Traffic Derived Particulate Matter Triggers Ocular Surface Inflammation in Murine Model

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Extended Abstract

Objective: According to the recent industrialization and urbanization, air pollution has been identified as a major risk factor for respiratory, cardiovascular, and skin diseases and increased morbidity and mortality worldwide in these days [1, 3]. Polluted air causes subclinical inflammation and dryness on the ocular surface. Previous studies have focused on air pollution from fine particles (PM2.5; particulate matter less than 2.5 mm in aerodynamic diameter) [3,4,5]. The aim of this study is to evaluate the mechanism of environmental eye disease caused by the exposure to airborne PM2.5 using animal experiment models.

Methods: 7-8 week old C57BL/6 mice were exposed with topical application of PM2.5, PM10, and saline vehicle to induce experimental environmental eye disease model. Corneal fluorescein staining and the number of corneal CD11b+ cells were assessed in the different groups. Expression of IL-1β, IL-6, tumor necrosis factor (TNF)-α, IL-17A, and MUC5AC, were evaluated by real-time PCR in the corneas at day 5 or 14. TUNEL assay was used to evaluate apoptosis of corneal epithelial cells.

Results: Treatment with PM2.5 and PM10 showed a significant increase in corneal fluorescein staining compared with the vehicle, but not statistically different in between PM2.5 and PM10 group. A significant increase in the number of CD11b+ cells was observed in PM2.5 and PM10 treated eyes, compared with the vehicle in the central corneas. Exposure with PM2.5 and PM10 was associated with a significant increase in the corneal expression of IL-1β, IL-6, IL-17, and TNF compared to the vehicle, and increased maturation of APCs in drainage LNs. In addition, cellular toxicity to the corneal epithelium was observed with PM2.5 and PM10.

Conclusions: Exposure to traffic derived PM induced ocular surface damage and inflammation, which induce the maturation of APCs in drainage cervical LNs.

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References


