

The environment, the airway, and the athlete

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Objective: To review the interaction of environmental factors with host conditions, including atopy, the potential resulting impaired upper and lower airway function, and diagnostic and therapeutic considerations in the athlete.

Data Sources: OVID, MEDLINE, and PubMed searches were performed cross-referencing the keywords: *asthma, athlete, atopy, bronchospasm, exercise, pollution, and rhinitis*.

Study Selection: Articles were selected based on relevance to the subject matter.

Results: Recent studies have yielded significant advances in our understanding of how intrinsic and extrinsic factors can potentially result in impaired function of the airways of athletes. Extrinsic factors include environmental exposure to temperature, humidity, aeroallergens, irritants, and pollution. Intrinsic factors include atopy, allergic rhinitis, asthma, and anatomical variants. These intrinsic and extrinsic factors can affect both the athlete's quality of life and athletic performance. However, uncertainty remains regarding relative contributions of these factors in explaining the high degree of bronchospasm seen in various populations of athletes with and without asthma, and no consensus exists regarding the most appropriate diagnostic and therapeutic modalities.

Conclusions: Great variability exists in the presentation, laboratory findings, diagnostic maneuvers, and response to therapeutic measures among populations of athletes in different sports and among individuals. An improved understanding of the unique exposures faced by athletes in different disciplines, of the available tests for pursuing the appropriate diagnosis, and of the available therapies will allow the allergist to provide clinical improvement and allow the athlete to find relief and achieve his/her full potential.

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INTRODUCTION

In the athlete a variety of host and environmental factors can potentially affect the upper and lower airway, leading to impaired function. Environmental factors include exposure to ambient temperature, humidity, aeroallergens, irritants, and pollution. Host factors include atopy, allergic rhinitis (AR),

asthma, and anatomical variants. These factors can affect both the athlete's quality of life and athletic performance. It is important that the allergist understand the various factors and their impact on the athlete, as well as the appropriate diagnostic and therapeutic measures to allow this unique population to achieve improved quality of life, symptom control, and maximum athletic potential.

This article reviews the interaction of environmental factors with host conditions, including atopy, the potential resulting impaired upper and lower airway function, and diagnostic and therapeutic considerations in the athlete. OVID, MEDLINE, and PubMed searches were performed cross-referencing the keywords: *asthma, athlete, atopy, bronchospasm, exercise, pollution, and rhinitis*. Articles were selected based on relevance to the subject matter.

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THE UPPER AIRWAY

Under normal circumstances the nasal airway is responsible for filtering, warming, and humidifying inspired air up to a ventilatory rate of 30 L/min.^{1,2} The nasal airway has been shown to respond differently to exercise than the lower airways in both healthy individuals and those with rhinitis and asthma. In healthy individuals during exercise the nasal airway becomes more patent and leads to a decrease in nasal airway resistance.³ Rhinitis, whether allergic, nonallergic, or exercise induced, can cause congestion and/or rhinorrhea, resulting in increased nasal resistance.⁴ This sense of nasal airway obstruction has been termed *nasal dyspnea*.⁵ In addition, the symptoms of AR and non-AR (NAR) can significantly affect an individual's quality of life, leading to sleep disturbance, fatigue, headache, and cognitive impairment.⁴

Role of Allergens

AR is thought to affect 10% to 30% of the population, with most studies estimating the ratio of AR to pure NAR as 3:1.^{4,6} It is reported that 44% to 87% of individuals with chronic rhinitis have mixed rhinitis. An increased prevalence of AR of up to 40% in endurance athletes has been reported. In the athlete this can translate not only to decreased quality of life but also to suboptimal athletic performance.^{7,8} In 1 study evaluating Australian athletes, 56% reported symptoms consistent with AR, 41% reported symptoms and had at least 1 positive skin prick test result, and 29% were diagnosed as having seasonal AR. In this cohort athletes from aquatic sports were more likely to report symptoms. Compared with the NAR athletes, the athletes with AR reported increased symptom scores and decreased quality of life during the pollen season.⁷ In this and other studies, it was observed that AR appeared to be underrecognized and undertreated in athletes.⁷⁻⁹

