Allergy and asthma in elite summer sport athletes

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Exercise may increase ventilation up to 200 L/min for short periods of time in speed and power athletes, and for longer periods in endurance athletes, such as long-distance runners and swimmers. Therefore highly trained athletes are repeatedly and strongly exposed to cold air during winter training and to many pollen allergens in spring and summer. Competitive swimmers inhale and microaspirate large amounts of air that floats above the water surface, which means exposure to chlorine derivatives from swimming pool disinfectants. In the summer Olympic Games, 4% to 15% of the athletes showed evidence of asthma or used antiasthmatic medication. Asthma is most commonly found in endurance events, such as cycling, swimming, or long-distance running. The risk of asthma is especially increased among competitive swimmers, of which 36% to 79% show bronchial hyperresponsiveness to methacholine or histamine. The risk of asthma is closely associated with atopy and its severity among athletes. A few studies have investigated occurrence of exercise-induced bronchospasm among highly trained athletes. The occurrences of exercise-induced bronchospasm vary from 3% to 35% and depend on testing environment, type of exercise used, and athlete population tested. Mild eosinophilic airway inflammation has been shown to affect elite swimmers and cross-country skiers. This eosinophilic inflammation correlates with clinical parameters (ie, exercise-induced bronchial symptoms and bronchial hyperresponsiveness). Athletes commonly use antiasthmatic medication to treat their exercise-induced bronchial symptoms. However, controlled studies on their long-term effects on bronchial hyperresponsiveness and airway inflammation in the athletes are lacking. Follow-up studies on asthma in athletes are also lacking. What will happen to bronchial hyperresponsiveness and airway inflammation after discontinuation of competitional career is unclear. In the future, follow-up studies on bronchial responsiveness and airway inflammation, as well as controlled studies on both short- and long-term effects of antiasthmatic drugs in the athletes are needed. (J Allergy Clin Immunol 2000;106:444-52.)

Key words: *Asthma, athletes, bronchial hyperresponsiveness, chlorine derivatives, maximal exercise capacity, sports medicine, summer events, swimming*

Abbreviations used
DSCG: Disodium cromoglycate
ECT: Exercise challenge test
EIB: Exercise-induced bronchospasm

Team physicians became interested in asthma in elite athletes after the 1972 Olympic Games in Munich. The winner of the men's 400 m freestyle swimming race was disqualified because a significant amount of a banned drug, ephedrine, was found in his postrace urinalysis sample. The swimmer had used a combination preparation of theophylline and ephedrine for the treatment of his asthma. Since then both US and Australian Olympic Committees have put special emphasis on the accurate diagnosis and treatment of asthma in their Olympic teams.[1,2]

The first athlete group whose bronchial responsiveness was assessed were American football players who frequently complained of exercise-induced respiratory symptoms.[3] Half of the 151 football players showed increased bronchial responsiveness in a methacholine challenge test, but 41% of the medical students serving as controls were also hyperresponsive to methacholine. A significant association was found between methacholine hyperresponsiveness and asthma symptoms, although only 30% of the 151 players reported at least 1 exercise-induced chest symptom.

TRAINING ENVIRONMENT OF ELITE ATHLETES

Exercise may increase ventilation up to 200 L/min for short periods of time in speed and power athletes and for longer periods in endurance athletes, such as long-distance runners and swimmers. During winter in countries with 4 distinct seasons, athletes train outdoors in cold weather or indoors where air quality may be poor.[4] Inhalation of cold air during exercise aggravates exercise-induced asthma in people with asthma.[5] Exercise alone increases bronchial responsiveness to methacholine in patients with asthma.[6] Inhalation of cold air during rest somewhat increases bronchial responsiveness to histamine in subjects with and without asthma.[7] Inhalation of cold air during light exercise induces an increased number of granulocytes and macrophages in the lower airways of healthy subjects when compared

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with exercise in normal temperature.[8] No data are available concerning air quality in indoor sports halls. However, athletes report more exercise-induced respiratory symptoms during indoor training than during outdoor training at subzero temperatures.[4]

