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Original Article

The Role of Gut Microbiota in Pediatric Obesity: A Systematic Review and Meta Analysis of Microbiota Profiles in Obese versus Normal Weight Children

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Abstract:

Background: Pediatric obesity is increasingly acknowledged as a significant public health issue with the gut microbiome identified as a potential contributing factor. Increasing evidence indicated that the gut microbiome is integral to metabolic health and the etiology of obesity. Nonetheless, data pertaining specifically to pediatric populations is still limited and underexplored. This study compared the composition of gut microbiota between obese and normal-weight children and to identify microbial patterns associated with pediatric obesity.

Methods: This study adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A thorough literature search was performed across various databases. We looked at eligible studies and then rated their quality and analyzed them with Newcastle–Ottawa Scale (NOS) and Review Manager (RevMan) 5.4.

Result: This systematic review and meta-analysis included ten studies involving 562 children, utilizing cross-sectional and case-control methodologies. The meta-analysis, which included two studies with 124 participants (64 obese and 60 normal-weight), showed that the Firmicutes to Bacteroidetes (F/B) ratio was much higher in obese children than in normal-weight (mean difference = 5.15; $p < 0.00001$). Taxonomic analysis showed obese children had more members of the phylum Firmicutes, such as *Lactobacillus*, *Clostridium*, and *Megamonas*. On the other hand, Bacteroidetes, especially *Prevotella* and *Bacteroides*, were usually less abundant.

Conclusion: The results indicate that dysbiosis in gut microbiota may contribute to pediatric obesity. These results underscore the potential of gut microbiota modulation as a treatment for childhood obesity. Research is necessary to clarify causal mechanisms and investigate microbiota-based-interventions.

Keyword: gut microbiota, normal weight, obese, pediatric

Introduction

Obesity in children has become a major health problem around the world. The World Health Organization says that the number of overweight kids between the ages of 5 and 19 has gone up by 6.3 percent in 2022.[1] Childhood obesity is when a child stores too much fat, which can lead to serious long-term health problems like a higher risk of diabetes, early death, and heart disease.[2, 3] A child's socioeconomic status, the place they live, their behavior, and their genetic profile can all lead to obesity.[4]

Recent evidence indicates that gut microbiota significantly influences the onset and advancement of obesity. The gut microbiota significantly influences critical metabolic processes, including metabolic health, energy balance, and inflammation.[5] Advanced gene sequencing technologies, such as 16S rRNA, have produced numerous findings from case-control and cross-sectional studies of children, demonstrating that compositional alterations in the gut microbiota (dysbiosis) are associated with the onset of obesity.[6]

Although the precise compositional variations contributing to pediatric obesity are not consistently defined., the ratio of the phylum Firmicutes to Bacteroidetes (F/B) has been suggested as a potential indicator of dysbiosis. Additionally, changes in the gut microbiome, such as a rise in Firmicutes and a decrease in Bacteroidetes, have been associated in children with subsequent metabolic disorders, including impaired insulin signaling, and non-alcoholic fatty liver disease (NAFLD).[7, 8] For instance, an increased F/B ratio has been noted in obese individuals with hepatic steatosis, indicating that microbial dysbiosis may serve as a mechanistic target for intervention in the metabolic consequences of childhood obesity.[8, 9]

This review sought to compare the gut microbiota composition between obese and normal-weight children, identifying microbial patterns linked to pediatric obesity.

Method

Data Source and Search Strategy

We conducted the study in accordance with PRISMA flow diagrams. Four investigators independently searched studies from EBSCO, Epistemonikos, PubMed, and Science Direct. The search was limited to English-language, peer-reviewed studies published up to May 11th, 2025. The following indicated databases performed the search strategy: (Pediatric OR Child* OR Infant) AND (Obese OR Obesity OR Childhood Obesity) AND (Normal Weight AND Gut Microbiota OR Microbiota OR Microbiome OR Biological Community OR Biotic Community).

Inclusion and Exclusion Criteria

The inclusion criteria included: (1) Children under 18 years as the study population, (2) Children with obesity, (3) Studies reporting gut microbiota composition related to obesity, including but not limited to the (F/B) ratio, or other relevant bacteria taxa, and (4) Cross-sectional and case-control studies. The exclusion criteria included: (1) Irrelevant title/abstract, (2) Non-english literature, (3) Inaccurate PICOs component, and (4) Irretrievable full text.

