PĄCZEK, Adrian Jan, KOWALCZYK, Aleksandra, STANISZEWSKA, Wiktoria, DYCZEK, Paweł and HOFMAN, Julia. Gut Microbiota and Its Role in Obesity and Metabolic Syndrome: A Narrative Review. Journal of Education, Health and Sport. 2025;85:66499. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2025.85.66499
https://apcz.umk.pl/JEHS/article/view/66499

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 08.11.2025. Revised: 16.11.2025. Accepted: 16.11.2025. Published: 24.11.2025.

Gut Microbiota and Its Role in Obesity and Metabolic Syndrome: A Narrative Review

Adrian Pączek (corresponding author)
ORCID: https://orcid.org/0009-0009-5151-7556
E-mail: adrjanalan@gmail.com
Zagłębiowski Clinical Hospital in Czeladź, Poland

Aleksandra Kowalczyk
ORCID: https://orcid.org/0009-0002-9523-2367
E-mail: olakowalczyk2001@wp.pl
Medical University of Silesia in Katowice, Poland

Wiktoria Staniszewska
ORCID: https://orcid.org/0009-0002-8964-404X
E-mail: wiktoria.staniszewska@gmail.com
Wojewódzki Hospital in Bielsko-Biała, Poland

Paweł Dyczek
ORCID: https://orcid.org/0009-0008-6607-6231
E-mail: dyczek@onet.eu
Wojewódzki Hospital in Bielsko-Biała, Poland

Julia Hofman
ORCID: https://orcid.org/0009-0008-2609-8292
E-mail: juliahofman2001@gmail.com
Medical University of Silesia in Katowice, Poland

Abstract

Background. Obesity and metabolic syndrome represent major global health challenges, with steadily increasing prevalence worldwide. Beyond caloric intake and physical activity, the gut microbiota has emerged as an important modulator of host metabolism and metabolic homeostasis.

Objective. To summarize current evidence regarding the role of gut microbiota in obesity and metabolic syndrome and to discuss potential therapeutic interventions targeting the microbiota.

Methods. This narrative review was based on a literature search in PubMed using the following keywords: *gut microbiota*, *obesity*, *metabolic syndrome*, *probiotics*, *prebiotics*, *synbiotics* and *fecal microbiota transplantation*. Preference was given to systematic reviews, meta-analyses, and randomized controlled trials.

Results. Individuals with obesity typically exhibit reduced gut microbial diversity and altered relative abundances of beneficial taxa such as *Akkermansia muciniphila* and *Bifidobacterium longum*. Evidence from clinical and translational studies indicates that obesity is associated with decreased microbial diversity and functional alterations affecting short-chain fatty acid (SCFA) production, bile acid metabolism, and inflammatory signaling. Experimental and clinical findings suggest that probiotics and fecal microbiota transplantation (FMT) may improve selected metabolic parameters, although results remain heterogeneous. Moreover, baseline gut microbiota composition may influence the effectiveness of lifestyle and dietary interventions.

Conclusions. The gut microbiota plays a significant role in the pathogenesis of obesity and metabolic syndrome. Interventions such as probiotics, FMT, and high-fiber diets show therapeutic potential but require larger, standardized, long-term clinical trials to confirm their efficacy and safety.

Keywords: gut microbiota, obesity, metabolic syndrome, probiotics, prebiotics, synbiotics, fecal microbiota transplantation, short-chain fatty acids.

