



IMPACT OF BARIATRIC SURGERY ON GUT MICROBIOTA

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Abstract

Obesity is associated with gut microbial dysbiosis, reduced microbial diversity and metabolic disturbances. Bariatric surgery (notably Roux-en-Y gastric bypass, sleeve gastrectomy and biliopancreatic diversion) is an effective treatment for morbid obesity and its comorbidities. Increasing evidence suggests that beyond mechanical restriction and malabsorption, bariatric procedures exert profound effects on the gut microbiota, altering microbial composition, metabolic function, and host–microbe interactions. These shifts may contribute to improved insulin sensitivity, inflammation modulation, altered energy harvest, and appetite regulation. However, the temporal dynamics, mechanistic mediators, and long-term stability of microbiota changes remain incompletely understood. This article reviews current knowledge on how different bariatric procedures affect gut microbiota (taxonomic, functional, metabolomic), the plausible mechanistic pathways (bile acids, pH, gut transit, hormonal shifts), and the clinical significance of such microbial modulation for metabolic outcomes.

Keywords: Bariatric surgery, gut microbiota, microbial diversity, metabolic outcomes, bile acids, gut hormones



Introduction

Obesity remains a leading global health challenge linked to numerous comorbidities such as type 2 diabetes mellitus (T2DM), cardiovascular disease, nonalcoholic fatty liver disease, and increased mortality. Conventional treatments—diet, lifestyle, pharmacotherapy—often fail to deliver sustained weight loss in patients with severe obesity. Bariatric surgery has emerged as the most effective long-term intervention for significant and durable weight reduction, remission of metabolic diseases, and improved quality of life.

Historically, the beneficial effects of bariatric surgery were attributed to mechanical restriction of food intake, malabsorption of nutrients, hormonal changes (e.g., GLP-1, PYY, ghrelin), and alterations in energy expenditure. However, accumulating evidence reveals that **gut microbiota**—the complex community of microorganisms in the gastrointestinal tract—plays a pivotal role in obesity pathophysiology and may mediate some of the metabolic benefits conferred by bariatric procedures.

Gut microbiota influences host energy balance, glucose and lipid metabolism, immune/inflammatory states, gut barrier integrity, and gut–brain signaling through microbial metabolites (short-chain fatty acids, bile acids, lipopolysaccharides) and cross-talk with host receptors. Obesity is often associated with gut dysbiosis, characterized by reduced microbial richness, alterations in Firmicutes/Bacteroidetes ratio, expansion of pro-inflammatory taxa, and endotoxemia from microbial products.

Thus, bariatric surgery provides a “natural experiment” in which dramatic anatomical, physiological, and dietary changes may restructure the gut biochemical milieu and thus reshape the microbial ecosystem. By studying how surgery modulates gut microbiota, we can better understand mechanisms of metabolic improvement and perhaps develop adjunctive microbial therapies. This article addresses how different bariatric procedures affect the gut microbiota (taxonomically and functionally), the key mechanistic drivers of those changes, and the clinical significance of the microbiota shifts in metabolic outcomes.



Main Body

1. Microbiota Alterations After Bariatric Surgery: Taxonomic and Functional Shifts

1.1 Baseline Microbiota in Obesity and Dysbiosis

Before diving into post-surgical changes, it is critical to summarize the typical dysbiotic signatures observed in obese individuals. Many studies report that obesity is associated with **lower microbial diversity (α -diversity)** and alterations in key phyla composition. In particular:

- An increased **Firmicutes/Bacteroidetes (F/B)** ratio is frequently described, though with heterogeneity across cohorts and methods.
- Overrepresentation of certain Firmicutes genera (e.g., some *Clostridium* clusters) and reduction in beneficial taxa such as ***Akkermansia muciniphila*** and ***Faecalibacterium prausnitzii*** have been observed.
- Elevated presence of endotoxin-producing gram-negative bacteria (e.g., Proteobacteria) may contribute to systemic low-grade inflammation via lipopolysaccharide (LPS) translocation (metabolic endotoxemia).
- Functional shifts in microbial metabolism toward increased energy harvest (fermentation of polysaccharides) and altered bile acid biotransformation are often reported.

However, heterogeneity is large: geographic, dietary, methodological (16S rRNA sequencing vs metagenomics), and confounding by medications (antibiotics, proton-pump inhibitors) all blur the consistency of signatures.

1.2 Taxonomic and Alpha/Beta Diversity Changes After Surgery

Post-bariatric surgery, numerous human studies and animal models consistently report a **rise in microbial diversity (α -diversity)** and shifts in β -diversity (community structure). For example, bariatric surgery “seems to entail an improvement in gut dysbiosis, leading to an increased diversity of the gut microbiota.”[1] Several systematic reviews and meta-analyses confirm this trend across procedures (RYGB, SG)[2].

