Annals of Pharmacy Practice and Pharmacotherapy
ISSN: 3062-4436
2025, Volume 5, Page No: 194-200

Available online at: www.galaxypub.co/page/journals

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Drug-Based Strategies for Weight Reduction and Their Interaction with the Human Gut Microbiota

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Received: 07 September 2025; **Revised:** 29 November 2025; **Accepted:** 07 December 2025

ABSTRACT

At the onset of the twenty-first century, obesity has emerged as a major global health concern due to its widespread prevalence in modern populations. It is a metabolic disorder characterized by excessive accumulation of adipose tissue, primarily driven by increased caloric intake, reduced energy expenditure, and dysregulation of hunger signals. Obesity is a chronic systemic condition that contributes to numerous health complications, with the most common being type 2 diabetes mellitus, arterial hypertension, atherosclerosis, sleep apnea, various forms of cancer, and more.

Over the past decade, considerable progress has been made in the pharmacological management of endocrine and metabolic disorders, and several of these new therapies have demonstrated efficacy in treating obesity. Growing evidence suggests that such medications may influence the human gut microbiota—a complex community of commensal bacteria residing in the gastrointestinal tract—which can impact appetite regulation, mucosal integrity, and nutrient absorption. Understanding this interaction may be pivotal for elucidating the mechanisms underlying obesity and optimizing its treatment.

Keywords: Weight reduction, Antiobesity drugs, Obesity, GI tract microorganisms

How to Cite This Article: Lee V, Kim E, Singh J. Drug-Based Strategies for Weight Reduction and Their Interaction with the Human Gut Microbiota. Ann Pharm Pract Pharmacother. 2025;5:194-200. https://doi.org/10.51847/OobVHFcZjv

Introduction

Obesity, along with the related metabolic syndrome, is increasingly recognized as a critical public health problem worldwide, posing significant health risks in both developed and developing nations, with prevalence rising among adults and children alike [1–4]. This chronic systemic condition is marked by excessive fat accumulation in the body, primarily driven by a combination of high-energy diets—rich in calorie-dense foods, simple sugars, and saturated fats—disrupted appetite regulation, and reduced physical activity associated with sedentary lifestyles [1–4]. The development of obesity is multifactorial, involving social, economic, environmental, genetic, and metabolic contributors, as well as endocrine disturbances [2–4]. Hormonal imbalances in the central nervous system, affecting molecules such as ghrelin, leptin, proopiomelanocortin (POMC), and cocaine-amphetamine-regulated transcript (CART), further influence hunger and energy regulation [2–4].

The consequences of obesity extend to multiple organs and systems, increasing the risk for cardiovascular diseases (including endothelial dysfunction, atherosclerosis, heart attack, and stroke), kidney impairment, liver disorders like non-alcoholic fatty liver disease (NAFLD), certain cancers (e.g., liver and pancreatic cancer), and metabolic disorders such as type 2 diabetes [2, 4, 5]. Obesity is also associated with persistent low-grade systemic inflammation, reflected by elevated circulating pro-inflammatory markers, including interleukins IL-1 and IL-6, tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP) [2, 4, 5]. This chronic inflammatory state activates immune cells, increases vascular permeability, and promotes immune infiltration into tissues such as adipose tissue, skeletal muscles, liver, and pancreas, ultimately resulting in oxidative stress, free radical production, and cellular damage, which manifest as organ dysfunctions [2, 4, 5].

Conventional approaches to obesity management

Given obesity's widespread impact, implementing effective prevention and treatment strategies is essential. Management should begin with a comprehensive evaluation of the patient's individual factors, including dietary habits, physical activity levels, genetic predisposition, family history of obesity, prior weight loss attempts, and medical conditions or medications that may contribute to weight gain. This personalized assessment helps identify the primary drivers of obesity in each patient [6, 7].