1. Introduction

Obesity and metabolic syndrome (MetS) are among the leading causes of morbidity and mortality worldwide [1]. According to the World Health Organization (WHO), more than one billion individuals currently live with obesity, and projections indicate a further increase in the coming decades, driven primarily by urbanization, sedentary lifestyle, and a shift toward Western dietary habits [1]. Both conditions are major contributors to cardiovascular disease, type 2 diabetes mellitus, non-alcoholic fatty liver disease (NAFLD), and certain cancers, representing a substantial global public health and socioeconomic burden [1]. Despite progress in prevention and treatment, long-term effectiveness of current strategies remains limited, as many patients experience weight regain or incomplete metabolic improvement despite adherence to standard interventions. Traditional risk factors—excess caloric intake, insufficient physical activity, and genetic predisposition—do not fully explain interindividual variability in metabolic response, suggesting the existence of additional modulatory mechanisms [2,3]. In this context, the gut microbiota, comprising trillions of microorganisms that inhabit the gastrointestinal tract, has emerged as a critical determinant of energy homeostasis, glucose regulation, and systemic inflammation [3–5]. Experimental and clinical studies have revealed that individuals with obesity exhibit distinct microbial signatures characterized by reduced α-diversity and decreased abundance of beneficial taxa such as Akkermansia muciniphila (a mucin-degrading bacterium associated with improved metabolic profiles rather than direct butyrate production) and Bifidobacterium longum, alongside enrichment of pro-inflammatory species associated with metabolic endotoxemia [3,5,6]. Mechanistically, the gut microbiota influences host metabolism through multiple interconnected pathways, including modulation of short-chain fatty acid (SCFA) production, bile acid signaling, appetite regulation via the gut–brain axis, and integrity of the intestinal barrier [3–5,8]. Altered SCFA profiles and availability, including changes in butyrate and propionate, impair insulin sensitivity, alter lipid metabolism,

and promote low-grade systemic inflammation [3,4]. Furthermore, dysbiosis-induced disruption of the gut barrier leads to translocation of bacterial components, particularly lipopolysaccharides (LPS), into the bloodstream, initiating chronic metabolic inflammation—a process termed "metabolic endotoxemia" [6,7]. Such inflammation is now recognized as a key factor linking intestinal dysbiosis with insulin resistance and adipose tissue dysfunction. Accumulating evidence also suggests a bidirectional relationship between the gut microbiota and lifestyle factors. Diet composition, caloric restriction, physical activity, and pharmacotherapy can modulate microbial diversity and function, influencing metabolic outcomes [2,8,9]. Conversely, baseline microbiota composition may determine individual responsiveness to dietary or probiotic interventions [10,11]. These findings highlight the therapeutic potential of targeting the microbiota as an adjunct to traditional lifestyle and pharmacological strategies in the management of obesity and MetS. In light of this, the present review aims to synthesize current evidence on the relationship between the gut microbiota and metabolic dysregulation in obesity and MetS. The analysis focuses on mechanistic insights, recent clinical and translational findings, and the therapeutic implications of microbiota modulation—through diet, probiotics, prebiotics, and fecal microbiota transplantation—as emerging tools in metabolic medicine [10,12–14].

2. Methods

A narrative literature review was conducted to summarize current evidence on the role of the gut microbiota in obesity and metabolic syndrome (MetS). The PubMed database served as the primary source of scientific literature. The search was performed for English-language articles using combinations of the following keywords and MeSH terms: *gut microbiota*, *obesity*, *metabolic syndrome*, *probiotics*, *prebiotics*, *synbiotics*, *short-chain fatty acids (SCFA)*, and *fecal microbiota transplantation (FMT)*. Boolean operators "AND" and "OR" were used to broaden or refine the search strategy depending on context. Priority was given to systematic reviews, meta-analyses, randomized controlled trials (RCTs), and high-quality observational or translational studies involving human participants. Seminal earlier studies were also included to provide historical and mechanistic context, particularly in areas where foundational discoveries established key physiological or microbiological mechanisms (e.g., metabolic endotoxemia, SCFA signaling, host–microbiota energy exchange). Articles were selected based on their relevance, methodological quality, and clinical applicability. Studies limited solely to animal or in vitro models were excluded from the clinical synthesis; however, pivotal animal experiments were cited to provide mechanistic background where relevant. Case reports,

editorials, or expert opinions without empirical data were also excluded, as well as papers not peer-reviewed or published in predatory journals. Each article was assessed for its contribution to one or more thematic domains: (1) alterations in microbiota composition and diversity in obesity and MetS, (2) mechanistic pathways linking dysbiosis with metabolic dysfunction (e.g., inflammation, insulin resistance, intestinal permeability), and (3) potential microbiota-targeted therapeutic interventions, including probiotics, prebiotics, synbiotics, dietary modulation, and FMT. Data synthesis focused on identifying consistent trends, reproducible findings, and existing research gaps, rather than quantitative meta-analysis. This methodological framework ensured a comprehensive and integrative overview of both clinical and mechanistic evidence, aligning with contemporary standards for narrative reviews in metabolic and microbiome research.

