrhea
Congestion
Sinus headache
Mouth breathing
Post-nasal drip
Sleep disturbance
Cough
Fatigue

infectious, hormonal, occupational, and other factors are causative agents in nasal mucosal edema [35]. Peak presentations of allergic rhinoconjunctivitis occur between 10 to 25 years, which includes the age range of most elite athletes [3].

Allergic rhinitis and athletic performance

Although exercise may increase nasal airway patency, likely by way of increased sympathetic nerve discharge [36], there are several reasons why AR may interfere with athletic performance. Rhinitis often causes changes in sleep patterns because of nasal obstruction, rhinorrhea, and sinus pressure that add to tiredness and fatigue and impair athletic performance [18]. Additionally, short bursts of sprinting require nasal breathing for optimal performance [36], implicating nasal obstruction as a deleterious factor in performance. AR has been associated with alteration in central nervous system (CNS) function, which may significantly affect the athlete's ability to perform. Using a standardized computer-based battery of cognitive processing tests, subjects who have AR experienced decrements in reaction time, attention, and vigilance when exposed to pollen [13]. Others associated AR with increased fatigue [11], depression [11,37], and anxiety in women [38], which all pose a potential hindrance to performance.

The mechanism by which seasonal environmental exposures may affect these cognitive and emotional parameters has not been elucidated. Reichenberg et al [39] has shown activation of the innate immune response in humans by low-dose endotoxin exposure and subsequent release of pro-inflammatory cytokines (eg, IL-1, IL-6, TNF- α), some of which are also released by IgE-mediated responses in the nasal mucosa (see Fig. 1) [40,41]. The exposure to endotoxins and secretion of cytokines induced depressive symptoms, anxiety, and cognitive impairment in the absence of constitutional symptoms (eg, decreased energy, anorexia, drowsiness) [39,42]. Circulating cytokines may also induce activation of the HPA-axis [43], and elevation of corticotrophin-releasing factor (CRF)

and cortisol have been shown to contribute to major affective illness [44]. Furthermore, cytokines in the respiratory tract may stimulate specific receptors present in the vagus nerve and affect brain function through this pathway [45]. As such, cytokine release could contribute to decrements in cognition and to the onset or exacerbation of depression or anxiety, thus having detrimental effects on athletic performance.

Management of allergic rhinitis

Prevention

Athletes must prepare themselves for the various climatic conditions they will encounter in their training locale and the competition destinations. Knowledge of the temperature, humidity, sunlight, altitude, season, and type and concentration of pollen they will be exposed to can help athletes achieve peak performance through the use of prophylactic measures [21].

Nonpharmacologic therapy

Various nonpharmacologic therapies have been effective under certain conditions. For the treatment of AR, saline nasal irrigation is a safe and effective method of cleansing the nasal mucosa of allergens, and improving nasosinus disease [46]. External nasal dilators have been used by athletes [47] to increase nasal valve area, which is the narrowest area in the nasal canal [46,48], and to significantly decrease submaximal exercise-perceived exertion, heart rate, ventilation, and volume of oxygen use [47].

Pharmacologic treatment and doping concerns

Beginning with the earliest Olympic games, athletes have enhanced athletic performance through the use of foreign substances. In 1967, the International Olympic Commission developed a list of prohibited methods and substances to protect the health of, and foster equality for, all competing athletes [49]. In the realm of allergic diseases, these restrictions have generated significant challenges. For example, the overall Olympic champion in women's gymnastics at the 2000 Olympics was disqualified and her gold medal withdrawn because she used a medication for AR which contained pseudoephedrine, allegedly without the intent of doping. Pseudoephedrine is no longer on the Prohibited List (Table 2). In 1972, the winner of the 400-meter men's Freestyle was disqualified because significant levels of the banned drug ephedrine were detected in his post-race urinalysis. The swimmer had used a combination ephedrine/theophyl-

