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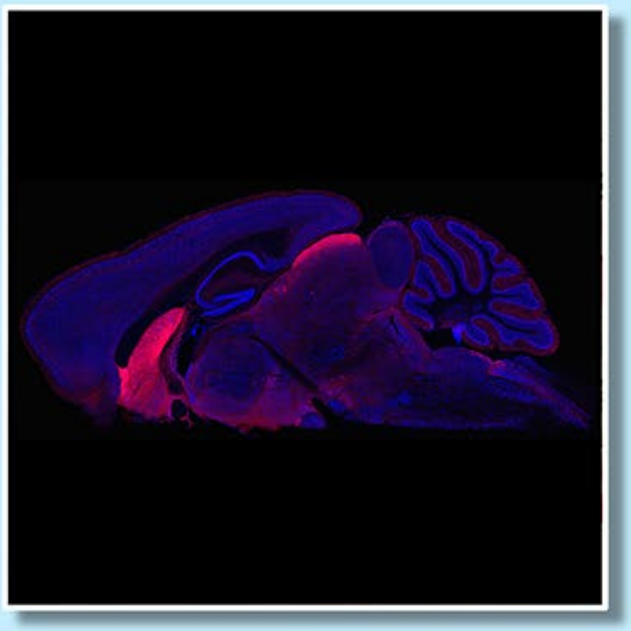

Prepronociceptin (red) in the
Mouse Brain (blue: nuclei)

Prepronociceptin-expressing Neurons in Central Amygdala Mediate Palatable Food Reward Independent of Anxiety

Binge eating behavior is a pathological feeding pattern affecting 4.5% of Americans in their lifetime and present in a number of eating disorders, including binge eating disorder, bulimia nervosa, and the binge-purge subtype of anorexia nervosa. Despite its prevalence and detrimental physical and psychological effects, the factors that drive binge eating are not fully understood. Particularly, the anxiolytic and rewarding properties of binge eating highly palatable food (HPF) that are proposed to motivate this behavior require further characterization, as do their neurobiological correlates. In this study, we first used a mouse model to investigate the behavioral effects of acute and chronic intermittent access to HPF on feeding and anxiety-like behaviors. We then identified a functional neural circuit activated during intermittent access to HPF, and performed pathway specific optogenetics to determine the role of this circuit in feeding, anxiety-like, and reward-related behaviors. Specifically, we investigated a circuit involving projections from pre-pronociceptin expressing neurons in the central nucleus of the amygdala (CeA^{NOC}) to the parabrachial nucleus (PBN). While binge-like consumption of HPF resulted from an intermittent access schedule, neither acute nor extended intermittent access affected anxiety-like behavior. Similarly, activation of the CeA^{NOC} projection to the PBN did not impact anxiety-like behavior or feeding. However, this activation did produce reward-related phenotypes. Together, these results suggest that binge-like eating of HPF in mice is promoted by reward, which is partially encoded by the projection of CeA^{NOC} to the PBN, but does not impact anxiety-like behavior.