3. Results

3.1. Altered microbiota diversity in obesity

Recent human studies and meta-analyses have consistently demonstrated that obesity is associated with a reduction in gut microbial diversity—both within-sample (α-diversity) and between-sample (β-diversity)—compared to lean individuals [2,3,5]. Reduced α-diversity is widely interpreted as a marker of microbiome instability and impaired ecological resilience. In particular, large-scale metagenomic analyses have revealed a lower ratio of Bacteroidetes to Firmicutes, although this finding is not universal across populations due to dietary and genetic variability [3,5]. Obese individuals also display a depletion of beneficial taxa such as Akkermansia muciniphila, and of key SCFA-producing genera including Faecalibacterium prausnitzii and Roseburia, which play crucial roles in maintaining mucosal integrity and energy homeostasis [3,5,11]. Conversely, taxa linked to energy-harvest efficiency—such as certain Ruminococcus and Clostridium species—are often enriched [5,6,18]. However, some members of Clostridium cluster XIVa also produce butyrate and exert anti-inflammatory effects, highlighting the complexity of taxonomic classifications. These compositional changes are functionally reflected in reduced microbial gene richness, altered carbohydrate metabolism, and enhanced production of LPS-containing Gram-negative bacteria, which together predispose to weight gain and insulin resistance.

3.2. Short-chain fatty acids (SCFA)

Short-chain fatty acids—acetate, propionate, and butyrate—are among the most biologically active metabolites generated by gut microbiota through fermentation of dietary fibers. They

exert pleiotropic effects on host metabolism: regulating appetite and satiety through G-protein-coupled receptors (GPR41/43), improving insulin sensitivity, and modulating lipid oxidation in liver and adipose tissue [3,4,17]. In obesity and MetS, alterations in SCFA-producing bacteria such as Faecalibacterium, Eubacterium, and Akkermansia have been reported. While several studies describe lower fecal and circulating SCFA levels, others note increased fecal concentrations, likely reflecting differences in microbial fermentation and host absorption [4,15]. Functionally, these changes disrupt intestinal gluconeogenesis, compromise gut barrier integrity, and promote chronic low-grade inflammation. Butyrate, in particular, supports colonocyte energy metabolism and suppresses pro-inflammatory cytokine expression via inhibition of NF-κB activation [4,17]. Experimental supplementation with SCFA or prebiotic fibers that increase endogenous SCFA production has been shown to improve glucose tolerance, reduce hepatic steatosis, and lower systemic inflammation, underscoring their therapeutic relevance [3,4].

3.3. Increased gut permeability and endotoxemia

Obesity-related dysbiosis induces disruption of intestinal barrier integrity, characterized by reduced expression of tight junction proteins such as occludin and claudin, which increases permeability of the gut epithelium [7]. This “leaky gut” facilitates translocation of bacterial endotoxins—particularly LPS—into the bloodstream, a phenomenon termed metabolic endotoxemia [7]. Circulating LPS binds to Toll-like receptor 4 (TLR4) on immune and adipose cells, triggering activation of pro-inflammatory signaling pathways and secretion of cytokines such as tumor necrosis factor α (TNF-α) and interleukin-6 (IL-6) [4,15]. The resulting low-grade systemic inflammation interferes with insulin signaling and promotes lipid accumulation within hepatocytes and adipocytes [4]. Clinical data indicate that plasma LPS levels are significantly higher in individuals with obesity and type 2 diabetes, correlating with markers of insulin resistance and dyslipidemia [7,15]. Restoration of gut barrier function—through dietary fiber, probiotics, or specific microbial metabolites—has been shown to attenuate LPS translocation and improve metabolic outcomes, reinforcing the gut barrier as a therapeutic target.

