# Does air pollution increase risk of AD in a genetically susceptible animal model?

https://neurodegenerationresearch.eu/survey/does-air-pollution-increase-risk-of-ad-in-a-genetically-susceptible-animal-model/

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# Contact information of lead PI Country

USA

# Title of project or programme

Does air pollution increase risk of AD in a genetically susceptible animal model?

# Source of funding information

NIH (NIA)

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01/06/2016

# **Total duration of award in years**

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# **Keywords**

traffic-related air pollution, Alzheimer's disease risk, Air Pollution, Alzheimer's Disease, Vehicle Emissions

# **Research Abstract**

? DESCRIPTION (provided by applicant): Alzheimer's disease (AD) is the most prevalent agerelated neurodegenerative disease in the United States. More than 90% of cases are idiopathic and there is growing consensus that environmental factors interact with genes of susceptibility to influence the age of onset and progression of this disease. Recent epidemiological studies have reported a positive correlation between exposure to traffic-related air pollution and the occurrence of the hallmark clinical characteristics of AD, including increased expression of proinflammatory markers in the brain, diffuse amyloid plagues, neuronal cell loss, and impaired cognition. We hypothesize that traffic- related air pollution emitted from motor vehicles triggers inflammatory responses in the brain that initiate or accelerate the progression of AD. To test this, we will use a unique animal model: the TgF344 rat, which expresses human genes that confer susceptibility to AD. We will expose male and female TgF344 rats and their wildtype littermates to polluted air sampled directly from the Caldecott tunnel in the San Francisco area beginning at postnatal day 28 to up to 12 months of age. Caldecott tunnel air will be delivered to animals housed in a portable vivarium parked adjacent to the tunnel. Control animals will be exposed to clean filtered air. Caldecott tunnel air will be collected every three weeks for gas and particle chemical characterization, and both Caldecott tunnel air and clean filtered air will be monitored continuously for particle size distribution and concentration. Learning and memory as well as neuroinflammation and AD-like pathology will be assessed in animals at 3, 6, 9 and 12 months of age. These studies will provide proof-of-concept data identifying functionally relevant interactions between AD-linked genetic susceptibilities and a ubiquitous environmental risk factor.

## Further information available at:

Types:

Investments < €500k

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United States of America

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**Database Categories:** 

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