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EDITORIAL

Asthma, airway inflammation and epithelial damage in elite athletes

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In recent years there has been special focus on the increased occurrence of asthma and bronchial responsiveness among top athletes within endurance sports. As early as 1989, an increase in nonspecific bronchial responsiveness after heavy endurance training was found in young competitive swimmers [1]. Later, reports were made concerning increased prevalence of asthma and bronchial hyperresponsiveness to metacholine among top cross-country skiers [2, 3]. Reports came from Trondheim (Norway) regarding inflammatory changes in bronchial biopsies appearing during a winter season in young competitive cross-country skiers [4, 5]. These studies confirmed that both bronchial responsiveness and airway inflammation increased through heavy endurance training [1–5].

In the Olympic arena, such reports were confirmed by VOY [6], who reported a prevalence of exercise-induced asthma of 11% among the American summer Olympic athletes of 1984; this increased to >20% among the American participants in the 1996 summer Olympic Games, and was especially high among cyclists and mountain bikers [7]. K. Fitch (who supplied some of the data presented in [8]) reported that the use of asthma drugs and, in particular, inhaled β_2-agonists, was highest in cross-country skiing and speed-skating followed by cycling, Nordic combined (both cross-country skiing and ski jumping) and swimming during the last three summer Olympics and the last three winter Olympics [8].

In the present issue of the *European Respiratory Journal*, BOUGAULT *et al.* [9] report findings from a study of induced sputum and cell counts of eosinophils and neutrophils in swimmers and cold-air athletes (cross-country skiers, speed skaters and biathletes), as compared to healthy controls and controls with asthma. The study participants had a mean age of 19–22 yrs; the two groups of athletes had a mean of 10 yrs of training. Details are not given as to how the athletes were recruited, other than that they were high-level athletes. BOUGAULT *et al.* [9] could not find any correlation between bronchial hyperresponsiveness (BHR) to metacholine and age and number of years of training; however, the athletes in the study were still at an early stage in their career as compared with other studies, which showed increased prevalence of asthma [2] and increased prevalence of BHR to metacholine [10] among elite athletes with increasing age and duration of training. BOUGAULT *et al.* [9] reported that 69% of swimmers and 28% of cold-air athletes suffered from BHR to metacholine. An increased number of bronchial epithelial cells and neutrophils was found in induced sputum, with neutrophil counts being independent of the presence of BHR. In contrast, eosinophils were only increased among both swimmers and cold-air athletes with the presence of BHR, and correlated with BHR in swimmers only. It is of great interest that the sputum neutrophil counts correlated with number of training hours per week.

The study by BOUGAULT *et al.* [9] clearly demonstrates the effect of heavy endurance training upon the respiratory tract. The findings of the study show both increased bronchial epithelial cells and an increased number of neutrophils among the athletes, as well as a rise in the number of eosinophils in athletes with BHR. These findings demonstrate the wear-and-tear effect that may be caused by increased ventilation during endurance training, as well as increased airways inflammation due to training. These findings are in line with results from bronchial biopsies from skiers [4, 5], as well as with the results of the study by HELENIUS *et al.* [11], who also reported increased levels of eosinophils and neutrophils, as well as increased concentrations of sputum eosinophil peroxidase and human neutrophil lipocalin in induced sputum from swimmers. The findings of BOUGAULT *et al.* [9] are also supported by animal studies. CHIMENTI *et al.* [12] compared the bronchial mucosa of trained mice with that of sedentary mice. Bronchiolar epithelium of trained mice showed progressive loss of ciliated cells, and increased apoptosis and proliferation. In another study, DAVIS *et al.* [13] performed bronchoscopy and bronchoalveolar lavage in elite racing sled dogs 24–48 h after completion of a 1,100-mile endurance race in Alaska (USA). Of the 59 dogs examined, 48 showed abnormal accumulations of intra-luminal debris, and bronchalveolar lavage obtained after the race showed significantly higher nucleated macrophage and eosinophil counts as compared with sedentary control dogs. These studies suggest, in the same way as BOUGAULT *et al.* [9], that increased ventilation during repeated heavy exercise causes stress to the airway epithelium and increases airway mucosal inflammation. Environmental agents, such as exposure to cold air and organic chlorine products, will probably increase this stress, increasing
bronchial responsiveness.

The environment in which the sports performance takes place is important for the development of bronchial hyperresponsiveness as measured by the reaction to methacholine or

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histamine, airways inflammation and respiratory epithelial damage in endurance athletes. This is the case for several types of sports [14] but particularly for cross-country skiers and swimmers [8], as in the study of BOUGAULT *et al.* [9]. Actively competing swimmers are particularly exposed to organic chlorine products used to disinfect the water in indoor swimming pools [15]. BERNARD *et al.* [16] have also focused upon exposure in indoor swimming pools and found that there is a relationship between the number of hours spent in indoor swimming pools when growing up and the risk of asthma development in pre-school children and schoolchildren [17]. Serum levels of surfactant proteins A and B [16] as well as of Clara cell proteins [18] have been related to lung damage induced by pool attendance during childhood. BERNARD *et al.* [19] also reported an increased risk of asthma related to attendance at outdoor chlorinated pools.

