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Key words: Asthma, elite athletes, Olympic Games, bronchial hyperresponsiveness

Pierre de Coubertin, the first president of the International Olympic Committee, chose “Citius, Altius, Fortius” (“Faster, Higher, Stronger”) as the Olympic motto in 1894. Today, this motto, stating the need for continuous improvement in physical performance, should also be considered from the perspective of the increase in asthma prevalence among elite athletes that has occurred in recent decades. Asthma is presently the most common chronic condition found among elite athletes participating in the Olympic Games.¹

In 1988, bronchial responsiveness was shown to increase in young swimmers after 3000 m of exertion at an exercise load approximately meeting the anaerobic threshold. The increase in bronchial responsiveness correlated significantly with the increase in serum lactate levels reflecting the exercise load, and it was observed in both asthmatic and nonasthmatic swimmers.² Soon afterward, 79% of Swedish elite cross-country skiers from 6 ski clubs were reported to have asthma or bronchial hyperresponsiveness (BHR) compared with 14% of control subjects.³ Increased prevalence of asthma and BHR is now reported in athletes who participate in a number of summer and winter endurance sports: speed skaters, figure skaters, ice hockey players, biathlon athletes, cyclists, swimmers, triathlon athletes, rowers, and long-distance runners. Specifically among Olympic athletes, increased prevalence has been reported in those who participate in cross-country skiing, biathlon, cycling, triathlon, and swimming.¹

In particular, asthma develops in endurance athletes and is believed to be related to daily training sessions and frequent competitions with heavily increased ventilation. This has been shown to cause respiratory epithelial damage and apoptosis in nonasthmatic half-marathon runners.⁴ Daily training under such conditions can cause repeated respiratory water loss and subsequent respiratory mucosal dehydration. The ensuing

mediator release in respiratory mucosal tissue leads to respiratory inflammation and exercise-induced bronchoconstriction in susceptible subjects.⁵ By means of natural warming of the inspired air to 37°C during exercise, heat loss can also occur through increased ventilation, causing parasympathetic stimulation that contributes to bronchoconstriction and increased cholinergic inflammation.⁶ Endurance training has been shown to cause increased parasympathetic tone and parasympathetic modulation, as measured by heart rate variation related to physical fitness.⁷ Environmental elements also contribute to increased inflammation, including cold air exposure during winter sports, chlorine exposure during swimming in indoor swimming pools, and exposure to ultrafine particles in indoor ice rinks.⁸

Fig 1 outlines a hypothesis concerning the role and interplay of factors in the development of asthma and BHR among elite athletes. A study on Portuguese and Norwegian Olympic athletes using latent class analysis identified 2 main asthma phenotypes in athletes: atopic asthma, which is defined by allergic sensitization, rhinitis, allergic comorbidities, and increased exhaled nitric oxide levels, and sports asthma, which is defined by exercise-induced respiratory symptoms and airway hyperresponsiveness without allergic features. Risk of the sports asthma phenotype was significantly increased in athletes practicing water (odds ratio [OR], 2.87) and winter (OR, 8.65) sports.⁹

A differential diagnosis of asthma should include a number of conditions in athletes with respiratory exercise limitations. Most important and most frequent is exercise-induced laryngeal obstruction, which is also called exercise-induced vocal cord dysfunction and is classified as either supraglottic or glottic. Exercise-induced laryngeal obstruction is marked by inspiratory stridor that occurs during maximum physical intensity. It is most often found in young, well-trained female athletes. Additional potential causes of less than optimal physical performance include other chronic respiratory disorders and cardiac disorders and poor physical fitness. Dysfunctional breathing during exercise, exercise-induced arterial hypoxemia, and, rarely, swimming-induced pulmonary edema should be considered in the diagnosis of sports asthma as well.

Shortly after the introduction of inhaled β_2 -agonists, their use was restricted in 1972 by the medical commission of the International Olympic Committee because of a fear that they could improve performance among competitive athletes. A major increase in notifications on the use of inhaled β_2 -agonists during the Olympic Games from 1996 to 2000 led the medical commission to require objective diagnostic tests before use shortly before the Salt Lake City Olympic Games in 2002. Approval requires documentation of either bronchial dilator reversibility (increased FEV₁ $\geq$ 12%) or BHR by means of methacholine bronchial challenge, exercise tests, or eucapnic voluntary hyperpnea¹ in addition to case history and physical examination.