Eligibility Criteria

The identified studies were uploaded to Rayyan.id, and any duplicate records were eliminated before screening. Four reviewers independently assessed the titles, abstracts, and full texts. If disagreements arose, a fifth reviewer (FRS) was involved to resolve discrepancies and finalize study selection.

Data Extraction

The extracted data were arranged in a Google Spreadsheet, with the first step being to record study details like the names of the authors, the year of publication, the design of the study, the population (criteria and amount), the exposure, the comparison, the outcome, the diversity (methods and result), the abundance of bacterial species (methods and result), the ratio of Bacteroidetes (methods and result), the differences between bacterial taxa (methods and result), the exposure to define obese, the control to define non-obese, the result, and the conclusion. The next step was to collect data that was specific to the outcome, such as the number of samples, the means, and the standard deviations for the exposure and control groups. All results were shown as odds ratios (OR) with their 95% confidence intervals (CI).

Quality Assessment

We implemented the Newcastle–Ottawa Scale (NOS) tools to assess the risk of bias for the included cross-sectional and case-control study.

Statistical Analysis

A random-effects model was used to combine study results for continuous variables like the F/B ratio. This model took into account the fact that study results might not be the same across studies. The results were reported as mean differences (MD) along with the corresponding 95% confidence interval (CI). The I2 statistic was used to measure heterogeneity, and an I2 value over 50% was seen as a sign of significant heterogeneity. In addition to the meta-analysis, outcomes unsuitable for quantitative pooling, such as variations in the relative abundance of particular bacterial taxa, were analyzed via a descriptive narrative synthesis. This method involved combining and comparing data from multiple studies to identify comparable trends or discrepancies in gut microbial alterations associated with obesity.

Result

Study Selection

A total of 602 records were identified. After removing 17 duplicates using Rayyan software, 585 records remained. Screening of titles and abstracts resulted in the exclusion of 536 irrelevant studies. Twenty-seven full-text articles were unavailable. Following full-text assessment based on the inclusion and exclusion criteria, 10 studies were included in the final analysis. The study selection process is presented in **Figure 1**.

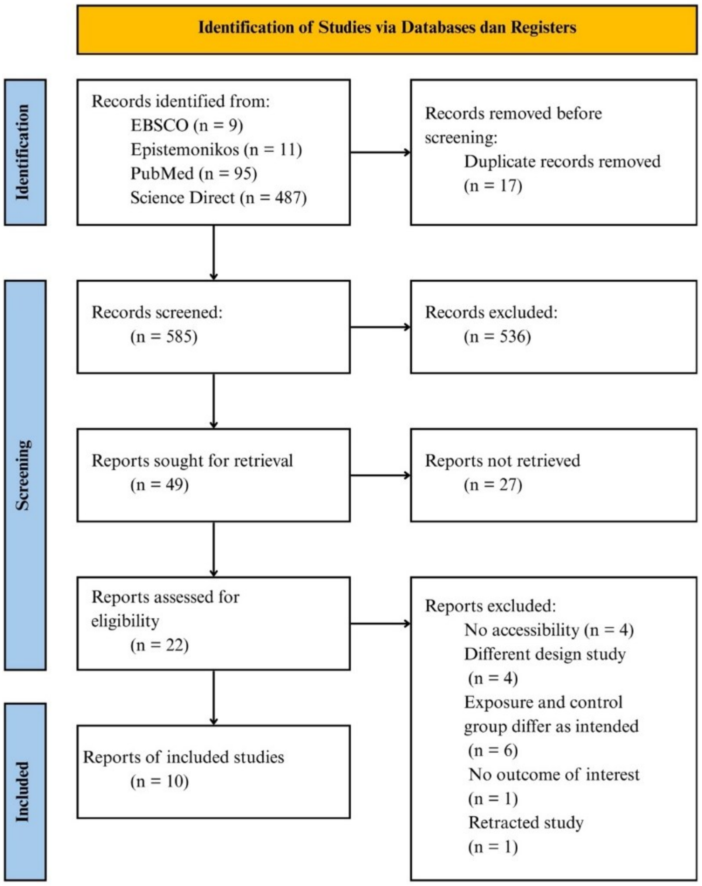


Figure 1. PRISMA Flowchart

Quality Assessment

NOS was used to assess and classify the methodological quality of the included studies, as summarized in **Table 1** for cross-sectional studies and **Table 2** for case-control studies. Based on the NOS, 3 of the 10 studies (30%) were rated as “good quality,” while 7 (70%) were rated as “fair quality.” The scores ranged from a minimum of 6 to a maximum of 8. The lower NOS scores of the case-control study by Borgo et al. were mainly due to limitations in case and control selection and exposure ascertainment.

