CHAPTER 5

The role of the environment and climate in relation to outdoor and indoor sports

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Outdoor and indoor environment

The number of hours spent in training at high intensity levels by athletes is progressively increasing. At the same time, a growing number of recreational athletes are more concerned about the intensity of their training schedules. Physical exercise imposes a certain stress on the respiratory tract as it has to eliminate carbon dioxide and to supply the muscles with oxygen for energy production. This process requires increased ventilation of the airways that may go as high as 10–15 times above the normal resting frequency. This, for the asthma patient, represents the most important stimulus for the onset of exercise-induced asthma (EIA).

Increased ventilation rate and oral breathing displaces pulmonary uptake of pollutants to more distal sites in the lung, thereby further increasing the effect of ambient exposure of pollutant deposition in the distal airways. Without doubt, this alteration will affect the athlete who suffers from asthma or bronchial hypersensitivity. Cross-sectional studies have demonstrated that competitive athletes have a high prevalence of asthma and EIA or bronchial hyperreactivity. Mechanisms for this association include increased inhalation of cold air and allergens, increased response to respiratory infections and air pollutants, and increased parasympatethic tone [1–8]. The purpose of this chapter is to provide a review of the outdoor and indoor environmental factors that can affect the airways of the asthmatic athlete.

Cold and dry air

It is assumed that the amount of drying of the lining of the airway wall during exercise is the main factor in the early reaction, modulated only by the inspired air temperature [9]. The stimulus by which hyperpnoea induces an attack of asthma is a loss of water from the respiratory mucus [10, 11]. The mechanism by which evaporative water loss induces an attack of asthma is thought to result from an increase in the osmolarity of the airway surface liquid of the respiratory tract due to a concentration of ions [12]. This provokes the EIA acute phase, the mechanism being the osmolarity change due to the dry air, and this is more important than heat loss for determining EIA [13]. This may be obvious in the asthmatic athlete, but in the healthy athlete the combined effect of the drying and temperature stimuli to the airways, stimulating vagal nerve activity, can gradually cause increasing minor bronchoconstriction [14]. This situation was also

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evaluated in animals practicing sports, such as Alaskan sled dogs [15, 16], showing that 81% have abnormal accumulation of intraluminal debris, and 46% classified as moderate to severe, indicating significant accumulation of exudate. In contrast, in cold environments, the hotter it is when asthmatic patients stop exercising the more intense the bronchoconstriction [17]. Hence, the importance to the athlete of "cooling down" slowly after ending an exercise session, which allows the airways to recover the initial temperature progressively. This is the reason for dyspnoea in sensitive individuals when entering a hot environment following moderately intense exercise in cold surroundings.

Air pollutants

There are a large number of chemical compounds present in the polluted atmosphere that, alone or in combination, are important to consider for their potential effect on the respiratory system and impact on athletic performance [18]. These substances will have a varying impact on the development or worsening of the inflammatory process in the asthmatic athlete's respiratory tract. A number of factors will contribute to it, including: 1) concentration of pollutants; 2) ventilation level; 3) previous state of the respiratory tract; and 4) a combination with other atmospheric factors, such as temperature or humidity. There is an estimated mean increase of 3% in the prevalence of lower respiratory symptoms with each 10 $\mu g \cdot m^{-3}$ increase in the daily mean concentrations of airborne particulate matter. Furthermore, it is estimated that there is a 0.7% increase in the prevalence of upper respiratory symptoms for each 10 $\mu g \cdot m^{-3}$ increase in airborne particulate matter <1 μm [19].

Polluting agents can be classified as primary or secondary. The primary agents originate from a source and do not undergo chemical changes. Even if changes take place, these are irrelevant from the clinical point of view, *e.g.* CO, CO_2, Sulphur dioxide (SO_2), nitric oxide, and metals, such as lead, graphite or coal. Secondary agents are produced through chemical reactions from natural precursors or they are emitted by artificial sources, such as ozone (O_3), nitric acid, sulphuric acid, nitrate peroxiacetyl and a great number of inorganic compounds, which can exist in gas or particulate form. The most important source of both types of agents is from products originating from oil combustion in cities and industrial areas.