Track and field athletes and, to some extent, swimmers are extensively exposed to many pollen allergens in spring and summer.[4] When the ventilation level exceeds about 30 L/min there is a shift from nose breathing to combined mouth and nasal breathing.[9] This shifting results in a greater deposition of airborne allergens and other inhaled particles to the lower airways; in addition, incompletely conditioned air may reach the mucous membranes of the lower airways.[10] Exercise-induced bronchospasm (EIB) increases significantly during the pollen season in patients with asthma who are allergic to pollen, but not in patients who are allergic to pollen but do not have asthma.[11] The number of eosinophils and the amount of eosinophil cationic protein in bronchoalveolar lavage fluid increased in patients with asthma who were sensitive to pollen from preseason to pollen season.[12] Thus symptoms of asthma may develop in athletes who are repeatedly and strongly exposed to pollen allergens.

When swimming up to 30 hours per week, competitive swimmers inhale large amounts of air that floats just above the water surface. They may also microaspirate water in the trachea and bronchi (Fig 1).[13] Swimming pools are disinfected with either chlorine gas or hypochlorite liquid.[13,14] Chlorination is used also in smaller amounts if ozonation of water is the main disinfecting method. Exposure to chlorine gas and its derivatives can produce inflammation and edema of the mucous membranes.[14] At the pH found in pool water (7.2 to 7.8), chlorine is completely hydrolyzed to hypochlorous acid (HClO) or hypochlorite ion (OCl^-). Other compounds known to be present are free chlorine, chloramines, chloroform, formaldehyde, acetaldehyde, and a number of halogenated hydrocarbons.[13-16] Most of these compounds are highly volatile and readily come out of the solution in the form of an aerosol or a gas. Thus competitive swimmers are exposed to high quantities of these chemicals. Previously it has been shown that the concentration of chloroform in alveolar air and serum varies directly with that found in the swimming pool water, the number of swimmers in the pool, the length of time spent in the pool, and the intensity of training.[16] Drobnik et al[15] measured chlorine gas 10 cm above the surface of water from 4 swimming pools in Spain. They found a mean chlorine gas concentration of 0.42 mg/m^3 of air and calculated that during a 2-hour training period, the swimmer may be exposed to an amount of chlorine (4 g to 6 g) that exceeds the US recommendation for a worker with 8-hour exposure.[15] Repeated, strong, or accidental exposure to chlorine gas may cause asthma symptoms, airflow obstruction, and increased bronchial responsiveness to methacholine without a latency period.[17] In these cases, the histologic analysis of the bronchial mucosa has shown a thickened basement membrane, eosinophilic inflammation, and a relative lack of T-lymphocytes.[17]

PREVALENCE OF ASTHMA AND ALLERGY IN SUMMER SPORT EVENTS

Asthma and allergy in Olympic Games

In the 1976 and 1980 Olympic Games, 9.7% and 8.5% of the Australian Olympic athletes reported asthma in a physical examination (Table I).[2] Most of the athletes with asthma were swimmers. Allergy was reported by 10% of the athletes in both Olympic Games. In 1984, 26 (4.3%) of the 597 US Olympic team athletes had physician-diagnosed asthma, 41 (6.9%) used bronchodilator medication for symptoms compatible with EIB, and 42 (7.0%) had a history of exercise-induced bronchial symptoms.[1] These 67 athletes won 41 Olympic medals in the Los Angeles Olympic Games. In the 1992 Spanish Olympic team, 4.4% of the athletes reported asthma.[19] Recently, Weiler et al[22] reported that 107 (15.3%) of the 699 US Olympic team athletes in 1996 Summer Olympic Games had a previous diagnosis of asthma, and 97 (13.9%) had used antiasthmatic medication. Interestingly, they found that the occurrence of asthma was highest in athletes with endurance sports, such as cycling, swimming, or rowing.

