

The effect of bariatric surgery on gut hormones that alter appetite

D.-J. Pournaras^{a,b}, C.-W. Le Roux^{b,*}

^a Department of Bariatric Surgery, Musgrove Park Hospital, Taunton, UK.

^b Imperial Weight Centre, Imperial College London, W6 8RF, UK.

Abstract

Bariatric surgery is the only effective treatment for morbid obesity in the long term. Gut hormones are key players in the metabolic mechanisms causing obesity. Furthermore gut hormones are involved in the signalling process of hunger and satiety which leads to the control of nutrient intake. In this review, the role of these hormones as facilitators of appetite control after bariatric and metabolic surgery will be explored.

© 2009 Published by Elsevier Masson SAS.

Keywords: Bariatric surgery; Gut hormones; Appetite; Review

Résumé

Effets de la chirurgie bariatrique sur les hormones digestives qui contrôlent l'appétit

La chirurgie bariatrique est le seul traitement de l'obésité morbide efficace à long terme. Les hormones digestives ont un rôle clé dans les mécanismes métaboliques responsables de l'obésité. En outre, ces hormones sont impliquées dans les processus de signalisation de la faim et de la satiété qui conduisent au contrôle de la prise alimentaire. Dans cet article, sera passé en revue le rôle de ces hormones en tant que facilitateurs du contrôle de l'appétit après chirurgie bariatrique et métabolique.

© 2009 Publié par Elsevier Masson SAS.

Mots clés : Chirurgie bariatrique ; Hormones digestives ; Appétit ; Revue générale

1. Introduction

Surgical procedures are currently the most effective therapy for long-term weight loss [1]. Furthermore, some of these operations lead to the rapid remission of type 2 diabetes in a weight loss independent manner [2]. The mechanism that leads to sustained weight loss as well as diabetes remission after bariatric operations remains to be fully elucidated.

Gut hormones cause hunger and satiety effects. Therefore they play an integral role in the appetite-signalling process and are key element of the gut-brain axis. They have been implicated to play an important role in the successful outcomes

after gastric bypass surgery [3]. It is becoming evident that bariatric procedures modulate the gut-brain axis by altering the anatomy of the gut and affecting gut hormones [3]. In fact some of these procedures are now considered suitable models for the study of the gut brain axis.

Bariatric procedures were designed to promote weight loss due to the reduction of stomach volume (laparoscopic adjustable gastric banding, laparoscopic sleeve gastrectomy, malabsorption of nutrients (biliopancreatic diversion, duodenal switch) or a combination of both (Roux-en-Y gastric bypass). Although there is no evidence of calorie malabsorption, (with the exception of the biliopancreatic diversion and duodenal

* Corresponding author.

E-mail address: c.leroux@imperial.ac.uk (C.-W. Le Roux)

switch), the effects of bariatric procedures cannot be fully attributed to the reduced gastric volume. A number of studies have shown that changes in gut hormones after bariatric and metabolic surgery may be responsible for the appetite control and the resulting weight loss experienced post-operatively [4]. We review the most important peptides in terms of appetite control after bariatric and metabolic procedures; peptide YY (PYY), glucagon-like peptide-1 (GLP-1), ghrelin and cholecystokinin (CCK).

2. Peptide YY

Peptide YY is 36-amino-acid peptide, member of the PP-fold peptide family. Y is the abbreviation for tyrosine. It is released postprandially by endocrine L-cells of the gut in response to the calories ingested, however it is not affected by gastric distension [5,6]. Although PYY is present in the whole length of the intestinal, the concentration gets higher distally [5]. PYY inhibits gastrointestinal motility and the gastric, pancreatic and intestinal secretion [7,8]. It induces satiety and reduce nutrient intake in both the obese and the non-obese, however obese individuals appear to have a PYY deficiency that could affect satiety signals and could thus reinforce obesity [9-11].

