Title *

Inhaled ultrafine particulate matter affects microglial morphology and learning and memory behavior in an Alzheimer's disease mouse model

Abstract *

Alzheimer's disease (AD) is a progressive neurodegenerative disease with hypothesized causal links to environmental factors such as air pollution. Ambient ultrafine particles (UFPs, <100 nm in diameter) deposit efficiently in the respiratory tract, translocate to secondary target organs like the brain, and induce inflammation. We hypothesize that exposure to UFPs can accelerate AD progression and cognitive decline by enhancing inflammatory processes in the brain. Cohorts of young and old transgenic male AD mice (3xTgAD; 2.5-3 or 12.5-13 mos) were exposed to concentrated ambient UFPs (HUCAPS) or filtered air for 2 weeks (4 hours/day, 4 days/week). Over several months post-exposure, young mice underwent radial arm maze (RAM), novel object recognition (NOR), and locomotor function testing. HUCAPS exposure diminished the rate of spatial learning independent of genotype across 43 sessions of RAM testing. While NOR testing did not show exposure-related changes in recognition memory, basal locomotor activity is different in NTg and 3xTgAD mice and HUCAPS exposure diminished exploratory activity specifically in 3xTgAD mice. In the old mice, HUCAPS exposure was found to affect the morphology of amyloid plaque-associated microglia in the subiculum (Sholl analysis) of 3xTgAD mice such that they became more ramified and closer in morphology to cells from NTg mice. There was also a trend towards higher levels of insoluble amyloid-β42 in hippocampi from HUCAPS-exposed as compared to air-exposed 3xTgAD mice. These findings suggest that UFP exposure may impact microglial function and cognition in an AD mouse model.

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Affiliations and Authors *

Author Information
Alison Elder (Presenting)

Affiliations
Deparment of Environmental Medicine, University of Rochester, Rochester, USA

Author Information
Katrina Jew

Affiliations
Deparment of Environmental Medicine, University of Rochester, Rochester, USA

Author Information
Denise Herr

Affiliations
Deparment of Environmental Medicine, University of Rochester, Rochester, USA

Author Information

Andrea Kennell

Affiliations
Deparment of Environmental Medicine, University of Rochester, Rochester, USA

Author Information
David Chalupa

Affiliations
Deparment of Environmental Medicine, University of Rochester, Rochester, USA

Author Information
Robert Gelein

Affiliations
Deparment of Environmental Medicine, University of Rochester, Rochester, USA

Author Information
Deborah Cory-Slechta

Affiliations
Deparment of Environmental Medicine, University of Rochester, Rochester, USA

Author Information
Jack Walter

Affiliations
Deparment of Neuroscience, University of Rochester, Rochester, USA

Author Information
M. Kerry O'Banion

Affiliations
Deparment of Neuroscience, University of Rochester, Rochester, USA

Author Information
Günter Oberdörster

Affiliations
Deparment of Environmental Medicine, University of Rochester, Rochester, USA