A link therefore remains between respiratory symptoms, bronchial hyperresponsiveness to metacholine/histamine and airway inflammation in athletes, occurring in particular environmental conditions and documented in several studies [8], and the risk of developing asthma in physically active children. However, this is so much more obvious in top athletes due to their intensive, repetitive training and high ventilation rates. This underlines the need to understand the pathogenesis of asthma and bronchial hyperresponsiveness occurring in elite athletes, as exemplified in the paper of BOUGAULT *et al*. [9] in the present issue of *European Respiratory Journal*.

REFERENCES

1 Carlsen KH, Oseid S, Odden H, Mellbye E. The response to heavy swimming exercise in children with and without bronchial asthma. *In*: Morehouse CA, ed. Children and Exercise XIII. Champaign, Human Kinetics Publishers, Inc.; 1989; pp. 351–360.

2 Heir T, Oseid S. Self-reported asthma and exercise-induced asthma symptoms in high-level competetive cross-country skiers. *Scand J Med Sci Sports* 1994; 4: 128–133.

3 Larsson K, Ohlsen P, Larsson L, Malmberg P, Rydstrom PO, Ulriksen H. High prevalence of asthma in cross country skiers. *BMJ* 1993; 307: 1326–1329.

4 Sue-Chu M, Karjalainen EM, Altraja A, *et al.* Lymphoid aggregates in endobronchial biopsies from young elite cross-country skiers. *Am J Respir Crit Care Med* 1998; 158: 597–601.

5 Karjalainen EM, Laitinen A, Sue-Chu M, Altraja A, Bjermer L, Laitinen LA. Evidence of airway inflammation and remodeling in ski athletes with and without bronchial hyperresponsiveness to methacholine. *Am J Respir Crit Care Med* 2000; 161: 2086–2091.

6 Voy RO. The U.S. Olympic Committee experience with exercise-induced bronchospasm, 1984. *Med Sci Sports Exerc* 1986; 18: 328–330.

7 Weiler JM, Layton T, Hunt M. Asthma in United States Olympic athletes who participated in the 1996 summer games. *J Allergy Clin Immunol* 1998; 102: 722–726.

8 Carlsen KH, Anderson SD, Bjermer L, *et al.* Exercise-induced asthma, respiratory and allergic disorders in elite athletes: epidemiology, mechanisms and diagnosis: part I of the report from the Joint Task Force of the European Respiratory Society (ERS) and the European Academy of Allergy and Clinical Immunology (EAACI) in cooperation with GA2LEN. *Allergy* 2008; 63: 387–403.

9 Bougault V, Turmel J, St-Laurent J, Bertrand M, Boulet L-P. Asthma, airway inflammation and epithelial damage in swimmers and cold-air athletes. *Eur Respir J* 2009; 33: 740–746.

10 Stensrud T, Mykland KV, Gabrielsen K, Carlsen KH. Bronchial hyperresponsiveness in skiers: field test *versus* methacholine provocation? *Med Sci Sports Exerc* 2007; 39: 1681–1686.

11 Helenius IJ, Rytila P, Metso T, Haahtela T, Venge P, Tikkanen HO. Respiratory symptoms, bronchial responsiveness, and cellular characteristics of induced sputum in elite swimmers. *Allergy* 1998; 53: 346–352.

12 Chimenti L, Morici G, Paterno A, *et al.* Endurance training damages small airway epithelium in mice. *Am J Respir Crit Care Med* 2007; 175: 442–449.

13 Davis MS, McKiernan B, McCullough S, *et al.* Racing Alaskan sled dogs as a model of ''ski asthma''. *Am J Respir Crit Care Med* 2002; 166: 878–882.

14 Belda J, Ricart S, Casan P, *et al.* Airway inflammation in the elite athlete and type of sport. *Br J Sports Med* 2008; 42: 244–248; discussion 248–249.

15 Drobnic F, Freixa A, Casan P, Sanchis J, Guardino X. Assessment of chlorine exposure in swimmers during training. *Med Sci Sports Exerc* 1996; 28: 271–274.

16 Bernard A, Carbonnelle S, Michel O, *et al.* Lung hyperpermeability and asthma prevalence in schoolchildren: unexpected associations with the attendance at indoor chlorinated swimming pools. *Occup Environ Med* 2003; 60: 385–394.

17 Bernard A, Carbonnelle S, de BC, Michel O, Nickmilder M. Chlorinated pool attendance, atopy, and the risk of asthma during childhood. *Environ Health Perspect* 2006; 114: 1567–1573.

18 Lagerkvist BJ, Bernard A, Blomberg A, *et al.* Pulmonary epithelial integrity in children: relationship to ambient ozone exposure and swimming pool attendance. *Environmental Health Perspectives* 2004; 112: 1768–1771.

19 Bernard A, Nickmilder M, Voisin C. Outdoor swimming pools and the risks of asthma and allergies during adolescence. *Eur Respir J* 2008; 32: 979–988.