The World Anti-doping Agency made some changes in 2010 and 2012, presently allowing free use of all inhaled corticosteroids and the inhaled β_2 -agonists salbutamol (maximum allowed

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Disclosure of potential conflict of interest: K.-H. Carlsen has received payment for lectures from Boehringer Ingelheim and Novartis. K. C. Lødrup Carlsen has a board membership with Sanofi and has received grants from the Regional Health Board South East, the Norwegian Research Council, Oslo University Hospital, the University of Oslo, Health and Rehabilitation Norway, Hospital Trust Østfold, and the European Union (MeDALL project), as well as unrestricted grants from the Norwegian Association of Asthma and Allergy; the Kloster Foundation; Thermo Fisher, Uppsala, Sweden (allergen reagents); Furst Medical Laboratories (analyses of IgE); the Norwegian Society of Dermatology and Venerology, and Arne Ingel's legacy.

Received for publication May 5, 2016; accepted for publication May 12, 2016.

Available online June 23, 2016.

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J Allergy Clin Immunol 2016;138:409-10.

0091-6749/\$36.00

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<http://dx.doi.org/10.1016/j.jaci.2016.05.025>

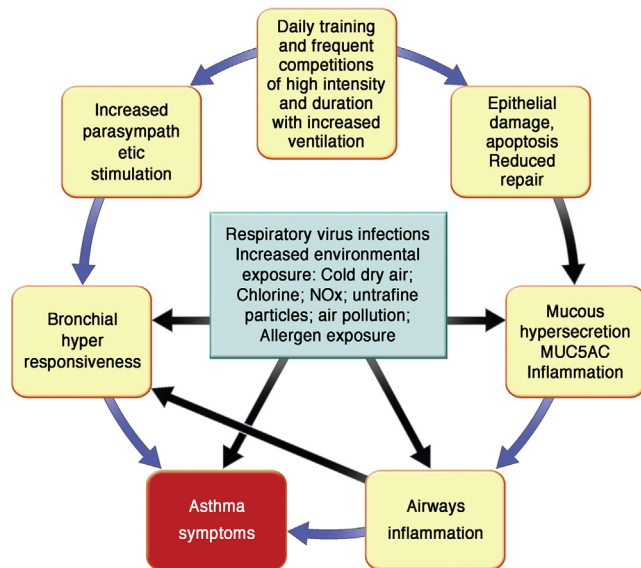


FIG 1. Overview of probable pathogenic mechanisms for the development of asthma in athletes. NOx, Nitrogen oxide.

daily dose, 1.6 mg), formoterol (maximum allowed daily dose, 54 µg), and salmeterol in recommended doses. Urinary concentrations of salbutamol and formoterol are measured in doping controls. Salbutamol concentrations of greater than 1000 ng/mL and formoterol concentrations of greater than 40 ng/mL are presumed not to be an intended therapeutic use of the substance and will be considered an Adverse Analytical Finding unless the athlete proves, through a controlled pharmacokinetic study, that the abnormal result was the consequence of the use of the therapeutic inhaled dose up to the maximum indicated above. However, it should be mentioned that in a recent study combining the maximum daily dose of inhaled salbutamol (1.6 mg) with

physical exercise, 20 of 32 athletes had urinary concentrations of greater than the allowed limit (1000 ng/mL) and the decision limit of 1200 ng/mL.¹⁰

It is of particular interest that asthmatic Olympic athletes have won significantly more medals than nonasthmatic competitors in every Olympic Games since the year 2000. It has been speculated that the harder an athlete trains, the better the performance, simultaneously increasing asthma risk.¹

Because asthma is the most common chronic disease in Olympic athletes, it is important that the present available treatment according to international guidelines enables those with asthma to compete on an equal level and with equal success as other healthy athletes.

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