Alzheimer's disease (AD) is a neurodegenerative disease, neuropathologically characterized by extracellular amyloid plaques and intracellular neurofibrillary tangles. Numerous data suggest that inflammatory processes in the brain may have an important role in AD. Allergy is a chronic inflammatory disease affecting more than 20% of the Western population. The effects of allergic conditions on brain functions are largely unknown, but a recent longitudinal study in humans showed an association between a history of allergy and development of dementia.

The aim of this study was to investigate whether chronic peripheral inflammation associated with allergy affects the expression of AD-related proteins and inflammatory markers in the brain, using a model for chronic airway allergy in Balb/c mice, with ovalbumin as allergen. Airway inflammation was evaluated by presence of eosinophilia in the bronchoalveolar lavage fluid, BAL.

Allergic animals were found to have increased phosphorylation of tau protein in the parietal cortex and hippocampus, as determined by immunoblotting with both AT8 and AT180 antibodies. Also, brain levels of tau-kinases, ERK and cdk5, were increased in the allergic animals. Moreover, allergic mice have increased brain levels of both immunoglobulin (Ig) G and IgE, and increased levels of the cytokines IL-5 and IFNγ in hippocampus, and IL-10 in parietal cortex.

The present data supports that allergy-dependent chronic peripheral inflammation modifies the brain inflammatory status and influences phosphorylation of AD-related proteins, indicating that allergy may be yet another factor to be considered for the development and/or progression of neurodegenerative diseases such as AD.