



Apr 13th, 9:00 AM - 10:30 AM

Immunolesions of the Cholinergic Medial Septal Area Induced by Injections of 192 IGG Saporin Have No Appreciable Effect on Spatial Learning in the Male Rat

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Poster Presentation 33

IMMUNOLESIONS OF THE CHOLINERGIC MEDIAL SEPTAL AREA
INDUCED BY INJECTIONS OF 192 IGG SAPORIN HAVE NO
APPRECIABLE EFFECT ON SPATIAL LEARNING IN THE MALE
RAT

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Alzheimer's Disease (AD) is a progressive, neurodegenerative disorder that has already reached epidemic proportions. Indeed, AD is the fourth leading cause of death in adults, after heart disease, cancer and stroke, and is the most common form of dementia. Although most people diagnosed with AD are older than 65, AD can occur in people in their 40s and 50s. Over the past 2 years research in this laboratory has focused on developing an animal model of AD by mimicking the loss of the basal forebrain cholinergic projection using a variety of lesion techniques, and to determine how the loss of the fibers affect learning and memory in the rat. While a considerable amount of evidence has implicated the loss of cholinergic basal forebrain (CBF) input to the hippocampus and cortex as being one of the major neuropathological components characteristic of AD, the exact role the CBF system plays in the cognitive deficits observed in Alzheimer's disease is still uncertain. One factor that has contributed to this ambiguity is that until recently the lack of a specific cholinergic neurotoxin has hindered attempts to selectively destroy the cholinergic input to the cortex and hippocampus. As a result, there is considerable excitement regarding the introduction of the new cholinergic toxin, 192 IgG saporin as a potential new tool in generating an animal model that mimics the profound memory impairment that characterizes Alzheimer's disease. Unilateral injections of 192 IgG saporin into the lateral ventricles induces within 3–5 days, a 80–90% reduction of acetylcholine levels in the cortex and hippocampus. In this study animals received three stereotaxic injections of 192 IgG saporin into the medial septal area and the effects of these injections on a variety of spatial learning tasks were assessed. Our preliminary results indicate that intracerebral injections of 192 IgG saporin failed to appreciably effect the retention or acquisition of a spatial learning task despite a considerable depletion of acetylcholine. The significance of these results will be presented at the conference.