Repeated gastric distension alters food intake and neuroendocrine profiles in rats
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Bulimia nervosa (BN) is an eating disorder characterized by a pattern of uncontrolled ingestion (binging) and expulsion (purging) of food. Bulimics have increased gastric capacity, delayed gastric emptying, blunted post-meal ghrelin and insulin responses, disproportionately low leptin levels, resistance to α-melanocyte stimulating hormone, and increased neuropeptide Y (NPY) expression. Binge eating induces gastric distension and the stimulation of gastric mechanoreceptors to a greater degree than experienced by non-binging individuals. To test the effects of repeated gastric distension (RGD) without nutrient absorption on the neuroendocrine factors involved in energy homeostasis, a permanent intra-gastric balloon was implanted in rats, and inflated daily for 4 weeks. Though body weights and daily food intakes remained equivalent in RGD and control rats, a significant delay in the onset of feeding was present during the first and second, but not the third and fourth weeks of inflations. Leptin levels were decreased after RGD (p < 0.05); insulin and ghrelin were unaffected. Expression of proopiomelanocortin was unaffected by RGD. Fasting acute NPY levels in RGD rats were suppressed significantly more than control animals following food intake (control and RGD decreases from baseline were 184.95% and 257.42%, respectively). NPY expression in the nucleus of the solitary tract followed a similar pattern. These data suggest a role for RGD in multiple factors associated with BN.
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Leptin—TRH interactions in the Solitary Nucleus. An in vitro calcium imaging study
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Thyrotropin releasing hormone [TRH] is at the center of endocrine and autonomic thermoregulation and thermogenesis is gated by leptin. We observed that leptin injected into the fourth ventricle increases the potency of TRH to increase brown adipose tissue [BAT] temperature. This effect is order specific; leptin applied before TRH produced a much larger increase in BAT than when leptin is applied after TRH. This synergy suggests that leptin “gates” TRH transduction [e.g., PLC-mediated ER calcium release which, in turn, triggers changes in membrane excitability]. Rapid leptin signaling depends on the activation of a PI3kinase. Studies in culture systems suggest that PI3P [product of PI3kinase] potently upregulates PLC. We recently showed that NST neurons possess both LepRβ and TRHR1 receptors, making these cells likely loci for leptin-TRH interactions [Hermann et al., 2006, 2009; Rogers et al. 2009]. In vitro calcium imaging of the medullary slice was used to test the hypothesis that leptin modulates TRH transduction in NST neurons. Calcium green 1AM [calcium indicator] was injected into the NST; medullary slices containing the NST were harvested and transferred to the recording chamber of a confocal microscope. Slices were subjected to TRH alone, leptin alone, leptin followed by TRH or leptin plus wortmannin [PI3kinase inhibitor] followed by TRH. The results show that while leptin alone did not produce an increase in NST neuronal calcium, leptin pretreatment significantly increased the NST activation caused by TRH. The combination of wortmannin with leptinblocks the interaction with TRH.
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Effects of portion size and social modeling on food intake of young women
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People eat more or less when their eating companions eat more or less. Moreover, they eat more when being served a larger portion compared to a smaller portion. The current study was conducted to examine the potential influences of both types of situational norms on young women’s intake. The experiment involved a 3 (confederate’s eating condition: low-intake, normal-intake, large-intake) by 2 (portion size condition: small vs. large) factorial design. One hundred females participated. Participants’ intake was observed during a 20-min break. The total quantity of food consumed (in g) was used as our dependent variable. An ANOVA was used to examine the main and interactive effects of the modeling and portion size conditions. Results show a significant interaction between modeling condition and portion size condition on participants’ intake. Closer inspection revealed a modeling effect in the normal-portion size conditions, but not in the small-portion size conditions. Our results suggest that if women are served a portion of food that is too small to serve as a complete meal, they eat regardless of what their eating companion is eating. However, if they are served a normal portion of food, then their intake is subject to social modeling processes.
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Sex and photoperiod regulate central and peripheral endocannabinoid signaling
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Siberian hamsters (Phodopus sungorus) adapt to seasonal changes in environment with marked changes in body mass, primarily in the form of adiposity. Winter-like conditions (e.g., short days) are sufficient to decrease body mass by ~30%, with corresponding changes in food intake. The neuroendocrine mechanisms responsible for these changes are not well understood, and homeostatic orexigenic/anorectic peptides provide little explanation. We investigated the potential role of endocannabinoids, known modulators of appetite and metabolic profiles, as mediators of seasonal changes in energy balance. Specifically, we housed hamsters in long or short days for 0, 3, or 9 weeks (n=6 per group) and measured endocannabinoid levels in the hypothalamus, liver, and retroperitoneal white adipose tissue (RWAT). Levels of the endocannabinoid 2-AG were significantly elevated in RWAT of short-day animals by week 9. No photoperiodic changes were seen in the hypothalamus or liver; however, sex differences were found in the liver (M>F) and RWAT (F>M). Ongoing analyses will determine whether photorefractory hamsters (i.e., short-day-housed hamsters reverting back to long-day phenotype) demonstrate a return of 2-AG RWAT levels comparable to those seen in long days. Brainstem endocannabinoid levels will also be examined as previous work has demonstrated effects of photoperiod on cannabinoid receptor (CB1) levels in brainstem nuclei. Together these findings will shed light on mechanisms that defend dynamic states of energy balance and provide important implications for those that contribute to human states of obesity and leanness.
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