Silvers et al¹⁰ examined the prevalence and nature of nasal symptoms occurring during both indoor and outdoor exercise in a population of athletes with and without AR. They reported that exercise-induced rhinitis occurred in 61% of indoor athletes and 56.1% of outdoor athletes. Rhinorrhea was the primary symptom, followed by nasal congestion and postnasal drip. Forty percent indicated that their symptoms adversely affected their athletic performance, and this occurred more frequently in the 21% of the population with AR. Silvers et al concluded that exercise-induced rhinitis was common and adversely affected athletic performance in both allergic and nonallergic individuals. They theorized that this might represent a form of vasomotor rhinitis; however, they found that those with AR were affected more frequently and were more likely to have their performance adversely affected.¹⁰

Role of Irritants

Because the prevalence of atopic disease has increased worldwide among athletes, recent attention in the medical literature has focused on the role of pollution as a potential cause with particular attention on nitrogen dioxide and particulate matter (PM). Wang et al¹¹ demonstrated that in patients with a

history of AR, exposure to nitrogen dioxide at concentrations similar to those found at curbside in heavy traffic followed by allergen challenge resulted in increased levels of eosinophilic cationic protein (ECP) in nasal lavage fluid. The authors suggested that the exposure to nitrogen dioxide may "prime" eosinophils for subsequent activation in susceptible individuals, possibly enhancing the upper airway response to inhaled allergens and exacerbating their disease. In a study examining junior ice hockey players exercising in indoor ice arenas with fossil fuel-powered surfacing equipment, increased exposure to nitrogen dioxide was significantly associated with the increased prevalence of rhinitis symptoms.¹² Additional evidence of the effect of pollutants on the airway comes from studies of diesel exhaust particles (DEPs). Patients challenged nasally with DEPs have exhibited IgE isotype switching and increased total IgE levels. DEPs have also been shown to interact with allergens to augment allergen-induced responses and to increase allergen specific IgE levels up to 50-fold greater in allergic patients. Also, DEPs administered with allergen were shown to induce a T_H2 cytokine profile with decreased interferon γ levels in the nasal mucosa, to have direct effects on mast cells and basophils, and to have increased allergic response.¹³

Swimming pool attendance, both during childhood and later in life, has been implicated as a risk factor for AR with repeated exposure to chlorine thought to be the mechanism.¹⁴ In a study comparing competitive swimmers training an average of 32 hours per week with nonswimming controls, Zwick et al¹⁵ demonstrated increased AR and subclinical aeroallergen sensitization in the swimmers. They concluded that frequent exposure to chlorine may facilitate sensitization to aeroallergens.

Treatment

Treatment of rhinitis in the athlete should be based on accurate diagnosis and symptoms, with special attention to the adverse effects on performance and regulatory guidelines and requirements for the sport.^{4,8} Although standard practice includes avoidance of relevant exposures, often it is not practical for athletes engaged in training that requires prolonged exposure to indoor and/or outdoor triggers. With limitations to avoidance, pharmacotherapy is often the mainstay of management. In keeping with the practice parameters,⁴ topical nasal corticosteroids and antihistamines are considered first-line therapy for both AR and NAR, with oral antihistamines and leukotriene receptor antagonists also options. An open-label treatment study of 977 athletes diagnosed 24% of participants as having AR and offered treatment with intranasal budesonide. Comparisons of baseline symptom scores, overall quality of life, and performance scores demonstrated statistically significant improvements in symptoms, quality of life, and performance scores in athletes using the intranasal budesonide.¹⁶ Because of their clinically significant rapid onset of action, topical nasal antihistamines are also recommended for as-needed use in episodic or intermittent rhinitis. However, care should be taken to avoid the first-generation

antihistamines because their central nervous system effects can result in sedation with a potential for impaired cognition and decreased reaction times.^{4,8} In individuals in whom the primary concern is rhinorrhea, the use of a nasal anticholinergic such as ipratropium can provide relief.^{1,4} Isotonic and hypertonic nasal saline irrigation has been demonstrated to improve symptom control and quality of life in rhinitis patients.⁴ An additional benefit of obtaining optimal control of AR in the upper airway may include reducing the risk of increased airway hyperresponsiveness in the lower airway. Work by Bonay et al¹⁷ demonstrated the presence of airway hyperresponsiveness to methacholine challenge and increased amounts of eosinophils and ECP in the sputum of nonasthmatic patients after nasal allergen challenge outside pollen season.