The most important pollutants, for athletes suffering from asthma, are found in large cities with high rates of contamination. These are industrial cities, with heavy car traffic and atmospheric conditions, which contribute to the contamination.

It seems that the incidence of new diagnoses of asthma is associated with heavy exercise in communities with high concentrations of O_3, thus, air pollution and outdoor exercise contribute to the development of asthma in children [20]. Although no effect of sports on asthma has been seen in communities with high concentrations of pollutants (other than O_3), it is difficult to evaluate the real power of other pollutants alone, in terms of the development of newly diagnosed asthma, or to identify the interaction between sports and other pollutants, other than O_3 [20]. There is even evidence of the effect of nitrogen dioxide (NO_2) in the exacerbation of bronchial asthma in adults [21].

The role of atopy in sports-related asthma is unclear. For example, atopy did not modify the risk of asthma associated with nordic skiing [1], but those children with bronchial hyperresponsiveness and relatively high concentrations of serum Immunoglobulin E are susceptible to air pollution [22].

Ozone. O_3 is a gas that can be produced by an electric or a photochemical reaction of ultraviolet radiation of a certain wavelength. At high concentrations, O_3 can be very

harmful due to its oxidative power. This is the most potent oxidative gas found in atmospheric pollution and is the most significant component of photochemical smog.

Exposure to O_3 provokes nose and throat irritation, shortness of breath, and shallow, rapid breathing, as well as alteration of the breathing function at rest, even at low intensity exercise [23]. Due to the fact that people living in areas with relatively high O_3 levels, including temporarily high levels, present a lower response than those who live in lesser polluted areas, it was suggested that a certain degree of adaptation may occur [24, 25]. After 2 days of O_3 exposure, the time that respiratory function is worst, there are symptoms and respiratory function stability [26]. However, this adaptation disappears 1 week after the exposure or when the O_3 concentration increases [27]. In contrast, although O_3 can affect the lung tissue, the alveolar gas exchange is not affected. Diffusion and transport of oxygen and CO_2 is not altered [28]. The inflammatory and lung function responses to O_3 differ between individuals and are reproducible, but not related to each other, and are similar in healthy or asthmatics subjects. Therefore, these responses appear to represent two independent factors underlying the airway response to O_3 [29].

Athletes, even if well trained and in good health, when exposed to high levels of environmental pollutants and when breathing high volumes of air, as is required when exercising, are at risk for the problems mentioned above. The need for a high ventilation volume contributes to disconnection of the nose as a filtering air barrier. This causes the amount of toxic substances penetrating into the respiratory tract to be higher. In healthy people, airway reactivity is increased after 5 h of exercise, which is equivalent to a day of moderate-to-heavy work or play during exposure to 0.08 ppm of O_3 [30]. O_3 also increases responses to other allergens present in ambient air [31]. In communities with high O_3 concentrations, the relative risk of developing asthma in children playing three or more sports is 3.3-times higher compared with children not playing any sports. Also, the time spent outside was associated with a higher incidence of asthma in those areas with high O_3 [20]. The increased pulmonary dose of ambient O_3 resulting from heavy exercise, combined with exposure to outdoor and indoor allergens, is one possible mechanism for inducing new-onset asthma or for exacerbating existing asthma [20]. Fortunately for asthmatic athletes, response to O_3 is similar to that of healthy adults, and there is no increase in the bronchoconstriction response to subsequent exercise after exposure to O_3 [32]. However, it does increase the response to subsequently encountered allergens, and this is of special importance to those athletes who may encounter both airborne allergens and O_3 during exercise [33].

The vasoconstriction produced by O_3 seems to diminish after administration of topical atropine [34] and a cyclooxygenase inhibitor [35]. However, the efficacy of β-agonists is still unclear [36].

Sulphur dioxide. Coal and oil combustion produce SO_2. SO_2 is a common environmental pollutant produced mainly from oil derivatives and also from the paper, varnish and enamel manufacturing industries and products containing sulphur. It is readily absorbed by epithelial fluid and bronchial mucus forming the acidic compounds involved in inflammation. As a consequence, there is a decrease in the basal pulmonary function and an increased bronchial hyperreactivity [37]. This feature of SO_2 explains why exercise increases the effect of this gas [38]. These physiological effects are responsive to pharmacological intervention with β-agonists, cromolyn and atropine but are unresponsive to theophylline and steroids [39]. Spontaneous recovery has been reported after 30 min of challenge, with a refractory period of up to 4 h. Repeated exposures to a low concentration of SO_2 over a short period can induce tolerance to the bronchomotor effect of SO_2 [40, 41]. However, responsiveness is restored within 6 h.