Table 1. Newcastle-Ottawa Scale Risk of Bias for Cross-Sectional Study

Study Cross-sectional	Selection	Comparability	Outcome	Score NOS	Standard AHRQ
Da Silva et al., 2020[10]	★★★	★★	★★★	8	Fair quality
Riva et al., 2017[11]	★★★★	★★	★★	8	Good quality
Maya-Lucas et al., 2018[12]	★★★	★★	★★	7	Fair quality
Chen et al., 2020[13]	★★★	★★	★★★	8	Fair quality
Shin et al., 2020[14]	★★★	★★	★★★	8	Fair quality
Barczynska et.al., 2018[15]	★★★	★★	★★	7	Fair quality
Ma et al., 2023[16]	★★★★	★★	★★	8	Good quality

Table 2. Newcastle-Ottawa Scale Risk of Bias for Case-Control Study

Study Case Control	Selection	Comparability	Exposure	Score NOS	Standard AHRQ
Wang et al., 2024[17]	★★★★	★★	★★	8	Good quality
Borgo et al., 2016[18]	★★	★★	★★	6	Fair quality
Li et al., 2025[3]	★★	★★	★★★	7	Fair quality

Study Characteristics

Seven cross-sectional studies and three case-control studies were included, originating from China, Korea, Italy, Poland, Mexico, and Republic of Trinidad and Tobago. In total, 562 children were enrolled, comprising 287 obese children and 275 with normal weight. The definitions of obesity and normal weight varied across studies and are summarized in **Table 3**. Most studies measured bacterial outcomes analysis.

Table 3. Characteristics of Included Studies

Author, Year	**Country**	**Study Design**	**Population**		**Exposure**	**Comparison**	**Outcome**
			Age (years)	**N**			
Wang et al., 2024[17]	China	Case-control study	6–16	66 children; 47 obese and 19 normal-weight	Chinese Society of Pediatric Endocrinology and Metabolism (2022) and WHO growth-reference standards	WHO growth-reference standards	Obese children had a higher abundance of Firmicutes and Actinobacteriota and a lower abundance of Bacteroidota, leading to an increased Firmicutes/Bacteroidota ratio. Significant differences in gut microbial composition and predicted metabolic pathways were observed, suggesting microbial dysbiosis associated with obesity.
Borgo et al., 2016[18]	Italy	Case-control study	8–12	61 children; 28 obese and 33 normal-weight	WHO criteria (BMI z-score; obesity defined by WHO growth standards)	Age- and sex-matched children within WHO normal range	Obese children exhibited lower levels of *Akkermansia muciniphila*, *Faecalibacterium prausnitzii*, *Bacteroides/Prevotella* group, *Candida* spp., and *Saccharomyces* spp. compared to normal-weight children, indicating reduced bacterial and fungal diversity and a potential microbial imbalance related to obesity.
Barczyńska et al., 2018[15]	Poland	Case-control study	6–15	40 children; 10 overweight, 10 obese, and 20 normal-weight	International Obesity Task Force (IOTF) criteria (Cole et al., 2012)	IOTF reference range (BMI 18.5–22.38)	Obese children showed a predominance of Firmicutes (*Clostridium*, *Lactobacillus*) and a reduction in Bacteroidetes (*Bacteroides*, *Prevotella*) compared with normal-weight children. Fecal samples from obese children also contained lower levels of short-chain fatty acids (SCFAs), indicating altered microbial metabolism associated with obesity.
Chen et al., 2020[13]	China	Case-control study	6–11	51 children; 28 obese and 23 normal-weight	BMI cutoffs for overweight and obesity in Chinese children and adolescents (Li et al., 2010)	BMI within normal range per Li et al., 2010	Obese children exhibited significantly lower gut microbial diversity and richness than normal-weight children. The relative abundance of Bacteroidetes was reduced, while Firmicutes and Actinobacteria were more abundant. *Faecalibacterium*, *Phascolarctobacterium*, *Lachnospira*, *Megamonas*, and *Haemophilus* were elevated in obese children, whereas *Oscillospira* and *Dialister* were reduced, indicating gut microbiota dysbiosis associated with obesity.

