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Review Article

The Effect of Cholecystokinin (CCK) on Obesity: Evaluation of Hormonal Changes Following Roux-en-Y Gastric Bypass Surgery

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Abstract: Obesity is a chronic disease linked to increased risks of type 2 diabetes, cardiovascular disorders, and metabolic dysfunction. As global obesity rates continue to rise, bariatric surgery, particularly Roux-en-Y gastric bypass (RYGB), has become a well-established intervention for achieving sustained weight loss and improving obesity-related comorbidities. Cholecystokinin (CCK) is a gut hormone released by enteroendocrine cells in response to food intake. It plays a key role in appetite regulation, promoting satiety, slowing gastric emptying, and stimulating digestive enzyme secretion. Acting through CCK1 receptors and signaling via the vagus nerve, CCK has been shown to reduce food intake in various experimental models. This study focuses on evaluating hormonal changes following RYGB, with an emphasis on the role of CCK. While research findings vary—due to differences in sample timing, patient variability, and methodologies—evidence supports the involvement of CCK in the improved satiety observed after surgery. Factors such as diet composition, meal timing, and individual health status significantly influence CCK response. The findings suggest that CCK contributes to the metabolic benefits of RYGB and may serve as a potential target for therapeutic interventions in obesity treatment. However, further standardized, well- controlled studies are needed to clarify its mechanisms and interactions with other gut hormones.

Keywords: Obesity, Cholecystokinin (CCK), Roux-en-Y Gastric Bypass (RYGB), Satiety, Gut Hormones, Appetite Regulation.

INTRODUCTION

Obesity is a chronic complex disease defined by excessive fat deposits that can impair health. Obesity can lead to increased risk of type 2 diabetes and heart disease, it can affect bone health and reproduction, it increases the risk of certain cancers. Obesity influences the quality of living, such as sleeping or moving. In 2022, 43% of adults aged 18 years and over were overweight and 16% were living with obesity.

In 2024, 35 million children under the age of 5 were overweight. Over 390 million children and adolescents aged 5–19 years were overweight in 2022, including 160 million who were living with obesity [1].

Recent findings published in the Special Issue entitled “Molecular Research in Obesity” of Biomedicines provide crucial insights into the multifaceted nature of obesity. Highlights include the identification of specific genetic variants associated with increased risk, as well as novel biomarkers that could serve as targets for early intervention. Moreover, new research underscores the significant role of adipose tissue dysfunction in driving chronic inflammation and metabolic disturbances, offering potential therapeutic pathways for restoring energy homeostasis and reducing the burden of obesity-related comorbidities [2].

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Many hormones act on the hypothalamus to control hunger and satiety through various pathways closely associated with several factors. When food is present in the gastro intestinal (GI) tract, enteroendocrine cells (EECs) emit satiety signals such as cholecystokinin (CCK), glucagon like peptide-1 (GLP-1) and peptide YY (PYY), which can then communicate with the vagus nerve to control food intake [3], in 1973, Gibbs *et al*., discovered that exogenous CCK inhibits food intake [4]. The effect mimicked the satiety induced by food and was not seen with other gut peptides known then. The effect could be demonstrated in several mammals. Vagotomy studies indicate that peripheral CCK induces satiety via CCK_1 receptors relaying the effect into afferent vagal fibers [5].

With the growing prevalence of obesity in the global population, alternative measures for weight loss and treatment of comorbidities must be considered due to the increasing difficulty of conservative management alone. Bariatric surgery serves as an efficacious alternative for treatment of obesity and comorbidities [6]. Roux-en-Y gastric bypass (RYGB) was first introduced in 1966 by Mason, and after significant evolution, it is now accepted as a reliable bariatric surgery method with long-term results. Developments in laparoscopy across all fields of abdominal surgery have led to laparoscopic bariatric procedures being accepted as the standard of care. The low morbidity and mortality associated with laparoscopic procedures have led to the introduction of day-case surgery for bypass and gastrectomy procedures, establishing bariatrics as a cost-effective intervention [7].

Cholecystokinin

Cholecystokinin is a gut hormone released after a meal, which helps digestion and reduces appetite, used to be known as pancreozymin due to its actions on the pancreas but now it is commonly abbreviated to CCK; CCK-PZ [8].