Asthma and hay fever in competitive swimmers

A questionnaire survey of 738 competitive swimmers, including 165 international level swimmers, showed a high prevalence of asthma, exercise-induced respiratory symptoms, and allergies to dust (Table I).[14] The prevalence of asthma was highest in the group of the international level swimmers (21%). Additionally, hay fever was reported by 19% and use of an inhaled β_2-agonist by 9% in that group. According to a logistic regression analysis, the most important risk factors for asthma were increasing age and the amount of training in a swimming pool. A large proportion of the swimmers complained of irritant-induced symptoms, such as sore eyes, headache, and sore throat, when swimming in pools. Both the respiratory and toxic symptoms ameliorated when swimming was not performed for several days.

A few studies have investigated occurrence of increased bronchial responsiveness among swimmers. Zwick et al[23] studied the occurrence of atopy and bronchial responsiveness to methacholine in a group of 14 competitive swimmers and compared their results with those of age- and sex-matched control subjects. Atopy was observed in 9 of 14 swimmers in skin prick tests. The swimmers were significantly more hyperresponsive (11 of 14) than the control subjects (5 of 14) in a methacholine challenge test. In another study, bronchial hyperresponsiveness to methacholine was found in 21 of 35 competitive swimmers, with no difference between the symptomatic and the symptom-free swimmers; however, while only 2 of 17 nonswimming athletes showed increased bronchial responsiveness to methacholine.[14] No risk factors for bronchial respon-

FIG 1. During training and competition, highly trained swimmers inhale large amounts of air that floats just above the water surface. Possibly they also microaspirate water in the trachea and bronchi. Therefore they are repeatedly and strongly exposed to disinfecting agents (eg, chlorine derivatives). (Courtesy of Finnish Swimming Association).

TABLE I. Prevalence of asthma among highly-trained summer sports athletes.

Study year, athlete group	Method	Prevalence of asthma (%)	Reference No.
1976 Australian Olympic team (N = 185)	Physical examination	9.7	2
1980 Australian Olympic team (N = 106)	Physical examination	8.5	2
1984 US Olympic team (N = 597)	Questionnaire, treadmill exercise test in selected athletes	4.3	1
1986 Football players from Unversity of Iowa (N = 156)	Questionnaire, methacholine challenge	11.5	3
1986 Swiss athletes from various sport events (N = 2,060)	Questionnaire	3.7	18
1992 Spain Olympic team (N = 495)	Questionnaire	4.4	19
1993 US Track and Field Championship games (N = 73)	Exercise test (competition)	15.1	20
1994 Runners from Finnish national teams (N = 103)	Questionnaire	15.5	4
1995 Swimmers from United States (N = 738)	Questionnaire	13.4	14
1996 Track and field athletes, swimmers (N = 162)	Questionnaire, spirometry, histamine challenge	22.8	21
1996 US Olympic Team (N = 699)	Questionnaire	15.3	22

siveness were identified in either of the studies.[14,23] A study of young Norwegian swimmers suggested that a swim of 1000 m below, at, and over anaerobic threshold decreased provocative concentration causing a decrease of 20% in FEV_1 values in swimmers both with and without asthma.[24]

We studied 162 highly trained athletes (42 swimmers, 49 speed and power athletes, 71 long-distance runners) and 45 control subjects by use of respiratory symptom questionnaires, skin prick testing, a resting flow-volume spirometry, and a histamine challenge test.[21] Of the swimmers, 21 (50%) had atopy (at least 1 skin prick test reaction), and 15 (36%) showed increased bronchial responsiveness to histamine. Current asthma (increased bronchial responsiveness to histamine and exercise-induced bronchial symptoms monthly during the last year) was found in 11 (26%) swimmers, and total asthma (current asthma or asthma diagnosed previously by a physician) in 12 swimmers (29%). Hay fever (positive skin test reaction to pollen associated with symptoms of rhinoconjunctivitis during spring or summer) was observed in 12 swimmers (29%). The adjusted risk of total asthma in swimmers was nearly 6-fold as compared with control subjects. Atopy was significantly associated with increased bronchial responsiveness to histamine, as well as with current and total asthma in the multivariate analysis. When the 2 risk factors atopy and swimming were combined in the multivariate analysis, the risk of asthma in a swimmer with atopy was 96-fold as compared with a control subject without atopy.

