

ScholarWorks@GSU

A Novel Role for Non-Classical Peptide Release in the Social Behavior of Syrian Hamsters

Authors	Perry, Daniel
Download date	2026-04-13 02:52:05
Link to Item	https://hdl.handle.net/20.500.14694/7819

TITLE: A Novel Role for Non-Classical Peptide Release in the Social Behavior of Syrian Hamsters

AUTHORS & AFFILIATIONS: Daniel M. Perry, James C. Walton, Ph.D., Zhimin E. Song, M.S., Tony E. Larkin, B.A., H. Elliott Albers, Ph.D. Georgia State University, Neuroscience Institute & Center for Behavioral Neuroscience

Background:

Current views of brain function emphasize the importance of individual neurons communicating with each other at synapses. Recent evidence shows, however, that non-synaptic somatodendritic neurotransmitter release plays a role in neuronal communication, and we investigated if this novel mode of neuronal communication was involved in controlling social behavior. Syrian hamsters communicate social information via flank marking, which hamsters do in captivity and in the wild to express territoriality. Flank marking is driven by arginine vasopressin (AVP) in the anterior hypothalamus (AH). While both the supraoptic nucleus (SON) and paraventricular nucleus (PVN) within the AH release vasopressin axonally into the pituitary gland, they do not project axons to the region of the AH that drives this behavior. We hypothesized that, if flank marking was somatodendritic vasopressin release-dependent, inhibiting somatodendritic release of vasopressin from PVN and SON neurons by blocking intracellular calcium release would inhibit flank marking.

Methods:

Male Syrian hamsters were anesthetized and surgically implanted with a cannula projecting to the AH. After recovery, hamsters were injected with drug [8-(N,N-diethylamino)-octyl-3,4,5-trimethoxybenzoate (TMB8), an inhibitor of intracellular calcium release] or vehicle (saline), placed in a test cage that was recently flank marked by a novel stimulus male, and flank marking behavior was recorded for 15 minutes. In the first experiment we established a dose-response curve for TMB8, and in the second that effective dose was used in a larger cohort of animals. Animals were sacrificed and brain tissue verified for accurate cannula placement.

Results:

Microinjection of the experimentally determined effective dose of TMB8 (182.5nM) into the AH inhibited flank marking and increased latency to initial flank mark, without affecting general activity levels.

Conclusions:

Somatodendritic peptide release in the AH is necessary for flank marking in Syrian hamsters, opening the possibility that this mode of neuronal communication may be involved in other social behaviors. Future directions include further refinement of the mechanism underlying somatodendritic vasopressin release, including manipulations of the SERCA pump and the IP3 receptors, which are responsible for calcium storage and release, as well as expanding these results to other brain regions and social behaviors within other animal models.