In the brain, the dorsomedial hypothalamus (DMH) is important in appetite regulation and neurons in this region neurons express receptors that respond to stress hormones. The DMH is therefore an ideal site to study to effect of stress on appetite. Very little is known, however, how stress affects neuronal communication and excitability in the DMH.

Because DMH neurons stimulate appetite, and appetite is generally suppressed during an acute stressor, we hypothesized that acute stress would inhibit DMH neurons. We used whole-cell patch clamp electrophysiology to record from living DMH neurons from young, female Sparge-Dawley rats who experienced a single 30-minute restraint stressor. To determine if stress affects DMH neurons, we measured (1) transmission of the excitatory neurotransmitter, glutamate, onto DMH neurons by measuring glutamate current amplitude and (2) action potential (AP) parameters before, to access initial stress-induced changes, and after a high frequency stimulation (HFS), to access long-lasting changes.

Acute stress decreased evoked current amplitudes, AP amplitude, and AP frequency at glutamate synapses after HFS. Recordings obtained in the presence of an endocannabinoid-CB1 receptor blocker (AM251; 5 µM) showed no change in evoked current amplitudes, AP amplitude, or AP frequency after HFS, suggesting the endocannabinoid systems is required for the change in communication and excitability seen under acute stress.

As the global food system becomes increasingly saturated with processed and ultra-processed foods, the obesity epidemic grows in parallel, with New Brunswick having one of the highest obesity levels in Canada. The mechanisms that respond to stress have not adapted to our high stress society and landscape of high calorie, highly palatable foods. Women are particularly vulnerable to disordered eating behaviours when stressed, for which the neurophysiological basis is unclear. Yet, female research subjects remain underrepresented.