Table 2
Medical treatment of seasonal allergic diseases related to athletes

Drug	Mode of action	Use	Comments	WADA status ^a
Antihistamines Oral first-generation Oral second-generation Intranasal Topical (optical)	Histamine antagonist at H1 receptor site	Allergic rhinitis and conjunctivitis, urticaria, allergic asthma	First-line therapy for mild to moderate symptoms Often combined with oral decongestant Very effective for symptoms of rhinorrhea, sneezing, and nasal and ocular itch First-generation formulations have significant CNS adverse effects, second-generation preferred	Not prohibited
Corticosteroids Oral Inhaled Intranasal Topical (skin)	Antiinflammatory	Treatment of many allergic diseases: allergic rhinitis and conjunctivitis, asthma, urticaria, and atopic dermatitis	Potent antiinflammatory intranasal — first-line therapy for moderate to severe symptoms of rhinitis oral — severe exacerbations of asthma, urticaria	Systemic ^b uses are prohibited ^c ; inhaled and intranasal only require abbreviated TUE ^d ; topical are not prohibited
Decongestants Oral Intranasal	α -adrenergic agonist Causes nasal vasoconstriction	Rhinitis	Reduces nasal congestion May cause insomnia, loss of appetite, and nervousness Intranasal may cause rebound nasal congestion (rhinitis medicamentosa)	Ephedrine is prohibited ^e , phenylephrine, phenylpropanolamine, pseudoephedrine, and synephrine are on monitoring list ^f
Cromolyn/nedocromil sodium Oral Inhaled Intranasal	Mast cell stabilizer Inhibits degranulation	Asthma, allergic rhinitis	Nonsteroidal antiinflammatory Minimal adverse effects Requires multiple daily dosing	Not prohibited

Anticholinergic Inhaled Intranasal	Muscarinic receptor antagonist	Asthma, rhinitis	Effectively reduce rhinorrhea Role in acute bronchospasm	Not prohibited
Leukotriene inhibitors Oral	Inhibit phospholipid metabolism	Asthma	Nonsteroidal antiinflammatory Very effective in preventing EIA Steroid sparing controller	Not prohibited
Allergen immunotherapy	Th2 response suppression Th1 response stimulated	Allergic rhinitis and conjunctivitis, asthma	Very effective for allergic rhinitis	Not prohibited
Anti-IgE antibody IM injection	Reduces serum IgE	Severe asthma, possibly allergic rhinitis	Approved for severe asthma Requires multiple IM injections Expensive	Not prohibited
β_2 agonist Inhaled Short-acting Long-acting	Bind β_2 -adrenergic receptor, \uparrow cAMP, relaxes bronchial smooth muscle	Asthma	First-line therapy in preventing EIA	Prohibited in general, some formulations with specific clinical indications require only an abbreviated TUE ^g
Theophylline Oral Inhaled	Inhibits phosphodiesterase, causing bronchodilation	Asthma	Long-term controller Required serum level monitoring	Not prohibited
Epinephrine	α - and β -adrenergic agonist	Anaphylaxis	Universally recommended drug of choice for acute anaphylaxis Adult dose: SQ or IM, 0.2–0.5 ml of 1/1000 (wt/vol) dilution	During competition requires a TUE

Abbreviations: cAMP, cyclic adenosine monophosphate; EIA, exercise-induced asthma; IM, intramuscularly; SQ, subcutaneously; TUE, therapeutic use exemption.

^a Based on the WADA 2005 Prohibited List International Standard, see www.wada.com for current updated information.

^b Orally, rectally, IV, or IM.

^c In competition only. Any substance or method that is on the Prohibited List must be granted a Therapeutic Use Exemption (TUE) for use.

^d nonsystemic routes require the completion of an abbreviated TUE application. Dermatological preparations are not prohibited.

^e Prohibited when its concentration in urine is >5 mcg/mL.

^f These stimulants are no longer on the Prohibited List, but are on the 2005 monitoring program “in order to detect patterns of misuse in sports.”

