

# A CHAMBER STUDY OF AIRWAY AND SYSTEMIC EFFECTS AFTER EXPOSURE TO TWO KINDS OF WOOD SMOKE

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**Background:** Air pollution increases cardiovascular morbidity and mortality. One hypothesis is that local airway inflammation is a pathway to systemic inflammation, and in the long term atherosclerosis and cardiovascular events. Wood smoke contributes significantly to indoor and outdoor air pollution. The composition is complex, depending on e.g. combustion technique. We examined health effects of two kinds of wood smoke in a controlled chamber study of human subjects.

**Methods:** Thirteen subjects were exposed to filtered air and two sessions of wood-smoke, one week apart, with smoke from the start-up or the burn-out phase of the wood-burning cycle. The particle mass concentrations were 295  $\mu\text{g}/\text{m}^3$  and 146  $\mu\text{g}/\text{m}^3$ ; number concentrations 140 000/ $\text{cm}^3$  (68% ultrafine) and 100 000/ $\text{cm}^3$  (40% ultrafine) respectively. Blood, urine and breath condensate were collected before and on several occasions after exposure, and analyzed for biomarkers of inflammation, coagulation and oxidative stress. Exhaled nitric oxide (FENO) was measured. Net effects of wood smoke compared to filtered air were evaluated.

**Results:** Clara Cell protein 16 (CC16) increased in serum four hours after exposure to smoke from the start-up phase, and in urine the next morning. There was a net increase of FENO<sub>270</sub> after wood smoke in both sessions, but mainly due to a decrease after filtered air. Other markers of airway inflammation were not affected by exposure. No consistent increase was seen for markers of systemic inflammation and coagulation. Urinary isoprostane and plasma-fibrinogen unexpectedly decreased after exposure.

**Conclusions:** The results indicate that airway effects appear at relatively low levels of exposure to wood smoke, while signs of systemic inflammation, previously shown at higher exposures, do not. Effects could only be shown after smoke from the start-up phase. CC16 is a sensitive marker in both serum and urine for effects of air pollution, but its function and significance need to be clarified.