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Metabolic actions of the gut microbiota in obesity: a systematic review

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Abstract

Introduction: Obesity is a multifactorial disease that is difficult to manage and causes several comorbidities, such as physiological and mental disorders, diabetes, stroke, and depression. Worldwide, more than 2.3 billion people are overweight or obese. The gut microbiota interacts with several organs, including the brain, and can regulate metabolism, adiposity, homeostasis, energy balance, and central signaling of appetite and food reward. **Objective:** To explore and describe the metabolic actions of the gut microbiota and probiotics in the management of patients with obesity. **Methods:** The systematic review guidelines of the PRISMA Platform were followed. The search was conducted from August to November 2025 in the Scopus, PubMed, Science Direct, Scielo, and Google Scholar databases. Study quality was based on the GRADE instrument, and risk of bias was analyzed according to the Cochrane instrument. **Results and Conclusion:** A total of 125 articles were found. A total of 32 articles were fully evaluated, and 21 were included and developed in this systematic review. Using the Cochrane risk of bias tool, the overall assessment resulted in 23 studies with a high risk of bias and 25 studies that did not meet the GRADE and AMSTAR-2 criteria. Most studies presented homogeneous results, with X^2=74.8%>50%. It was concluded that bidirectional signaling occurs within the gut-brain axis in the pathophysiology of obesity, mediated by metabolic, endocrine, neural, and immune system mechanisms. Fecal microbiota transplantation and supplementation with probiotics and prebiotics may be potential treatments for obesity. Diet is a determining factor for healthy colonization of the gut microbiota. Studies in obese humans also found a lower proportion of Bacteroidetes compared to those in normal-weight individuals. After weight loss, the

proportion of Firmicutes is reduced and becomes more similar to that of lean individuals. Inulin supplementation can significantly promote intestinal bacterial diversity and improve gut microbiota dysbiosis in obese patients. Low-carb diets and time-restricted feeding are effective in weight management and produce profound changes in the gut microbiome and metabolome, in addition to caloric restriction.

Keywords: Obesity. Gut microbiota. Dysbiosis. Metabolism.

Introduction

Obesity is a multifactorial disease that is difficult to manage and causes several comorbidities such as physiological and mental disorders, diabetes, stroke, and depression. Worldwide, there are more than 2.3 billion people who are overweight and obese **[1]**, and Brazil has about 18.0 million people, tending to reach 70.0 million individuals **[2]**. It is estimated that in 2050, there will be 15.4 million deaths worldwide due to chronic non-communicable diseases, and the Brazilian population will contribute significantly to this scenario **[1]**.

In the context of obesity, the gut microbiota interacts with various organs, including the brain. The gut microbiota and its metabolites can reach the brain directly through vagal stimulation (vagus nerve) or indirectly through immunoneuroendocrine mechanisms, and can regulate metabolism, adiposity, homeostasis, energy balance, and central signaling of appetite and food reward. Therefore, there is bidirectional signaling within the gut-brain axis (GBI) in the pathophysiology of obesity, mediated by metabolic, endocrine, neural, and immune system mechanisms **[3]**.

In this sense, the human microbiome has about 3 million genes in the gastrointestinal tract, corresponding to 150 times more than the human genome **[4-8]**. In recent years, new technologies in genomics, transcriptomics, and proteomics have allowed researchers to phylogenetically identify and/or quantify the components of the gut microbiota by analyzing nucleic acids (DNA and RNA) directly extracted from feces. Most of these techniques are based on DNA extraction and amplification of the 16S ribosomal RNA (rRNA) gene **[9]**. 16S rRNA sequencing has become the most useful technique for highlighting the diversity and abundance of the microbiome. 16S rRNA gene sequences can be explored with polymerase chain reaction (PCR) and metagenomic sequencing to characterize strains **[9]**.

The gut microbiota is essential for the host to ensure digestive and immunological homeostasis. However, in the presence of dysbiosis, the malfunction of the epithelial barrier leads to intestinal and systemic disorders, mainly obesity **[3,10]**. Therefore, the present study explored and described the metabolic actions of the gut microbiota and probiotics in the management of patients with obesity.

METHODS

Study Design

This study followed an international systematic review model, following the PRISMA (preferred reporting items for systematic reviews and meta-analysis) guidelines. Available at: http://www.prisma-statement.org/?AspxAutoDetectCookieSupport=1. Accessed on: November, 07, 2025. The methodological quality standards of AMSTAR2 (Assessing the methodological quality of systematic reviews) were also followed. Available at: https://amstar.ca/. Accessed on: November, 07, 2025.