3.4. Interaction with diet and host metabolism

Diet remains a primary driver of microbiota composition. High-fiber, plant-based diets enrich microbial taxa that produce SCFAs, particularly Bifidobacterium and Akkermansia muciniphila, leading to improved insulin sensitivity and lipid profiles [8,9]. Conversely, Western-style diets,

typically high in saturated fats and refined sugars, may foster dysbiosis by promoting growth of bile-tolerant species (e.g., Bilophila wadsworthia, Alistipes putredinis) and reducing butyrate-producing bacteria, although dietary context and fiber intake can modify these effects [5,6,8]. This diet-induced microbial shift increases intestinal permeability, oxidative stress, and LPS production—mechanisms central to metabolic inflammation and adipose dysfunction [4,6]. Moreover, recent randomized controlled trials have shown that individual baseline microbiota composition influences response to caloric restriction and exercise interventions, with certain microbial profiles predicting greater improvements in body weight and glycemic control [2,8,9]. Physical activity itself appears to enhance microbial diversity, increasing the abundance of butyrate-producing taxa and supporting anti-inflammatory signaling pathways [9]. Altogether, these findings emphasize a dynamic bidirectional relationship between diet, microbiota, and metabolic health.

3.5. Microbiota-targeted interventions

Emerging therapeutic strategies focus on modulating the gut microbiota to restore metabolic homeostasis. Probiotics. Randomized controlled trials and meta-analyses demonstrate that certain probiotic strains (notably Lactobacillus gasseri, L. rhamnosus, and Bifidobacterium breve) can modestly reduce body weight, waist circumference, and homeostatic model assessment of insulin resistance (HOMA-IR), although results remain heterogeneous due to strain-specific effects and differences in study design [13,14]. Fecal microbiota transplantation (FMT). Studies transferring fecal microbiota from lean donors to obese or insulin-resistant recipients report transient improvements in insulin sensitivity and lipid profile [10,12]. However, long-term benefits are inconsistent and may depend on compatibility between donor and recipient microbiota composition. The long-term engraftment of donor microbial taxa appears to depend strongly on post-transplant diet and host immune compatibility, emphasizing the importance of controlled dietary protocols following FMT [10,11]. Prebiotics and synbiotics. Dietary fibers such as inulin and resistant starches selectively stimulate beneficial bacteria, enhancing SCFA production and reducing intestinal inflammation [8,15]. Clinical studies show that prebiotic supplementation improves glycemic parameters and body composition when combined with caloric restriction [8]. Although lifestyle and dietary interventions remain the cornerstone of management, modulation of the gut microbiota represents a promising adjunctive strategy, offering potential for individualized, microbiome-informed therapy in obesity and metabolic syndrome [2,13,15].

Table 1. Mechanistic links between gut microbiota and metabolic dysfunction in obesity and metabolic syndrome

Mechanism	Description	Representative microbiota alterations	Consequences for host metabolism	Key References
Reduced microbial diversity (α-diversity)	Lower richness and evenness of gut species in obesity compared to lean controls.	↓ *Akkermansia muciniphila*, *Faecalibacterium prausnitzii*; ↑ *Ruminococcus*, *Clostridium* spp.	Impaired resilience, energy imbalance, inflammation.	[3], [5], [6]
Altered SCFA production	Decrease or dysregulation of short-chain fatty acids (acetate, propionate, butyrate).	↓ *Faecalibacterium*, *Roseburia*, *Eubacterium*; ↑ fermenters with reduced SCFA efficiency.	Reduced insulin sensitivity, increased inflammation, impaired lipid oxidation.	[3], [6], [17]
Increased gut permeability and metabolic endotoxemia	Disruption of tight junction proteins leads to LPS translocation into circulation.	↑ Gram-negative LPS-producing bacteria.	Low-grade systemic inflammation, insulin resistance, hepatic steatosis.	[6], [7], [10], [16]
Bile acid signaling dysregulation	Gut bacteria alter bile acid pools and FXR/TGR5 signaling.	Dysbiosis of bile-metabolizing species (*Clostridium*, *Bacteroides*).	Altered glucose and lipid metabolism, impaired thermogenesis.	[3], [6], [17]

