

ORAL PRESENTATION

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Mechanisms of A β induced synaptic toxicity

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Increasing evidence favors the synapse as one of the initial sites of neuronal damage by amyloid β -protein (A β) and such synaptic damage is thought to underlie the cognitive deficits seen in Alzheimer's disease. A decade ago, Roberto Malinow and colleagues published a seminal paper proposing that neuronal activity augments processing of the amyloid precursor protein (APP) by β - and γ -secretases to enhance A β production and release. In turn, A β then depresses synaptic activity. Thus, an interesting positive feed forward and negative feedback loop is created during periods of neuronal activation. Since publication of this report, a number of elements within this intriguing pathway have been confirmed by other laboratories in both the *in vitro* and *in vivo* settings. Specifically, there is compelling evidence that neuronal activity is associated with enhanced A β generation and subsequent amyloid deposition. Similarly, A β has been shown by multiple investigators to depress synaptic activity and synaptic plasticity. In this presentation, I will summarize some recent work examining the mechanisms that underlie both pathways. In particular, among the many proposed pathways of A β toxicity, I will discuss the potential involvement of APP in A β -initiated synaptic damage.

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