Cholecystokinin (CCK) was discovered in 1928 in jejunal extracts as a gallbladder contraction factor. It was later shown to be member of a peptide family, which are all ligands for the CCK_1 and CCK_2 receptors. CCK peptides are known to be synthetized in small intestinal endocrine I-cells and cerebral neurons. But in addition, CCK is expressed in several endocrine glands (pituitary cells, thyroid C-cells, pancreatic islets, the adrenals, and the testes); in peripheral nerves; in cortical and medullary kidney cells; in cardial myocytes; and in cells of the immune system. CCK peptides stimulate pancreatic enzyme secretion and growth, gallbladder contraction, and gut motility, satiety and inhibit acid secretion from the stomach [9].

The cellular effects of CCK peptides are mediated *via* two receptors, the “alimentary” CCK-A or CCK_1 receptor [10], mediates gallbladder contraction, relaxation of the sphincter of Oddi, pancreatic growth and enzyme secretion, delay of gastric emptying, and inhibition of gastric acid secretion *via* fundic somatostatin [11]. CCK_1 receptors have been found also in the anterior pituitary, the myenteric plexus, and areas of the midbrain [12, 13].

The CCK-B or CCK_2 receptor (the “brain” receptor) is the predominant CCK receptor in the brain, it is less specific than the CCK_1 receptor and binds also non-sulfated CCK, gastrin [14].

Potential Role for Abnormalities of Cholecystokinin in Obesity

Based on the evidence that CCK plays a role in the acute regulation of appetite and energy intake, it has been suggested that abnormalities in CCK, or its actions, on gastrointestinal function and appetite, may con-tribute to the development and maintenance of obesity. There is a body of literature supporting the potential role for CCK in obesity, in animal studies. In particular, rats genetically lacking functional CCK1 receptors rapidly develop obesity, consistent with the concept of a role for CCK. For example, Otsuka Long-Evans Tokushima Fatty (OLETF) rats, which lack CCK1 receptors as a result of a mutation, become markedly hyperphagic and overweight early in life [15]. OLETF rats consume much larger meals, and are completely resistant to the inhibitory effects of exogenous CCK, as well as gastric or intestinal nutrient infusion, on energy intake [16]. In contrast, in receptor knockout mice, while intraperitoneal administration of CCK fails to inhibit food intake, as in OLETF rats, normal body weight is maintained well into adult life [17]. This latter observation argues against a role of CCK in the long term maintenance of body weight. A role for CCK in the pathogenesis of human obesity is, however, less well defined. In humans, polymorphisms in the promoter region of the gene for the CCK1 receptor have been reported to be associated with a higher percentage of body fat [18], and, accordingly, may contribute to increased body weight. However, mutations of the CCK1 gene appear to be infrequent in humans [19]. Therefore, it is unlikely that genetic mutations of the CCK1 receptor are of major relevance. In obese subjects fasting plasma concentrations of CCK have been reported to be increased, rather than decreased [20]. This contrasts with observations in healthy older people in whom fasting plasma CCK concentrations are increased, and associated with diminished hunger [21], as well as increased sensitivity to the inhibitory effects of exogenous CCK on energy intake, when compared with healthy young subjects [22]. There is only limited information about the release of CCK following nutrient ingestion in obesity; one study has reported that following a high-fat meal, CCK secretion was higher in obese, than in lean, subjects despite comparable rates of gastric emptying [23]. As obese subjects commonly consume a diet high in fat and therefore high in energy, the prior exposure of the gut to nutrients that stimulate the release of CCK may account for this increase in plasma CCK concentrations. The satiating effect of acute intravenous infusion of exogenous CCK in obese