Diet-induced dysbiosis	Western diets reduce beneficial taxa and SCFA producers.	↓ *Bifidobacterium*, *Akkermansia*; ↑ *Bilophila wadsworthia*, *Alistipes putredinis*.	Increased permeability, oxidative stress, LPS release.	[9], [10], [14], [18], [19]
Microbiota–host interaction with lifestyle and therapy	Baseline microbiota influences response to diet, exercise, or FMT.	Variable by individual microbial profile.	Personalized response in weight loss and glycemic control.	[2], [11], [12], [14], [18], [21]

4. Discussion

The current body of evidence demonstrates a robust and reproducible association between gut microbiota composition and the development of obesity and metabolic syndrome (MetS) [3,5,6]. Across multiple clinical cohorts and meta-analyses, obesity is linked to decreased microbial diversity, reduced abundance of beneficial commensals, and enrichment of pro-inflammatory species capable of altering energy harvest and immune signaling [3,5]. Mechanistic studies have provided strong support for several interrelated pathways: altered SCFA profiles and host utilization, increased intestinal permeability, chronic metabolic endotoxemia, and sustained low-grade inflammation [5–7,15]. These factors interact to create a self-perpetuating cycle of metabolic dysfunction—where inflammation further disrupts microbial homeostasis, and dysbiosis, in turn, aggravates insulin resistance and lipid accumulation. Nevertheless, causality remains difficult to establish. Differences in sequencing technologies, analytical pipelines, dietary backgrounds, and host genetics contribute to heterogeneity across studies [2,3,5]. Moreover, most clinical data are derived from cross-sectional or short-term interventional designs, limiting the ability to infer direct cause-and-effect relationships. Even though animal models have shown that microbiota transfer from obese donors can induce weight gain in germ-free mice, extrapolating such results to humans is complex due to interspecies variation and the multifactorial nature of human obesity [6,18].

Interventional research exploring microbiota-targeted therapies has produced encouraging but heterogeneous outcomes. Probiotic supplementation demonstrates modest but statistically significant improvements in insulin sensitivity, inflammatory markers, and body

composition, particularly with strains such as Lactobacillus gasseri, L. rhamnosus, and Bifidobacterium breve [13,14]. These effects vary markedly between strains and host microbiota profiles, underscoring the need for precision in strain selection and dosing strategies. However, inconsistencies across trials reflect strain-specific effects, variable dosages, and differences in baseline microbiota composition. Fecal microbiota transplantation (FMT) from lean donors has shown temporary improvements in glucose homeostasis and lipid metabolism, but the benefits often diminish after several months, and interindividual responses vary widely [10–12]. The donor–recipient microbial compatibility, diet during follow-up, and host immune status appear to influence the durability of these effects. Prebiotic and synbiotic strategies, emphasizing fermentable fiber intake, have demonstrated more consistent results by enhancing SCFA production and supporting beneficial taxa such as Akkermansia and Bifidobacterium [8,15]. Despite these advances, the long-term safety, sustainability, and clinical utility of microbiota modulation remain open questions requiring standardized protocols and multicenter trials.

Additionally, emerging data indicate that obesity-related dysbiosis can also affect neurobehavioral and affective pathways through microbiota–gut–brain signaling, further linking metabolic and mental health disturbances [16,21]. This psychometabolic overlap, described in recent microbiota–gut–brain studies, may partly account for interindividual variability in weight trajectories and treatment responses.

An emerging concept is the individualization of microbiome-targeted therapy. Integrative “omics” approaches—including metagenomics, metabolomics, and transcriptomics—may enable stratification of patients based on microbial signatures predictive of treatment response [19,20]. Personalized interventions tailored to an individual's microbial and metabolic profile could improve efficacy and minimize variability observed in current studies. In addition, combining microbiota modulation with lifestyle interventions—caloric restriction, increased physical activity, and pharmacologic agents such as GLP-1 receptor agonists—may yield synergistic benefits by simultaneously addressing both microbial and host metabolic pathways [2,9,13]. Longitudinal multi-omic studies integrating metagenomics, metabolomics, and dietary tracking will be essential to disentangle causality and identify biomarkers predicting therapeutic response. Personalized nutrition guided by microbial and metabolic profiling represents a promising frontier in metabolic medicine [19,20].

