

Correspondence

Reply

To the Editor:

Inoue and Takano¹ rightly emphasize the importance of macrophages in determining the overall epithelial response to environmental particulate matter (PM). Because alveolar macrophages (AMs) phagocytose both carbonaceous PM and *Streptococcus pneumoniae*,² we speculate that adding AMs to our *in vitro* model will attenuate the capacity of PM to stimulate epithelial platelet-activating factor receptor (PAFR) expression. However, this interaction is likely to be complex because phagocytosis of carbonaceous PM by AMs *per se* impairs their ability to kill internalized pneumococci.³ We agree that the way in which PM is delivered to epithelial cells is also an important variable. Indeed, in a previous study we found that very low concentrations of discrete metal nanoparticles causes significant DNA damage to human macrophages when cells are exposed to particles at an air-tissue interface.⁴ Given this complexity, studies of PAFR expression in human subjects exposed to PM *in vivo* are essential for establishing the role of this receptor in mediating vulnerability to pneumococcal infection. From our results,⁵ we predict that subjects with very high exposure to environmental PM (eg, cyclists and smokers) will have increased PAFR expression in lower airway cells. A study of epithelial PAFR expression in bronchial

biopsy specimens obtained from healthy smokers and healthy nonsmokers is currently being done by our group.

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