In the appropriate patient for whom pharmacotherapy measures have failed to provide relief, immunotherapy has been shown to be effective in providing control of rhinoconjunctival symptoms, decreased seasonal pulmonary symptoms with decreased bronchial hyperreactivity (BHR) in pollen season, and improved quality of life that may be beneficial to the athlete.¹⁸ Although omalizumab has been demonstrated to be effective in treating AR and this may conceivably have a role in athletes, it is currently approved by the Food and Drug Administration only for treatment of allergic asthma.⁴ The external nasal dilator (eg, Breathe Right) is used by many athletes and has been shown to significantly increase nasal valve area by an average of 25% as measured by acoustic rhinometry. External nasal dilators have also been shown to improve submaximal exercise economy by decreasing oxygen uptake, ventilation, heart rate, and perceived exertion.¹⁹ These benefits illustrate the important contribution of a patent nasal airway to an athlete's ability to achieve his/her best performance and highlight the significant adverse impact of nasal dyspnea.

THE LOWER AIRWAY

Exercise-induced bronchospasm (EIB) is defined as the transient narrowing of the airways occurring with exercise.²⁰ The pathophysiology of EIB is thought to be due to the dehydrating effect of evaporative water loss from the airway surfaces as they humidify large quantities of inspired air. This evaporative water loss leads to an increase in the osmolarity of the airway surface, with water loss from the airway cells causing cell shrinkage. Cell shrinkage, through loss of water volume, is believed to result in the release of inflammatory mediators, including histamine, leukotrienes, and prostaglandins, from airway epithelial and mast cells.^{20,21} This effect can be magnified by breathing cold dry air that leads to recruitment of additional generations of airways in an effort to warm and humidify the inspired air. For similar reasons, exercises that require greater minute ventilation and/or increased intensity and duration are associated with more severe EIB.²² Recent work by Anderson et al²¹ suggests that the pathogenesis of EIB in elite athletes results from airway epithelial injury due to repeatedly breathing poorly conditioned air at high respi-

ratory rates for prolonged periods with possible contribution from large volumes of irritant particles or gases. Over time, these exposures were thought to increase sensitivity of the bronchial smooth muscle to various triggers of bronchoconstriction. Anderson et al supported this concept with data demonstrating that when competitive athletes discontinued competition or high-level training their airways exhibited decreased reactivity and decreased numbers of inflammatory cells and their pulmonary symptoms demonstrated improvement or even resolution.²³

Although EIB has been estimated to occur in up to 90% of patients with underlying asthma, it also occurs in individuals with no prior history of asthma and no symptoms outside exercise.²⁴ The prevalence of EIB in athletes has been estimated to range from 8% to 50%.²⁵⁻²⁸ The prevalence rate of EIB varies, depending on the athletic activity. The highest rates of EIB are noted in swimmers; endurance athletes, such as runners, cyclists, and cross-country skiers; and in speed and power athletes, such as track and field, ice hockey, and ice skaters.^{25-27,29,30} Although Wilber et al²⁷ found EIB to be more prevalent in female (26%) vs male (18%) athletes, no sex difference was noted in a more recent study of college athletes by Parsons et al.²⁸ The prevalence of BHR and asthma has also been shown to be significantly increased in athletes with atopy. Combining high-risk sports and atopy results in further increases in the relative risk of asthma. Atopic speed and power athletes, atopic long-distance runners, and atopic swimmers have been found to have relative risks of 25-, 42-, and 96-fold, respectively, when compared with nonatopic controls.²⁹

Like asthma, EIB has a syndromic clinical definition that may represent several different phenotypes.³¹ EIB occurring in athletes with a history of preexisting asthma may represent poor control of the underlying asthma and exhibits the classic eosinophilic airway inflammation associated with asthma.^{24,31} EIB in athletes without a prior history of asthma exhibits airway inflammation suggestive of an irritant phenomenon with a predominantly neutrophilic pattern or a mixed pattern of eosinophilic and neutrophilic components suggestive of a combined irritant and allergic process.^{21,24,26} Increased airway inflammatory cells found in endurance athletes also occur in athletes without respiratory symptoms or evidence of EIB. This suggests that increased airway neutrophils may result from hyperventilation associated with endurance training, whereas the increased airway eosinophils may result from a response to specific environmental exposures.³²