Author, Year	Country	Study Design	Population		Exposure	Comparison	Outcome
			Age (years)	N			
Ma et al., 2023[16]	China	Cross-sectional case-control study	3–5	60 preschool children; 30 obese and 30 normal-weight	Centers for Disease Control and Prevention (CDC) criteria (BMI ≥ 95th percentile)	BMI between 5th and 85th percentile (CDC reference)	Children with obesity had significant alterations in both oral and gut microbiota composition, with higher Firmicutes/Bacteroidetes ratios than controls. *Faecalibacterium*, *Tyzzerella*, and *Klebsiella* were more abundant in obese children's fecal samples, while *Filifactor* and *Butyrivibrio* were enriched in oral samples. A total of 148 bacterial pathways differed significantly, mainly in biosynthetic functions, suggesting functional and compositional dysbiosis linked to obesity.
Shin and Cho, 2020[14]	South Korea	Cross-sectional observational study	5–13	46 children; 22 obese and 24 normal-weight	Korean National Growth Chart 2017 (BMI ≥ 95th percentile)	BMI 5th–85th percentile (Korean Growth Chart)	Obese children showed a significant decrease in Bacteroidetes abundance, which correlated negatively with BMI z-score, while Firmicutes and Actinobacteria proportions increased. Beta diversity differed significantly between groups. Predicted microbial functions related to carbohydrate and lipid metabolism were enriched in normal-weight children, while obese children exhibited distinct clustering of genera associated with inflammation and metabolic risk.
Riva et al., 2017[11]	Italy	Case-control study	6–16	78 children; 42 obese and 36 normal-weight	BMI z-score ≥ 2 SD (WHO criteria)	BMI z-score between −2 SD and +1 SD (WHO criteria)	Obese children exhibited a distinct gut microbiota composition, characterized by increased Firmicutes (72.1%) and decreased Bacteroidetes (16.6%), resulting in a significantly higher Firmicutes/Bacteroidetes ratio (7.7 vs. 2.6; $p < 0.0001$). SCFA concentrations (acetate, propionate, butyrate) were significantly higher in obese children ($p < 0.05$), and BMI z-scores positively correlated with SCFA levels and Firmicutes abundance, indicating that microbial shifts and elevated fermentation activity are associated with pediatric

Author, Year	Country	Study Design	Population		Exposure	Comparison	Outcome
			Age (years)	N			
							obesity.
Maya-Lucas et al., 2018[12]	Mexico	Case-control metagenomic study	5–11	20 children; 10 obese and 10 normal-weight	BMI percentile ≥ 95th (WHO criteria)	BMI percentile 5th–85th	Obese Mexican children had altered gut microbiome composition, with a *Prevotella*-dominated enterotype (Enterotype 2) in 50% of obese children, while normal-weight children mainly exhibited a *Ruminococcus*-dominated enterotype (Enterotype 3). *Megamonas* spp. were significantly enriched, and Oscillospiraceae members were depleted in obese children, correlating negatively with serum cholesterol. Functional pathway analysis revealed that glycolysis pathways (Glycolysis I & II) were overrepresented in normal-weight children, while obese children showed enrichment of catabolic genes (α-amylase) related to starch metabolism, indicating functional and compositional dysbiosis linked to lipid metabolism and obesity.
Da Silva et al., 2020[10]	Brazil	Cross-sectional case-control study	6–14	51 children; 21 obese/overweight and 30 normal-weight	BMI ≥ 97th percentile (WHO criteria)	BMI 15th–85th percentile	Obese children presented reduced microbial diversity and higher abundance of Firmicutes and Actinobacteria, with lower Bacteroidetes compared to controls. Lachnospiraceae and Ruminococcaceae were predominant in obese subjects, while *Bacteroides* and *Prevotella* were enriched in normal-weight children. Functional prediction indicated increased carbohydrate metabolism pathways in obese children, suggesting that gut microbiota composition contributes to enhanced energy harvest and obesity risk.
Li et al., 2025[3]	China	Cross-sectional case-control study	7–14	99 children; 49 obese and 50 normal-weight	Chinese BMI-for-age reference ≥ 95th percentile	5th–85th percentile (Chinese standard)	Obese children exhibited decreased microbial richness and diversity, with a significant increase in Firmicutes and Proteobacteria, and reduction in Bacteroidetes and Verrucomicrobia. The Firmicutes/Bacteroidetes ratio