Data Sources and Research Strategy

The literature search process was conducted from August to November 2025 and developed based on Scopus, PubMed, Lilacs, Ebsco, Scielo, and Google Scholar, addressing scientific articles from various periods to the present. The following descriptors (DeCS/MeSH Terms) were used: "Obesity. Gut microbiota. Dysbiosis. Metabolism", and using the Boolean operator "and" between MeSH terms and "or" between historical findings.

Study Quality and Risk of Bias

Quality was classified as high, moderate, low, or very low regarding the risk of bias, clarity of comparisons, precision, and consistency of analyses. The most evident highlight was systematic review articles or meta-analyses of randomized clinical trials, followed by randomized clinical trials. Low-quality evidence was attributed to case reports, editorials, and brief communications, according to the GRADE instrument. The risk of bias was analyzed according to the Cochrane instrument through the analysis of the Funnel Plot (Sample size versus Effect size), using Cohen's d test.

Results and Discussion

Summary of Findings

A total of 125 articles were found and submitted to eligibility analysis, with 21 final studies selected to compose the results of this systematic review. The listed studies presented medium to high quality (Figure 1), considering the level of scientific evidence of studies such as meta-analyses, consensus, randomized

clinical trials, and prospective and observational studies. Biases did not compromise the scientific basis of the studies. According to the GRADE instrument, most studies showed homogeneity in their results, with X^2=74.8%>50%. Considering the Cochrane tool for risk of bias, the overall assessment resulted in 23 studies with a high risk of bias and 25 studies that did not meet the GRADE and AMSTAR-2 criteria.

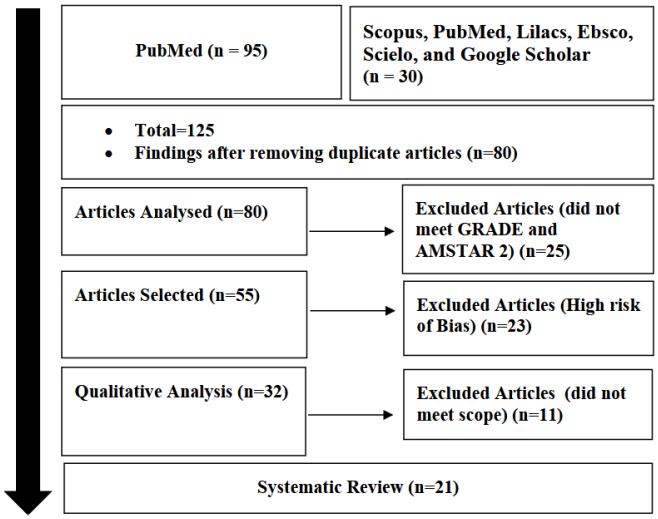

Figure 1. Flowchart showing the article selection process. Source: Own authorship.

Figure 2 presents the results of the risk of bias of the studies using the Funnel Plot, showing the calculation of the Effect Size (Magnitude of the difference) using Cohen's Test (d). The precision (sample size) was determined indirectly by the inverse of the standard error (1/Standard Error). This graph had a symmetrical behavior, not suggesting a significant risk of bias, both between studies with small sample sizes (lower precision) that are shown at the bottom of the graph and in studies with large sample sizes that are presented at the top.

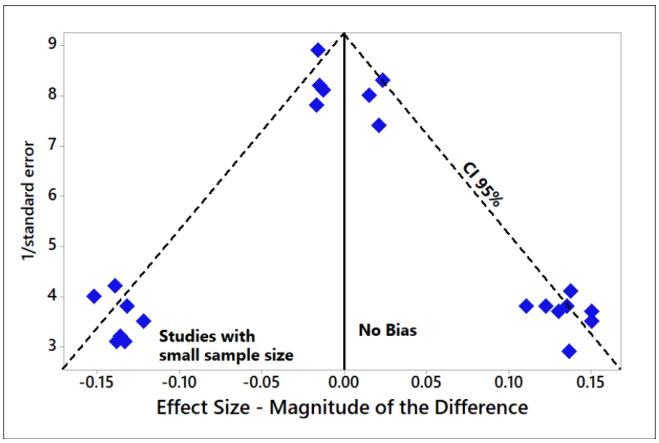

Figure 2. The symmetrical funnel plot does not suggest a risk of bias between the small sample size studies that are shown at the bottom of the graph. High confidence and high recommendation studies are shown above the graph (n=21 studies). Source: Own authorship.