Asthma and asthmalike symptoms in track and field athletes

When 103 runners from Finnish national teams replied to a respiratory symptom questionnaire, 16 (16%) reported having asthma documented by a physician.[4] Twenty-four (28%) of the remaining 87 runners reported allergies. A much larger proportion of the athletes (53 of 103; 51%) reported having exercise-induced, asthma-like symptoms at least monthly. The asthmalike symptoms were significantly associated with allergies. Training indoors caused respiratory symptoms more often than training in cold, dry weather or during the pollen season.

The occurrences of atopy, increased bronchial responsiveness, and asthma were studied in a group of 120 track and field athletes (71 long-distance runners and 49 speed and power athletes) and 45 control subjects.[21] Atopy was found in 35 (49%) of long-distance runners and 21 (43%) speed and power athletes, as compared with 16 (36%) of the control subjects. Increased bronchial responsiveness to histamine occurred in 6 (9%) long-distance runners and in 9 (18%) speed and power athletes. Current asthma (increased bronchial responsiveness to histamine and exercise-induced bronchial symptoms monthly during the last year) was found in 5 (7%) long-distance runners and in 6 (12%) speed and power athletes, and total asthma (current asthma or asthma diagnosed previously by a physician) was found in 17 (24%) long-distance runners and in 8 (16%) speed and power athletes. The adjusted risk of total asthma was 6-fold in long-distance runners and 3.6-fold in speed and power athletes, as compared with control subjects. The risk of asthma was significantly associated with atopy and increased significantly when the number of positive reactions increased in the skin prick tests. The adjusted risk of hay fever was 3.2-fold in long-distance runners and 1.5 in speed and power athletes, as compared with control subjects.

Asthma and allergic rhinitis in former elite athletes

Kujala et al[25] studied 1282 former Finnish elite athletes (including 205 long-distance runners or cross-country skiers) who represented Finland between 1920-1965 at least once in international competitions, and 777 control subjects who were matched according to age, sex, and area of residence. Of the athletes, 2.4% (30 of 1,282) and 3.5% (27 of 777) of the control subjects reported physician-diagnosed asthma. The occurrence of asthma was 2.9% in long-distance runners and cross-country skiers. Pulmonary diseases were associated with age, smoking, occupation, and a history of exposure to chemicals. The prevalence of allergic rhinitis was 11% in both former athletes and control subjects. The apparent discrepancy between these results in former athletes and athletes of today may be explained in many ways. The prevalence of asthma has increased 8-fold within the last 30 years in Finland,[26] training volumes have increased, records have improved, training environment has changed (air pollution, urbanization, indoor and swimming pool training), and diagnostic approaches to asthma have improved.

EXERCISE CHALLENGE TESTS IN ELITE ATHLETES

Although many studies have shown that increased bronchial responsiveness to nonspecific stimuli is common in elite athletes, few studies have investigated the occurrence of EIB. It is a unique or pathognomonic feature of asthma unlike methacholine or histamine hyperreactivity.[27] The interpretation of the exercise challenge test (ECT) result in athletes with respect to postexercise lung function is unclear because only a few studies have investigated normal exercise responses in athletes.