When the ventilatory rate exceeds 30 L/min the nasal airway is no longer capable of meeting the demand, so combined nasal and oral breathing is necessary. The additional oral airflow bypasses the nasal filtration and conditioning mechanisms.^{1,2} Oral airflow is increased in the presence of nasal congestion, which may place the lower airway at increased risk.³³ During exercise, ventilation can increase up to 200 L/min for short periods in speed and power athletes and for longer periods in endurance athletes. Airflows of these magnitudes and duration may lead to an increased

deposition of aeroallergens and other particles in the lower airway.²

Role of Allergens

Aeroallergen exposure in nonasthmatic individuals with AR resulted in increased airway inflammation marked by eosinophils, ECP in sputum, and increased BHR. Furthermore, higher levels of eosinophils and ECP correlated with greater methacholine responsiveness.¹⁷ Indoor aeroallergen levels in some training facilities have been shown to exceed the threshold for sensitization,³⁰ potentially contributing to the observation that athletes training indoors experienced respiratory symptoms more often than when training outdoors in cold, dry weather or during pollen season.² Pollen exposure has been demonstrated to result in seasonally increased EIB in sensitized athletes exercising outdoors during pollen season.^{34,35} Helenius et al²⁹ found that seasonal AR occurred more frequently in athletes vs controls and that the severity of the seasonal AR significantly correlated with BHR and severity of atopy.

Role of Irritants

Cold air is a nonspecific airway irritant in asthmatic and healthy individuals both at rest and with exercise.^{36,37} Inhalation of cold air increased airway responsiveness to histamine in asthmatic and healthy individuals at rest;³⁶ it also increased inflammatory cells in the bronchoalveolar lavage fluid of healthy individuals after exercise.³⁷

Athletes are exposed to significant levels of pollutants in both indoor and outdoor settings during exercise, resulting in lower airway inflammation, respiratory symptoms, increased bronchial reactivity, and impaired exercise performance.^{12,38–41} Pollutants, including nitrogen dioxide, ozone, sulfur dioxide, and PM, occur either directly or indirectly primarily as a result of the combustion of fossil fuels.^{12,38,41} In a study examining 793 Finnish junior hockey players, the respiratory effects of increased nitrogen dioxide exposure were associated with increased cough prevalence, asthma prevalence, nonspecific airway hyperresponsiveness, and decreased lung function.¹² Levels of fine and ultrafine PM in indoor ice arenas have been shown to reach levels nearly 30 times greater than corresponding outdoor values. These high-level exposures may contribute to the airway inflammation and BHR seen in indoor winter athletes.³⁸ A study demonstrating the protective effect of a single dose of montelukast taken 6 to 8 hours before an exercise challenge in a high-PM environment suggests that leukotrienes may act as mediators of the EIB noted to occur in high-PM environments.³⁹ The detrimental effect of fine and ultrafine PM inhalation on exercise in healthy individuals has been demonstrated, with exposure to high PM resulting in diminished exercise performance.⁴⁰ With nasal breathing, sulfur dioxide is almost completely absorbed by the nasal mucosa. However, with exercise and increased oral breathing a greater amount of sulfur dioxide is delivered to the lower airways.⁴¹ Even low levels of sulfur dioxide and ozone have been shown to result in de-

creased forced vital capacity and forced expiratory volume in 1 second (FEV₁) in competitive runners and cyclists.⁴¹ In the lower airway, DEPs resulted in increased numbers of airway inflammatory cells, increased levels of inflammatory cytokines, and increased airway resistance in asthmatic and healthy subjects. Similarly, DEPs increased BHR in asthmatic patients.¹³

Chlorine is a toxic gas with known airway irritant and inflammatory properties used to disinfect swimming pools. Swimmers are exposed to high levels of chlorine and chlorine-containing compounds when they inhale air just above the water surface during training sessions. These exposure levels potentially exceed recommended occupational limits.^{2,42} Repeated chlorine exposure in competitive swimmers has been associated with increased atopy as evidenced by sensitization to aeroallergens on skin prick and serologic testing, increased allergic rhinoconjunctivitis, and increased BHR.¹⁵ The high prevalence of asthma in elite swimmers is thought to be due to an irritant effect of the chlorine and chlorine byproducts, resulting in a form of occupational asthma. The most important risk factors for developing asthma are increasing age and amount of training time in a swimming pool.² In a study comparing elite swimmers with age-matched controls, Helenius et al²⁶ demonstrated that swimmers had increased numbers of sputum eosinophils and neutrophils, exercise-induced pulmonary symptoms, atopy, and BHR.²⁶ Only 1 of 29 swimmers in the study had asthma before starting their competitive swimming career. A 5-year, prospective follow-up of elite swimmers examined the effect of continued intense training and competition. In those swimmers stopping intense training, there was a decrease in sputum eosinophilia, BHR, and exercise-induced symptoms. In swimmers continuing intense training, eosinophilic airway inflammation persisted or increased, whereas BHR and symptoms were unchanged.²³ A recent study compared elite adolescent swimmers, healthy adolescents, and adolescents with asthma. Among the 3 groups there were no differences in exhaled nitric oxide, sputum cellular composition, or prevalence of BHR. In addition, there was no difference in respiratory symptoms between the swimmers and healthy adolescent controls.⁴³ This lack of airway inflammatory markers in the elite adolescent swimmers supports the theory that elite swimmers develop symptoms, airway inflammation, and BHR as a result of repeated chlorine exposure during their careers rather than being predisposed when they take up the sport. Several studies have examined the relationship between chlorine exposure and risk of developing atopy and asthma with variable results. A study of Belgian schoolchildren examined the presence of asthma, exhaled nitric oxide levels, aeroallergen specific serum IgE, and total serum IgE in relation to cumulative swimming pool attendance. The probability of developing asthma increased with increased pool attendance only in those children with elevated total serum IgE levels, whereas the exhaled nitric oxide level as a surrogate for pulmonary inflammation was elevated with increased pool attendance independently of serum IgE level.⁴⁴

