What is the role of lung inflammation in mediating particulate matter and ozone exposure effects on health and disease?

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Compelling evidence suggests that exposure to particulate matter and airborne toxicants profoundly affects human health globally. Particulate air pollution not only induces lung inflammation but is also associated with systemic inflammation. Potentially, the local and systemic inflammation may increase healthcare utilization for cardiovascular and pulmonary disease. Although traffic-related particulate matter and ozone are associated with an increase in hospitalization rates, it is unclear whether the inflammation alone mediates these effects or whether the inflammation may serve as a negative homeostatic signal to mitigate further injury. We and others have demonstrated that ozone, formaldehyde and particulate matter exposure enhances airway hyperresponsiveness, a physiological surrogate for asthma or chronic osbstructive lung disease. Curiously, the inflammation which is predominantly neutrophilic in nature is uncoupled from the induction of airway hyperresponsiveness. Evidence also suggests that the airway epithelium and other structural cells play a pivotal role in mediating the oxidative stress-induced signals from the lung to the periphery. Our data also suggests that current approaches in the management of airways disease, specifically inhaled corticosteroids, are ineffective in mitigating the neutrophil-driven inflammation. Collectively, this session will review current evidence supporting a central hypothesis that air pollution-induced local and systemic inflammation may, in part, increase morbidity and mortality. In some inhaled toxicants, however, there exists an uncoupling of lung inflammation from the systemic and organ-specific physiologic consequences of air pollution (supported by P30-ES013508).