^g All β_2 -agonists including their D- and L-isomers are currently (2005) prohibited in and out of competition and require a TUE for use. Formoterol, salbutamol, salmeterol, and terbutaline are permitted by inhalation to prevent and or/treat asthma and exercise-induced asthma/bronchoconstriction, with the completion of abbreviated TUE application.

line preparation for the prerace treatment of his asthma [22]. These examples highlight the importance for athletes, coaches, and team physicians to understand the potential that a particular medication, apparently innocuous, may contain or be contaminated by a particular banned substance. To establish a universal internationally standardized Olympic antidoping code, the World Anti-Doping Agency (WADA) was established in 1999. The WADA disseminates current information on substances prohibited for use by competitive athletes. WADA maintains and updates the World Anti-Doping Code Prohibited List, which includes substances that are prohibited only during competition, others that are always prohibited, and others that are on the monitoring list (see Table 2). More and current updated information regarding antidoping rules, regulations, and prohibited substance listings can be found at www.wada-ama.org.

Pharmacologic therapies

Oral decongestants, such as pseudoephedrine and phenylpropanolamine, are effective in reducing nasal congestion from rhinitis, although they can cause insomnia, loss of appetite, and excessive nervousness [3,50]. These decongestants are no longer on the Prohibited List, but have been placed on the 2005 Monitoring Program to detect patterns of misuse in sports (see Table 2). Topical α -adrenergic nasal decongestants are commonly used, but may cause rhinitis medicamentosa, the syndrome of rebound nasal congestion, when overused [3]. Antihistamines are commonly used, have long been standard therapy for seasonal and perennial AR, and are effective in relieving symptoms of itching, sneezing and rhinorrhea, and allergic conjunctivitis [6,51]. The older first-generation antihistamines are significantly limited by their sedative and anticholinergic adverse effects and may compromise important psychomotor skills (eg, reaction time, visual discrimination) [52], and therefore should be avoided. The newer second-generation antihistamines are preferred because of their longer duration of action and minimal, if any, CNS adverse effects [53,54]. Intranasal steroids are likely the most effective therapy for AR and allergic conjunctivitis. Their efficacy is maximized with continuous use [53], but there are also proven as-need benefits [55]. They have the benefit over antihistamines of down-regulating numerous steps in the inflammatory process, including reducing the release of cytokines and chemokines [56]. Their use in Olympic athletes requires the application for an abbreviated therapeutic use exemption (TUE). Several studies have shown concurrent reduction with use of intranasal corticosteroids in bronchial hyperresponsiveness [5]. Leukotriene modifiers have confirmed benefit in allergic rhinitis comparable to antihistamines [57]. Other effective intranasal agents include intranasal antihistamines, ipratropium bromide, and cromolyn sodium [3]. To date, there have been no published reports of immunotherapy specifically targeting elite athletes [18]. However, allergen immunotherapy significantly decreases the severity of AR, reduces the requirement for pharmacotherapy, and improves quality of life [53].

Asthma

Asthma in athletes

Asthma is characterized by variable airflow obstruction, bronchial hyper-responsiveness, and airway inflammation. Respiratory viral infections (most common), allergens, irritants, drugs, climatic conditions, exercise, and other stimuli can induce asthmatic exacerbations. In a survey of 214 athletes representing 12 Olympic sports, 21% reported having experienced asthma [23]. The reason for the higher prevalence of asthma among athletes is because of their exposure to various climatic conditions (eg, cold in winter), pollen content (eg, grass, weed, trees), and various pathophysiologic changes that occur during exercise that may promote bronchial hyperresponsiveness [17,19,58–63]. When strenuous exercise results in resistance to airflow, the phenomenon is called exercise-induced bronchospasm or exercise-induced asthma (EIA) [19].

Exercise-induced asthma

Pathogenesis and presentation

Various factors have been described in the development of EIA. The generally accepted pathogenesis of EIA involves the loss of heat and water from airway mucosa [59,64–66]. During exercise and increased respiratory rate, large volumes of dry, cold air reduce airway cooling as it travels along the tracheobronchial tree, which may cause bronchial constriction through a reflex stimulation of airway receptors. This response may lead to changes in mucosal osmolarity and promote mast cell degranulation, consequently furthering bronchoconstriction [67]. Rapid airway rewarming after exercise may cause vascular congestion, increased permeability, and edema, leading to obstruction [19,68].

