

WOOD DUST-INDUCED INFLAMMATORY RESPONSE IN MOUSE MACROPHAGES

Juha Määttä, Marja-Leena Majuri, Antti Lauerma, Kirsti Husgafel-Pursiainen, Antti Tossavainen, Jaakko Sääntti, Harri Alenius, and Kai Savolainen

Finnish Institute of Occupational Health, Topeliuksenkatu 41 a A, 00250 Helsinki, Finland

In addition to nasal and sino-nasal adenocarcinomas, wood dust exposure can induce several nonmalignant, mainly respiratory diseases such as allergic rhinitis, chronic bronchitis, and asthma. To find out whether wood dust is able to influence the development of inflammatory process through macrophages, we have elucidated the effects of wood dust exposure on the cytokine and chemokine expression of mouse macrophage cell line cells (RAW 264.7). The cells were exposed to graded doses of selected hardwood (birch, beech, oak, and teak) and softwood dusts (pine and spruce). TiO₂ and LPS were used as controls. The mRNA expression of major proinflammatory cytokines (IL-1 β , TNF- α , and IL-6), an anti-inflammatory cytokine (IL-10), and several chemokines (CCL2, CCL3, CCL4, CCL5, and CCL24) were assessed by real time PCR at several time points after wood dust exposure. TNF- α , IL-6, and CCL2 expression was studied also at the protein level using the ELISA method. Wood dust had in general more effects on cyto- and chemokine expression than inorganic dust TiO₂. All wood dusts induced TNF- α , IL-6, CCL3, and CCL4 expression and inhibited IL-1 β and CCL24 expression. However, many differences were detected in the strength of induction or inhibition between different wood dusts. In the case of CCL2, birch, beech, pine, and spruce induced CCL2 production, but oak and teak dusts had no effect. Oak dust, that has been previously shown to be carcinogenic, appears to be a weaker inducer of inflammatory response than the other wood dusts. Our results show that exposure to different wood dusts elicits dose-dependent changes in the levels of inflammatory mediators in mouse macrophage cells. These findings suggest that exposure to wood dust may significantly influence development of inflammatory process in the airways by modulating the expression of proinflammatory cytokines and chemokines.