


23 Thelin A, Tegler O, Bylander R. Lung reactions during poultry handling related to dust and bacterial endotoxin levels. Eur J Respir Dis 1984; 65:206-71


32 Olenchock SA, Mull JC, Major PC. Extracts of airborne grain dusts activate alternative and classical complement pathways. Ann Allergy 1980; 44:23-8


35 Lewis DM, Mentench MS. Extracts of airborne grain dusts stimulate interleukin-1 (IL-1) production by alveolar macrophages [abstract]. Am Rev Respir Dis 1984; 129:A161


Cigarettes Are a Rich Source of Bacterial Endotoxin*

Jeffrey Hasday, MD, FCCP; Wendy Dahlin, MD; Thomas Fitzgerald, BS; and Rebecca Bascom, MD, MPH

Chronic bronchitis in cigarette smokers and workers with environmental lung diseases attributed to bacterial endotoxin (LPS) inhalation (eg, byssinosis, swine farmers lung) share many clinical and histologic features. Inhaled LPS mimics many of the acute effects of cigarette smoke in the lower airway. We reasoned that cigarette tobacco and cigarette smoke may contain bioactive LPS.

We used a Limulus amebocyte lysate (LAL) assay to measure biologically active LPS in extracts from tobacco and smoke. In human volunteers, we found that cigarette smoke contains significantly more LPS than cigarette tobacco. Cigarette smoke contains 10-20 fold more bioactive LPS than cigarette tobacco.

*From the Division of Pulmonary and Critical Care Medicine, University of Maryland, Baltimore.
cigarette filters in 1R4F experimental cigarettes and commercially available “lite” cigarettes. Cigarette smoke was generated with an automated smoking machine, and mainstream (MS) and sidestream (SS) smoke was collected on spun polyester ventilator filters and extracted in 0.01% triethylamine.

LPS was detected in the tobacco portion of cigarettes (1R4F: 17.8±1.0 [mean±SE]; lite: 26.8±7.3 μg) and in the cigarette filter tips (1R4F: 0.67±0.55; lite: 0.7±0.39 μg). LPS was detected in both MS (1R4F: 37.7±22.2; lite: 11.3±5.7 ng) and SS smoke (1R4F: 1.4±0.53; lite: 21.6±17.5 ng). The LAL activity of MS and SS smoke extracts was attenuated by >80% by alkalization, indicating it was predominantly LPS rather than glucans.

The current workplace permissible exposure limit for endotoxin is 20 ng/m³; assuming 6 L/min per minute ventilation and an 8 h workday, smoking one pack of cigarettes would provide an LPS dose equivalent to 13-day exposure at these LPS levels. We suggest that the bioactive LPS in cigarette smoke may be a major contributor to the pathogenesis of smoking-related lung diseases in susceptible individuals.

Inflammatory Responses in the Lung and Cell Activity Indicators*

Lena Beijer, PhD; Robert R. Jacobs, PhD; and Ragnar Rylander, MD, PhD

There is increasing evidence that symptoms and disease caused by inhalation of organic dust are related to airways inflammation. To evaluate if cellular parameters are related to airways inflammation, subjects were exposed to cotton dust, an organic dust containing bacterial endotoxin (LPS). The subjects were defined as atopic (n=17) or nonatopic (n=25) using a questionnaire. Measurements were made of FEV₁. Responsiveness of blood mononuclear cells was measured by the in vitro LPS-induced production of procoagulant activity (PCA). A series of different subsets of T cells was identified and expressed as percent of the total blood lymphocytes. Thereafter, they were exposed in an experimental cardroom for 5 h. FEV₁ was measured immediately after exposure and a questionnaire was administered to obtain information on subjective symptoms induced by the exposure.

Atopic persons had a more pronounced decrease in pulmonary function after the exposure, in accordance with previous reports (-7.8%±5.5 vs -4.8%±4.2, p<0.05). The LPS-induced PCA production in mononuclear cells correlated to the decrease in FEV₁ (rₓ=0.30, p<0.05) in both atopics and nonatopics. The PCA production was significantly larger in subjects reporting chest tightness. The atopic group had a larger proportion of a specific T-cell subset in the blood, CD8⁺ cells, negative for the monoclonal antibody S6F1, binding an epitope of the LFA-1 adhesion complex. The proportion of this cell related to the dust-induced decrease in FEV₁ (rₓ=0.73, p=0.001). The atopics all had an increased serum level of IgE, specific for inhalant antigens (Phadiatop; Pharmacia; Uppsala, Sweden). In this group a negative relationship was found between the number of CD8⁺ S6F1 cells and IgE (rₓ=−0.49, p=0.05) as well as a negative correlation between the decrease in FEV₁ and IgE (rₓ=−0.49, p=0.05).

The results suggest that functional changes induced by environmental agents with a potential to cause inflammation are related to cell responsiveness and specific cell function in both atopic and nonatopic subjects.

*From the Department of Environmental Medicine, Gothenburg University, Sweden (Drs. Beijer and Rylander); and the University of Alabama, Birmingham (Dr. Jacobs).