The response of the asthmatic athlete to SO_2 depends more on the concentration of the

gas than the humidity and temperature [42]. However, previous exposure to cold or dry air also exacerbates the bronchospastic effect of SO_2 in asthmatics [43]. Other symptoms include nasal irritation, conjunctivitis, pharyngitis and an unpleasant sulphur smell. Serious symptoms in nonreactive subjects are rarely produced if the concentrations are not very high.

Nitrogen dioxide. Nitrogen oxide, as a contaminant, is partially oxidised and transformed into NO_2. A precursor of photochemical smog, it is found in ambient outdoor air in urban and industrial regions. NO_2 is formed by oil combustion and from its derivatives, which are used by cars. There are two daily concentration peaks in cities: one early in the morning and the other in the afternoon. Other places where NO_2 is found are smoking areas and areas heated with kerosene and gas stoves. Like O_3, NO_2 produces oxidation of cell membranes of the respiratory airways and free radicals with an inflammatory response [44], but the mechanism by which NO_2 alters airway function is unclear.

In asthmatic subjects this inflammation is evident by an increased bronchial reactivity to stimuli, such as cold or exercise [45], despite the fact that methacholine test results are negative [46]. Moreover, in relation to sport activities, the high prevalence of asthma in competitive figure skaters might be related to NO_2 generated by ice-grooming equipment [6]. However, controlled chamber studies reveal no consistent effect of NO_2 on airway function of either normal subjects or those with asthma [47].

Chlorine. Chlorine is a greenish-yellow gas used in the sterilisation of water supplies and in swimming pools. It is a potent irritant to the mucous membranes, eyes and skin, and its exposure causes pulmonary irritation [48]. Accidental exposures of humans to high concentrations during work and sports activities have been reported [49–51]. There is insufficient evidence to conclude that there is chronic impairment of pulmonary function after acute or chronic exposure in athletes but some observations indicate possible effects of chlorine on the respiratory airway. Swimmers inhale high amounts of chlorine during training and competition throughout their sporting season [52]. The sudden onset of reversible airway obstruction in young swimmers [53], increased sensitisation to aeroallergens [54], high rate of bronchial responsiveness to methacholine in swimmers [55], increased lung epithelium permeability [56] or epithelial integrity [57], or change in the antioxidant status of the respiratory airways [58] suggest that high exposure to chlorinated products in indoor pools might be an important cause in the rising incidence of childhood asthma, allergic diseases or respiratory problems in the athlete.

Smoke from cigarettes. Parental tobacco smoking is associated with lower airway function and an increased incidence of wheezy respiratory illnesses in infants [59]. Increased bronchial reactivity [60], even from prenatal cigarette smoke [61], increases the tendency for upper respiratory infections and induces small airway damage [62], and may even induce exercise-induced narrowing in children [63]. Cigarette smoking produces a 10% increase of the basal metabolic rate and an almost inverse relationship between the levels of carboxyhaemoglobin (COHb) and the capacity to achieve the maximal oxygen consumption, influencing not only the work capacity of the athletes but also the recovery from exercise.

Cannabis. Cannabis was banned by the World Anti-Doping Agency in 1986 and is considered as a positive dope test. Due to this, its consumption by athletes is inexcusable [64]. It is important not to forget that smoking marijuana has some points that enhance

the problems of tobacco in athletes and in asthmatics. Tetrahydrocannabinol (THC) in cannabis has been shown to have a short-term bronchodilator effect [65]. This has lead to suggestions that THC may have therapeutic benefits in asthma. However, the noxious gases, chronic airway irritation or malignancy after long-term use associated with smoking would seem likely to negate these benefits [66]. Other noxious effects of the cannabis smoking habit include: 1) an increase in the fixation of COHb (more than five times that obtained from tobacco); 2) a weakening of the inmune system; 3) increased deposits of tar; and 4) production of greater increases in cellular abnormalities, indicating a cumulative effect of smoking [67, 68]. In contrast, alterations on the psyche and social behaviour of the subjects are no less important [69, 70].

