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AN ANIMAL MODEL OF ALZHEIMER'S DISEASE I : BEHAVIORAL AND HISTOLOGICAL ASSESSMENT FOLLOWING INTRAHIPPOCAMPAL INJECTIONS OF A B-AMYLOID 25-35 IN THE RAT

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Perhaps no other pathology has caused more interest in the neuroscientific community in recent years than Alzheimer's disease (AD). Alzheimer's disease is a neurodegenerative disease and the major cause of dementia in North America. Moreover, it is presently the 4th major cause of death in the U.S. The total annual cost to the nation for the care of AD patients is estimated at 90 billion, including medical and nursing home care, social services, and early death. More importantly, for victims and their loved one, the dementias are devastating. Thus AD represents a formidable challenge for the research community. While the mechanism of neuronal atrophy in AD is unknown, pathologically AD is characterized by extracellular deposition of neuritic plaques (NP) and a generation of neurofibrillary tangles typically found in the cerebral cortex, hippocampus and basal forebrain. Accumulating evidence suggests that the major constituent of NP, a beta-amyloid protein composed of 39-42 amino acids, possesses neurotoxic properties. Presently, nothing is known regarding the effects of intracerebral injections of beta-amyloid on memory. In this study the effects of a variety of doses of beta-amyloid on spatial memory were assessed following bilateral injections into the hippocampus. Memory deficits (working and reference) were assessed in a 8-arm radial arm maze. Preliminary results indicate that there is a clear disruption of learning in the experimental animals. These results, along with a preliminary analysis of the cytoarchitecture of hippocampal and basal forebrain neurons will be presented.