In contrast, a similar study from Germany failed to show any effect of swimming pool attendance on the development of asthma or atopic disease.⁴⁵

Diagnosis

The signs and symptoms of EIB are variable and commonly include cough, wheeze, dyspnea, chest tightness, and excess mucous production.⁴⁶ Other nonspecific symptoms include poor performance, “feeling out of shape,” muscle cramps, fatigue, chest pain, and a sensation of “heavy legs.”²⁴ It has been shown that symptoms alone are not reliable in identifying EIB.^{46–48} Rundell et al⁴⁷ noted in elite cold weather athletes that 91% of the EIB-positive athletes had at least 1 symptom, whereas 48% of the EIB-negative athletes also had at least 1 symptom. A study comparing self-reported EIB symptoms to exercise challenge testing demonstrated no difference in reported symptoms between those with positive and negative exercise challenge results.⁴⁶ Exercise-induced dyspnea is a common presenting symptom in athletes, and many of these individuals are presumptively diagnosed as having and empirically treated for EIB. However, it has been shown that many of these EIB patients have other conditions that cause or contribute to their symptoms, including exercise-induced vocal cord dysfunction, exercise-induced laryngomalacia, gastroesophageal reflux, exercise-induced hyperventilation, cardiac abnormalities, or poor conditioning.^{24,49} Flattening of the inspiratory loop with spirometry can identify patients with vocal cord dysfunction and laryngomalacia, both of which can ultimately be diagnosed by flexible fiberoptic laryngoscopy.^{24,49} Abu-Hasan et al⁴⁹ reviewed 117 cases of presumptive EIB in patients referred to a pediatric pulmonary clinic and identified EIB as the cause of symptoms in only 11 patients. Bronchospasm or upper airway edema may also occur with exercise-induced anaphylaxis, which may be medication or food dependent.⁵⁰ Although exercise-induced anaphylaxis should be included in the differential diagnosis of an athlete with bronchospasm, most patients will exhibit cutaneous symptoms in addition to airway involvement.

The initial evaluation of patients with suspected EIB should include a detailed history and physical examination and lung function measurements both before and after administration of a short-acting inhaled β_2 -agonist.²⁴ Various methods of testing for EIB have been validated, including bronchoprovocation via pharmacologic (methacholine, histamine, saline, and mannitol) challenge, exercise challenge, and eucapnic voluntary hyperventilation (EVH).^{31,51,52} Although there is not universal agreement, positive bronchoprovocation challenge results are generally defined as a decrease in FEV₁ from baseline of 20% in the methacholine challenge test, 15% in mannitol or saline, and 10% in EVH.⁵³ In exercise challenge a decrease in FEV₁ of 10% to 15% is considered a positive result,⁵⁴ although some authors suggest a decrease as low as 6.5% should be considered a positive result in elite athletes.^{46,47}

