Hypothalamic nociceptin neurons modulate stress-induced binge eating behaviors

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Homeostatic regulation of food consumption and stress is critical to survival, yet certain environmental conditions facilitate aberrant feeding behaviors. Nociceptin and its receptor, nociceptin opioid peptide (NOP) receptor have a vast neuromodulatory network spanning multiple stress and reward-related brain nuclei and have been demonstrated to modulate feeding and stress regulation. Previous data has shown that stimulation of nociceptin-expressing neurons in the arcuate nucleus of the hypothalamus (ARC) increases food consumption. Similarly, stimulation of these neurons afferent projections within the bed nucleus of the stria terminalis (BNST) also increases food consumption. Here, we examined how this nociceptinergic neurocircuitry controls palatable food consumption in a model of stress-induced binge eating behavior where animals are given intermittent food restriction and palatable food access similar to "yo-yo" dieting. We used inhibitory opsin, parapinopsin (PPO) in Pnoc-Cre mice to inhibit nociceptin-expressing ARC projections in the BNST during food stress and palatable food access. We found that BNST-afferent ARC Pnoc neuron inhibition reduces stress-induced food consumption in animals previously exposed to cycles of food restriction and ad libitum palatable food access.

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