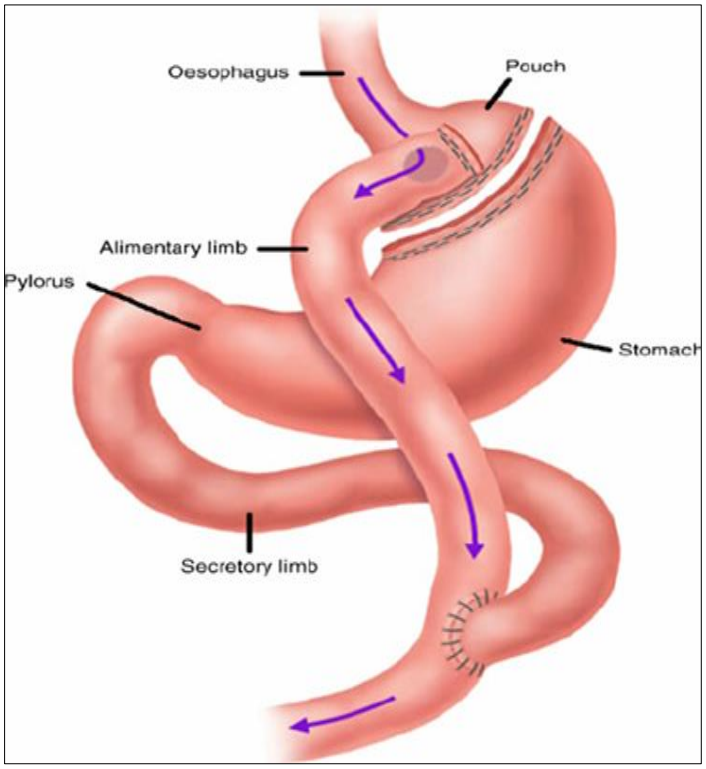
subjects does not appear to differ from that observed in healthy lean subjects [24], indicating that the sensitivity to the appetite-suppressant effects of CCK is not affected by increased body weight. Therefore, it seems unlikely that CCK plays a major role in an etiology of human obesity.

Roux-en-Y Gastric Bypass (RYGB) – Mechanism and Advantages

Roux-en-Y Gastric Bypass (RYGB) is a type of bariatric surgery used to treat severe obesity. It works by both restricting food intake and altering the digestive process. During the procedure, a small pouch is created from the upper part of the stomach and connected directly to a lower part of the small intestine, bypassing most of the stomach and the first segment of the intestine (duodenum) [25]. This surgery promotes weight loss through Restriction: The small stomach pouch limits the amount of food that can be consumed. Malabsorption: Bypassing part of the intestine reduces calorie and nutrient absorption. Hormonal changes: RYGB alters gut hormones like GLP-1, PYY, and ghrelin, which reduce hunger and improve blood sugar regulation [26].

Clinical and Metabolic Benefits of Roux-en-Y Gastric Bypass (RYGB)

1- Significant and long-term weight loss Improvement or resolution of type 2 diabetes Reduced appetite and enhanced satiety Improvement in obesity-related conditions like hypertension, sleep apnea, and high cholesterol RYGB is considered one of the most effective and well-studied bariatric procedures available today [27].



Visual description of anatomical changes following RYGB [28]

The effect of RYGB on CCK Level

Roux-en-Y Gastric Bypass (RYGB) significantly alters gastrointestinal hormone secretion, including cholecystokinin (CCK). After RYGB, CCK levels often increase postprandially (after meals), which contributes to enhanced satiety and reduced food intake. This rise is believed to be due to greater stimulation of the remaining small intestine by undigested nutrients reaching more distal segments, as well as potential adaptation and proliferation of CCK-producing enteroendocrine cells. Although the bypassed duodenum contains many CCK-secreting cells, the hormonal response is preserved or even enhanced due to these compensatory mechanisms [29].

Is There a Link Between Increased CCK Levels After RYGB and Weight Loss?

Following Roux-en-Y Gastric Bypass (RYGB), elevated levels of cholecystokinin (CCK) have been observed postprandially. CCK is an anorexigenic hormone that promotes satiety by stimulating vagal afferent pathways and delaying gastric emptying. The anatomical changes induced by RYGB lead to altered nutrient flow, which enhances stimulation of CCK-producing enteroendocrine cells in the distal small intestine. This heightened CCK response is thought to contribute to reduced appetite and lower food intake, thereby supporting postoperative weight loss. However, while CCK plays a role

in appetite regulation, it acts synergistically with other gut hormones such as Glucagon Like Peptide-1 & 2[GLP-1 & GLP-2] and Peptide YY [PYY], and is not considered the sole driver of weight loss after RYGB [29].

Inconsistency in CCK Response after RYGB Surgery

Several studies have reported inconsistent findings regarding the levels of cholecystokinin (CCK) following Roux-en-Y gastric bypass (RYGB). While some investigations observed a significant postprandial increase in CCK levels after surgery, others reported either no significant change or even a decrease. For example, a prospective study comparing hormonal changes after gastric banding and RYGB demonstrated increased CCK levels in non-diabetic patients' post-surgery, whereas other research failed to confirm this elevation or observed a decline instead [30].

