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MINIREVIEWS

Microbiota remodeling after bariatric surgery: Procedure-specific dynamics and metabolic implications

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Abstract

Bariatric surgery is recognized as the most effective surgical strategy for achieving sustained weight loss and improving metabolic disorders in patients with severe obesity. Beyond anatomical restriction and caloric malabsorption, increasing evidence suggests that surgery-induced remodeling of the gut microbiota plays a critical role in mediating postoperative metabolic benefits. Initial studies primarily described global alterations in microbial diversity; however, subsequent research has revealed complex, procedure-specific, and time-dependent changes in microbial composition and function. Nevertheless, findings across studies remain heterogeneous, and the clinical and mechanistic relevance of these microbial shifts is not fully established. Key unresolved issues include inconsistent trajectories of microbial diversity from the early postoperative period to long-term follow-up, debated differences between Roux-en-Y gastric bypass and sleeve gastrectomy, and limited integration of microbial functional changes with host metabolic regulation. In particular, the causal links between microbiota remodeling and alterations in carbohydrate metabolism, bile acid signaling, and vitamin absorption remain incompletely understood, limiting translational application and microbiome-targeted interventions. In this minireview, we synthesize current clinical and experimental evidence on gut microbiota remodeling following bariatric surgery, with emphasis on species-specific alterations, temporal dynamics, and procedure-dependent metabolic consequences. We compare microbial responses across major surgical techniques, summarize short-and long-term patterns of microbiota adaptation, and integrate mechanistic insights involving microbial metabolites, bile acid metabolism, and gut hormone signaling. By consolidating longitudinal and multi-omics data, this review aims to clarify existing contro-

versies, highlight surgery-specific microbial signatures, and identify future research directions relevant to optimizing metabolic outcomes and postoperative management in bariatric surgery.

Key Words: Bariatric surgery; Gut microbiota; Roux-en-Y gastric bypass; Sleeve gastrectomy; Metabolic regulation

Core Tip: Emerging evidence indicates that bariatric surgery reshapes the gut microbiota in a procedure-specific and time-dependent manner, extending its metabolic benefits beyond anatomical restriction and caloric malabsorption. This minireview synthesizes current clinical and experimental data to highlight surgery-specific microbial signatures, longitudinal remodeling patterns, and key mechanistic links to bile acid signaling, carbohydrate metabolism, and micronutrient handling. By integrating species-level and functional insights, we emphasize existing controversies and translational considerations relevant to optimizing postoperative metabolic outcomes.

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INTRODUCTION

Obesity has emerged as one of the most pressing global health challenges, closely associated with type 2 diabetes (T2D), cardiovascular disease, nonalcoholic fatty liver disease, and increased all-cause mortality. Among available therapeutic strategies, bariatric surgery remains the most effective intervention for achieving sustained weight loss and long-term metabolic improvement in patients with severe obesity. Traditionally, the benefits of bariatric procedures have been attributed to anatomical restriction, malabsorption, and caloric reduction. However, growing evidence suggests that these mechanisms alone cannot fully explain the rapid and durable metabolic improvements observed after surgery, particularly the early resolution of insulin resistance and T2D.

Over the past decade, the gut microbiota has emerged as a key mediator linking bariatric surgery to host metabolic regulation. Early studies demonstrated that Roux-en-Y gastric bypass (RYGB) induces profound alterations in gut microbial composition, characterized by shifts in dominant phyla and enrichment of specific bacterial taxa associated with improved metabolic profiles[1,2]. Subsequent investigations expanded these observations to sleeve gastrectomy (SG), revealing that different surgical procedures produce distinct yet overlapping microbial signatures[3,4].

Importantly, bariatric surgery-induced microbiota remodeling is not merely a passive consequence of weight loss. Experimental studies have shown that transplantation of post-RYGB microbiota into germ-free mice can partially reproduce metabolic benefits, supporting a causal role for microbial alterations in mediating surgery-associated outcomes [2]. These findings prompted a paradigm shift, positioning the gut microbiota as an active participant in post-surgical metabolic regulation.