Author, Year	Country	Study Design	Population		Exposure	Comparison	Outcome
			Age (years)	N			
							correlated positively with BMI and waist circumference. *Faecalibacterium*, *Blautia*, and *Clostridium* were enriched in obese children, whereas *Bacteroides* and *Akkermansia* were more abundant in normal-weight peers, indicating gut microbiota dysbiosis strongly associated with metabolic indicators of childhood obesity.

Microbiota Composition in Obese Children

Shown in **Table 4**, obese children exhibited modifications in gut microbiota, marked by an increase in Firmicutes and Proteobacteria, coupled with a decrease in Bacteroidetes. Multiple studies have also shown a lower diversity of microbes and a higher F/B ratio.

Phylum-level changes

Wang et al. and Riva et al. reported a predominance of Firmicutes, with Riva et al. documenting a significantly higher F/B ratio.[11, 17] Furthermore, Shin et al. noted a negative correlation between Bacteroidetes abundance and BMI z-scores, whereas Li et al. (2025) reported an enrichment of Proteobacteria, including *Enterobacter, Haemophilus, and Sutterella.*[3, 14]

Genus- and species-level changes

Barczyńska et al. observed elevated levels of Clostridium and Lactobacillus, accompanied by decreases in Prevotella and Bacteroides.[15] Similarly, Da Silva et al. identified increased *Lactobacillus* and decreased *Bifidobacterium.*[10] Ma et al. also found that *Fusobacterium, Butyrivibrio, Faecalibacterium, Tyzzerella*, and *Klebsiella* were important groups of bacteria linked to obesity.[16] Maya-Lucas et al. described higher abundances of *Megamonas*, *Ruminococcus*, *Clostridium citroniae*, *Streptococcus thermophilus*, and *Oscillibacter*, with reductions in *Bacteroides* and *Desulfovibrionaceae.*[12] In addition, Borgo et al. emphasized the depletion of *A. muciniphila* and *F. prausnitzii* as notable in obese children.[18]

Diversity and community structure

Da Silva et al. noted diminished overall microbial diversity, whereas Chen et al. validated a general reduction in gut microbiota, with no substantial alterations in Firmicutes.[10, 13]

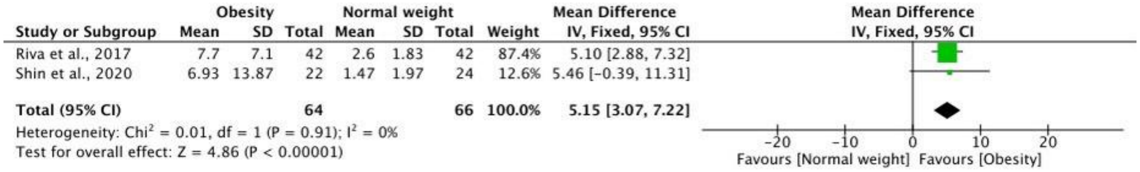
Table 4. Result of Microbiota Profile in Obese Children