Lifestyle modifications remain the foundation of obesity treatment, encompassing dietary changes, increased exercise, and behavioral therapy [6, 7]. Weight loss is achieved when caloric intake is lower than energy expenditure over time. Dietary strategies focus on creating a negative energy balance, typically with a daily deficit of 500–750 kcal, and may involve altering macronutrient composition through low-fat, low-carbohydrate, high-protein, or high-fiber diets [6, 7]. Regular physical activity complements dietary interventions by enhancing weight reduction and supporting long-term maintenance of weight loss [6, 7]. Cognitive behavioral therapy aids patients in adhering to these lifestyle changes but requires consistent effort and motivation [6, 7]. Combining dietary, physical, and behavioral strategies generally leads to the most sustainable outcomes in weight management **(Table 1)**.

Table 1. Step-wise approach in the treatment of obesity.

	Normal weight BMI = 20–24.9	**Overweight BMI = 25–29.9**	**Class I Obesity BMI = 30–34.9**	**Class II Obesity BMI = 35–39.9**	**Class III Obesity BMI > 40**
Balanced diet Physical activity	+	+	+	+	+
Cognitive Behavioral Therapy		+	+	+	+
Pharmacological Therapy		* In the presence of obesity-related complications; comorbidities	+	+	+
Surgical therapy			* If pharmacological and cognitive behavioral therapy proves ineffective; comorbidities	* In the presence of obesity-related complications; comorbidities	+

Legend: Body Mass Index (BMI), expressed in kg/m², is presented according to Nuttall (2015) [1]. Therapeutic strategies are indicated following Wirth *et al*. (2014) and Shukla *et al*. (2015) [6, 7].

When the initial multidisciplinary treatment approach fails to achieve the desired weight and health outcomes—typically in cases of persistent obesity that do not respond to the previously applied interventions and demonstrate early signs of obesity-related complications—additional pharmacological therapy may be necessary.

Emerging pharmacological strategies for obesity management

Recent studies indicate that excessive food intake is largely driven by disrupted communication between hormones and central nervous system (CNS) satiety centers. Specifically, obesity has been linked to an imbalance in orexigenic hormones (elevated ghrelin) and anorexigenic hormones (reduced leptin, cholecystokinin, peptide YY /PYY/, and glucagon-like peptide-1 /GLP-1/), leading to impaired signaling to hypothalamic centers that regulate satiety—a critical factor in the pathophysiology of obesity [8, 9].

In recent years, the incretin system has emerged as a promising pharmacological target. Incretins are small peptides secreted by enteroendocrine L-cells of the small intestine in response to glucose and include glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) [8, 9]. These molecules act as metabolic regulators, enhancing pancreatic insulin secretion while suppressing glucagon release, and also influence peripheral tissues and CNS regions involved in appetite, satiety, and food intake [8, 9]. By interacting with receptors in the CNS, gastrointestinal tract, liver, pancreas, skeletal muscle, and adipose tissue, incretins modulate energy balance, glucose metabolism, and insulin sensitivity. Their effects include reduced hunger, lower

caloric intake, and delayed gastric emptying. Notably, GLP-1 secretion from the intestinal epithelium is diminished in patients with type 2 diabetes and obesity, highlighting its role in the pathophysiology of these metabolic disorders [8, 9].

However, the therapeutic use of endogenous GLP-1 is severely limited due to its extremely short half-life (~2 minutes), as it is rapidly degraded by the enzyme dipeptidyl peptidase-4 (DPP-4) [8, 9]. To overcome this limitation and harness its clinical benefits, GLP-1 receptor agonists have been developed. These agents mimic the actions of natural GLP-1—primarily stimulating insulin secretion from pancreatic beta-cells—but are resistant to DPP-4 degradation [8, 9]. Currently available GLP-1 receptor agonists include Exenatide, Liraglutide, Semaglutide, Dulaglutide, and Lixisenatide **(Table 2)**. While initially approved for managing type 2 diabetes, these drugs not only maintain effective glycemic control but also replicate the anorexigenic effects of endogenous incretins. By influencing both central and peripheral pathways, they reduce appetite, lower food intake, and support long-term weight reduction [8, 9].

