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Research report

Soluble oligomers of Î² amyloid (1-42) inhibit long-term potentiation but not long-term depression in rat dentate gyrus

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Abstract

The dementia in Alzheimer disease (AD) is usually attributed to widespread neuronal loss in conjunction with the pathologic hallmarks of intracellular neurofibrillary tangles and extracellular plaques containing amyloid (AÎ²) in fibrillar form. Recently it has been demonstrated that non-fibrillar assemblies of AÎ² possess electrophysiologic activity, with the corollary that they may produce dementia by disrupting neuronal signaling prior to cell death. We therefore examined the effects of soluble oligomers of $AÎ²_{1-42}$ on long-term potentiation (LTP) and long-term depression (LTD), two cellular models of memory, in the dentate gyrus of rat hippocampal slices. Compared with vehicle controls, slices pre-incubated 60 min in the presence of AÎ²-derived diffusible ligands (ADDLs) showed no differences in threshold intensity to evoke a synaptic response, slope of field

excitatory post-synaptic potentials (EPSPs), or the input/output function. Tetanus-induced LTP and reversal of LTD were strongly inhibited in ADDLs-treated slices whereas LTD was unaffected. These data suggest that soluble non-fibrillar amyloid may contribute to the pathogenesis of AD both by impairing LTP/memory formation at the cellular level and by creating â€˜neuroplasticity imbalanceâ€™ manifested by unopposed LTD in the setting of impaired capacity for neural repair via reversal of LTD or LTP.

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Keywords

Neuroplasticity; Long-term potentiation; Long-term depression; Dentate gyrus; AÎ²-derived diffusible ligand

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