Exercise challenge testing can use laboratory treadmill, cycle ergometry, a field free-run, or a sport-specific challenge

in the athlete's discipline.⁵² Although the laboratory exercise challenge may be adequate to demonstrate EIB in recreational athletes,³³ it results in a high number of false-negative test results in elite athletes. In 1 series the false-negative rate for laboratory exercise challenge was 78%.⁴⁷ Standardizing field free-run challenges and sport-specific challenges is problematic. Pharmacologic bronchoprovocation results in elevated rates of positive challenge results in individuals with conditions other than asthma. As a result EVH has evolved as a preferred surrogate method to identify EIB.⁵² The standardized EVH protocol of inhalation of a fixed ratio dry gas (5% carbon dioxide with the balance air) at 30 times FEV₁ for 6 minutes was developed by Argyros et al. It has a higher sensitivity and specificity than both methacholine and field exercise challenge.^{48,55} Holzer et al⁴⁸ found that the symptoms with the greatest positive predictive value for a positive EVH result were wheezing without a cold, nocturnal chest tightness causing awakening, and exercise-associated dyspnea. However, he noted that these symptoms exhibited only moderate sensitivity and suggested that neither symptoms nor methacholine challenge should be used exclusively to diagnose EIB. Rundell et al⁵⁵ noted that although EVH was better at identifying EIB than a field exercise challenge for both summer and winter athletes, it is possible that EVH could overdiagnose EIB in recreational athletes because of the higher ventilation rate than would normally be achieved during exercise in this population. Other challenges that have been suggested as surrogates for exercise in the diagnosis of EIB include hypertonic aerosols, such as 4.5% saline and dry powder mannitol. These agents are used to mimic the airway dehydration due to water loss seen with hyperventilation, although mannitol is not yet Food and Drug Administration approved for this purpose.^{51,52}

Treatment

In the athlete as in the nonathlete the focus of therapy is on obtaining control of underlying inflammation and providing relief from symptoms. Initial management is based on current national or international asthma treatment guidelines.⁵³ As with the treatment of asthma in nonathletes, inhaled corticosteroids (ICSs) are the most effective class of medications for both long-term asthma control and prevention of EIB.⁵³ Nevertheless, it has been demonstrated that ICSs do not result in improvement of airway inflammation, symptoms, or bronchial responsiveness in all athletes.⁵⁶ Both short- and long-acting inhaled β_2 -agonists have successfully been used to treat and prevent EIB. However, potentially significant safety concerns exist regarding long-acting β_2 -agonists and the development of tolerance.^{57,58} Studies have demonstrated that regular use of short- and long-acting inhaled β_2 -agonists results in a loss of the bronchoprotective effect against EIB.^{57,58} Furthermore, the loss of bronchoprotective effect occurs even with the use of ICSs.⁵⁸ As a result, most experts now recommend against the frequent use of inhaled β_2 -agonists, instead supporting the use of an ICS to treat underlying uncontrolled asthma if present or alternate agents.^{24,53}

Although β_2 -agonists are currently listed as prohibited by the World Anti-Doping Agency and International Olympic Committee,⁵⁹ multiple studies have demonstrated no effect of either short- or long-acting inhaled β_2 -agonists on maximal oxygen use, anaerobic threshold, strength performance, blood lactate value, perceived exertion, or psychomotor performance in asthmatic or healthy athletes.⁶⁰ Leukotriene receptor antagonists have been demonstrated to provide protection against EIB without issues of tolerance arising.^{24,61} In addition, cromolyn sodium and nedocromil have been shown to be effective in reducing EIB when used before exercise.^{33,62} The cromoglycates are no longer available in the United States in metered-dose inhaler form as of late 2009. Omalizumab has demonstrated efficacy in the treatment of asthma.⁴ However, there have been no specific studies of omalizumab in athletes, and the high cost of this therapy may be a limiting factor.

Nonpharmacologic measures that have shown benefit include a warm-up before exercise, which has been shown to decrease EIB,⁶³ environmental control measures to allow avoidance of irritant or allergic triggers when feasible, and limiting dietary salt intake, which has been demonstrated to decrease the severity of EIB and reduce the number of sputum inflammatory cells and mediators in a population with underlying asthma.⁶⁴

CONCLUSION

There have been great advances in our understanding of the effects of atopy, environmental conditions, aeroallergens, irritant gases such as chlorine, and pollutants on the airways of athletes. However, uncertainty remains regarding the relative contributions of these factors in explaining the high degree of bronchospasm seen in various populations of athletes with and without asthma. In addition, although there is no consensus regarding the most appropriate diagnostic and therapeutic modalities, the EIB practice parameters are expected to be published soon. There is great variability in the presentation, laboratory findings, diagnostic maneuvers, and response to therapeutic measures among populations of athletes in different sports and among individuals. However, an improved understanding of the unique exposures faced by athletes in different disciplines, of the available tests for pursuing the appropriate diagnosis, and of the available therapies will allow the allergist to provide clinical improvement and allow the athlete to find relief and achieve his/her full potential.

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