

A new neuronal target for beta-amyloid peptide in the rat hippocampus

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Abstract

In Alzheimer's disease, amyloid beta peptide (A) accumulation is associated with hippocampal network dysfunction. Intrahippocampal injections of A induce aberrant inhibitory septohippocampal (SH) network activity in vivo and impairment of memory processing. In the present study, we observed, after hippocampal A treatment, a selective loss of neurons projecting to the medial septum (MS) and containing calbindin (CB) and/or somatostatin (SOM). Other GABAergic neuronal subpopulations were not altered. Thus, the present study identifies hippocamposeptal neuron populations as specific targets for A deposits. We observed that in A-treated rats but not in controls, glutamate agonist application induced rhythmic bursting in 55% of the slow-firing neurons in the medial septum. This suggests that hippocampal A can trigger modifications of the septohippocampal pathway via the alteration of a specific neuronal population. Long-range hippocamposeptal GABA/calbindin neurons, targets of hippocampal amyloid deposits, are implicated in supporting network synchronization. By identifying this target, we contribute to the understanding of the mechanisms underlying deleterious effects of A, one of the main agents of dementia in Alzheimer's disease.

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