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DOES THE SEPTAL CHOLINERGIC PROJECTION TO THE AMYGDALA PLAY A ROLE IN THE SPATIAL LEARNING IN THE MALE RAT?

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Alzheimer's disease (AD) is a neurodegenerative disorder, currently affecting over four million Americans, with 100,000 new cases reported each year. The disease is characterized by cognitive impairments, most notably memory loss throughout the earlier stages and various emotional changes as well. These deficits, although predominant in cases of AD, have yet to be shown in a reliable animal model. Studies in humans and rats report a significant correlation between the memory loss found in AD and a decline in cholinergic markers such as choline acetyltransferase (ChAT) levels in the basal forebrain and hippocampus. Therefore, lesioning cholinergic projections to these areas seems a reasonable way of replicating the profound memory deficits found in AD. In a recent study by Dornan, McCampbell, Tinkler, Hickman, Bannon, Decker, and Gunther (submitted 1995), they reported that even with a reduction of ChAT activity as a result of 192 IgG Saporin injections into the medial septal area (MSA) and nucleus basalis magnocellularis (NBM), only marginal deficits were seen in the Morris water maze and radial arm maze tasks. One putative explanation for this dilemma may be the failure of 192 IgG Saporin to destroy the cholinergic projection from the nucleus basalis magnocellularis to the amygdala. Indeed, several studies have implicated the amygdala in learning and memory in the rat. In this study, we investigated this hypothesis by injecting both 192 IgG saporin and phthalic acid into the NBM. Following postsurgical recovery, all animals were assessed on spatial learning using the Morris water maze. The results of this study will be presented at the conference.