At the taxonomic level, salient changes include:

- **Decrease in Firmicutes** relative abundance in many studies, reducing the F/B ratio.



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- **Increase in Proteobacteria** (especially facultative anaerobes such as *Escherichia/Shigella*) after surgery. This may reflect a greater luminal oxygen tension or nutrient flow shift [3].
 - **Enrichment of Bacteroidetes**, or relative increase in *Bacteroides* and related taxa, in many cohorts [4].
 - **Rise in beneficial taxa** like *Akkermansia muciniphila*, *Bifidobacterium*, some *Veillonella* spp., *Prevotella* spp., or SCFA-producing genera in a subset of patients [5].
 - Decrease in certain *Clostridium* clusters, *Dorea*, *Blautia*, *Eubacterium rectale*, *Roseburia* in some reports [4].
 - Notably, some taxa respond variably depending on time post-surgery, patient diet, and surgical type, so inconsistencies exist.

One recent observational study (Amb Express) in obese patients undergoing RYGB or SG found significant shifts in *Bacteroides* and *Fusobacterium* by qPCR at 3 and 6 months, and correlations between BMI, weight loss, and bacterial groups like *Lactobacillus* and *Clostridium* cluster IV [2].

Another recent finding (preprint) reports that a **species of Coprococcus** correlates inversely with BMI pre- and post-operatively, and its abundance is modified by magnesium and thiamin intake after malabsorptive surgery [6].

In sum, bariatric surgery tends to remodel the gut microbiome toward greater diversity and more favorable taxa, though heterogeneity and inter-individual variation are considerable.

1.3 Functional and Metabolomic Alterations: SCFAs, Bile Acids, and Other Metabolites

Beyond taxonomy, the functional/metabolic outputs of the microbiome undergo substantial shifts post-surgery, which may mediate host metabolic benefits.

- **Short-chain fatty acids (SCFAs)** (acetate, propionate, butyrate) are generated by bacterial fermentation of dietary fibers. SCFAs modulate gut barrier, appetite regulation, and systemic metabolism. Some studies post-surgery show altered SCFA profiles—often a relative reduction in butyrate producers (e.g., *Roseburia*, *Faecalibacterium*) but also increased propionate/acetate in some contexts [7].



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- **Bile acid (BA) pool and biotransformation:** Microbiota deconjugate and convert primary BAs to secondary BAs (via dehydroxylation). Post-surgery, increased luminal bile acid flux and altered BA recycling have been documented, and microbial shifts in BA-transforming species (e.g. *7 α* -dehydroxylating *Clostridium* spp.) are observed. Changes in BA pool composition influence signaling via the farnesoid X receptor (FXR) and TGR5, modulating glucose metabolism, energy expenditure, and microbiota composition itself (feedback) [8].
 - **Metabolomic shifts:** Some shotgun metagenomics or metabolomics studies report altered microbial gene pathways (e.g., carbohydrate utilization, amino acid metabolism, lipopolysaccharide biosynthesis, vitamin biosynthesis) post-surgery [9]. For instance, enhanced microbial pathways for propionate production or reduced endotoxin-related gene potential have been reported.
 - **Microbial-derived secondary metabolites** (e.g. trimethylamine, branched-chain amino acids, tryptophan metabolites) may also shift, influencing systemic insulin sensitivity, inflammation, and gut–liver axis communication. A comprehensive schematic of how bacterial composition, metabolites, host receptors, and downstream metabolic effects interrelate has been proposed in recent reviews [5].

1.4 Dynamics Over Time and Stability

The time course of microbiota changes is an active area of research. Many studies examine early (weeks to months) changes; fewer follow patients into long-term (years).

- In early postoperative phases, abrupt shifts in diversity and taxa are common, likely driven by major changes in diet, gut transit, and anatomical rearrangement.
- Over weeks to months, some taxa show sustained changes (e.g., persistent increase in *Akkermansia*, *Proteobacteria*), whereas others reverse toward baseline (especially in patients with weight regain).
- Some studies suggest that the preoperative microbiome may predict postoperative success in weight loss or metabolic response [10].



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- However, long-term sustainability of beneficial microbial changes remains uncertain, and in some patients, microbial drifting or relapse of dysbiosis may occur.

Thus, characterizing the stability and resilience of the post-surgical microbiome, and whether it becomes a new “set point,” is essential for linking microbiota changes with durable metabolic benefits.

