Letter to editor

Does exercise affect lung diseases with lungkine (CXCL15)? Clinical benefits of high intensity interval training

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Dear Editor-in-Chief

The chemokines are a superfamily of small, secreted proteins that regulate leukocyte migration. Several of these chemokines have been associated with various diseases. Some chemokines have been reported to be expressed in the lung, including interleukin (IL)-8/CXCL8, eotaxin/CCL11, macrophage-inflammatory protein (MIP)-3α/CCL20, and DC-CK1/pulmonary- and activation-related chemokine (PARC)/alternative macrophage activation-associated CC-chemokine (AMAC)-1/CCL18 (Homey et al., 2000). It is interesting that there is even one reported chemokine that is specifically expressed in the lung, lungkine/CXCL15 (Rossi et al., 1999). Lungkine is an important mediator of neutrophil migration from the lung parenchyma into the airspace.

Chemokine CXCL15, which absorbs neutrophils during pulmonary inflammation, is also known as lungkine because of its reported exclusive expression in the lung. CXCL15, previously reported as the only lung-specific chemokine, is also highly expressed in other mucosal organs and endocrine glands of mice. The functional role of CXCL15 is unclear with precise exercise training, and little research has addressed this issue. Files et al. (2015) evaluated the effects of therapeutic exercise on lung disease and also measured CXCL15 levels (Files et al., 2015). In this study, although CXCL15 was down regulated in response to exercise therapy, they stated that exercise therapy improved both alveolar neutrophil lung damage and skeletal muscle atrophy in the animal model of ARDS. They stated that more studies are needed to identify mechanisms underlying exercise and its benefits in the lungs, which may indicate new molecular targets for the treatment of ARDS.

Clinical and experimental studies have shown that regular aerobic exercise can prevent or even eliminate a number of diseases, especially in patients in the intensive care unit. This beneficial effect of exercise is associated with anti-inflammatory and antioxidant protection. Despite the apparent benefits, the dose of exercise intensity is still unknown. Balducci et al. (Balducci et al., 2010), showed that people with type 2 diabetes who performed intense exercise had a significant improvement in their inflammatory status. High-intensity exercise reduced pneumonia and improved oxidative status in experimental models of allergic pneumonia and exposure to contaminants (Ávila et al., 2015). Due to the mechanism involved in changes in epithelial pathogenesis, strenuous exercise with hyperventilation can affect the airway epithelium by altering the viscosity, elastic force, or amount of airway fluid. In an animal model trained with an intense exercise protocol, an increase in the infiltration of leukocytes into the bronchial wall was observed (Chimenti et al., 2007). Therefore, it is believed that performing intense exercise may cause inflammatory cells to invade the airways, epithelial changes, and defective remodeling. However, studies in this area are also contradictory. In a study high-intensity swimming showed a protective effect on ALI, decreasing inflammatory processes and preventing disturbances in antioxidant defenses into the lungs (Cardoso et al., 2018). However, little is known about the possible protective effects of HIIT on lungkine CXCL15 and its mechanisms of action.

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