

Understanding the molecular mechanisms underlying Alzheimer's disease

10 June 2013

The accumulation of amyloid- β (A β) in the brains of Alzheimer's disease (AD) patients is known to be associated with memory loss and neuronal degeneration, but the mechanism of A β pathogenesis is not fully understood.

In this issue of the *Journal of Clinical Investigation*, researchers led by Yong-Keun Jung at Seoul National University demonstrate that A β binds to a [cellular protein](#) known as FC γ R11b.

Greater levels of FC γ R11b were detected in the brains of AD patients. Binding of A β to FC γ R11b activated cell stress and death pathways. In a mouse model of AD, depletion of FC γ R11b ameliorated memory impairment.

This study demonstrates that FC γ R11b plays a critical role in AD pathogenesis.

More information: Fc γ R11b mediates amyloid- β neurotoxicity and memory impairment in Alzheimer's disease, *J Clin Invest*.
[doi:10.1172/JCI66827](https://doi.org/10.1172/JCI66827)

Provided by Journal of Clinical Investigation

APA citation: Understanding the molecular mechanisms underlying Alzheimer's disease (2013, June 10) retrieved 6 September 2022 from <https://medicalxpress.com/news/2013-06-molecular-mechanisms-underlying-alzheimer-disease.html>

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