Future success eradicating drug usage in sport will only result from increased efforts directed at the development of strict policies dealing with those athletes who use banned substances, refinement of drug testing procedures and enhancement of athlete education [71, 72].

Aeroallergens. Subjects suffering from allergy to certain aeroallergens will be more exposed if their sport requires them to run long distances, travel frequently to diverse places or exercise during certain times of the year [5, 73]. Moreover, if their sport requires an important respiratory debt, the aeroallergens will perhaps reach the respiratory tract through unusual channels. This will produce a positive skin-prick test result in atopic athletes who will be prone to EIA. With one or two positive responses, the risk is 3.1, and with five or more, the risk is 4.7 [2]. The practice of a sports activity combined with sustained allergenic inhalation, as occurs in diving, is unfortunate and at the same time avoidable [74]. This situation should bring us to reconsider the control mechanisms of this sport and the environment in which it is carried out.

Upper respiratory airway infection. Upper respiratory tract infection (URTI), pollution, exercise and asthma are increasingly being related. Epidemiological studies suggest that intensive training is associated with an increased risk of URTIs, with elite athletes at greater risk than those undertaking more moderate training regimes [75]. The single precipitating factor most frequently associated with acute asthma is respiratory virus infection when compared with aeroallergens [76]. The existence of an association between respiratory infections and exacerbation of asthma has been acknowledged for many centuries, evidenced epidemiologically by case reports [77], time series studies [78], identification of viruses during exacerbations of asthma [79] or following prospective longitudinal studies of individual asthma [80].

The experimental studies performed in allergy and sport has lead to a more general aspect being considered, *i.e.* the effects of exercise on the immune system. In fact, it has been observed that exercise can induce significant and measurable immunological changes involving a transient immune suppression (changes in the number and activity of neutrophils, lymphocytes and macrophages, and the secretion of cytokines) [81]. Atypical bacterial pathogen infections were linked with prolonged asthmatic symptoms. JOHNSTON *et al*. [82] confirmed the high incidence of viral infection in acute exacerbation of asthma, especially enteroviruses or rhinoviruses. Persistent clinical features are more frequently associated with atypical bacterial infections, suggesting these infections should be investigated and treated in cases of persistent asthmatic symptoms [83]. Respiratory tract infection and bronchial responsiveness has been observed in elite athletes and sedentary control subjects, and when it is related to environmental conditions, seasonal variations and training intensity [7, 8]. Sport can be important in the development of URTIs, and not just in elite athletes; for example, baby swimming increases the risk of recurrent respiratory tract infections and otitis media [84]. In fact, *Chlamydia pneumoniae*

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infection has been reported as a possible aetiological agent in asthma, which in primary care settings often appears to be initiated by acute respiratory infections [7, 8]. Serological markers of *C. pneumoniae* infection were associated with acute bronchitis and with asthma (which first became symptomatic following respiratory illness). Serological responses to *C. pneumoniae* may be useful in the classification and diagnosis of asthma [85], mostly in those who have a variant of the mannose-binding lectin alleles, who seem to have a susceptibility to asthma when infected with *C. pneumoniae* [86]. Some attempts to identify any relationship between *C. pneumoniae* and *Mycoplasma pneumoniae* infection in athletes has been made [87], and its impact on incidence of EIA symptoms, but a low correlation was found. [84, 88].

Impact of the sport activity

The prevalence of asthma and bronchial responsiveness is greater amongst elite athletes than in the general population [89]. This prevalence is even higher amongst certain groups of these (*e.g.* long distance runners, cyclists, cross-country skiers and swimmers) than amongst athletes in general. The underlying inflammation in such athletes is quite similar to the pattern seen in asthmatics, involving a predominance of eosinophils, and has been related to the presence of atopy, type of sport, seasonal activity and duration of training [90]. Inflammation can be reversed, at least partially, when an athlete's period of active training comes to an end [91], suggesting that it is at least partly caused by participating in sport (table 1).

