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Effects of Guanfacine on Mnemonic Processing Following Lesion to Rat Medial Septum: A Novel Treatment Approach to Alzheimer's Memory Type Deficits

Mark D. Opal, '07

Illinois Wesleyan University

Renee Countryman, Faculty Advisor Illinois Wesleyan University

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Poster Presentation P62

EFFECTS OF GUANFACINE ON MNEMONIC PROCESSING FOLLOWING LESION TO RAT MEDIAL SEPTUM: A NOVEL TREATMENT APPROACH TO ALZHEIMER'S MEMORY TYPE DEFICITS

Mark D. Opal and Renee Countryman*
Psychology Department, Illinois Wesleyan University

Cognitive deficits associated with Alzheimer's disease (AD) are known to result from decreases in acetylcholine (ACh) within the cholinergic system of the medial septal area (MSA), which projects to the hippocampus (HPC). Recent studies underline the significance of norepinephrine (NE) within the context of decreased memory functioning following lesioning to the MSA. However, this finding has not been studied as thoroughly. Research shows that a decrease in NE leads to a decrease in memory processing. Following a chemical lesion to the cholinergic system of the MSA, axonal cholinergic arborization is compensated for by an increase of NE afferent fiber projection to the HPC. Consequently, increased NE projection to the HPC might increase NE transmitter levels, thereby increasing processing. As a result, the NE agonist Guanfacine will be administered following lesion in an attempt to increase NE levels in the HPC following lesions to the MSA.