The inhalation of cold air during exercise aggravates EIB in subjects with asthma,[5] whereas in normal subjects exercise causes no effects on lung function even at low temperatures.[28] Eucapnic hyperventilation in the levels that exceed those seen with exhausting exercise in nonathletic subjects, however, can cause a decrease in postchallenge lung function even in subjects without asthma.[29] In older age groups, cold-induced facial cutaneous reflex mechanisms may also affect lung function during exercise challenges.[30] Therefore a whole-body or naturally occurring cold air exposure is necessary to evaluate lung function during and after an ECT. Walking at a low exercise level (70% of the maximal heart rate) combined with a whole-body exposure to cold air did not affect lung function in healthy young students.[31] A positive effect on postexercise lung function was observed in 8 athletes without asthma on a high-intensity treadmill exercise test combining the inhalation of cold air and a whole-body exposure to cold wind.[32]

When 32 elite runners without asthma (main events from 400 m to marathon) performed a heavy ECT outdoors and without warm-up at subzero temperatures, 8 (25%) showed EIB (defined as mean –2 SD of the postexercise change of FEV_1 in runners without atopy).[33] In runners without atopy or symptoms, postexercise changes in FEV_1 were small, but in runners with atopy but no symptoms, significant reductions in postexercise lung function were observed. In a larger group of 58 runners, EIB was shown to correlate significantly with atopic disposition and its severity in athletes.[34] Additionally, seasonal variability was shown to affect the occurrence of EIB in athletes, and therefore exercise should be carried out in both cold winter air and the pollen season to detect EIB in elite runners.

Paul et al[35] measured peak expiratory flow in 10 adult speed skaters during a 1,500 m World Cup speed skating race. Significant decreases in peak expiratory flow were found at 10, 90, and 120 minutes after the race. The mean changes ranged from –3% at 10 minutes to –8% at 90 minutes. Mannix et al[36] measured lung function combined with a simulated long program (4 to 5 minutes, rink-side temperature 7°C to 10°C [39°F to 42°F]) in 124 professionally coached figure skaters. Thirty-five percent of the figure skaters had a 10% or greater decrease in postexercise FEV_1, which indicates EIB.

Recently, Mannix et al[37] compared ECT on ice with eucapnic hyperventilation test in figure skaters to identify EIB. They observed EIB in 16 out of 29 skaters, of whom

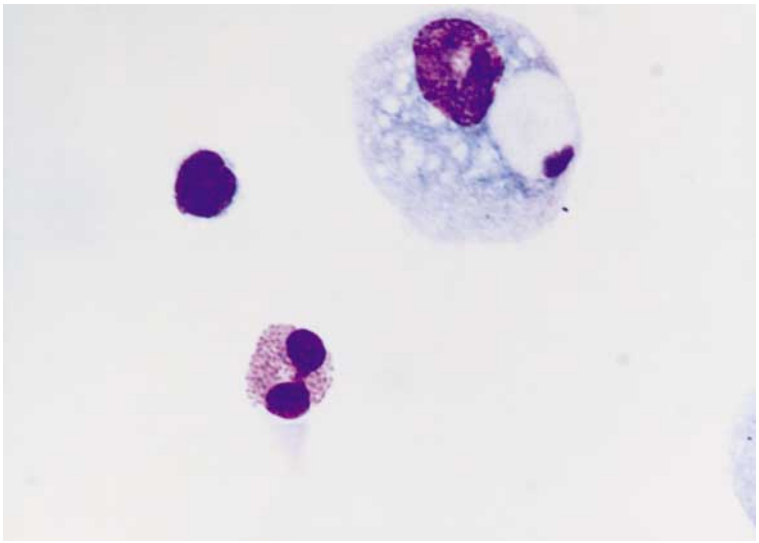

FIG 2. Eosinophilic airway inflammation (differential cell count of eosinophils 4.3%) in an induced sputum sample from a 20-year-old, national team, female swimmer.[38] She reported exercise-induced cough, wheeze, and breathlessness repeatedly in connection to swimming pool training. Additionally, she showed moderate bronchial hyperresponsiveness in a histamine challenge test (provocative dose causing a 15% decrease in FEV_1 = 0.36 mg).

9 were positive on ice testing and 12 by hyperventilation testing. Thus ice testing missed 7 skaters and hyperventilation test missed 4 skaters with EIB. The authors suggest that evaluation for EIB in athletes should include both tests to increase sensitivity.