Despite substantial progress, the field remains marked by heterogeneity and unresolved questions. Reported trajectories of microbial diversity vary widely across studies, differences between RYGB and SG are inconsistently defined, and the functional relevance of observed taxonomic changes remains incompletely understood[5]. Moreover, the mechanisms linking microbiota remodeling to carbohydrate metabolism, bile acid signaling, gut hormone secretion, and micronutrient absorption remain under active debate.

In this minireview, we synthesize current clinical and experimental evidence on gut microbiota remodeling following bariatric surgery, with a focus on procedure-specific dynamics, temporal patterns, and metabolic implications. By integrating longitudinal and functional insights, we aim to clarify existing controversies and identify future research directions relevant to microbiome-informed optimization of bariatric surgery outcomes. The conceptual framework linking bariatric procedures, gut microbiota remodeling, and metabolic regulation is illustrated in Figure 1.

GLOBAL GUT MICROBIOTA REMODELING AFTER BARIATRIC SURGERY

Bariatric surgery induces rapid and sustained restructuring of the gut microbial ecosystem. Across multiple cohorts, both RYGB and SG are associated with reproducible changes in microbial composition, including enrichment of Proteobacteria and Verrucomicrobia and reductions in certain Firmicutes taxa. These alterations reflect profound changes in the intestinal environment, including nutrient flow, luminal pH, bile acid exposure, and oxygen availability. Key procedure-specific patterns of gut microbiota remodeling and their metabolic implications are summarized in Table 1.

Early postoperative periods are characterized by pronounced shifts in beta diversity, indicating rapid community restructuring, while alpha diversity often declines transiently following preoperative crash diets and early surgical stress [6]. It should be noted that perioperative antibiotic exposure may act as an additional confounder during this early phase, potentially contributing to short-term microbiota perturbations independent of surgical anatomy or long-term remodeling. Over time, microbial communities partially recover, though they rarely return to their preoperative config-

Table 1 Summary of gut microbiota remodeling after bariatric surgery and associated metabolic implications

Aspect	Roux-en-Y gastric bypass	Sleeve gastrectomy	Metabolic implications
Overall microbiota remodeling	Profound and extensive restructuring	Moderate but consistent remodeling	Contributes to sustained metabolic improvement
Dominant taxonomic shifts	Enrichment of facultative anaerobes and aero-tolerant taxa; reduction of obligate anaerobes	Enrichment of mucin-degrading and lactic acid-producing taxa	Alters microbial metabolic capacity
Microbial diversity dynamics	Often reduced early, followed by partial recovery or long-term reconfiguration	Generally stable or mildly increased over time	Diversity alone not predictive of outcomes
Bile acid-microbiota interaction	Markedly altered bile acid pools and signaling	Moderate changes in bile acid metabolism	Activation of FXR/TGR5 pathways improves glucose homeostasis
Carbohydrate metabolism	Reduced intestinal glucose absorption and altered fermentation	Improved glycemic regulation *via* microbial-host interaction	Enhances insulin sensitivity
Gut hormone modulation	Strong stimulation of GLP-1 and PYY secretion	Moderate incretin response	Supports appetite control and metabolic regulation
Vitamin and micronu-trient handling	Greater disruption of microbial vitamin utilization	Less pronounced alterations	May contribute to postoperative deficiencies
Long-term microbiota stability	Persistent, surgery-specific microbial signatures	Relatively conserved long-term profiles	Influences durability of metabolic benefits

GLP-1: Glucagon-like peptide-1; PYY: Peptide YY.

