

## Stephen C. Benoit, PhD

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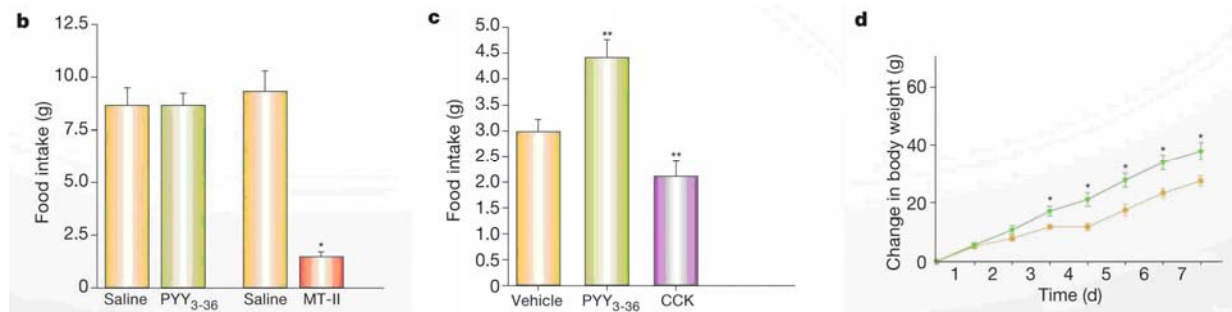
### Description of Research:

Under normal circumstances, caloric intake is matched closely to caloric expenditure such that stored calories in the form of adipose mass remains relatively stable. Considerable evidence indicates that hormonal, neuroendocrine, and learning mechanisms are integrated in the complex control of this system. Dr. Benoit's research has centered on the molecular, physiological and behavioral effects of several hypothalamic neuropeptides that influence energy homeostasis, and how these neurotransmitters are modified by signals proportional to the amount of fat in the body (e.g., the hormones, leptin and insulin) by using a diet-induced obesity rat model. Related research projects are designed to identify the anatomical circuits, as well as the functional relationships, among these neuropeptides. Dr. Benoit is also examining the role that learning has in the control of energy homeostasis. Influences on food intake can be categorized as homeostatic (e.g., related to body weight) and non-homeostatic (e.g., related to visceral illness, hedonics or the incentive value of food). The long-term goal is to determine precisely *what is learned* about food, as well as *which specific learning processes are critical* for the maintenance of energy balance. All of these studies should be especially important to the understanding of how regulatory mechanisms interact with learned patterns of food intake, and how these interactions might result in obesity.

### Collaborations:

Dr. Benoit works with Drs. D'Alessio, Seeley, Tschop, Tso, and Woods studying the crosstalk between high-fat diets and obesity. As a new member, Dr. D'Alessio has not yet used DHC cores.

### Representative Figure:



Lack of inhibitory effect of PYY<sub>3-36</sub> on food intake in rodents. Testing of human PYY<sub>3-36</sub> (b, d) and rat PYY<sub>3-36</sub> (c). B. The melanocortin-receptor agonist MT-II (3 mg/kg), but not PYY<sub>3-36</sub> (100 µg kg<sup>-1</sup>), decreased 4-hr food intake in Wistar rats ( $n=18$ ). C. PYY<sub>3-36</sub> (5 µg/kg) increased food intake after overnight fasting (30 min,  $P<0.05$ ,  $n=10$ ) in Sprague–Dawley rats; cholecystokinin (CCK; 6 µg/kg) decreased food intake ( $n=14$ ). D. PYY<sub>3-36</sub> (2x day at 50 µg/kg green line) increased body weight compared with saline control (orange line) over a 7-day treatment period in Sprague–Dawley rats ( $n=8$ ). Error bars show SEM; \*  $P<0.001$ ; \*\*  $P<0.05$ . Fig 1 from Nature, 2004; 430:1-4.