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Letter to the Editor

Impact of Particulate Matter Exposure Duration and Intensity on Circulating Pro-Inflammatory Cytokines

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Dear Editor-in-Chief

Air pollutants, such as particulate matter (PM), ozone, and carbon monoxide, are global burdens that threaten human health and negatively affect the natural environment and ecosystem. Among the total suspended particles in the atmosphere, PM is an inhalable air pollutant of variable composition and source, classified according to size as coarse (PM10, with a diameter of ≤10 μm), fine (PM2.5, with a diameter of ≤2.5 μm), or ultrafine (PM0.1, with a diameter of ≤0.1 μm) PM (1). The mechanism by which PM adversely affects health remains unclear, but inflammatory responses and the induction of high oxidative stress have been described as the major pathways (1-3).

Dust particles larger than 10 μm are naturally filtered out by the nose or pharynx. In contrast, the immune system is activated in response to the infiltration of smaller sized PM, with immune and/or airway cells secreting cytokines and chemokines that will promote the inflammatory response and oxidative stress, and negatively affect various organs of the body, such as the heart and brain, as well as the respiratory system (2-3). In addition, the secretion of pro-inflammatory cytokines can be increased by the physical influence of PM10 particles themselves or endotoxins, and that PM10 metal components, such as iron, vanadium, and copper, are potentially involved in oxidation-reduction reactions in the body, thereby promoting the secretion of pro-inflammatory cytokines (4). However, it is not clear how much exposure (duration and intensity of exposure) to PM induces an inflammatory response.

Tumor necrosis factor (TNF)-α is mainly produced by activated macrophages and plays an important role in the host response to bacterial infection or malignant tumors. Commonly working along with interleukin (IL)-1β, TNF-α acts as a pro-inflammatory cytokine in response to infection or tumors (5).

Therefore, the purpose of this study was to verify the effect of PM exposure for different durations and intensities on serum TNF-α and IL-1β levels.

Four-week-old C57BL/6 male mice (n=63), from Samtako Bio Korea (Osan, South Korea), were bred in a constant environment of 22 °C, 55 % humidity, and 12-h light/dark cycle, and were used in the experiment after one week of adaptation. Feeding and drinking water were provided *ad libitum* during the experiment. The mice were randomly divided into the saline administration (CON), low-dose PM administration (low-PM), and high-dose PM administration (high-PM)

groups. PM treatment was performed using PM10-like particles (ERM-CZ120; Sigma-Aldrich, St. Louis, MO, USA) with similar composition and concentration to that of environmentally occurring PM10 particles, according to European Reference Materials, which were injected into the tail vein as described by Bai and van Eeden (6).

In the low- and high-PM groups, 7.5 and 15 μg of PM was suspended in 200 μL saline, and 0.25 and 0.5 μg was injected per gram body weight, respectively. For the CON group, 200 μL saline solution was injected. At 4, 6, and 8 weeks of treatment, seven mice per group were anesthetized with ethyl ether, and blood was collected from the abdominal inferior vena cava for TNF-α and IL-1β analysis using ELISA kits (R&D Systems, Minneapolis, MN, USA).

The study protocol was approved by the animal Ethics Committee of the National Research Foundation of Korea (NRF-2020S1A5B5A16083863).

Differences between the experimental groups were verified through one-way analysis of variance using SPSS software (version 26.0; IBM Corp., Armonk, NY, USA). Statistical significance was set to α=0.05.

Overall, serum TNF-α levels were significantly higher at 8 weeks than at 4 and 6 weeks in both the low- and high-PM groups (P<0.05) (Fig. 1). In addition, serum IL-1β levels were significantly higher at 8 weeks than at 4 weeks in the high-PM group (P<0.05).

These results suggest that the secretion of circulating pro-inflammatory cytokines may induced by PM exposure in a dose- and time-dependent manner.

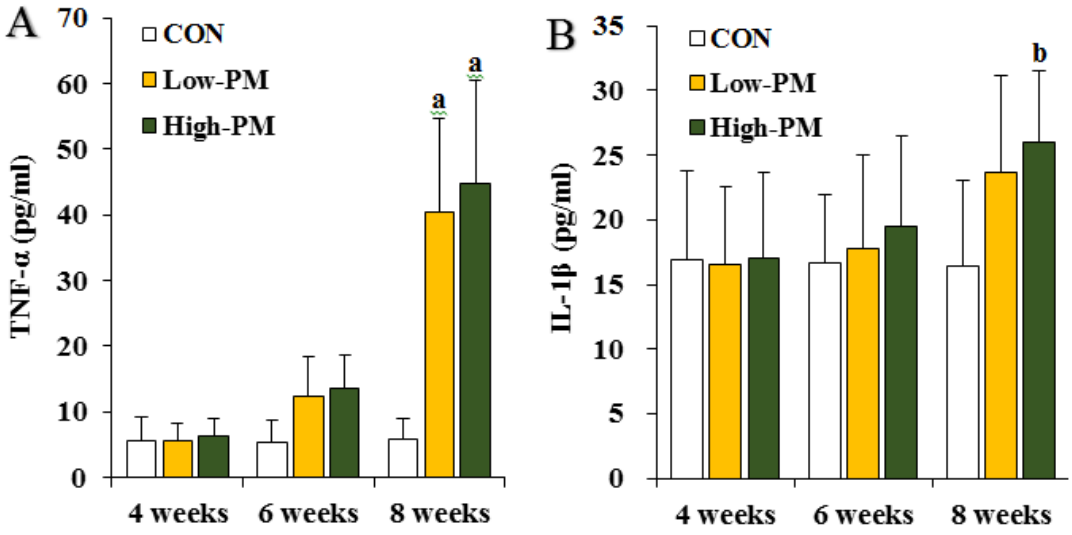

Fig. 1: Changes in serum TNF-α (A) and IL-1β (B) levels induced by different PM exposure durations and intensities. Data are presented as mean ± standard deviation. CON, saline administration group; Low-PM, low-dose PM administration group; High-PM, high-dose PM administration group; [a]Significant difference with 4 and 6 weeks (P<0.05); [b]Significant difference with 4 weeks (P<0.05)

Conflict of interest

The authors declare that there is no conflict of interest.

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