Effects of oxidative stress and inflammation induced by ultrafine particles on Alzheimer disease onset

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Objectives: The aim of this work was to evaluate the activation of oxidative stress and inflammation in mice exposed to ultrafine particles (UFPs), and to elucidate putative physiopathological correlations with neurodegeneration.
Materials: UFPs derived from two different anthropogenic sources were used: DEP, from EURO 4 diesel engine emission, and BC, from pellet boiler biomass emission (ENEA).
Method: Male BALB/c mice were submitted to single or repeated intratracheal instillation of DEP and BC (50Âµg); in parallel, control mice were always considered (sham). Cortex, cerebellum and hippocampus from sham and treated mice were screened for markers of inflammation and oxidative stress (iNOS, HO-1), endoplasmic reticulum stress (Hsp70), a putative pro-carcinogenic marker (Cyp1B1) and markers related to the onset of Alzheimer Disease (AD) (P-Tau, Tau, P-APP, APP, BACE1).
Results: After both single and repeated DEP and BC instillation, we observed an increase in iNOS, HO-1 and Hsp70 levels in all districts, while Cyp1B1 and markers of neurodegeneration changed only after repeated treatment.
Discussion: The single DEP and BC instillation induced oxidative stress, endoplasmic reticulum stress and inflammation, while typical AD-related proteins seemed not affected. The repeated instillation was able to sustain oxidative stress and inflammation; moreover, it induced increase of BACE1 levels and changes in Tau and APP total levels and phosphorylation. Finally, BC seemed to be less effective than DEP.
Conclusions: Our results suggest UFPs or inflammatory mediators translocation from lungs, and their effects on the brain, after both acute and repeated DEP and BC treatment.
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Topic: neurobiology

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