How do combustion related products cause/induce asthma and what can we do about it?  

Presenters: I Gilmour1, D.B. Peden2  

Contributing Organizations: ORD: 1NHEERL, NERL; 2University of North Carolina at Chapel Hill

Traffic is a major source of CRPs that exacerbate asthma. Two major epidemiology studies performed in two urban areas Detroit and in S. California suggest traffic can impact clinical endpoints of asthma. In Detroit elevated exposures are associated with traffic intensity for those living close to a freeway. Similarly in S. California children living close to a freeway show decreased lung function.

Exposure to traffic and PM2.5 can override the protective effects of high lung function and increase incident asthma rates.

Dipal exhaust, an important component of traffic, induces inflammation and promotes allergic control. There are distinct gene and pathway responses between different diesel particles and between species. However, we have identified 4 toxicological pathways (endothelium, eosinophils, and asthma) in common between particles and models. A novel pathway has been identified that may regulate diesel effects on allergic asthma through epigenetic modifications.

A subgroup of asthmatics may acquire increased sensitivity to allergens after exposure to ozone. Ozone exposure will increase antigen presentation markers in the human airway. Similarly, diesel particles can activate dendritic cells in vivo and in mouse models. CRPs may also enhance responses to viruses by a key trigger of asthma by altering the innate immune response. Anti-oxidant genes such as GSTM1 and IEPX1 can modify asthma risk, this in turn is modified by traffic exposure.

GSTM1 is a protective factor for the pro-allergic and pro-inflammatory effects of diesel particles. Recruitment of inflammatory cells following ozone exposure is modified by GSTM1. Exogenous or induction of endogenous anti-oxidants can reduce lung injury and inflammation by CRPs in vitro and in animal asthma models. Endogenous anti-oxidants can be induced in the airway of humans subjects by dietary interventions.

Air pollution is a huge health, economic and societal burden. It affects over 20 million Americans and is estimated to cost nearly $18 billion and account for more than 14 million school days per year. Asthmatics and especially children are more sensitive to the adverse health effects of air pollutants. The research goals of this program are therefore centered on:

1. Identifying individual components or defined mixtures that produce health effects in asthmatics
2. Understanding upstream pathways and susceptibility factors that may predict those who will be at increased risk
3. Developing methods to minimize risk

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