Organic dust and inflammatory markers

Relations between respiratory symptoms, lung function and inflammation of the airways

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ABSTRACT

The studies were carried out at the Institute of Public Health, Department of Environmental and Occupational Medicine, Faculty of Health Sciences, University of Aarhus.

The aims were: 1) to study nasal mucosal reactions in humans exposed to office dust alone or combined with a β-glucan or a mixture of aldehydes, 2) to study airway inflammation in relation to organic dust exposure and in relation to markers of sensitivity, and 3) to compare the cytokine release to selected organic dust in a whole blood assay (WBA).

Two surveys were performed in fish-processing factories. An experimental exposure study was conducted with volunteers. Finally, immunological investigations of dust from a swine confinement building (SCB) were performed.

Questionnaires, lung function measurements including bronchial responsiveness, acoustic rhinometry, rhinostereometry, nasal lavage, and CO diffusion capacity were applied. In the double blinded, randomized, crossover, experimental exposure study, 36 volunteers were exposed to 500 μg dust/m^3, with or without 300 μg aldehydes/m^3 or 5 μg glucan/m^3. The WBA was carried out by adding the samples in question directly to the fresh blood, incubating, and measuring the cytokine concentrations after 18 h.

In the fish processing industry, an elevated prevalence of respiratory symptoms and occupational asthma was observed, accorded by poorer lung function. The WBA revealed that rinsing water from the filleting station showed greater inflammatory potential than pure lipopolysaccharide (LPS) from gram-negative bacteria or pure glucan. The WBA release of IL-8 in response to this rinsing water was elevated in factory workers compared with control subjects. The samples of rinsing water from the first and second survey in the fish factories resulted in lower IL-1β release than the SCB dust or pure LPS but higher or equal IL-8 release than LPS. The SCB dust caused the highest IL-8 release. A consequence of the exposures in the climate chamber was nasal mucosal swelling and enhanced release of nasal IL-8 after office dust spiked with glucan or aldehydes. The gas transfer was reduced 24 h after the spiked dust exposures, but no other effects were observed on the lower airways. The observed effects tended to be greater after dust with glucan than after dust with aldehydes. This was particularly true for nasal mucosal swelling observed in atopic volunteers after 3.5 h of dust with glucan.

In conclusion, we found evidence of both allergic and non-allergic airway reactions in relation to herring filleting. Mixed dust from the studied work environments can induce greater cytokine release in the WBA than pure LPS. Short time exposure with dust spiked with glucan appears to enhance the inflammatory potential of office dust in the upper airways.