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An Animal Model of Alzheimer's Disease II: Assessment of the Aggregational Properties of Beta-Amyloid (25-35) on Learning Following Intrahippocampal Injections of Long Evans Male Rats

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Alzheimer's Disease is a neurodegenerative disorder that is behaviorally typified by dementia, memory loss, and learning impairment. Presently, Alzheimer's Disease is the fourth major cause of death in the United States. Although the cause of Alzheimer's remains unknown, there is widespread agreement that the cortex and hippocampus seem to be selectively targeted. For example, neurons in the hippocampus degenerate and are lost in significant numbers as Alzheimer's progresses. On a neurohistological level, neuritic plaques and neurofibrillary tangles are often found in postmortem examination of Alzheimer's patient's brains. Recent research has implicated a protein called beta-amyloid as a core component of the neuritic plaques. One interesting aspect of beta-amyloid is that it has been reported to possess both neurotrophic and neurotoxic effects *in vivo* and *in vitro*. The neurotoxic effects appear to be potentiated by the high self-aggregational property of beta-amyloid. A study being run concurrently that will be reported at this conference (see David Kang's abstract) is investigating the role of beta-amyloid and its effects on learning performance in Long Evans male rats. The purpose of this investigation will be to extend the findings of the above study by comparing the behavioral effects of beta-amyloid (25-35) and non-incubated beta-amyloid (25-35). Animals received bilateral injections into the hippocampus of incubated beta-amyloid (25-35). The rats were assessed behaviorally using two valid measures of memory performance, the radial arm maze and the Moris water maze. The results of these tests will be presented at the conference.