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Perspective

Effects of particulate matter on allergic respiratory diseases

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Abstract

The health impact of airborne particulate matter (PM) has long been a concern to clinicians, biologists, and the general public. With many epidemiological studies confirming the association of PM with allergic respiratory diseases, an increasing number of follow-up empirical studies are being conducted to investigate the mechanisms underlying the toxic effects of PM on asthma and allergic rhinitis. In this review, we have briefly introduced the characteristics of PM and discussed its effects on public health. Subsequently, we have focused on recent studies to elucidate the association between PM and the allergic symptoms of human respiratory diseases. Specifically, we have discussed the mechanism of action of PM in allergic respiratory diseases according to different subtypes: coarse PM ($PM_{2.5\text{-}10}$), fine PM ($PM_{2.5}$), and ultrafine PM.

Keywords: Particulate matter; Allergic respiratory diseases; Coarse PM; Fine PM; Ultrafine PM

Physical and chemical characteristics of particulate matter

The growing population density and the rapid urbanization lead to the deterioration of air quality worldwide, especially in China.[1] Atmospheric particulate matter (PM; pl. "particulates"), consisting of both primary and secondary particles, is one of the major air pollutants. Primary PM is generated from road transport, combustion (mainly coal burning), and other industrial processes, whereas secondary PM is generated through chemical reactions among different primary particulates in the atmosphere (e.g., sulfates

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and nitrates, formed from the conversion of SO_2 and NO_2 into their respective acids).[2]

Particulates are classified into three categories according to their aerodynamic diameters: coarse PM ($PM_{2.5-10}$, aerodynamic diameter ranging from 2.5 to 10 μm), which deposits primarily in the primary bronchi[2]; fine PM ($PM_{2.5}$, ranging from 0.1 to 2.5 μm), which can penetrate the alveoli and terminal bronchioles[2]; and ultrafine PM ($PM_{0.1}$ or UFP, less than 0.1 μm), which can cross cell membranes and interact directly with cellular structures.[3,4]

PM toxicity arises from two aspects. First, the particulates can penetrate the gas-exchange region of the lung and thereby infiltrate the circulatory system through layers of alveolar obstruction.[5,6] Second, particulates can absorb many other airborne toxic substances on their surface area, such as heavy metals, polycyclic aromatic hydrocarbons, and organic and inorganic ions.[7]

PM as a risk factor to public health

PM has become a public health concern, having being implicated as the cause of 4.24 million deaths in 2015, 7.8% higher than that in 2005.[8] In 2015, PM ranked 6th on the list of 10 most hazardous factors contributing to global disability-adjusted life-years.[8] Many epidemiological studies worldwide have addressed the correlation between the concentrations of PM and hospital visits due to respiratory diseases (Table 1).[9–16]

PM interacts directly with the human body. Although particulates can be detected in many organs such as the lungs, liver, kidneys, heart, and brain, their deposition patterns show that lungs are the primary site.[17] Because of the heterogeneity in their chemical and physical properties, there is no standard toxic dose for PM. According to Fann et al,[18]even exposure to PM at a concentration below the US national standards poses a significant risk to health.

Because of its substantial health impacts, the World Health Organization Air Quality Guidelines (WHOAQG) have established $PM_{2.5}$ as the indicator of pollution caused by particulates in 2006.[7]

Effects of PM on allergic respiratory diseases

Asthma and allergic rhinitis (AR) are the major types of allergic respiratory diseases, characterized by their similar pathophysiological changes and inflammatory responses.[19] The major risk factors of allergic respiratory diseases are genetic (germline risk loci) and environmental factors (including PM).[20] Although many studies have confirmed that exposure to particulates has significant effects on asthma and AR, the mechanism by which PM influences these diseases is not fully understood. In the following sections, the latest advances that have helped in elucidating the pathogenicity of different PM subtypes are summarized (Fig. 1).

Coarse PM ($PM_{2.5-10}$)

Coarse PM is deposited in extra thoracic airways and induces various symptoms of pulmonary inflammation.[2] Studies have shown the pulmonary toxicity of coarse PM both *in vivo* and *in vitro*.[21–23] We discuss three detailed mechanisms of such toxicity induced by coarse PM (Fig. 1A,B, and E) below.

$PM_{2.5-10}$ activates neutrophils and eosinophils

Diesel exhaust particles (DEPs), a type of coarse PM, exacerbate the symptoms of AR by increasing the levels of the proinflammatory cytokines interleukin (IL)-8, IL-1β, granulocyte-macrophage colony stimulating factor (GM-CSF), and tumor necrosis factor-alpha (TNF-α) in nasal epithelial cells.[24,25] Terada et al[26] claimed that DEPs activate proinflammatory cytokines by upregulating histamine H1 receptor (H1R) expression. The proinflammatory cytokines further induce neutrophils and eosinophils, leading to inflammatory responses.[27] In addition, $PM_{2.5-10}$ was shown to increase the number of IL-5-induced eosinophils in exposed allergic mice.[27]

$PM_{2.5-10}$ induces antigen-presenting cell-mediated inflammatory responses

Becker et al[4] demonstrated that $PM_{2.5-10}$ modulates airway macrophage host defenses by significantly increasing IL-6 levels and suppressing cluster of differentiation (CD) $11b^+$ macrophages in human alveoli. In asthmatic patients exposed to $PM_{2.5-10}$, an inflammatory response was observed with decreased expression of the innate immune receptors CD11b/complement receptor 3 (CR3) and CD64/FcγRI, and the antigen-presenting receptors CD40 and CD86/B7-2, and concomitantly increased expression of the inflammatory receptor CD16/FcγRIII and the low-affinity IgE receptor CD23 in macrophages.[28] DEPs induced murine dendritic cell activity, causing their migration to the mediastinal lymph node, which strengthened the immune response via the nuclear factor-erythroid 2-related factor 2 (Nrf2) signaling pathway.[29]