

Effects of PM2.5 Exposure on Cerebral Circulation and Prognosis of Ischemic Stroke.

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[Abstract]

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It has recently become epidemiologically clear that exposure to PM2.5 exacerbates the prognosis of cerebral infarction. PM2.5 exposure has been reported to increase poststroke hospital stay by 2-5 days or increase mortality within 1 year after ischemic stroke. The purpose of this study was to clarify the effect of PM2.5 exposure on stroke prognosis from a toxicological point of view and to challenge the mechanistic insight. Because inflammation and edema are observed in the acute stage of cerebral infarction and because polycyclic aromatic hydrocarbons (PAHs) are known to induce inflammation, we focused on PAHs contained in PM2.5.

Nasal exposure of mice to Beijing air dust CRM28 activated microglia in the cerebral cortex, leading neuroinflammation. Neuroinflammation, edema and motor dysfunction after ischemic stroke was exacerbated by CRM28 exposure. Exposure to PM2.5 collected in Yokohama also exacerbated the prognosis of ischemic stroke. Exposure of aromatic hydrocarbon receptor (AhR) KO mice to CRM28 did not induce inflammation as well as motor dysfunction after ischemic stroke. Given that PAHs attached to CRM28 are associated with exacerbation of prognosis of ischemic stroke.

PM2.5 collected in Yokohama contained more endotoxin than that collected in Fukuoka. Exposure to PM2.5 collected in Yokohama exacerbated the prognosis of ischemic stroke, while exposure to PM2.5 collected in Fukuoka did not. When Yokohama PM2.5 was mixed with an endotoxin-neutralizing reagent, polymyxin B, the aggravation of the prognosis observed with Yokohama PM2.5 exposure was greatly suppressed. Therefore, endotoxin contained in PM2.5 is also considered to be involved in worsening the prognosis of ischemic stroke as similar to PAHs.

Collectively, PM2.5 exposure causes neuroinflammation and aggravates the prognosis of ischemic stroke. We identified PAHs and endotoxins in PM2.5 as aggravating factors. It is known that the PAH concentration in PM2.5 has seasonality, and in addition, this study revealed that the endotoxin concentration in PM2.5 has regional characteristics. To evaluate the neurological effects of PM2.5, it is necessary to consider the PAHs and endotoxin contents in PM2.5; the regional and seasonal characteristics of PM2.5 can determine the neuronal health impact.

[References]

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