Indoor practice

Swimming and other water sports. Water sports are indicated in asthmatics. The adaptation to the effort of heavy swimming in children with asthma is basically the same as that observed in nonasthmatics, with the exception of that related to the bronchial response [92]. There is no satisfactory explanation for the advantage of exercising in water for asthmatic patients. In contrast, an increase in bronchial sensitivity in asthmatic and nonasthmatic swimmers has been reported when compared with a reference population

Table 1. – Recommendations for athletic competition in polluted environments

Organisers of the sport event
Investigation of the sport premises
Evaluation of training areas
Establish the time of day for the sport event on the basis of lowest pollution levels
Establish proper measures to limit pollution during physical activities (factories, cars, *etc.*)
Assessment of the ventilation of sport facilities under standard limits
Assessment of the temperature for outdoor events
Athletes team (technical personnel, athletes)
In close collaboration with the above mentioned points
Modifying the above mentioned points is difficult
Try to anticipate the arrival of the athletes $\geq$3 days before competition
Recommend the ingestion of antioxidant supplementation
Use a preventive medication for cough post-exercise in sensitive subjects
Evaluate the presence of other possible contaminants and establish preventive measures in hyperreactive subjects (asthmatics or nonasthmatics)
If the sports area is highly contaminated consider cancelling the events mainly in those sports of moderate-to-high intensity and long duration
Accommodation and living conditions for athletes
Check pollen charts for the competitive area for the season for the competition

and with other athletes [93, 94]. If an EIA crisis is triggered by dry air and significant ventilation, undoubtedly the best place for asthmatic subjects will be indoor pools where the temperature and humidity is 24–30°C and 60–70%, respectively.

Even by changing the air composition [95] it is suggested that, unlike running, swimming is of low asthmogenicity, even when the inspired air is dried to 25–30% at neutral temperatures [96]. The reason for this beneficial effect [97] is difficult to explain as body posture on land has no meaningful effect on the severity of bronchoconstriction in asthmatic children [98]. Also, airway hyperactivity is not alleviated by whole-body prone immersion [99]. Therefore, there is no satisfactory explanation for the advantage of exercising in water. In contrast, an increase in bronchial sensitivity in asthmatic and nonasthmatic swimmers has been reported when compared with a reference population and with other athletes [3]. Respiratory and other health-related complaints also become evident when swimmers exercise for long periods of time at high intense or minute ventilation [93, 100]. This situation can be maintained with an alteration of the cellular and inflammatory response of these athletes [57], probably modulated by excess of chorine inhalation throughout the sporting life of the athlete [52]. Elite athletes who practice water sports have mild neutrophilic inflammation, whether or not asthma is present, which is related to the degree of bronchial reactivity and the duration of training in swimming pool water [4, 101]. In contrast, if the possible infectious influences in the development of asthma are considered, asthma may be triggered in this particular type of sport [55, 102]. Differential diagnosis has to be made with swimming-induced pulmonary oedema (observed in some swimmers and divers), which can masquerade as EIA or can be observed in those asthmatic subjects, or athletes, who practice this sport thinking it is one of the safest for patients with EIA.

Ice hockey players and skaters. In a study by LUMME *et al.* [103], the presence of a positive histamine challenge test and atopy (determined by skin-prick test), was 24% and 58%, respectively, in a total of 88 ice hockey players. Similar results were obtained for methacholine tests (34.6%) and prevalence of asthma (19.2%) in this sport by other authors [104], and in those related with ice arenas [105]. A mixed neutrophilic and eosinophilic airway inflammation showed in these athletes and is associated with exposure to cool air and indoor inhaled pollutants, such as NO_2, during intensive training [103, 106].

Cycling. Indoor cycling is a strenous exercise practiced by a lot of athletes in the Nordic countries, mostly during the winter season. However, indoor cyclists usually perform part of their season and training outdoors. Respiratory problems were mostly related to those observed in other outdoor endurance athletes, as explained below. Even though the places of competition and training are large and spacious, ventilation and smoke exits have to be taken into account in acute cases.