Table 2. Glucagon-like peptide-1 receptor agonist drugs.

GLP-1 Receptor Agonist Generic name	GLP-1 Receptor Agonist Trademark name (Authorized for use in the EU)	Method of administration	Pharmacological effects (identical for all medications in the group)	Side effects (identical for all medications in the group)
Exenatide	Byetta ®	subcutaneous	↑ insulin	GI disturbance
	Bydureon ®	subcutaneous	secretion	- nausea
Liraglutide	Victoza ®	subcutaneous	↓ glucagon	- vomiting
	Saxenda ®	subcutaneous	secretion	- diarrhea
Semaglutide	Ozempic ®	subcutaneous	↓ blood glucose	Hypoglycemia
	Wegovy ®	subcutaneous	levels	/rare/
	Rybelsus ®	oral tablets	↓ weight	(?) ↑ risk of
Dulaglutide	Trulicity ®	subcutaneous	↓ hunger	pancreatitis
Lixisenatide	Lyxumia ®	subcutaneous	↓ appetite	pancreatic
			↓ food intake	cancer

Legend: Glucagon-like peptide-1 (GLP-1) receptor agonists are listed with their generic and trademark names (for drugs approved by the European Medicines Agency /EMA/), along with additional data relevant to their clinical applications, according to Ryder (2013), Consoli and Formoso (2014), Sharma *et al*. (2018), and Popoviciu *et al*. (2023) [8–11].

GLP-1 receptor agonists are generally considered safe and well tolerated. The most commonly reported adverse effects are gastrointestinal in nature, including nausea, vomiting, and diarrhea; in severe cases, these may necessitate discontinuation of therapy. Hypoglycemia is very rare, typically occurring only at higher doses when combined with sulfonylurea-class antidiabetic medications, and is extremely uncommon when the drugs are used alone. For subcutaneously administered formulations, mild local inflammatory reactions at the injection site may occur [9–11]. Some data suggest that long-term use of GLP-1 receptor agonists could be linked to a higher risk of acute pancreatitis or pancreatic cancer, though these associations have not been conclusively confirmed in large-scale clinical trials. Therefore, caution is recommended, including periodic monitoring of pancreatic enzymes such as amylase and lipase in patients undergoing prolonged therapy [9–11].

Beyond their known therapeutic effects, emerging research indicates that GLP-1 receptor agonists may also interact with the gut microbiota—the complex community of microorganisms residing in the gastrointestinal tract—which itself has become an important target for interventions in endocrine and metabolic disorders, including obesity and type 2 diabetes.

Human gut microbiota – composition and physiological roles

The gut microbiota represents a diverse ecosystem of commensal bacteria inhabiting the human gastrointestinal tract, particularly the intestines and colon. Over the past two decades, extensive research has provided insights into its composition, physiological functions, and involvement in disease pathogenesis [12]. The human GI tract

contains over 100 trillion bacterial cells, predominantly from the phyla Bacteroidetes (genera Bacteroides, Prevotella), Firmicutes (genera Clostridium, Enterococcus, Lactobacillus), with smaller populations from Actinobacteria (genus Bifidobacterium) and Proteobacteria (genera Helicobacter, Escherichia) [12, 13].

These microorganisms contribute to numerous local and systemic physiological processes, including inhibiting pathogenic bacterial growth, maintaining intestinal epithelial barrier integrity, aiding digestion and fermentation in the colon, modulating nutrient absorption and energy balance, and supporting overall metabolic and endocrine homeostasis [12, 13]. Consequently, the gut microbiota exerts a profound influence on multiple physiological systems, and alterations in its composition have been implicated in conditions such as obesity. Experimental studies and human evaluations demonstrate that obese individuals often exhibit marked deviations in microbiota composition [12, 13].

