

Correspondence

Particulate matter–induced hypersusceptibility to infection

To the Editor:

We read with great interest a recent research article by Mushtaq et al.,¹ in which particulate matter (PM) increased the adhesion and invasion of *Streptococcus pneumoniae* to human airway epithelial cells, likely through oxidative stress and platelet-activating factor receptor, implicating PM-induced vulnerability against bacterial infection. We would like to add some comments.

First, as they pointed out in the final paragraph of their discussion, it should be important to assess the effects of inhalable foreign compounds on respiratory immune systems covering global cell populations. In particular, alveolar macrophages are critical for the outcome. Although some studies have implied inhibitory effects of diesel exhaust particles (ie, representative PM in urban areas) on the responsiveness of alveolar macrophages against bacterial endotoxin (LPS),^{2,3} we have previously shown that carbon nanotubes (ie, other types of PM) tend to promote activated alveolar macrophages by LPS to produce/release proinflammatory cytokines.⁴ Thus the *in vitro* effects of inhalable PM on susceptibility against microbial infection should be globally elucidated.

We have experienced a biphasic phenomenon concerning diesel exhaust exposure on lung inflammation induced by LPS *in vivo*: intratracheal administration of diesel exhaust particles exacerbates the acute lung inflammatory response characterized by effector granulocyte infiltration into air spaces with enhanced proinflammatory cytokine expression in the lung,⁵ whereas inhaled diesel exhaust exposure attenuates this response.⁶ These contrast results might be explained by features of the experimental setting, such as exposure type and degree of inflammation. Taken together, the effects of PM on host defense should be investigated carefully and multidimensionally both *in vivo* and *in vitro*.

Another interesting issue is the particle size dependency of the effects. In Fig 5, it seems that there were no size-dependent effects between PM_{2.5} and PM₁₀ in the context of the adhesion of the bacteria to A549 cells. However, we have recently shown that

nanosize particle (average size, 20 nm)–rich diesel exhaust particles can activate both hematopoietic and nonhematopoietic cells *in vitro*,⁷ implicating eradicating potential against xenobiotics.

Therefore, further insight into PM toxicology is required to clarify the size-dependent effects of PM, including nano-level particles, on respiratory immune system responses.

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