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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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AN ANIMAL MODEL OF ALZHEIMER'S DISEASE I: BEHAVIORAL AND
HISTOLOGICAL ASSESSMENT FOLLOWING INTRAHIPPOCOMPAL
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.