AIRWAY INFLAMMATION

Airway inflammation in athletes has been studied only in swimmers and cross-country skiers. Twenty-nine elite swimmers and 19 healthy, symptom-free control subjects gave induced sputum samples to investigate airway inflammation.[38] Of these, 14 (48%) swimmers and 3 (16%) control subjects showed increased bronchial responsiveness to histamine. Sputum from swimmers showed significantly higher differential cell counts of eosinophils and neutrophils compared with control subjects. Sputum eosinophilia (sputum differential eosinophil count of >4%) was observed in 6 (21%) swimmers and in none of the control subjects. Sputum from swimmers contained also a significantly higher mean concentration of eosinophil peroxidase and human neutrophil lipocaline than sputum from control persons.

Airway inflammation was shown to correlate significantly with clinical data. The swimmers with exercise-induced bronchial symptoms had significantly higher sputum eosinophil cell counts (mean 7.6%) than the symptom-free swimmers (mean 0.7%). Those 6 swimmers with sputum eosinophilia showed significantly greater bronchial hyperresponsiveness to histamine than swimmers without eosinophilia (Fig 2).

Airway inflammation has been studied also in cross-country skiers by use of endobronchial biopsies. Karjalainen et al[39] studied 40 young elite skiers and 12 healthy controls. Bronchial hyperresponsiveness to methacholine was found in 30 of the skiers, and one third had asthmalike symptoms. They observed that T-lymphocytes, macrophages, and activated eosinophils were more abundant in skiers than in control subjects. No correlation was observed between the degree of bronchial hyperresponsiveness to methacholine and inflammatory cell counts, and atopy did not have any influence on airway inflammatory cell counts. In bronchoalveolar lavage fluid, skiers had greater total cell, lymphocyte, and macrophage counts than control subjects.[40] No differences were observed in eosinophil or neutrophil cell counts. Sue-Chu et al[41] investigated occurrence of lymphoid aggregates (a folliclelike cluster of more than 50 cells) through use of endobronchial biopsies in 44 skiers and 12 controls. Lymphoid aggregates were found in 28 (64%) skiers and in 3 (25%) control subjects. The aggregates were more frequent in skiers who were using β_2-agonists and who had bronchial hyperresponsiveness, whereas they were not associated with a history of respiratory allergy or asthmalike symptoms.

Why do exercise-induced bronchial symptoms and signs of airway inflammation develop in healthy swimmers? Irritant-induced asthma is a variant of occupational asthma, and it occurs in subjects who are acutely or repeatedly exposed to high concentrations of an irritant product, commonly a gas.[17] Respiratory symptoms develop in the patients without a latency period.[17] Asthma symptoms, airflow obstruction, and increased bronchial responsiveness can arise as a result of repeated exposure to chlorine gas.[17] In these cases, histologic analysis of the bronchial mucosa has shown a thickened basement membrane, eosinophilic inflammation, and a relative lack of T-lymphocytes.[17] The much increased

TABLE II. Use of allergy and asthma medication among Finnish summer sports elite athletes*

Medication	Speed and power athletes (N = 106)	Long-distance runners (N = 107)	Swimmers (N = 42)
Asthma medication	11 (10.4%)	23 (21.5%)	9 (21.4%)
Inhaled corticosteroid	5 (4.8%)	6 (5.6%)	6 (14.3%)
Inhaled β_2-agonist	10 (9.4%)	17 (15.9%)	6 (14.3%)
Nedocromil sodium	0 (0%)	1 (0.9%)	2 (4.8%)
Disodium cromoglycate	0 (0%)	3 (2.8%)	1 (2.4%)
Ipratropium bromide	0 (0%)	1 (0.9%)	1 (2.4%)
Antihistamine	3 (2.8%)	5 (4.7%)	6 (14.3%)

Adapted from Helenius.[13]

ventilation during training and competition results in long-term and repeated exposure to chlorine derivatives from swimming pool disinfectants in elite swimmers.[13-16] The eosinophilic and neutrophilic inflammation observed in sputum from the elite swimmers could represent a form of irritant-induced asthma.