An exaggerated postprandial PYY response after gastric bypass has been demonstrated [12]. This may contribute to the initial weight loss as well as the sustained long-term maintenance of this weight loss [12]. Another study of a human model of gastric bypass and a rodent model of jejuno-intestinal bypass showed increased PYY levels postprandially associated with increased satiety [13]. In a mechanistic investigation using the animal model an additional to the food intake effect of gastric bypass on weight loss was shown, suggesting that enhanced energy expenditure may play a role [13]. A recent prospective study of patients undergoing gastric bypass confirmed an increased postprandial PYY compared to patients undergoing gastric banding [14]. This finding suggested that differences in levels of gut hormones may play a role in promoting greater weight loss with gastric bypass compared to gastric banding [14].

Recently, we demonstrated a causative relationship between the enhanced PYY and GLP-1 response and the increased satiety following gastric bypass [15]. In this study increased postprandial PYY and GLP-1 responses were detected as early as the first week after gastric bypass, before any significant weight loss has occurred [15]. In the second part of this study good and poor responders to gastric bypass in terms of weight loss were investigated. Lower PYY and GLP-1 postprandial responses were associated with inferior weight loss [15]. Finally a comparative study of patients after gastric bypass and gastric banding was performed using a randomised double-blind saline controlled design [15]. Blockade of the gut hormone response with the somatostatin analogue octeotide increased nutrient intake and reduced satiety in the gastric bypass group, but not in the gastric banding group [15]. This

finding supports the hypothesis that the enhanced gut hormone response might play a key role in the reduced food intake after gastric bypass [15]. The longer term effect of this procedure on appetite and PYY was investigated in another study, in which the enhanced response as well as the reduced appetite was sustained for 24 months postoperatively [16].

Comparative studies of patients after gastric banding and gastric bypass showed a reduced PYY response in the gastric banding group on a number of occasions [13,14,17]. A prospective study of patients undergoing vertical banded gastroplasty compared to non-obese controls demonstrated a significantly lower PYY in the preoperative, obese group [18]. Following vertical banded gastroplasty, PYY gradually increased to the control levels [18].

In a comparative study of laparoscopic sleeve gastrectomy and gastric bypass using a randomised, double-blind design, both fasting and postprandial PYY levels, were increased similarly postoperatively [19]. The markedly reduced ghrelin levels in addition to increased PYY levels after sleeve gastrectomy are associated with greater appetite suppression and excess weight loss compared with gastric bypass [19]. The authors hypothesised that the reduced ghrelin after sleeve gastrectomy has an additive to the PYY response on appetite control [19]. An animal study supports this hypothesis by demonstrating that ghrelin attenuates the inhibitory effect of PYY and GLP-1 on food intake and gastric emptying in a dose-dependent manner [20]. However the long term effects of sleeve gastrectomy remain to be elucidated.

3. Glucagon-like peptide-1 (GLP-1)

GLP-1 is released postprandially by endocrine L-cells of the gut [21]. The inhibitory effect of GLP-1 and PYY on food intake is additive [22]. Furthermore sustained GLP-1-receptor activation is associated with weight loss in both preclinical and clinical studies [23].

As in the case of PYY the postprandial GLP-1 response is enhanced after gastric bypass, but not after gastric banding [13,15]. Both fasting and postprandial levels of GLP-1 remain elevated even 20 years after jejuno-ileal bypass [24].

GLP-1 plays an important role in glucose metabolism in addition to the effect on appetite control. It is a potent incretin. GLP-1 enhances the insulin response to nutrients, delays gastric emptying and inhibits the glucagon response in a glucose-dependent manner [23].

4. Ghrelin

Ghrelin is a 28-amino acid peptide produced from the fundus of the stomach and the upper intestine [25,26]. Central and peripheral administration increases energy intake and remains the only known orexigenic gut peptide known to date [27,28]. Ghrelin increases prior to meals and is suppressed rapidly by food intake proportionally to the amount of calories

Download English Version:

<https://daneshyari.com/en/article/3260481>

Download Persian Version:

<https://daneshyari.com/article/3260481>

[Daneshyari.com](https://daneshyari.com)