Apr 13th, 11:30 AM - 11:45 AM

Effects of Adrenergic and Cholinergic Pharmacological Challenges on Radial Arm Maze Performance in Male Rats with 192 IGG Saporin Induced Lesions of the Basal Forebrain

Lesley Hickman
Illinois Wesleyan University

Wayne Dornan, Faculty Advisor
Illinois Wesleyan University

Oral Presentation 1.5

EFFECTS OF ADRENERGIC AND CHOLINERGIC PHARMACOLOGICAL CHALLENGES ON RADIAL ARM MAZE PERFORMANCE IN MALE RATS WITH 192 IGG SAPORIN INDUCED LESIONS OF THE BASAL FOREBRAIN

Lesley Hickman and Wayne Dornan*, Dept. of Psychology, IWU

Alzheimer's Disease (AD) is a neurodegenerative disease that currently afflicts over 4 million people in the United States, with roughly 100,000 new cases reported every year. This disorder causes gradual deterioration of cognitive functions, particularly learning and memory. Pathologically, AD is manifested by the appearance of neuritic plaques and neurofibrillary tangles, and also by the progressive deterioration of the cortex and septohippocampal pathway, which supplies the hippocampus and cortex with cholinergic fibers. Currently, there is no effective treatment for this disease. Development of drug therapies is hindered by the lack of an animal model of AD that mimics both the pathological and behavioral deficits present in AD. The goal of our lab is to create such a model in the male rat by lesioning the areas that supply acetylcholine, thus imitating the cholinergic degeneration seen in AD. These areas are the nucleus basalis magnocellularis (NBM), which innervates the cortex and amygdala, and the medial septal area (MSA), which innervates the hippocampus. We used a highly specific neurotoxin called 192–IgG–Saporin, a toxin coupled to an antibody directed against a receptor found only on cholinergic neurons. The lesions appeared to have no effect on retention of a previously learned radial arm maze (RAM) memory task (5 arms baited), or acquisition of a task involving a 5–minute delay, when compared to controls who had received identical injections of the vehicle. Because the lack of effect may be due either to compensation by the brain to the loss of these fibers or incomplete lesions of the MSA and NBM, we challenged the cholinergic system and the adrenergic system with scopolamine, a muscarinic receptor antagonist, and an adrenergic antagonist and assessed the effects of these injections using the same RAM delay task. The results of this study will be presented at the conference.