PSYCHOLOGY

Can nicotine improve scopolamine-induced behavioral deficit? *Brianna K. Ward\*, Elizabeth A. Collins, Wesley White, and Ilsun M. White*. Neuroscience Program, Department of Psychology, Morehead State University, Morehead, KY 40351.

Nicotine, a psychostimulant, is a direct agonist of nicotinic receptors. Scopolamine, a direct antagonist of muscarinic receptors, impairs memory and is commonly used in animal models of Alzheimer’s disease (AD). This study examined the interaction between two cholinergic receptor subtypes in simple memory, focusing on the effects of nicotine on scopolamine-induced behavioral deficits. Rats were trained on a fixed ratio 20 (FR20), which required 20 lever-presses for each food-pellet reward. Once performance reached a behavioral criterion of 60-rewards for 2 consecutive sessions, the drug phase began. Response latency and task completion were measured under four different conditions: saline+saline, saline+nicotine, saline+scopolamine, and nicotine+scopolamine. Compared to saline-controls, scopolamine-alone impaired performance by increasing response latency and decreasing responses that earned rewards, whereas nicotine-alone did not impair performance. Moreover, nicotine partially reversed scopolamine-induced deficits.  Our results suggest that nicotine may play a role in improving memory deficits. Given the prevalence of smoking among patients being treated for AD, examining the interaction between the two cholinergic receptors, muscarinic and nicotinic, in memory function is warranted.