In healthy individuals, the gut microbiota is dominated by two major phyla, Firmicutes and Bacteroidetes, which together account for up to 90% of intestinal bacteria [14, 15]. In obesity, this balance is disrupted, with high-calorie diets rich in saturated fats and simple carbohydrates promoting an increase in Firmicutes and a decrease in Bacteroidetes [14, 15]. Because gut bacteria play a key role in nutrient absorption and energy homeostasis, such shifts can contribute to the development of metabolic and endocrine disorders, including obesity.

Recent scientific interest has focused on interactions between gut microbes and intestinal host cells, particularly enteroendocrine cells, which detect luminal chemical signals and release regulatory peptides that influence systemic homeostasis [16]. L-type enteroendocrine cells, for example, possess membrane-bound G-protein coupled receptors such as GPR41 and GPR43, which respond to microbial metabolites by secreting regulatory peptides, including GLP-1 and peptide YY—both anorexigenic hormones. This exemplifies a finely tuned mechanism by which the gut microbiota and host endocrine cells collaborate to regulate energy balance and metabolic function [16].

GLP-1 receptor agonists and the gut microbiota

Over the past decade, GLP-1 receptor agonists have demonstrated strong efficacy in treating both type 2 diabetes mellitus and obesity [17, 18]. Given their widespread use in endocrinology, researchers have increasingly explored a potential link between the weight-reducing effects of these drugs and alterations in the composition and function of gut microbiota. Emerging evidence suggests that such a connection is not only plausible but likely, providing valuable insight into the pathogenesis of metabolic disorders [17, 18].

Current understanding of how GLP-1 receptor agonists influence gut dysbiosis is primarily derived from studies employing PCR-based techniques such as 16S rDNA and 16S rRNA sequencing to identify bacterial operational taxonomic units (OTUs) in fecal samples of experimental subjects. More recently, metagenomic sequencing has been utilized, driven by decreasing sequencing costs [17, 18]. However, study outcomes can vary significantly due to differences in experimental models—despite general similarities between human and rodent gut microbiota, species-specific differences may influence results. Additionally, microbiota composition is not uniform along the gastrointestinal tract; sampling from different regions (ileum, cecum, colon) can yield varying results [17, 18]. Individual factors such as diet, age, comorbidities, ethnicity, and genetic background further contribute to variations in gut microbiota associated with obesity and related metabolic disorders, highlighting the relevance of investigating how GLP-1 receptor agonists may promote weight loss via modulation of gut microbial composition [17, 18].

In a study by Wang *et al*. (2016) using a mouse model, subcutaneous administration of the GLP-1 receptor agonist liraglutide was used to explore whether weight reduction was associated with changes in gut microbiota [19]. Analysis of fecal samples revealed substantial alterations in bacterial composition in liraglutide-treated mice compared to controls. Specifically, liraglutide decreased the relative abundance of obesity-associated genera such as Roseburia, Parabacteroides, Marvinbryantia, Candidatus, and Erysipelotrichaceae, while increasing the prevalence of lean-associated genera Blautia and Coprococcus. Notably, the effect on overall microbial structure was more pronounced in hyperglycemic mice than in normoglycemic mice. These findings suggest that liraglutide may shift gut microbiota toward a lean-associated profile, potentially contributing to its weight-lowering effects.

Similarly, Zhang *et al*. (2017) examined liraglutide's impact on gut microbiota in a rat model [20]. In untreated diabetic rats, the gut microbiome was dominated by Firmicutes and Bacteroidetes, with a markedly elevated Firmicutes/Bacteroidetes ratio compared to liraglutide-treated animals. Treatment with liraglutide significantly increased the relative abundance of genera positively associated with weight reduction, including Bacteroides, Bifidobacterium, Lachnospiraceae, Lachnoclostridium, Tenericutes, and Flavonifractor. Conversely, the

prevalence of Prevotella—a genus elevated in type 2 diabetes and obesity—was reduced in the treated group. These results indicate that liraglutide may facilitate weight loss by promoting structural changes in gut microbiota composition.