EIA symptoms of chest tightness, wheezing, and shortness of breath generally follow a brief period of bronchodilation that occurs at initiation of exercise. Bronchoconstriction peaks 8 to 15 minutes after exercise and resolves in about 60 minutes. A refractory period of up to 3 hours after recovery, during which time repeat exercise causes less bronchospasm, has been observed.[68]. Although EIA generally commences following the cessation of physical exertion, it may also appear during sustained activity [69].

Seasonal consideration in exercise-induced asthma

Track and field athletes are extensively exposed to seasonal allergens. When the rate of breathing exceeds 30 L/min, there is a shift from nose breathing to combined mouth and nasal breathing, thereby increasing deposition of airborne allergens and other inhaled particles into the lower airway [20]. Asthmatic subjects who are allergic to birch pollen were shown to have aggravated EIA

responses during the pollen season when compared with the response of asthmatic individuals who are not allergic to birch pollen [63].

Challenges in the diagnosis of exercise-induced asthma

It has been reported that 40% to 90% of asthmatics have EIA [61,67,70–72]. Before the 1984 Summer Olympics, 67 of 597 (11%) athletes screened for performance experienced symptoms of EIA, yet only 26% of these athletes had a history of asthma [60]. However, 42 of 67 reported symptoms of asthma during strenuous exercise, thus emphasizing the importance of screening for EIA in well-conditioned individuals who appear to be in excellent health [73].

EIA is a common and often unrecognized problem among school-age participants in sports [74]. In a cohort of 256 adolescent athletes, 9.4% were diagnosed with EIA. Yet screening for EIA through physical examination and history did not accurately predict it and would have detected only about half of the children who were diagnosed as having EIA using spirometry [75]. Furthermore, 9% of individuals in the general population who have EIA have no history of asthma.

Management of asthma

Prophylaxis

Detailed prior knowledge of environmental exposures can help prevent or minimize exacerbation. Once a pattern of symptoms has been established, prophylaxis is the key to therapy in patients who have EIA. Physical warm-up can reduce EIA. Continuous, low-intensity warm-up is more effective than interval warm-up in preventing EIA in athletes [76]. In certain cold conditions, such as those experienced by cross-country skiers, breathing filters are being used [67]. Although there is no clear evidence that physical conditioning decreases EIA or improves pulmonary function, it does delay the symptoms of EIA from reaching threshold [77].

Pharmacologic therapy

Long-term and more effective control is gained with pharmacologic intervention. The most common and effective prophylactic therapies used before exercise are β_2 agonists, cromolyn, and nedocromil (see Table 2) [78–80]. Inhaled corticosteroids may be helpful in reducing bronchial hyperresponsiveness when used for their long-term antiinflammatory effects. Theophylline preparations and anticholinergics are used as tertiary agents when other agents are proven ineffective. However, the requirement for blood-level monitoring of a narrow therapeutic window makes theophylline a less desirable alternative agent for athletes [61]. Short-acting β_2 agonists are considered first-line therapy and

provide protection in 80% to 90% of subjects with minimal adverse effects [81,82]. Long-acting β_2 agonists can prevent bronchial obstruction for 10 to 12 hours [83] and provide athlete with a full day of protection. Formoterol, salbutamol, salmeterol, and terbutaline are permitted by inhalation with the completion of an abbreviated TUE, but all other β_2 agonists, including their D- and L-isomers are prohibited in and out of competition, according to the 2005 Prohibited List (see Table 2). Regular use of β_2 agonists without antiinflammatory treatment may increase airway responsiveness to irritants and foster allergen-induced late bronchoconstriction and airway inflammation [84].

Given the central role of IgE in the pathogenesis of allergic disease, inhibiting IgE responses through the use of anti-IgE antibodies would decrease its sensitizing effects on mast cells and basophils. Several studies indicate that anti-IgE therapy is effective in the treatment of asthma and allergic rhinitis [85–88]. However, the implications for athletes are unknown.

In conclusion, evidence supports that seasonal allergy confers a seasonal vulnerability for performance decrements in competition and training. Specific interventions that prevent and directly address the allergic symptomatology, tailored to athletes' individual needs in specific circumstances, could prevent seasonal decompensation in performance or restore functioning to baseline levels.

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