**Editorial**
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# The Physiological Control of Eating in Humans: An Editorial

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## Editorial

Eating has significant physiological implications. Food and food components have been altered in experiments to activate numerous food-dependent physiological signals, revealing mechanisms for eating-induced satiety and thirst. Eating has also been shown to stimulate physiological signals for drinking in order to avert fluid balance difficulties. The signals produced by the gastrointestinal (GI) tract during meals that interact with the central nervous system to induce a feeling of fullness and satiety are discussed. Although the GI tract secretes dozens of enzymes, hormones, and other variables in response to food in the lumen, only a few are able to directly influence food intake.

The majority of these cause meals to end, and so are referred to be satiety signals, with CCK being the most researched. Only one GI signal, ghrelin, has been recognised as increasing meal size. Smaller meals are ingested when exogenous CCK or other satiety signals are administered, whereas inhibiting the effect of endogenous CCK or other satiety signals results in bigger meals being consumed. Satiety signals are sent to the hindbrain either indirectly through nerves from the GI tract, such as the vagus, or directly through the blood. The majority of factors that determine how much food is consumed during individual meals work through altering satiety signal sensitivity. This includes adiposity signals, habits and learning, social situations, and stresses, among other things.

The effectiveness of Roux-en-Y gastric bypass (RYGB) and other bariatric surgeries in the treatment of obesity and type 2 diabetes mellitus, as well as new developments in GI endocrinology, have reignited interest in the role of GI hormones in the control of eating, meal-related glycemia, and obesity. We examine the nutrient-sensing mechanisms that control the secretion of four of these hormones, ghrelin, cholecystokinin (CCK), glucagon-like peptide-1 (GLP-1), and peptide tyrosine tyrosine [PYY(3-36)], as well as their contributions to the regulation of GI motor function, food intake, and meal-related increases in glycemia in healthy. It is described their physiological roles as classical endocrine and locally acting signals.

The release of ghrelin, CCK, GLP-1, and PYY is influenced by gastric emptying, the recognition of specific digestion products by small intestinal enteroendocrine cells, and synergistic interactions among distinct GI loci (3-36). While CCK has been shown as an endogenous endocrine regulator of eating in healthy-weight people, the roles of the other three hormones in obesity and post RYGB are unknown. Similarly, only GLP-1 appears to play a role in endocrine regulation of meal-related glycemia. Local signalling is most certainly involved in these hormones' functions, but there are no ways for determining the physiological status of local signalling effects. To better understand ghrelin, CCK, GLP-1, and PYY(3-36) physiology, their involvement in obesity and bariatric surgery, and their therapeutic potentials, more study and new methodologies are needed [1-5].

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