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TOWARD AN UNDERSTANDING OF ALZHEIMER'S DISEASE VII:
THE EFFECTS OF β A(1-42) AND IBOTENIC ACID ON THE
RETENTION OF A SPATIAL LEARNING TASK IN RATS FOLLOWING
MULTIPLE INJECTIONS INTO THE HIPPOCAMPUS

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Neuropathologically, Alzheimer's disease (AD) is characterized by neuritic plaques and neurofibrillary tangles. Evidence has suggested that a protein called β -amyloid (β A) is a major component of the neuritic plaques and may play a role in the neurodegeneration seen in AD. The cellular mechanisms by which β A induces neurotoxicity, however, are still unclear. Recent evidence suggests that the aggregational state of β A may be relevant to its neurotoxicity. Whether portions of the β A protein or the entire sequence produces neurotoxicity in neurons, however, remains a controversy. Still another controversy is whether β A is directly neurotoxic to neurons or whether it increases the vulnerability of neurons. Recent evidence reported by Dorman, Kang, McCampbell and Kang, that injections of β A(25-35) with a low dose of ibotenic acid into the hippocampus did disrupt the acquisition of spatial learning in the rat, supports the vulnerability hypothesis. They suggest that the synergistic effect between β A and ibotenic acid may have produced the neurotoxic effect. In light of recent evidence, reported at this conference, that injections of β A(1-42) alone did not disrupt the retention of a spatial learning task, in this study we assessed the increased vulnerability hypothesis by coinjecting β A(1-42) with a subthreshold dose of ibotenic acid into the hippocampus of male rats. Another problem related to β A's neurotoxicity may concern the extent of hippocampal damage it produces. Therefore, we will assess the effects of multiple injections of β A(1-42) and ibotenic acid into the hippocampus of male rats. The results will be presented at the conference.