2. Mechanistic Mediators: How Bariatric Surgery Reshapes the Microbial Ecology

Understanding why the microbiota changes after surgery helps link microbial shifts to host metabolic effects. Key mechanistic mediators include:

2.1 Anatomical and Physiological Changes: Gastric Volume, Transit, pH, Oxygen

- **Altered luminal pH:** Reduction of gastric volume and acid-producing mucosa leads to higher gastric and proximal intestinal pH. This change can allow survival or colonization of bacteria ordinarily suppressed in low pH, and may permit small intestinal bacterial overgrowth (SIBO) or shifts in microbial ecology downstream [3].

- **Faster intestinal transit and altered nutrient flow:** Some surgical designs bypass segments of intestine or cause faster delivery of nutrients to distal gut, altering substrate availability for microbes [8].

- **Increased luminal oxygen or redox potential:** With anatomical rearrangement and reduced microbial density in portions of intestine, luminal oxygen diffusion may increase, favoring facultative anaerobes (e.g., Proteobacteria) at the expense of strict anaerobes [3].

- **Altered bile and pancreatic juice flow:** In bypass procedures, the mixing point of bile acids, digestive enzymes, and nutrients shifts distally, causing differential microbiota exposure to bile acids [8].

Thus, the physical restructuring of the gastrointestinal tract redefines the microbial “niche” (substrates, pH, oxygen, bile environment), compelling a reorganization of bacterial communities.

2.2 Bile Acid–Microbiota Interplay

One of the most compelling mechanistic links is via the **bile acid–microbiota–host axis**:



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- Bariatric surgery increases circulating and luminal bile acids (especially in RYGB).
 - Microbiota deconjugate, dehydroxylate, and convert bile acids; conversely, bile acids (especially secondary BAs) exert antimicrobial effects, shaping microbial composition [8].
 - Bile acids act as signaling molecules via FXR and TGR5. Activation of FXR modulates bile acid synthesis (feedback), lipid and glucose metabolism in the liver and intestine. TGR5 activation in enteroendocrine and other cells stimulates release of GLP-1, energy expenditure, and anti-inflammatory pathways [5].
 - Therefore, post-surgical changes in bile acid flux may “steer” the microbiome toward taxa adapted to bile stress, while the new microbiota further influences BA composition in a reciprocal feedback loop.

2.3 Gut Hormones, Appetite, and Host–Microbe Signaling

Bariatric surgery is well known to alter gut-derived hormones (e.g. GLP-1, PYY, ghrelin, CCK, leptin). These hormonal shifts may influence microbiota either directly or indirectly:

- GLP-1 and PYY may alter gut motility, transit time, and changes in nutrient absorption, thereby changing microbial substrate exposure [5].
- Ghrelin and leptin have been correlated with certain microbial taxa; for example, leptin level positively correlates with *Mucispirillum* and *Lachnospiraceae*, and negatively with *Bacteroides* [4].
- Some microbial metabolites (e.g., SCFAs) act on enteroendocrine L-cells or neurons of the gut–brain axis to influence appetite, satiety, and energy homeostasis. Therefore, a bidirectional interaction emerges: microbial changes modulate hormone secretion, which in turn influences microbial ecology.

2.4 Immune, Inflammatory, and Barrier Effects

Postoperative microbial shifts may influence gut barrier integrity, local immune tone, and systemic inflammation:

- A healthier microbiota (higher diversity, reduced endotoxin-producing taxa) may reduce **metabolic endotoxemia** (LPS translocation), lowering systemic inflammation and insulin resistance.



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- Enhanced production of SCFAs can strengthen tight-junction proteins, improve mucosal barrier, and modulate regulatory T cell populations.
 - Some taxa (e.g. Akkermansia) are associated with mucin degradation and enhanced mucosal health.
 - Microbiota-derived metabolites can modulate local immune cell activation (e.g. macrophages, ILCs) and cytokine profiles, thus indirectly influencing metabolic homeostasis.

2.5 Nutrient and Dietary Influences, Micronutrient Status

Postoperative diet (often highly restricted, protein-rich, fiber-poor initially) strongly influences microbial substrates. Over time, diet diversification gradually reintroduces fermentable fiber, shaping microbial recovery.

Moreover, bariatric surgery often causes **micronutrient deficiencies** (e.g., B vitamins, magnesium, iron). Since microbial communities synthesize or utilize micronutrients, such deficiencies may feedback on microbial ecology. The recent report linking Coprococcus abundance to magnesium and thiamin intake is one example [10].

In summary, the interplay of anatomical, chemical, hormonal, immunologic, and nutritional factors drives the post-surgical remodeling of the gut microbiome.

