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Wood smoke exposure induced IL-6 and eosinophil increase in human airways.

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Background: Ambient and residential exposure to wood smoke has implications on human health. Epidemiological studies have shown an association between exposure to wood smoke and worsening of chronic obstructive pulmonary disease (COPD) and asthma. A suggested mechanism is that wood smoke causes airway inflammation. The present aim was to study airway responses to short-term wood smoke exposure in healthy humans.

Materials and method: Fourteen healthy, non-smoking participants were included in a double-blinded randomised cross-over exposure study. Participants were exposed on two occasions, with at least 3 weeks apart, to filtered air and to sooty, polycyclic aromatic hydrocarbons-rich wood smoke at a concentration of 409±43 µg/m3 for 2 hours. Bronchoscopy with bronchial wash (BW) and bronchoalveolar lavage (BAL) was performed 6 hours after exposure. Differential cell counts and analysis of inflammatory markers with ELISA were performed in BW and BAL-fluid.

Results: Wood smoke increased eosinophil numbers, but no other cell influx in BW or BAL. This was associated with a significant increase in IL-6 in BW ($p=0.035$) and trend towards an increased IL-6 concentration in BAL-fluid ($p=0.051$).

Conclusion: Our findings suggest that wood smoke may cause airway eosinophilia and a minor pro-inflammatory airway response in healthy human airways. Further research is warranted in asthmatic subjects.