Gut Microbiota and Obesity

The gut-brain axis presents a bidirectional connection between the gut microbiota and the brain through three pathways. The neural pathway consists mainly of the enteric nervous system and the vagus nerve. The endocrine pathway, on the other hand, affects the brain's neuroendocrine system, particularly the hypothalamic-pituitary-adrenal axis and the immune pathway. Several alterations in the gut microbiome can lead to obesity, modulating metabolic pathways and host eating behaviors through the gut-brain axis **[11]**.

The authors Jiang et al. (2025) **[12]** showed an association between microbial aromatic amino acid metabolites in serum and body fat accumulation in a large Chinese longitudinal cohort. 4-hydroxyphenylacetic acid (4HPAA) and its analogues were found to effectively protect against obesity induced by a high-fat diet. These metabolites act on the intestinal mucosa to regulate the immune response and control lipid uptake, which protects against obesity.

In this sense, obesity participates in the genesis of insulin resistance as well as morbidities, such as glucose intolerance, dyslipidemia, and hypertension **[13]**. The cascade of intracellular mechanisms and systemic events involved in the aggregation of these risk factors continues to be the target of investigations. In this context, there is added interest in the role of the gut microbiota as an intermediary factor between environmental and behavioral components and the occurrence of obesity and metabolic disorders. Knowledge about the participation of intestinal bacteria in pathophysiological mechanisms has largely evolved with results obtained in animal models **[14]**.

The authors Visuthranukul et al. (2024) **[15]** analyzed through a randomized clinical study whether inulin supplementation improved the gut microbiota and microbial functional pathways in children with obesity. Children with obesity whose BMI was above the median + 2 standard deviations were recruited. Participants aged 7 to 15 years were assigned to counseling groups with supplementation of inulin extracted from Thai Jerusalem artichoke (intervention), maltodextrin (placebo), and dietary fiber. All participants received similar monthly conventional counseling and follow-up for 6 months. Fecal samples were collected for gut microbiome analysis using 16S rRNA sequencing. A total of 143 children were evaluated. A significant increase in alpha diversity was observed in the inulin group. Inulin supplementation substantially increased Bifidobacterium, Blautia, Megasphaera, and several butyrate-producing bacteria, including Agathobacter, Eubacterium coprostanoligenes,

and Subdoligranulum, compared to the other groups. The inulin group showed a significant difference in proteasome functional pathways and riboflavin metabolism.

The authors Li et al. (2024) **[16]** conducted a 12-week randomized controlled clinical trial with 28 weeks of follow-up among 96 overweight or obese participants. Isocaloric restricted feeding produces significant weight loss, ranging from 2.57 to 4.11 kg in different groups. In addition to caloric restriction, low-carbohydrate diets and timerestricted feeding enabled a further reduction in body mass index. Low-carbohydrate diets result in additional fat mass loss, while time-restricted feeding produces more lean mass loss. Furthermore, low-carbohydrate diets lead to a decrease in fecal branched-chain amino acids, and time-restricted feeding tends to produce an increased abundance of probiotic species involved in short-chain fatty acid synthesis. Overall, low-carbohydrate diets and time-restricted feeding are effective in weight management and produce profound changes in the gut microbiome and metabolome, in addition to caloric restriction.

The authors Zhang et al. (2025) **[17]** developed a randomized, double-blind, placebo-controlled clinical trial lasting 12 weeks, involving 58 participants with type 2 diabetes mellitus (T2D) who were overweight or obese, who received *Akkermansia muciniphila* (AKK-WST01) or placebo, along with routine lifestyle guidance. Both groups showed reductions in body weight and glycated hemoglobin (HbA1c), with no significant differences between the groups. In participants with low baseline levels of *Akkermansia muciniphila*, supplementation with AKK-WST01 demonstrated high colonization efficiency and significant reductions in body weight, fat mass, and HbA1c, which were not found in the placebo group. The metabolic benefits of *Akkermansia muciniphila* supplementation may depend on its baseline intestinal levels, supporting the potential of gut microbiota-guided probiotic supplementation.

In addition, the authors Graciliano et al. (2025) **[18]** investigated the association of gut microbiota composition and alpha diversity at the gender level with postprandial changes in satiety hormones and appetite measures in obese individuals who received either a meal rich in ultraprocessed foods or a meal without. Individuals were randomized into two groups, one meal without ultraprocessed foods and one meal with ultraprocessed foods. Blood samples for hormonal (ghrelin, leptin, and GIP) and appetite (hunger, satiety, fullness, and eating capacity) measures were collected using visual analog scales after a 12-hour fast and 90 minutes after the meal. Twenty individuals were included in the ultraprocessed food group and 19 in the control group, with no significant differences in gut microbiota composition or alpha diversity indices between the groups. Greater gut microbiota alpha diversity at the gender level was associated with increased postprandial satiety. The alpha diversity of the gut microbiota at the gender level appears to be associated with the subjective feeling of satiety after a meal, and this association may vary depending on the content of ultra-processed foods in the meal.

Another randomized controlled clinical trial was developed by the authors Bai et al. (2024) **[19]** and recruited 75 overweight or obese young adults, randomly assigned to a *Bifidobacterium breve* BBr60 (BBr60) group or a placebo group. Both groups received dietary guidance and took BBr60 (1 × 1010 CFU/day) or a placebo for 12 weeks. After 12 weeks, BBr60 significantly reduced weight and BMI compared to pre-treatment levels and outperformed placebo. The BBr60 group also showed improvement in blood biochemistry, with fasting glucose (FG) levels notably lower than the placebo group ($p < 0.05$). Furthermore, BBr60 influenced vital serum and fecal metabolites related to three amino acid metabolic pathways and regulated Dialister, Klebsiella, and Bacteroides bacteria, which correlated strongly with serum metabolites. These findings indicate that BBr60 can safely and effectively regulate BMI, body weight, serum glucose, lipids, and markers of liver function.

Adipose tissue hypertrophy leads to metabolic and hemodynamic disorders due to the production of various adipokines that play a role in the genesis of insulin resistance and atherosclerosis. Both processes are mediated by inflammatory cytokines, such as TNF-α, IL-6, IL-2, and IFN-γ, secreted by both adipocytes and monocytes that infiltrate this tissue. It is recognized that obese individuals have a chronic state of subclinical inflammation that favors insulin resistance, a central event in the generation of cardiometabolic risk **[20]**.

Studies in obese humans have also found a lower proportion of Bacteroidetes compared to eutrophic individuals. Moreover, when they lose weight, the proportion of Firmicutes decreases and becomes more similar to that of lean individuals **[14,21]**. Similarly, Turnbaugh et al. **[22]**, evaluating the gut microbiota of twins, found lower bacterial diversity in obese individuals, who presented 75.0% Actinobacteria, 25.0% Firmicutes, and 0.0% Bacteroidetes, while lean individuals presented 0.0% Actinobacteria, 58.0% Firmicutes, and 42.0% Bacteroidetes.

The products of a protein-rich diet, such as L-carnitine and phosphatidylcholine, can be metabolized into choline, which is converted into trimethylamine

(TMA) by the gut microbiota. TMA can be oxidized in the liver to form TMA-N-oxide (TMAO), which can promote the formation of atherosclerotic plaque **[6,7]**. On the other hand, diet-induced gut microbiota dysbiosis can result in bacterial translocation into the systemic bloodstream, where a blood microbiota (almost 90.0% Gram-negative bacteria) can establish itself. Subsequently, atherosclerotic plaques can develop and promote atherosclerosis and cardiovascular disease. Interestingly, there is a blood plaque-like microbiota, and it is dominated by Gram-negative bacteria (*Proteobacteria phylum*) as well **[8]**.

The BMI level below weight also revealed profound variations in the gut microbiota. Borgo et al. **[23]** performed a comprehensive data analysis comparing the gut microbiota and anthropometric characteristics of 15 anorexia nervosa (AN) women and healthy controls. The results showed that the gut microbiota showed a significant increase in Enterobacteriaceae and *Methanobrevibacter smithii* compared to controls.

Few studies have examined the interaction of the microbiome and obesity in relation to human geography. Thus, a study analyzed obese adult volunteers from France, Saudi Arabia, French Polynesia, and a traditional population in the village of Trois-Sauts, French Guiana. It was found that the French and Saudis had significantly less biodiversity richness in their gut microbiota compared to the others ($p<0.05$). The main coordinated analysis of the overall composition of genera communities revealed that the microbiomes of the participants from French Guiana clustered independently of the other obese individuals. Also, Polynesians had significantly lower relative abundance of Lactobacillus sp. than the French ($p<0.01$) and Saudis ($p<0.05$). Obese individuals with different origins exhibit modifications in their gut microbiota **[24]**.