Zhao *et al*. (2018) also investigated liraglutide's effects in both simple obese and diabetic obese rats [21]. They observed that treatment reduced obesity-associated microbial phenotypes and increased lean-associated ones. At the phylum level, Firmicutes (positively associated with obesity), Bacteroidetes (negatively associated), Tenericutes, and Proteobacteria dominated the gut microbiota. Liraglutide administration decreased Firmicutes and increased Bacteroidetes, resulting in a lower Firmicutes/Bacteroidetes ratio in treated groups compared to controls. High-fat diets were found to reduce Bacteroidia (phylum Bacteroidetes) and increase Clostridia (phylum Firmicutes), but these changes were reversed following liraglutide treatment. The authors propose that liraglutide may modulate the gut microbiota through mechanisms such as delayed gastric emptying and slower gastrointestinal transit, which could alter the gut lumen environment, including pH and nutrient composition. Importantly, similar microbiota shifts were observed regardless of baseline blood glucose levels, indicating that liraglutide's effects on gut microbial composition are consistent in both simple and diabetic obesity models.

Moreira *et al*. (2018) conducted experiments in two mouse models, administering subcutaneous injections of the GLP-1 receptor agonist liraglutide [22]. Their findings demonstrated that liraglutide alters gut microbiota diversity in both high-fat diet-induced obese mice and genetically obese mice. Specifically, treatment reduced the abundance of the Proteobacteria phylum while promoting the growth of Akkermansia muciniphila, Parabacteroides, and Oscillospira—changes associated with weight loss, improved glycemic control, and reduced inflammatory responses in both models.

In a rat model, Zhang *et al*. (2020) examined the effects of subcutaneous liraglutide in high-fat diet-induced obese rats [23]. The study showed that liraglutide significantly reshaped gut microbiota composition: phyla Bacteroidetes, Tenericutes, Cyanobacteria, Elusimicrobia, and Fusobacteria were increased, whereas Firmicutes, Actinobacteria, and Proteobacteria were decreased. These alterations resulted in a microbiota profile resembling that of lean control rats, suggesting that liraglutide can partially restore a healthy gut microbial balance.

Similarly, Zhao *et al*. (2022) conducted experiments in mice receiving subcutaneous liraglutide [24]. They observed a significant decrease in the relative abundance of Firmicutes and an increase in Bacteroidetes, reducing the Firmicutes/Bacteroidetes ratio. Liraglutide treatment also promoted the growth of beneficial microbes, including Akkermansia, Lactobacillus, Parabacteroides, Oscillospira, Sutterella, and Allobaculum, while suppressing potentially harmful genera such as Shigella and Proteobacteria. The authors proposed that modulation of gut microbiota, particularly microbes linked to lipid metabolism, may contribute to improved lipid profiles and reduced fat accumulation in adipose tissue.

In a clinical study, Ying *et al*. (2023) treated 15 patients with type 2 diabetes using daily subcutaneous injections of liraglutide [25]. Fecal samples analyzed via 16S rRNA sequencing revealed that liraglutide significantly increased both the diversity and richness of the gut microbiota. The treatment elevated the relative abundance of Bacteroidetes, Proteobacteria, and Bacillus, shifting the microbiota composition toward a profile resembling that of healthy individuals.

Conclusion

Overall, recent evidence indicates that GLP-1 receptor agonists, such as liraglutide, can modulate the gut microbiome, representing a promising avenue for understanding obesity pathogenesis. Observed effects include increased microbial diversity and richness, enrichment of lean-associated microbial phenotypes, reduction of obesity-related taxa, and normalization of the Firmicutes/Bacteroidetes ratio. These effects have been documented in both animal models and patients with obesity and/or type 2 diabetes. Nevertheless, current knowledge is still limited, primarily derived from preclinical studies. Further in-depth clinical research is necessary to clarify the precise mechanisms by which liraglutide alters gut microbiota and to determine the extent to which these microbial changes contribute to its weight-reducing effects.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

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