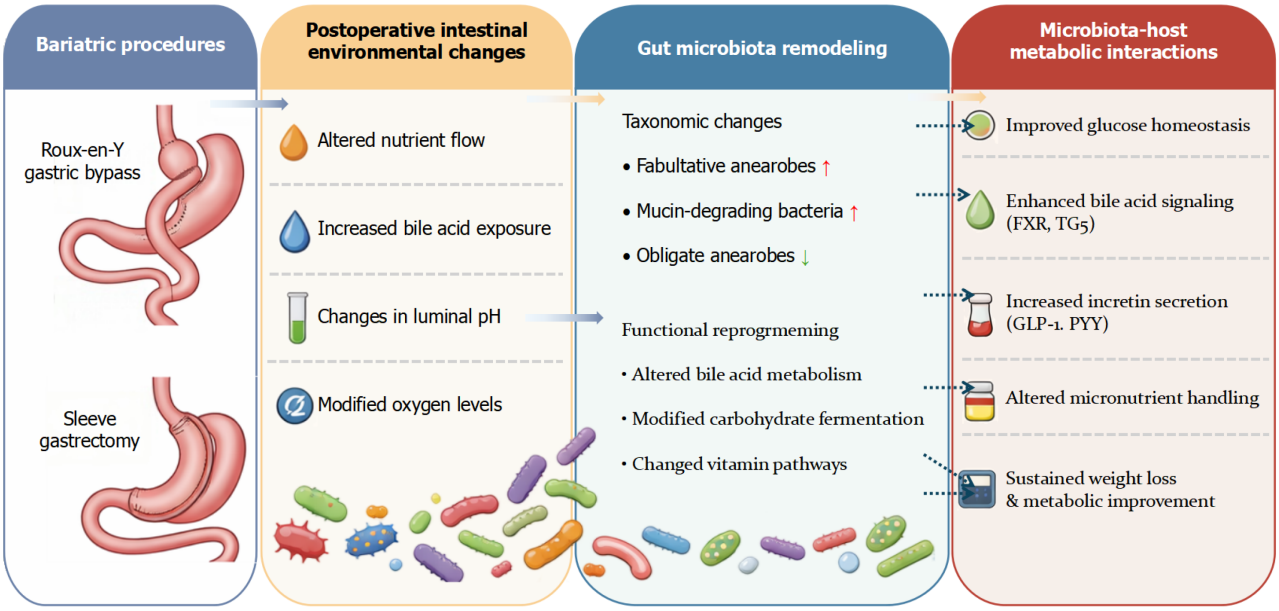

Figure 1 Conceptual framework of gut microbiota remodeling after bariatric surgery. The schematic illustrates proposed links between bariatric procedures, postoperative intestinal environmental changes, gut microbiota remodeling, and host metabolic outcomes. Solid arrows represent relationships supported by experimental or mechanistic evidence and are hypothesized to be causal, whereas dashed arrows indicate associations primarily derived from human observational studies, for which causality has not been firmly established. GLP-1: Glucagon-like peptide-1; PYY: Peptide YY.

uration, suggesting long-term ecological reprogramming rather than temporary dysbiosis[5].

PROCEDURE-SPECIFIC MICROBIOTA SIGNATURES

RYGB

RYGB consistently induces more extensive microbiota remodeling than SG. Compared with SG, RYGB has been associated with larger shifts in overall community structure, as reflected by greater postoperative changes in beta diversity metrics reported across multiple cohorts[7]. Studies report enrichment of facultative anaerobes and aero-tolerant taxa, including Enterobacteriaceae, Streptococcaceae, *Veillonella*, and *Escherichia*, alongside reductions in obligate anaerobic and butyrate-producing bacteria[1,8]. In several metagenomic and 16S rRNA-based analyses, these taxa exhibit

higher relative abundance increases after RYGB than after SG, supporting a more pronounced ecological restructuring.

These compositional changes are accompanied by functional reprogramming. Functional analyses further demonstrate that RYGB reshapes microbial gene content, with broader alterations in metabolic pathway representation, including pathways related to amino acid metabolism, vitamin utilization, and bile acid transformation[9]. Importantly, while the magnitude of microbiota remodeling varies substantially across studies, the overall pattern of larger beta diversity shifts and more marked taxonomic redistribution after RYGB compared with SG is consistently observed, supporting the characterization of RYGB as inducing more extensive microbiota remodeling[10].

SG

In contrast, SG induces subtler but metabolically relevant microbial changes. Several studies report enrichment of *Akkermansia muciniphila* and *Lactobacillus spp.*, taxa associated with mucin degradation, gut barrier function, and metabolic regulation[11,12]. SG tends to preserve anaerobic microbial communities to a greater extent than RYGB, consistent with its more limited anatomical rearrangement[3].

Despite inducing fewer taxonomic shifts, SG alters key metabolic pathways, particularly those related to bile acid signaling and short-chain fatty acid production, underscoring that modest compositional changes can still exert meaningful functional effects[13,14].

