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Description of Research:

Neuropeptide Y (NPY) is a 36 amino acid peptide present in high concentration in mammalian brain. Injection of NPY into hypothalamic regions elicits a powerful feeding response in rats, and NPY has been strongly implicated in diabetes and obesity. The peripheral satiety factor, leptin, has been shown to exert satiety stimuli in mice and rats by inhibiting the synthesis and release of NPY in hypothalamic sites that are known to regulate eating behavior. Most importantly, PYY 3-36, a peripheral homologue of NPY released from the L-cells of the gut into the circulation after a meal, is reported to act as a satiety signal. Administration of PYY 3-36 in humans significantly decreased appetite and attenuated food intake by 33 percent for up to 24 hours. In an attempt to understand the causes of eating disorders and obesity, Dr. Sheriff is investigating the molecular mechanisms regulating NPY-induced feeding in the hypothalamus. Activation of Y1 and Y5 receptors of NPY in the hypothalamic PVN by NPY trigger the hunger signal by inhibiting cAMP accumulation and mobilizing intracellular Ca⁺⁺. These two prominent second messengers activate a transcription factor, CREB, the activation of which regulates many CREB-dependent genes. These biochemical events translate into a hunger signal. PYY 3-36 activates hypothalamic Y2 receptors to turn off the hunger stimuli. Dr. Sheriff's overall goal is to investigate the signal transduction cascade and the regulatory genes involved in maintaining energy homeostasis.

Collaborations:

Dr. Sheriff collaborates with Dr. Waltz investigating the role of NPY-Y1 receptor and its interaction with Ron kinase signaling pathways. As a new member, Dr. Sheriff has not used DHC Cores.

Representative Figure:

