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A model explaining protracted weight loss after Roux en Y gastric by-pass bariatric surgery

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Introduction:

Despite evidence that Roux en Y gastric by-pass (RYGB) does not generally lead to a sustained decrease in dietary intake or appetite loss, this form of treatment is the most effective method of producing sustained weight loss in morbidly obese subjects. Animal and clinical studies have shown that RYGB induces metabolic changes enhancing energy expenditure at rest and particularly in the post-prandial period, so-called â€œdiet-induced thermogenesisâ€• (DIT) and also to increased faecal energy loss via both fat and undigested protein. A key question is what is the cause of the enhanced DIT following RYGB? Saeidi et al and Cavin et al. have reported that after RYGB in obese rats, the alimentary limb (AL) becomes hyperplastic and hypertrophic showing up-regulation of the glucose transporter GLUT1 in enterocyte basolateral membranes, also cytoplasmic hexokinase 2 and increased intraluminal metabolism of glucose as demonstrated by 2-deoxy-2-[18F] fluoro-D-glucose (18FDG) positron emission tomography. Sustained weight loss due to Roux en Y gastric bypass surgery (RYGB) may either be the result of improved incretin response to or a change in energy balance. A recent computer model of human glucose absorption and metabolism has been adapted to provide an explanation of both the increased DIT and weight-loss after RYGB. There are three likely mechanisms explaining the RYGB induced sustained weight loss: Increased sensitivity to incretins gastric inhibitory polypeptide GIP and glucagon-like peptide 1 GLP-1; increased leakage of glucose from the splanchnic circulation back into the small intestinal lumen via either transcellular route. This results from increased expression of glucose transporters at both the enterocyte basolateral and apical membranes, i.e. GLUT1 and GLUT2, or to inflammation-induced increased paracellular leakage. Overgrowth of facultative aerobic bacteria within the small intestine leads to enhanced luminal conversion of glucose to CO2 with consequent depletion of net energy absorption. The computer model simulates all of the combinations of these conditions and the observed decreases in plasma glucose and insulin concentrations along with increase in post prandial and fasting metabolic rates following RYGB. A combination of all three factors is likely to be the explanation for the success of RYGB. This raises the interesting consideration as to whether a non-surgical intervention which simulates these effects might be as efficacious as RYGB in producing sustained weight loss in morbidly obese subjects.