Bronchial responsiveness and airway inflammation in trained subjects

We read with interest the paper by Shaaban and coworkers on the protective effect of physical activity against bronchial hyperreactivity (BHR) in the general population. The authors suggest that a beneficial effect of deep inspirations during exercise could account for the lower prevalence of BHR in physically active subjects compared with sedentary subjects, while the accompanying editorial favours an “anti-inflammatory” effect of exercise as the most plausible explanation.

We have studied lung function and airway cell biology in non-asthmatic amateur athletes and found that both modulation of airway responsiveness and downregulation of airway inflammation occur with training. At rest, the response to single-dose methacholine inhalation in the absence of deep breaths was significantly lower in amateur runners than in age-matched sedentary controls. Shortly after a marathon race the response to methacholine was further blunted, suggesting a causal relationship between endurance exercise and low bronchial responsiveness, possibly mediated by ventilation at increased lung volumes.

We have previously reported large numbers of neutrophils in induced sputum of runners. However, this finding was not associated with evidence of neutrophil activation after intense exercise, since expression of adhesion molecules by airway neutrophils decreased and the elastase concentration in sputum supernatants was unchanged after a marathon race compared with baseline. Similarly, inflammatory cell infiltration in the airways was not associated with activation of the NfkB pathway in endurance-trained mice, while airway inflammation was found to decrease strikingly in ovalbumin-sensitised trained mice compared with sedentary mice. Exercise therefore appears as a model of tightly regulated airway inflammation, possibly secondary to exercise-induced mild bronchial epithelial damage. Along the same line, physically active smokers appear to be protected against lung function decline and the risk of developing chronic obstructive pulmonary disease compared with sedentary smokers, supporting a role for regular exercise in blunting airway inflammation.

We acknowledge that athletes, even at the amateur level, do not represent the general population. On the other hand, a publication bias may have favoured preferential reporting of exercise-associated BHR in athletes, especially those training under standard laboratory conditions damages small airway epithelium in mice. Am J Respir Crit Care Med 2007;175:442–9.

Propionibacterium acnes in granulomas of a patient with necrotising sarcoid granulomatosis

Necrotising sarcoid granulomatosis (NSG) was first described by Liebow in 1973. It is defined by three pathological features: the presence of a conglomerate mass of sarcoid-like granulomas; varying degrees of necrosis within the confluent granulomas; and vasculitis with granulomas and giant cells involving the walls of muscular arteries and veins. The relationship between NSG and classic sarcoidosis is controversial. In NSG hilar lymphadenopathy is not seen as frequently as in sarcoidosis, extrapulmonary involvement is rare and serum levels of angiotensin-converting enzyme (ACE) are not necessarily raised.

The cause of sarcoidosis is unknown, but it has been hypothesised that it results from exposure of a genetically susceptible individual to specific environmental agents. Abe et al isolated Propionibacterium acnes (P acnes) in culture from sarcoidosis biopsy specimens, and recently the P acnes genome has been detected in sarcoid lymph nodes by
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