Amyloid Beta Postsynaptic Signaling through AKAP-anchored Calcineurin

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Research Abstract

Project Summary Abstract Amyloid Beta Postsynaptic Signaling through AKAP-anchored Calcineurin A? overproduction from APP is believed to contribute to impaired synaptic plasticity and decreased cognitive function in Alzheimer’s disease (AD). Individuals with Down syndrome (DS; trisomy 21) have an extra copy of APP that predisposes them to early-onset AD. Thus, elucidating how A? inhibits plasticity is important for understanding cognitive impairments

associated with the development of dementia in AD and DS and could identify novel drug targets, diagnostics, and therapies. Rodent model studies indicate that calcineurin (CaN) phosphatase signaling could contribute to altered LTP/LTD synaptic plasticity, dendritic spine loss, and learning and memory impairments in AD. A?-induced spine loss may be further linked to altered gene expression through CaN activation of the transcription factor NFAT. Here we propose to test the novel hypotheses that AKAP79/150-CaN anchoring is required for A? activation of CaN signaling that regulates the balance between LTP/LTD signaling and NFAT transcription associated with dendritic spine/synapse loss.

Further information available at:

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