Targeting inflammatory pathway reduces Alzheimer's disease in mice

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Alzheimer's disease (AD) is the most common form of dementia and is characterized by the formation of β-amyloid plaques throughout the brain. Proteins known as chemokines regulate inflammation and the immune response. In both patients with AD and mouse AD models, the chemokine CXCL10 is found in high concentrations in the brain and may contribute to AD.

A new study in the *Journal of Clinical Investigation* indicates that activation of the CXCL10 receptor, CXCR3, contributes to AD pathology. Using a murine model of AD, Michael Heneka and colleagues at the University of Bonn found that mice lacking CXCR3 had reduced β-amyloid plaque formation. Importantly, loss of CXCR3 signaling in AD mice attenuated behavioral deficits.

The results of this study suggest that CXCR3 should be explored as a potential therapeutic target for AD.

More information: CXCR3 promotes plaque formation and behavioral deficits in an Alzheimer's disease model, *J Clin Invest*. DOI: 10.1172/JCI66771

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