From a primary care perspective, traditional lifestyle modification remains the cornerstone of obesity and MetS management. Nutritional counseling focusing on increased dietary fiber, reduction of ultra-processed foods, and encouragement of regular exercise should

remain first-line strategies [8,9,13]. Microbiota-targeted therapies—whether through probiotic supplementation, dietary modulation, or controlled FMT—should currently be regarded as adjunctive approaches, considered on a case-by-case basis until stronger evidence supports routine clinical use [14]. For family physicians and general practitioners, awareness of the microbiota's role in metabolic health may aid in guiding patients toward sustainable dietary habits that naturally promote microbial diversity, rather than relying solely on commercial supplements.

5. Conclusions

Current evidence clearly indicates that gut microbiota dysregulation plays a pivotal role in the pathogenesis of obesity and metabolic syndrome (MetS) [3,5,19]. The most consistent microbiological features include a reduction in overall microbial diversity, loss of key SCFA-producing taxa, and an increased abundance of pro-inflammatory bacteria contributing to metabolic endotoxemia and insulin resistance [3,5,6]. These findings underline the integral role of the intestinal ecosystem in modulating host energy metabolism, immune homeostasis, and systemic inflammatory tone. Despite significant scientific progress, translating these mechanistic insights into effective clinical interventions remains challenging. Probiotic and prebiotic supplementation, as well as fecal microbiota transplantation (FMT), have demonstrated encouraging short-term effects on metabolic parameters, yet results across randomized controlled trials remain heterogeneous and often modest in magnitude [10,12–14]. The variability of treatment outcomes emphasizes the need for standardized study designs, longer follow-up periods, and precise strain-level characterization to determine which microbial manipulations yield reproducible metabolic benefits. The future of microbiota-based therapy likely lies in personalized medicine, where treatment decisions are guided by an individual's microbial, metabolic, and dietary profiles [19,20]. Integration of metagenomic and metabolomic data could help identify distinct patient subgroups most likely to benefit from targeted microbiome modulation, allowing more efficient and tailored interventions. For primary care and preventive medicine, the most practical and evidence-based strategy remains dietary modification aimed at supporting microbial diversity and SCFA production. Such dietary interventions should emphasize gradual increases in fermentable fiber and the inclusion of naturally fermented foods, rather than reliance on commercial supplements alone. Emphasis should be placed on high-fiber, minimally processed diets rich in whole grains, legumes, fruits, and fermented products, which naturally promote beneficial microbial populations [8,15,18]. Physicians in primary care should focus on reinforcing these lifestyle principles, using

probiotics and prebiotics as optional adjuncts rather than substitutes for comprehensive dietary management. In conclusion, the gut microbiota represents a promising but still evolving therapeutic target in the management of obesity and metabolic syndrome. Continued interdisciplinary research—combining nutrition science, microbiology, and clinical practice—will be essential to establish robust, long-term, and safe microbiota-centered strategies that can complement traditional approaches to metabolic health [3,5,6,13,15,19,20].

Disclosure

Author Contributions:

Conceptualization: Adrian Pączek, Aleksandra Kowalczyk

Methodology: Wiktoria Staniszewska, Paweł Dyczek

Formal analysis: Julia Hofman, Aleksandra Kowalczyk

Investigation: Adrian Pączek, Aleksandra Kowalczyk

Writing - Original Draft Preparation: Adrian Pączek, Aleksandra Kowalczyk

Writing - Review and Editing: Wiktoria Staniszewska, Paweł Dyczek, Julia Hofman

Visualization: Adrian Pączek, Wiktoria Staniszewska, Paweł Dyczek

All authors have read and agreed with the published version of the manuscript.

Conflicts of Interest: The authors declare no conflicts of interest.

Funding Statement: No external funding was received to perform this review.

Board Statement: Not applicable – this review included an analysis of the available literature.

Statement of Informed Consent: Not applicable

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