CHARACTERISTICS OF ASTHMA IN SUMMER AND WINTER SPORT ATHLETES

Athletes with summer events are intensively exposed to airborne allergens during training and competitions, whereas winter sport athletes are intensively exposed to cold air. Swimmers are exposed to chlorine compounds. In skiers[39,42,43] the association between atopy, respiratory allergy, and asthma is not as clear as in summer sport athletes.[21,34] Hay fever and atopy are more common in long-distance runners as compared with control subjects,[21] whereas the occurrence of atopy in skiers is similar as in control subjects.[42] Both summer sport athletes (swimmers) and winter sport athletes (cross-country skiers) show evidence of mild eosinophilic airway inflammation,[38-40] although the exercise symptoms are mostly mild.

The lack of correlation between respiratory allergies and asthmalike symptoms in skiers suggests that the mechanism of asthma may be mostly nonallergic (cold air exposure). The strong correlation between atopy, hay fever, and asthma in summer sport athletes suggests that allergen exposure and sensitization are strong predisposing factors to asthmatic symptoms in this group. In swimmers the chlorine exposure may enhance the allergic mechanisms.

MEDICAL AND NONMEDICAL TREATMENT OF ASTHMA IN ELITE ATHLETES

Highly trained athletes commonly use antiasthmatic medication to treat their exercise-induced bronchial symptoms. In a recent study, 17% of 253 Finnish elite summer sports athletes used antiasthmatic medication[13] (Table II). The most commonly used medication was inhaled β_2-agonist. Despite the common use of antiasthmatic medication among athletes, controlled studies on their long-term effects on bronchial responsiveness or airway inflammation in the athletes are lacking. Obviously such studies in athletes are needed.

Physical warm-up has reduced EIB significantly.[44] Continuous, low-intensity warm-up is more effective than interval warm-up in preventing EIB in athletes.[44] Cross-country skiers are accustomed to wearing breathing filters for protection against cold air.[45] Koskela[30] found that facial cooling during cold air challenge induces a cutaneous reflex mechanism, which results in marked bronchoconstriction in patients with asthma and chronic obstructive pulmonary disease. Therefore clothing worn over the whole face might protect athletes with asthma against cold air and wind. Ongoing viral infections have been shown to increase bronchial responsiveness more in exercising athletes than in sedentary controls.[46] Thus endurance sports should not be undertaken during ongoing viral respiratory tract infections.

The Committee on Sports Medicine and Fitness of the American Academy of Pediatrics has recommended careful baseline control of asthma and emphasized treatment of airway inflammation in young athletes with EIB.[47] Additionally, athletes with asthma should use either inhaled β_2-agonist or cromolyn before sports activities.[47] Eosinophilic airway inflammation is found even in patients who are newly diagnosed with mild asthma.[48] Inhaled steroids are also the first-line treatment in these patients, suppressing the inflammation and normalizing bronchial responsiveness.[49]

Mild eosinophilic airway inflammation affects athletes with exercise-induced bronchial symptoms.[38,39] Treatment of asthma symptoms should therefore be started with inhaled corticosteroids, even though direct evidence of their effect on airway inflammation in athletes is lacking. Inhaled steroids have no immediate effects on EIB, but regular use has been shown to reduce EIB in children with asthma.[45] Papalia[50] reported that 1 week after starting inhaled steroid (budesonide), well-trained athletes with asthma showed marked reduction in EIB. Orally active leukotriene antagonists are recent developments in the treatment of asthma. Both montelukast and zafirlukast have immediate protective effect on EIB.[51] Montelukast has also reduced eosinophilic inflammation in asthma.[52] Thus leukotriene antagonists are theoretically useful for athletes with asthma, although clinical studies on their effects in elite athletes are lacking.