3. Clinical Implications: Microbiota, Metabolic Outcomes, and Therapeutic Prospects

3.1 Association with Metabolic Improvements

A central question is whether microbial changes mediate or merely accompany metabolic benefits after bariatric surgery (e.g., weight loss, insulin sensitivity, lipid profile improvements). Evidence supporting causality is emerging:

- Some studies report correlations between the abundance of Akkermansia, Bacteroides, or other taxa and greater weight loss or improvements in insulin resistance [6].
- Changes in bile acid–microbiota signaling (e.g. increased secondary bile acids, raised FXR/TGR5 activation) are postulated to foster glucose homeostasis improvements [8].
- Rodent studies and human microbial transplantation experiments (from rodent models) suggest that transfer of a post-bariatric microbiome can confer



weight or metabolic benefits. While human fecal microbiota transfer (FMT) trials remain rare, this lends a mechanistic plausibility.

- Some studies suggest that preoperative microbiota composition might predict postoperative metabolic outcomes, potentially offering prognostic or stratification value [10].

Nevertheless, cause–effect proof in humans remains elusive, and inter-individual heterogeneity complicates the interpretation.

3.2 Risks, Unintended Effects, and Dysbiosis Relapse

While most attention focuses on positive shifts, there are potential risks or undesirable microbial consequences:

- Overgrowth of opportunistic bacteria or small intestinal bacterial overgrowth (SIBO) in bypassed or blind loop segments may lead to symptoms (bloating, diarrhea, malabsorption).
- In some patients, microbial drift over time may re-establish dysbiosis, especially if diet lapses or weight regain occurs.
- Alterations in microbial metabolism might negatively influence micronutrient absorption, vitamin biosynthesis, or drug metabolism.
- Some taxa enriched postoperatively (e.g. Proteobacteria) may carry inflammatory or pathogenic potential if not balanced.

Thus, continuous monitoring and understanding of microbial resilience is important in long-term care [12].

3.3 Microbiota-Targeted Adjuncts: Probiotics, Prebiotics, Synbiotics, FMT

Given the importance of microbiota in mediating benefits, researchers are exploring adjunct interventions to steer microbial remodeling:

- **Probiotics or live biotherapeutic products:** Several small trials have tested probiotic administration in bariatric patients to ameliorate gastrointestinal symptoms, reduce bacterial overgrowth, or modulate weight outcomes. Results are mixed; strain selection, timing, and doses vary [3].
- **Prebiotics (fiber, oligosaccharides):** Enriching fermentable substrates may support favorable taxa (e.g. SCFA producers). However, dietary tolerances post-surgery are restrictive initially.



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- **Synbiotics** (combination of prebiotics + probiotics) hold promise but remain under-investigated in bariatric cohorts.
 - **Fecal microbiota transplantation (FMT)**: Though speculative, FMT from metabolically healthy donors or post-bariatric donors may be a future approach to augment microbial remodeling in patients with suboptimal response.
 - **Personalized microbial therapeutics**: In the future, preoperative microbial profiling may guide tailored probiotic or dietary interventions to optimize post-surgical microbiome trajectories.

3.4 Practical and Clinical Considerations

From a clinician standpoint:

- Monitoring microbial health (e.g. stool diversity, marker taxa) is not yet standard, but may become part of precision bariatric care.
- Diet and fiber optimization postoperatively should consider microbiota support (gradual reintroduction of fermentable fiber as tolerated).
- Probiotic use should be evidence-informed and phenotype-matched; indiscriminate use may not yield benefit.
- Long-term follow-up is essential to detect microbiota-related complications (e.g. SIBO, dysbiosis).

Conclusion

Bariatric surgery constitutes a powerful intervention not only by mechanical and hormonal means but also by reshaping the gut microbial ecosystem. Postoperative increases in microbial diversity, reduction in unfavorable taxa (e.g. some Firmicutes), enrichment of beneficial taxa (e.g. Akkermansia, Bacteroides), and functional metabolic shifts (e.g. altered SCFA and bile acid profiles) all appear to contribute—directly or indirectly—to improved metabolic health, insulin sensitivity, and weight maintenance.

These microbial changes are driven by a confluence of anatomical, physiological, dietary, hormonal, and immunologic perturbations. Yet, considerable inter-individual variability and incomplete understanding of long-term stability, causality, and optimal microbial modulation strategies remain. Future directions include:



1. Longitudinal, large-cohort studies to define durable microbial traits and link them with clinical trajectories
2. Mechanistic human intervention trials (e.g. probiotic, prebiotic, FMT) to test causality and therapeutic potential
3. Integration of multi-omics (metagenomics, metabolomics, host transcriptomics) to resolve host–microbe interaction networks
4. Personalized microbiota-guided strategies to enhance bariatric success and mitigate adverse outcomes.

In sum, the gut microbiota represents both a mechanistic mediator and a potential therapeutic target in the context of bariatric surgery. Understanding and harnessing this dimension may refine surgical outcomes, personalize care, and possibly extend benefits to non-surgical obesity therapies.

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