

TITLE: The effects of blocking hippocampal acetylcholine on meal onset in male rats

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Background: Consuming food in bouts is common among animals. The mechanisms underlying meal termination have been extensively researched, yet little is understood about meal onset or the post-prandial intermeal interval (the interval between 2 meals). In our lab the working hypothesis is that the hippocampus inhibits meal onset during the post-prandial intermeal interval. Our recent research shows that reversible inactivation of the hippocampus following a meal decreases the post-prandial intermeal interval and accelerates meal onset. Acetylcholine is important in hippocampal-dependent learning and memory. We hypothesized that hippocampal acetylcholine is necessary to form a memory of a meal and inhibits meal onset during the post-prandial intermeal interval. We predicted that if hippocampal acetylcholine inhibits meal onset, then blocking acetylcholine with scopolamine would decrease the post-prandial intermeal interval and accelerate meal onset.

Methods: Adult male Sprague Dawley rats (N=10) underwent stereotaxic surgery to implant cannulas unilaterally into the dorsal hippocampus. Rats were trained to consume a 32% sucrose solution each day. On experimental day, scopolamine (0.0µg/µl, 5µg/µl, 15 µg/µl) was infused into the hippocampus following meal consumption. After the injection, rats were given access to the sucrose solution. The size of the post-infusion meal, post-prandial intermeal interval, and satiety ratio (an estimate of satiety accounting for the size of the pre-infusion meal) were measured.

Results: Rats that received the low dose of scopolamine had a longer post-prandial intermeal interval compared with vehicle and the high dose of scopolamine groups. No significant difference in the post-prandial intermeal interval between the vehicle and groups. Additionally, the low dose group had the highest satiety ratio relative to vehicle and high dose groups. The size of the second meal was largest in the low dose group, but no significant differences in the size of the second meal between the vehicle and high dose groups.

Discussion: To our surprise the data shows an increase in post-prandial intermeal interval with the low dose group. This may be due to presynaptic cholinergic auto receptors. We did not see significant effects in the high dose group and future

experiments will increase the scopolamine dosages to determine whether hippocampal acetylcholine inhibits meal onset.