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REFERENCES


To the Editor:

I thank Dr. Inoue1 for his thoughtful comments in the June issue of Chest. As he mentions, the exact chemoattractant factors that brought the eosinophils to the lung in this case are not known. Unfortunately, we do not have the ability to reprocess the biopsy and stain for the markers discussed (interleukin-4, interleukin-5, eotaxin). With regard to Dr. Inoue’s question about the biopsy and stain for the markers discussed (interleukin-4, interleukin-5, eotaxin), at least partly, in the pathogenesis of pulmonary toxicities of DEP. In the future, studies of several other components of air pollution may be needed to develop toxicology in the “Air Pollution” section.

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Air Pollution and Pulmonary Diseases

To the Editor:

We would like to add some comments to the review by Smith1 in CHEST (October 2004) demonstrating our recent work. In the

“Air Pollution” section of the article, Smith did not refer to the causal correlation between air pollution and pneumonia. Our in vivo studies2,3 have demonstrated that pulmonary exposure to diesel exhaust particles (DEP), a main contributor of air pollution, aggravates acute lung injury induced by intratracheal administration of bacterial endotoxin. The exaggerated lung inflammation caused by DEP is characterized by increased lung expression of intercellular adhesion molecule-1, interleukin (IL)-1β, macrophage chemoattractant protein-1, keratinocyte chemotactrant, macrophage inflammatory protein-1α, and Toll-like receptors.2 The results indicate that short-term exposure to air pollution has a harmful influence on people with predisposing factors such as pulmonary infections diseases. Ongoing studies3,4 has clarified that residual carbonaceous nuclei of DEP rather than the extracted organic chemicals predominantly contribute to the aggravation of endotoxin-related lung injury in vivo.

More recently, we have demonstrated that short-term pulmonary exposure to quinine, a component of DEP, can induce recruitment of inflammatory cells into the lung, at least partly, through the local expression of IL-5 and eotaxin in vivo.5 Our results indicate that exposure to quinone may play a role, at least partly, in the pathogenesis of pulmonary toxicities of DEP. In the future, studies of several other components of air pollution may be needed to develop toxicology in the “Air Pollution” section.

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