Temporal dynamics of microbial diversity and adaptation

Longitudinal studies highlight a biphasic pattern of microbiota remodeling. An initial decline in alpha diversity is frequently observed shortly after surgery, followed by gradual recovery or enhancement over subsequent months[15]. However, diversity trajectories vary substantially across individuals and procedures, and long-term follow-up studies reveal persistent enrichment of specific taxa years after surgery[16,17].

Importantly, microbial diversity alone does not consistently predict metabolic outcomes. Several studies report discordance between diversity metrics and clinical improvement, emphasizing that functional composition and metabolic capacity may be more informative than richness *per se*[18].

MICROBIOTA-METABOLISM INTERACTIONS

Carbohydrate metabolism and energy regulation

Surgery-induced microbiota remodeling alters carbohydrate metabolism through multiple mechanisms. Experimental studies demonstrate that post-surgical microbiota can reduce intestinal glucose absorption by modulating host transporters such as SGLT1, contributing to improved glycemic control independent of weight loss[19]. Altered fermentation patterns and short-chain fatty acids profiles further influence host energy harvest and metabolic signaling[17].

Bile acid signaling and gut hormones

Altered bile acid metabolism represents a central link between microbiota changes and metabolic benefit. Both RYGB and SG modify bile acid pools, increasing secondary bile acids that activate FXR and TGR5 signaling pathways, thereby enhancing insulin sensitivity, energy expenditure, and incretin secretion[20,21]. Microbiota-driven bile acid transformation also promotes secretion of glucagon-like peptide-1 and peptide YY, reinforcing microbial-endocrine crosstalk [22].

Vitamin and micronutrient metabolism

Bariatric surgery alters microbial pathways involved in vitamin synthesis and utilization, particularly following RYGB. Increased bacterial utilization of vitamins B1 and B12 has been reported, potentially contributing to postoperative deficiencies[23]. After RYGB, the enrichment of facultative anaerobes and changes in small intestinal transit and luminal nutrient availability may further exacerbate competition between host and microbiota for micronutrients, especially water-soluble vitamins such as thiamine (vitamin B1) and cobalamin (vitamin B12)[24]. These alterations are clinically relevant, as deficiencies in B1 and B12 are frequently observed after RYGB and are associated with neurological complications, anemia, and impaired energy metabolism.

In addition to vitamin metabolism, microbiota remodeling after RYGB may influence iron absorption through multiple mechanisms, including altered bile acid signaling, changes in intestinal pH, and shifts in microbial taxa involved in iron handling. Specific taxa such as *Lactobacillus spp.* have been implicated in modulating iron bioavailability *via* hypoxia-inducible factor-dependent pathways[12]. Disruption of these interactions, together with reduced absorptive surface and dietary intolerance, may contribute to the high prevalence of iron deficiency and iron-deficiency anemia observed in RYGB patients.

Collectively, these findings highlight that postoperative micronutrient deficiencies are not solely a consequence of anatomical bypass or reduced intake but may also reflect surgery-induced microbiota remodeling and functional reprogramming. This perspective underscores the need for procedure-specific and microbiota-informed supplementation strategies, as well as careful long-term monitoring of vitamin B1, vitamin B12, and iron status in patients undergoing RYGB.

CONTROVERSIES AND UNRESOLVED ISSUES

Despite extensive investigation, several key controversies remain. First, studies report inconsistent trajectories of microbial diversity following bariatric surgery, with some demonstrating long-term increases and others observing persistent reductions[5,25]. Differences in dietary protocols, sequencing methodologies, and sampling time points likely contribute to these discrepancies.

Second, the extent to which microbiota changes differ meaningfully between RYGB and SG remains debated. While many studies suggest more profound remodeling after RYGB, others report overlapping microbial profiles and comparable metabolic outcomes despite distinct taxonomic shifts[26,27].

Third, causality remains incompletely established. Although transplantation and animal studies support a contributory role of microbiota, many human studies remain associative, and disentangling microbial effects from anatomical, dietary, and hormonal factors remains challenging.

