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Association between PM2.5 exposure by inhalation and brain damages of Alzheimer's disease in transgenic mice

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Title

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ABSTRACT

Background: Fine particulate matter ($PM_{2.5}$) exposure increases the risk of neurological disorders. However, the relevance between $PM_{2.5}$ and Alzheimer's disease (AD) needs to be identified and the effect of $PM_{2.5}$ exposure on the brain in AD mice remains unclear.

Objective: To assess the effects of $PM_{2.5}$ exposure on AD and investigate the brain damage in AD transgenic mice exposed to $PM_{2.5}$.

Methods: We searched articles from the database of PubMed for meta-analyses on the association between $PM_{2.5}$ exposure and AD. Further, using a novel real-world whole-body inhalation exposure system, wild type (WT) and APP/PS1 transgenic mice (AD mice) were respectively exposed to filtered air (FA) or ambient $PM_{2.5}$ for 8 weeks in Taiyuan, China. The pathological and ultrastructural changes and levels of Aβ-42, TNF-α, and IL-6 in brains in FA-WT mice, FA-AD mice, FA-$PM_{2.5}$ mice, and $PM_{2.5}$-AD mice were measured.

Results: Long-term $PM_{2.5}$ exposure had the association with increased risks of dementia and AD by OR of 1.16 (95% CI 1.07–1.26) and 3.26 (95% CI 0.84–12.74) via meta-analysis. Both lightly- and heavily polluted countries showed such increased risks. In the open field test, the $PM_{2.5}$-AD mice

showed more significant degenerative symptoms of AD by the behavioral change in movement. Hematoxylin-eosin staining results showed that noticeable histopathological injury such as structural disorder, hyperemia, and sporadic inflammatory cell infiltration in the brain of $PM_{2.5}$-AD mice, and transmission electron microscope results displayed that serious damage in the brain in $PM_{2.5}$-AD mice, which maintained disorder of cristae and vacuolation of mitochondria, synaptic abnormalities, and loose myelin sheaths. Aβ-42, TNF-α and IL-6 levels in brains of $PM_{2.5}$-AD mice had raised more strongly than that of FA-WT or FA-AD mice.

Conclusion: This study indicated a strong association between $PM_{2.5}$ exposure and AD risks. $PM_{2.5}$ significantly aggravated the severity of neuronal pathomorphological changes and inflammation in AD mice when Aβ-42 levels in the brain were visibly increased.

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