Characterizing effects of glucocorticoids and nitric oxide in airway inflammation in a murine in vivo model

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Asthma is a chronic (long-term) disease that makes it hard to breathe. Asthma is a common inflammatory disease affecting one in twenty adults and one in ten children. It is one of the most common diseases today; around 300 million people in the world suffer from asthma and 250,000 people die from the disease every year. Asthma is characterized by airway narrowing, inflammation of the airways, and higher numbers of inflammatory cells into the airways.

Current guidelines of asthma therapy have focused on the use of anti-inflammatory therapy, particularly inhaled glucocorticoids. Glucocorticoids are the most potent anti-inflammatory agents. Two of the major mechanisms by which glucocorticoids act in asthma, is by reducing airway inflammation and by reducing activation of the immune system.

The gas nitric oxide may have beneficial effects on pulmonary function in asthmatic patients. Asthmatics have increased levels of nitric oxide in their exhaled air. Exhaled nitric oxide is a sensitive marker of airway inflammation. One well-documented function of nitric oxide is expanding of blood vessels, accumulation of fluid, and facilitation of cell recruitment to the tissue. Nitric oxide can act relaxing on bronchial muscle as well, but the effect is weaker than on the blood vessels.

The main purpose of this study was to investigate if glucocorticoids could blunt the airway inflammation in a murine asthma model. Another aim was to investigate if inhaled nitric oxide could attenuate the airway reactivity in mice with airway inflammation.

The results showed that glucocorticoids had an effect on airway inflammation. The effect of inhaled nitric oxide needs more investigation, if it was appropriate right method for nitric oxide delivery.

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