β_2-Agonists, disodium cromoglycate (DSCG), and nedocromil sodium have been shown to be effective against EIB.[45] Regular long-term use of β_2-agonists

TABLE III. International Olympic Committee list of permitted and prohibited asthma and allergy medications, 1999

Freely allowed to use
Disodium cromoglycate
Nedocromil sodium
Leukotriene antagonists
Ipratropium bromide
Systemic theophylline
Antihistamines
Permitted by notification
Inhaled salbutamol, terbutaline, salmeterol
Declaration required in Olympic and championship games
Inhaled corticosteroids
Prohibited
Systemic corticosteroids
Other inhaled β_2-agonists than above
Systemic β_2-agonists
Inhaled or systemic epinephrine (allowed when combined with local anaesthetic)

Adapted from the Medical Commission of the International Olympic Committee.[62]

without anti-inflammatory treatment may increase airway responsiveness to nonallergic stimuli and enhance allergen-induced late bronchoconstriction, as well as airway inflammation.[53] As summer sports athletes are repeatedly and strongly exposed to various airborne allergens, the regular use of these agents without anti-inflammatory treatment may be harmful.

Nedocromil sodium protects against EIB exacerbated by cold air.[54] Combination of β_2-agonist with nedocromil or DSCG may provide additional efficacy if either alone proves unsatisfactory.[55] DSCG and β_2-agonist inhalation reduced ventilation and energy consumption during running in children with asthma.[56] In highly trained athletes with asthma, salbutamol pretreatment decreased maximal heart rate in ECT compared with placebo.[57] This suggests that EIB may significantly affect the maximal exercise performance of highly trained athletes. If athletes with asthma are carefully evaluated and treated, they may acquire equal performance level as participants without asthma.

Hay fever allergy is common in summer sports athletes.[21] Topical nasal treatment with corticosteroids has decreased not only nasal but also asthma symptoms[58] and should be prescribed for athletes with hay fever allergies. Oral antihistamines have effectively reduced symptoms of seasonal and perennial allergic rhinitis.[59] Terfenadine has also protected against EIB.[60] External nasal dilators have recently been introduced among athletes. In a placebo-controlled trial, they decreased submaximal exercise perceived exertion, heart rate, ventilation, and volume of oxygen utilization in athletes.[61]

DOPING ASPECTS

The Medical Commission of the International Olympic Committee has listed the banned drugs (Table III).[62] It should be noted that of inhaled β_2-agonists, only salbutamol (albuterol), salmeterol, and terbutaline are permitted for use. When they are needed, a written notification of asthma by a respiratory or team physician is necessary for the relevant medical authority. Use of inhaled steroids should be declared to the relevant medical authority in Olympic and world championship games. The use of ipratropium bromide, DSCG, nedocromil, leukotriene antagonists, and oral theophylline is not restricted by the International Olympic Committee. There are no data that the permitted inhaled antiasthmatic drugs would affect maximal exercise capacity in healthy athletes.[63]

CONCLUSIONS

EIB affects maximal exercise capacity of the athletes by increasing ventilatory cost and decreasing maximal ventilatory capacity. Increased bronchial responsiveness and airway inflammation may predispose athletes to upper respiratory tract infections. Viral respiratory tract infections increase bronchial responsiveness more in exercising athletes than in sedentary controls.[46] The variable nature of asthma symptoms can partly explain the day-to-day variability in performance capacity of some elite athletes. Thus all highly trained athletes should be screened for the possibility of asthma, as well as for the main risk factor, atopy. If asthma has been shown, anti-inflammatory treatment should be started with inhaled steroids because the airway inflammatory reaction commonly shown in athletes with asthma. Follow-up studies on athlete's asthma are lacking. What will happen to increased bronchial responsiveness and airway inflammatory findings after discontinuation of competitional career, is unclear. In the future, follow-up studies on bronchial responsiveness and airway inflammation as well as controlled studies on both short- and long-term effects of antiasthmatic drugs in the athletes are needed.

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