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FcgRIIb mediates amyloid- β neurotoxicity and memory impairment in a model of Alzheimer's disease

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Amyloid- β ($A\beta$) induces neuronal loss and cognitive deficits and is believed to be a prominent cause of Alzheimer's disease (AD). However, the cellular mechanism of the pathogenesis is not fully understood. Here we report that Fcg-receptor IIb (FcgRIIb) mediates $A\beta$ neurotoxicity and neurodegeneration. We found that FcgRIIb is significantly up-regulated in the hippocampus of AD brains and neuronal cells exposed to $A\beta_{1-42}$. Neuronal FcgRIIb activates ER stress and caspase-12, and FcgRIIb knockout primary neurons are resistant to $A\beta_{1-42}$ -induced cell death in vitro. FcgRIIb deficiency ameliorates $A\beta_{1-42}$ -induced inhibition of long-term potentiation and inhibits the reduction of synaptic density by naturally secreted $A\beta$. Moreover, genetic depletion of FcgRIIb rescues the memory impairments in AD model mice. In an action mode of FcgRIIb in $A\beta$ neurotoxicity, we found that soluble $A\beta_{1-42}$ oligomers interact with FcgRIIb in vitro and in AD brains, and inhibition of their interaction blocks $A\beta_{1-42}$ neurotoxicity. Thus, we conclude that FcgRIIb has an aberrant but essential role in $A\beta_{1-42}$ -mediated neuronal dysfunction, providing insight into $A\beta$ neuropathology.

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