suggested a rapid and dynamic metabolic response to conventionalization, not observed in the ileum or colon, and lending further support to the unique role of the proximal intestine in response to the luminal microbiota-diet interplay (9). Thus, experimental RYGB, by exposing jejunal mucosa to increased bacterial load including fecal type microbiota through the stagnant loop–associated SIBO, may be close, in terms of the induced microbiota shift, to conventionalization of germ-free mice (9). Because SIBO is very frequently infraclinical, latent nutrient deficiencies may develop after RYGB surgery, a both restrictive and malabsorptive procedure (6, 10): if not prevented or sought for (after cessation of postoperative antibiotic treatment), deficiencies of vitamin B-12, due both to suppressed secretion of intrinsic factor and bacterial consumption, and of vitamin D, iron, copper, or thiamine, nutrients physiologically absorbed in the upper gut, may occur at least partly through their consumption by the stagnant loop syndrome–associated microbiota. Conversely, serum folate concentrations may be elevated in patients with SIBO syndrome as a result of bacterial synthesis within small intestinal lumen. After RYGB, severe sequelaes of nutrient deficiencies are uncommon (6), but may be elevated in patients with SIBO syndrome as a result of jejunoenteric anastomosis is formed a common channel that is shorter than 120 cm—specifically, those in whom the jejunoenteric anastomosis is formed <120 cm from the ileocecal valve (10); in such cases, the stagnant, diverted duodenojejunal limb is also longer and increases the incidence of SIBO syndrome.

The author had no conflicts of interest to disclose.

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REFERENCES


Reply to C Matuchansky
Dear Sir:

In consideration of the concerns indicated by Matuchansky, we address below the 2 specific points that he raised.

As a first point, the concern is that the fecal microbiota may not be an accurate reflection of what is happening within other microbial niches of the gut. We certainly agree that this point warrants discussion in the context of Roux-en-Y gastric bypass (RYGB) in which the gut anatomy is remodeled by surgery. Whatever the context, RYGB or control, the fecal microbiota is recognized to represent the luminal microbiota of the distal segments of the colon (essentially the descending colon). The distribution of microbial taxa (dominant, subdominant, and transit bacteria) will be the major differential element, such that facultative anaerobes are in healthy controls codominant with strict anaerobes in the proximal colon, with population densities of $10^9$/mL, whereas the 2-log increase in population until the distal colon is due to an increase in anaerobes reaching $10^{11}$/g, when facultative anaerobes normally remain at $10^7$/g (1). The microbiota of the jejunum and upper limbs will only reach far lower densities ($<10^7$/mL) and, with a much shorter transit time, will only show a minor contribution to metabolic bioconversions. Hence, if we cannot exclude a contribution via glucagon-like peptide-1 (GLP-1) breakdown, the effect observed could still be mediated by the cross-talk between colonic bacteria and enteroendocrine cells, which still remains very poorly understood at present but would be consistent with GLP-1 being produced by L-cells in the ileum and colon (2). This would also be consistent with the mouse model of RYGB for which alterations of the gut microbiota are mainly observed in the distal segments of the gut. Observations on jejunal Roux-limb glucose metabolism are quite complex to interpret in human RYGB patients, especially in view of a potential jejunal bacterial overgrowth (see the second point below). It could be associated with an absorptive adaptation such as that observed in short bowel syndrome (3).

As a second point, the concern raised is that of the possible bacterial overgrowth induced by the blind stagnant loop generated by RYGB. There is little doubt that a stagnant loop will constitute a totally novel ecological niche that will be occupied by a dense bacterial population as long as it can withstand the bile acid concentrations encountered. This would even, as indicated, promote the establishment of an anaerobic environment favoring a fecal-like bacterial community. Bacteria released from the stagnant loop into the jejunal limb would in such a context be exposed to food constituents at an unusually high density. This could be associated with a sequestration of simple sugars by bacteria that are obviously well adapted and highly potent to capture such energy-dense nutrient sources in a competitive environment. In turn, this would contribute to the energy-restriction typical of RYGB surgery. Although they are not health-threatening, the deficiencies mentioned for RYGB patients do stress the need to take a much closer look into the role of the stagnant loop–associated microbiota into the overall energy and vitamin balance. This would be especially relevant if bacterial overgrowth is not systematic, suggesting that the composition of the stagnant loop–associated microbiota may determine its occurrence. In our clinical context, however, vitamin deficiencies were systematically corrected by nutritional substitution.
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