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Apr 13th, 11:15 AM - 11:30 AM

Effects of Chronic Injections of the Amyloid Fragment, β A (25-35) into the Medial Septal Area on Learning and Memory in the Male Rat

Greg Tinkler

Illinois Wesleyan University

Wayne Dornan, Faculty Advisor

Illinois Wesleyan University

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Oral Presentation 1.4

**EFFECTS OF CHRONIC INJECTIONS OF THE AMYLOID
FRAGMENT, β A(25–35) INTO THE MEDIAL SEPTAL AREA ON
LEARNING AND MEMORY IN THE MALE RAT**

Greg Tinkler and Wayne Dornan*, Department of Psychology, IWU

Alzheimer's Disease (AD), a neurodegenerative disorder associated with loss of neurons in the brain, is the most frequent cause of dementia in the elderly, accounting for more than 20 million cases worldwide. Despite a 20 fold increase in the number of reported deaths between 1979 and 1993, presently there is no cure or treatment for AD. 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. In a previous study done in our laboratory (Neuroreport 1993), we reported that bilateral injections of β A(25–35) into the hippocampus together with a subthreshold dose of IBO (which by itself has no neurotoxic effects) produced a dramatic disruption in the acquisition of spatial learning in the rat. In contrast, bilateral injections into the hippocampus of two different doses of β A(25–35) or the incubated form of β A(25–35) failed to significantly affect maze acquisition in the rat. Therefore, research done in our laboratory has failed to reveal any effects on spatial learning and memory in the rat following intrahippocampal injections of β A(25–35) alone. Collectively, this suggests that β A(25–35) is not directly neurotoxic to hippocampal neurons, but either increases their vulnerability to further insult or acts upon other neurons which synapse upon the hippocampus. An alternative hypothesis is that since the accumulation of amyloid plaques is a gradual process, single injections of β A(25–35) might not be expected to induce significant hippocampal damage. Recently it has been reported medial septal injections of β A(25–35) induced a significant reduction of hippocampal choline acetyltransferase (ChAT) without significantly altering the number of non-cholinergic neurons projecting to the hippocampus. This depletion in ChAT was significant on the seventh day postinjection, but had disappeared by day 21. However, no behavioral tests were performed to determine whether any impairments in learning and memory could be correlated to the degree of depletion. The aim of this study was to assess whether multiple injections of β A(25–35) into the medial septal area would cause a sustained reduction in acetylcholine input to the hippocampus and produce a concomitant disruption in learning and memory.