Outdoor practice

Endurance sports. Endurance sports are usually performed outdoors, although there are competitions throughout the year which are performed indoors. This situation allows for some mechanisms that contribute to an inflammatory state of the airways *e.g.* chemical contamination, aeroallergens, respiratory infection, *etc*. Moreover, this happens with high respiratory ventilation. This is the reason why the prevalence of asthma in endurance athletes is ~17% as compared with that of power and speed sports, where it is ~8% [107]. Further research by the same investigators demonstrated that the risk of asthma was 6-times higher in endurance athletes and 3.5-times higher in power sport athletes compared

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with control subjects [3]. Regarding asthmatic prevalence in cyclists and long distance runners, it varies and ranges from 20–50% [94, 108]. In this regard, the diagnosis of asthma is based on the criteria of bronchial hyperreactivity, as assessed by any positive test, which complies with drug testing rules. In this way, hyperreactive subjects with symptomatology during exercise are allowed to continue their treatment and compete under the same conditions as their nonhyperreactive competitors. As a matter of fact, hyperreactive subjects of endurance sports can modify the prevalence and intensity of their complaints according to environmental conditions, season of the year and origin of asthma [109].

Power sports. Asthmatics that practice pure power sports, such as weightlifting, throws in track and field, or even gymnastics, seem not to be influenced by their activity. Other power sports that are combined with outdoor activity and with running as the basis of their physical fitness condition (*e.g.* speed running, wrestling, jumps, *etc*.) can develop symptoms related to asthma, as their ventilation increases and/or environmental and climactic conditions change.

Winter sports. Asthma prevalence in athletes participating in winter sports seems high compared with that of summer endurance athletes, being 5–17% in the Barcelona (Spain, 1992) and Atlanta (USA, 1996) Summer Olympic Games and 22% in the Nagano (Japan, 1998) Winter Olympic Games [108, 110, 111]. The intense cold and dry atmosphere and high hyperventilation during lengthy training and competition affects the respiratory tract. The inflammatory process in this situation is different from that observed in the classic asthmatic process, as can be observed in the bronchoalveolar lavage of athletes. An increase in the total number of cells was seen with a very marked increase in lymphocytes and mastocytes [112], and with bronchial remodeling defined by an increase in neutrophils [114], as occurs in other sports (swimming, cycling, ice hockey) [101, 103]. Approximately 80% of Swedish elite skiers suffer respiratory symptoms related to asthma [115]. It is noteworthy to observe that the prevalence of asthma between two countries with a great tradition in this sport is somewhat different, with Norway at 12% and Sweden at 40% [1]. According to the SUE-CHU *et al*. [1], the probable reason for this difference is the fact that one is located along the coast and the other is located inland.

Conclusion

After reviewing these concepts, the present authors believe that there is a need for further studies to better define the aetiological factors and mechanisms involved in the development of asthma, AHR or pathophysiological processes masquerading as asthma in athletes. Moreover, the authors propose that other relevant preventive and therapeutic measures are found for EIA in athletes. Also, official regulations incorporating the ranges of optimal climate and air contamination conditions (including poor or dangerous conditions) in which to play sport have to be created. At this moment, only cross-country skiing has one officially regulated body, the Fédération Internationale de Ski (http://www.fis-ski.com)

Summary

Physical exercise imposes a certain level of stress on the respiratory tract to eliminate carbon dioxide and to supply the muscles with oxygen for energy production. This process requires increased airways ventilation above the normal resting frequency. The number of hours spent in training at high intensity levels by athletes is progressively increasing, and the number of recreational athletes is also growing and they are more concerned about their training schedules and intensities. Increased ventilation rate and oral breathing displaces pulmonary uptake of pollutants to more distal sites in the lung, further increasing the effect of ambient exposure on the pollutants deposition in the distal airways. This will affect the athlete, whether recreational or professional, who suffers from asthma or bronchial hypersensitivity. In contrast, it is known that competitive athletes have a high prevalence of asthma, exercise-induced asthma or bronchial hyperreactivity. Mechanisms for this association include increased inhalation of cold air, air pollutants, allergens, an increased response to respiratory infections, and increased parasympatethic tone. The purpose of this chapter is to provide a review of outdoor and indoor sports practice and the environmental and climatological factors that can affect the airways of the asthmatic athlete.

Keywords: Asthma, aeroallergens, chlorine, doping, exercise, pollution.

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