These discrepancies may be attributed to heterogeneity in study designs, including variations in the composition of test meals, sample collection timing, patient characteristics, and follow-up duration. Standardizing measurement protocols and controlling for these variables is essential to enhance comparability and accuracy across future studies.

Standardization of Measurement Protocols and Timing for CCK Sampling

The variability in reported cholecystokinin (CCK) levels following Roux-en-Y gastric bypass (RYGB) underscores the necessity for standardized methodologies in both sample collection and analysis. Discrepancies in study outcomes may arise from differences in patient demographics, meal compositions, and postprandial sampling intervals. For instance, a prospective study comparing gut hormone and metabolic changes after adjustable gastric banding and RYGB highlighted that variations in sampling times and meal types could influence the observed hormonal responses, including CCK levels.

To enhance the reliability and comparability of future research, it is imperative to establish uniform protocols that specify optimal sampling times relative to meal intake, standardized meal compositions, and consistent analytical techniques. Such standardization would facilitate more accurate assessments of CCK's role in post-RYGB metabolic adaptations and its interactions with other gastrointestinal hormones [31].

Postoperative measurements of cholecystokinin (CCK) levels after Roux-en-Y gastric bypass (RYGB) can be significantly affected by a range of individual factors, including the macronutrient composition of meals, timing of food intake, and the patient’s overall health status. CCK secretion is highly responsive to dietary fat and protein; thus, studies utilizing carbohydrate-rich test meals may underestimate postprandial CCK responses. Additionally, the timing of meal administration—whether during fasting or in close proximity to previous meals—can influence baseline hormone levels and their subsequent fluctuations.

Furthermore, inter-individual variability such as insulin sensitivity, gastrointestinal function, and coexisting metabolic conditions (e.g., type 2 diabetes or obesity) may modulate enteroendocrine cell activity and hormone release. These physiological differences complicate direct comparisons across patient populations and may account for some of the inconsistencies in reported CCK dynamics after RYGB [32].

Therefore, it is critical for future research to control for and document these variables, ensuring homogeneity in study populations and standardization in experimental conditions to accurately interpret CCK responses in the post-RYGB setting.

Can CCK Be Therapeutically Targeted for Obesity?

Cholecystokinin (CCK) plays a critical role in appetite regulation by promoting satiety and delaying gastric emptying. Because of these properties, CCK has been investigated as a potential therapeutic target for obesity management. Pharmacological approaches aiming to enhance CCK activity could theoretically reduce food intake and contribute to weight loss. However, clinical application faces challenges such as the short half-life of endogenous CCK, the development of receptor desensitization, and limited effectiveness when administered peripherally. Therefore, while CCK remains a promising target, more research is required to overcome these obstacles before it can be effectively utilized as an anti-obesity therapy [33, 34].

Potential Development of CCK Mimetics or Secretagogues

Efforts have been made to develop drugs that either mimic the action of CCK or stimulate its endogenous secretion. CCK receptor agonists have shown some success in preclinical models by reducing meal size and promoting satiety. Additionally, certain dietary components and gut microbiota modulators may enhance endogenous CCK release. Nonetheless, challenges including receptor subtype specificity, potential side effects (e.g., nausea), and maintaining sustained efficacy limit the current clinical use of such agents. Future advancements in drug delivery systems and combination therapies may improve the viability of CCK-based treatments for obesity [35, 36].

CONCLUSION

In summary, cholecystokinin (CCK) is a pivotal gut hormone involved in the regulation of appetite and digestion, with particular significance in the metabolic adaptations following Roux-en-Y gastric bypass (RYGB). Despite inconsistencies in the literature regarding post-RYGB changes in CCK levels—attributable to methodological differences, timing of sampling, and patient variability—evidence supports its role in enhancing satiety and modulating gastrointestinal function. Furthermore, factors such as dietary composition, meal timing, and individual health status critically influence CCK secretion and action.

Given the potential of CCK as a therapeutic target for obesity, further well-designed, standardized research is essential to clarify its mechanisms and optimize clinical interventions. Continued investigation will deepen our understanding of CCK's interaction with other gut hormones post-RYGB, ultimately contributing to improved obesity management strategies.

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