Finally, the clinical relevance of microbiota changes for postoperative complications, including micronutrient deficiencies, small intestinal bacterial overgrowth (SIBO), and inflammatory responses, remains insufficiently defined[28,29].

Among these complications, SIBO represents an increasingly recognized but underexplored issue, particularly after RYGB. Altered gastrointestinal anatomy, changes in motility, reduced gastric acid exposure, and modified bile acid delivery may collectively predispose patients to bacterial stasis and proximal microbial expansion[30]. From a microbial perspective, RYGB-associated enrichment of facultative anaerobic and aero-tolerant taxa, such as Enterobacteriaceae and Streptococcaceae, may facilitate small intestinal colonization under altered luminal conditions[31]. Clinically, SIBO may contribute to gastrointestinal symptoms, malabsorption, and micronutrient deficiencies; however, its true prevalence and causal role in postoperative dysbiosis remain incompletely defined due to observational study designs and potential confounding[32]. This uncertainty underscores the need for increased clinical awareness and targeted surveillance for SIBO in the long-term management of post-RYGB patients.

DIETARY MODULATION OF POSTOPERATIVE MICROBIOTA TRAJECTORIES

Longitudinal studies highlight substantial inter-individual variability in postoperative microbiota trajectories, suggesting that factors beyond surgical anatomy contribute to long-term microbial adaptation. Among these factors, postoperative diet represents a key and modifiable determinant of microbiota composition and function[33,34].

Protein intake after bariatric surgery may influence microbial amino acid metabolism and nitrogen utilization, potentially favoring the expansion of proteolytic and facultative anaerobic taxa, particularly after RYGB[35]. In contrast, dietary fiber availability can promote short-chain fatty acid-producing bacteria and support the recovery or maintenance of anaerobic microbial communities, especially after SG. In addition, changes in meal patterns, including reduced meal size, altered feeding frequency, and modified nutrient timing, may further interact with surgery-induced alterations in gut transit and bile acid exposure, thereby shaping microbial succession over time[36].

Together, these observations indicate that postoperative dietary composition and eating behavior may modulate the magnitude and direction of microbiota remodeling, highlighting diet as a clinically actionable factor that may influence long-term microbial adaptation and metabolic outcomes after bariatric surgery.

FUTURE DIRECTIONS

Future research should prioritize large-scale, longitudinal studies with standardized methodologies to define durable microbiota signatures associated with optimal metabolic outcomes. As highlighted in the main text, substantial inter-individual variability in postoperative microbiota trajectories and metabolic responses represents a key challenge in the field, underscoring the need for study designs that explicitly account for baseline microbial heterogeneity. Integration of multi-omics approaches, including metagenomics, metabolomics, and host transcriptomics, will be essential to move beyond descriptive associations toward mechanistic understanding[37].

Procedure-specific studies directly comparing RYGB and SG under controlled conditions are needed to clarify true microbial and functional differences. In this context, growing evidence suggests that preoperative gut microbiota composition may partially predict postoperative microbial remodeling and metabolic response, supporting its potential role as a biomarker for patient stratification and personalized surgical decision-making.

Finally, translational efforts should explore microbiota-modulating strategies, including probiotics, dietary interventions, and bile acid–targeted therapies, as adjuncts to bariatric surgery. Such approaches hold promise for enhancing metabolic benefits, minimizing nutritional complications, and optimizing long-term outcomes in patients undergoing bariatric surgery, particularly when informed by individual baseline microbiota profiles.

CONCLUSION

Bariatric surgery induces profound, procedure-specific, and time-dependent remodeling of the gut microbiota that extends beyond anatomical restriction and caloric malabsorption to actively shape postoperative metabolic regulation.

Accumulating clinical and experimental evidence indicates that distinct microbial signatures, functional reprogramming, and altered microbe-host interactions contribute to improvements in carbohydrate metabolism, bile acid signaling, gut hormone secretion, and micronutrient handling. Nevertheless, substantial heterogeneity across studies and incomplete mechanistic resolution continue to limit translational application. A clearer understanding of surgery-specific microbial dynamics and their causal metabolic roles will be essential for integrating microbiome-informed strategies into perioperative management and for optimizing long-term outcomes after bariatric surgery.

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FOOTNOTES

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