

**SELECTIVE VAGAL RHIZOTOMY: A SURGICAL APPROACH TO ANALYSING SPECIFIC SENSORY AND MOTOR CONTRIBUTIONS TO THE CONTROL OF FEEDING BEHAVIOR.**

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Control of meal size is disrupted in eating disorders and obesity. Therefore elucidating neural circuitry involved in the detection of satiety signals and the control of meal size is a high priority. The hindbrain appears to be involved in the integration of sensory and motor signals, which influence control of meal size. For example, the nucleus of the solitary tract (NTS) is the site of the first synapses of vagal sensory neurons, which carry satiety signals from the stomach and intestines. In addition, vagal efferent fibers leave the dorsal motor nucleus of the vagus (DMV) to innervate similar areas of the GI tract.

Our previous work has shown that blockade of NMDA receptors on neurons in the hindbrain increases food intake. Experiments using the NMDA receptor antagonist MK-801 in conjunction with hindbrain lesions, capsaicin treatment, and total subdiaphragmatic vagotomy, suggest that vagal *sensory* neurons participate in the control of food intake via hindbrain NMDA receptors. However, evidence from pharmacological experiments also suggests a vagal *efferent* contribution to this effect.

In order to distinguish between the contributions of vagal afferent or efferent neurons to the control of meal size by NMDA receptors, our lab has successfully executed a surgical procedure, which selectively isolates and sections either vagal sensory or motor rootlets as they emerge from the brainstem. These two rhizotomy techniques will enable us to independently test the participation of sensory and motor neurons in NMDA-mediated controls of feeding behavior.