RESEARCH IN PROGRESS

Effect of Elevated Carbon Dioxide on Innate Immune Response in the Lung

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Purpose/Objective:
Workers in high intensity livestock facilities exhibit reduced lung function over time in the industry. Many symptoms have been attributed to barn dust exposure and bacterial components in these dusts that can activate innate immune responses. Under circumstances of decreased ventilation of barn facilities and increased pulmonary obstruction, the lungs of these workers may also be exposed to elevated levels of carbon dioxide (CO_2).

Methods/Efforts:
In recent years there have been conflicting reports on the effects of CO_2 on inflammation in the lung from studies of permissive hypercapnia. We proposed these changes would be reflected in lung epithelial cells. To address this we looked at several cell lines representing bronchial epithelium, alveolar epithelium, and alveolar macrophages and how they responded to stimulation with a variety of innate immune stimuli as well as barn dust under normal (5%) and elevated (9%) CO_2.

Results/Findings:
We show that all cell types tested showed an increase in production of inflammatory signaling molecules in response to higher CO_2, suggesting that CO_2 may enhance inflammation in the lung, or make these cells more responsive to potential inflammatory molecules in the environment. Interestingly, expression of anti-microbial molecules produced by these same cells showed reductions in several at elevated CO_2, suggesting that killing of microbes may at the same time be inhibited by CO_2. In fact barn dust exposure itself may be inhibiting this one microbial defense mechanism.

Application to Field Research:
The study of hypercapnia and its effect on innate immunity is a basic study that has immediate implications for work on smoking, workplace safety, ventilator lung strategy, and infection in cases of chronic obstruction.

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