Maintaining a healthy metabolism depends on a symbiotic consortium between bacteria, archaea, viruses, fungi, and host eukaryotic cells throughout the human gastrointestinal tract. Microbial communities provide the enzymatic machinery and metabolic pathways that contribute to food digestion, xenobiotic metabolism, and the production of a variety of bioactive molecules. These include vitamins, amino acids, short-chain fatty acids, and metabolites, which are essential for the interconnected pathways of glycolysis, the tricarboxylic acid/Krebs cycle, oxidative phosphorylation, and amino acid and fatty acid metabolism **[25]**.

Recent studies have elucidated how the nutrients that fuel metabolic processes impact how immune cells, particularly macrophages, respond to different stimuli under physiological and pathological conditions, becoming activated and acquiring a specialized function. The two main inflammatory phenotypes of macrophages are controlled through differential consumption of glucose, glutamine, and oxygen. The M1 phenotype is triggered by the polarization signal of bacterial lipopolysaccharides (LPS) and pro-inflammatory Th1 cytokines, such as interferon-γ, TNF-α, and IL-1β, or both, while the M2 phenotype is triggered by Th2 cytokines, such as interleukin-4 and interleukin-13, as well as anti-inflammatory cytokines, IL-10 and TGF. Utilization of glucose and production of chemical mediators, including ATP, reactive oxygen species (ROS), nitric oxide (NO), and NADPH-supporting effector activities of M1 macrophages **[3,25,26]**.

Gut microbiota dysbiosis is closely related to the occurrence of many important chronic inflammation-related diseases. To date, traditionally prescribed probiotics and prebiotics have not shown a significant impact on improving these diseases in general. Thus, the development of next-generation prebiotics and probiotics designed to target specific diseases is much needed. So, under the situation of gut microbiota dysbiosis, chronic inflammation develops. These have resulted in the development of many important diseases, such as obesity, type 2 diabetes mellitus, liver inflammation, and other diseases, such as colorectal cancer, obesity-induced chronic kidney disease, impaired pulmonary immunity, and some brain/neuro disorders. Although the efficacy of probiotics and/or prebiotics is promising, further studies are needed to establish recommendations for most clinical scenarios **[25]**.

Another study observed associations between phosphatidylglycerols (PG) and gut microbiota dysbiosis. Compared with other phospholipids, serum PG levels were highest in patients with low microbiota gene richness, which were normalized after a dietary intervention that restored gut microbial diversity. Serum PG levels were positively correlated with metagenomic functional capacities for LPS synthesis. Experiments in mice and cultured human-derived macrophages demonstrated that LPS induces PG release. Acute PG treatment in mice altered adipose tissue gene expression toward remodeling and inhibition of *ex vivo* lipolysis in adipose tissue, suggesting that PG favors lipid storage **[27]**.

Finally, many efforts have been made to understand the link between gut microbiota composition and obesity, as well as the role of dietary ingredients, such as probiotics and prebiotics, in modulating the gut microbiota. Studies involving the gut microbiota composition of obese individuals are still

controversial, hindering the treatment of obesity **[28]**.

Conclusion

It was concluded that bidirectional signaling occurs within the gut-brain axis in the pathophysiology of obesity, mediated by metabolic, endocrine, neural, and immune system mechanisms. Fecal microbiota transplantation and supplementation with probiotics and prebiotics may be a potential treatment for obesity. Diet is a determining factor for the healthy colonization of the gut microbiota. Studies in obese humans have also found a lower proportion of Bacteroidetes compared to eutrophic individuals. After weight loss, the proportion of Firmicutes is reduced and becomes more similar to that of lean individuals. Inulin supplementation can significantly promote intestinal bacterial diversity and improve gut microbiota dysbiosis in obese patients. Low-carbohydrate diets and time-restricted feeding are effective in weight control and produce profound changes in the gut microbiome and metabolome, in addition to caloric restriction.

CRediT

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No additional data are available.

Conflict of Interest

The authors declare no conflict of interest.

Similarity Check

It was applied by Ithenticate®.

Application of Artificial Intelligence (AI)

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Peer Review Process

It was performed.

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