Author, year	**↑ Microbiota in Obesity**	**↓ Microbiota in Obesity**	**Significance of Findings**
Wang et al., 2024[17]	Firmicutes (63.5%)	Bacteroidota (37.7%)	-
Borgo et al., 2016[18]	*Bifidobacterium* spp, *Lactobacillus* spp, Enterobacteriaceae, *Saccharomyces* spp, *Candida* spp	-	Significant*: A. muciniphila, F. prausnitzii, Bacteroides/Prevotella* group
Barczynska et.al., 2018[15]	Firmicutes (*Clostridium*, *Lactobacillus*)	Bacteroidetes (*Prevotella*, *Bacteroides*)	*Bifidobacterium* (p = 0.036), *Bacteroides* (p < 0.01)
Ma, et.al, 2023[16]	*Fusobacterium*, *Butyrivibrio*, Faecalibacterium, *Tyzzerella*, Klebsiella	-	Significant based on LEfSe
Chen et al., 2020[13]	-	Overall gut microbiota decreased	No significant change in Firmicutes
Shin et al., 2020	-	Bacteroidetes	Negative correlation with BMI z-score
Li et al., 2025[3]	Proteobacteria (*Enterobacter*, *Haemophilus*, *Sutterella*)	*Oscillibacter*, *Alistipes*	Proteobacteria (p = 0.018)
Da Silva et al., 2020[10]	Firmicutes, *Lactobacillus*	*Bifidobacterium*	Firmicutes (p = 0.001), *Lactobacillus* (p = 0.049), *Bifido* (p = 0.039), reduced diversity (p = 0.009)
Riva et al., 2017[11]	Firmicutes, Ruminococcaceae	Bacteroidaceae, Bacteroidetes	Firmicutes/Bacteroidetes ratio increased (p < 0.00001)
Maya-Lucas et al., 2018[12]	*Megamonas* spp, *Ruminococcus* spp, *Clostridium citroniae*, *Streptococcus thermophilus*, *Oscillibacter* spp	*Bacteroides* spp, Desulfovibrionaceae	Higher Human herpesvirus 4 in obese children (p = 0.017)

Quantitative Assessment

The forest plot shown in **Figure 2.** summarized a meta-analysis that compared a continuous outcome between children with obesity and those with normal weight across two studies (Riva et al. and Shin et al.). The pooled mean difference was 5.15 (95% CI: 3.07-7.22, p<0.00001), which means that the measured variable was much higher in the obesity group. The study by Riva et al. demonstrated a significant difference (MD = 5.10, 95% CI: 2.88-7.32), whereas the study by Shin et al. indicated a similar directional effect, albeit not statistically significant (MD = 5.46, 95% CI: -0.39-11.31).[11, 14]

There was no heterogeneity among studies ($I^2 = 0\%$, p = 0.91), indicating consistent findings across datasets. The overall effect, represented by the diamond to the right of the line of no effect, indicated that the analyzed variable was significantly higher in children with obesity compared to their normal-weight counterparts.



	Obesity			Normal weight				Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Fixed, 95% CI
Riva et al., 2017	7.7	7.1	42	2.6	1.83	42	87.4%	5.10 [2.88, 7.32]
Shin et al., 2020	6.93	13.87	22	1.47	1.97	24	12.6%	5.46 [−0.39, 11.31]
Total (95% CI)			**64**			**66**	**100.0%**	**5.15 [3.07, 7.22]**

Heterogeneity: $\text{Chi}^2 = 0.01$, df = 1 ($P = 0.91$); $I^2 = 0\%$

Test for overall effect: $Z = 4.86$ ($P < 0.00001$)

Figure 2. Meta-Analysis using Review Manager 5.4

Discussion

This systematic review and meta-analysis included ten studies that varied by demographic, study type, and country. The majority were cross-sectional, with populations ranging from 20 to 99 and children ages 3 to 16 years old.[3, 10-18] Exposures included children with obesity based on body mass index (BMI z-score) compared with children of normal weight. The risk of bias using NOS in **Table 1** and **Table 2** resulted in three studies with good quality and seven with fair quality. A forest plot showed that the obese group had a much higher F/B ratio than the normal-weight group.[11, 14] These findings reveal differences in the gut microbiome composition between obese and normal-weight children. Obese children exhibited a greater relative prevalence of Firmicutes bacteria compared to Bacteroidetes, leading to an elevated F/B ratio. This suggests that an imbalance in gut flora may elevate the risk of obesity.

Figure 3 illustrates the complex interplay between gut microbiota composition and the host's energy metabolism. It shows how the balance of microbes affects the processing of nutrients, the extraction of energy, and metabolic health. The gut microbiota plays a crucial role in regulating nutrition, energy metabolism, and the pathogenesis of diseases, including obesity.[19] The bacterial phylum Firmicutes has been associated with an increased ability to harvest energy among its members. More Firmicutes in the body helps the body get more calories from food, which leads to more energy intake and fat storage.[20] In the left pathway, gut bacteria break down carbohydrates that can't be digested through fermentation, which makes short-chain fatty acids (SCFAs). These SCFAs act as signaling molecules that affect the release of the satiety hormones Glucagon-Like Peptide-1 (GLP-1) and Peptide YY (PYY) which enhance fat metabolism, reduce hunger, and suppress inflammation. This biochemical signaling not only promotes efficient energy use but also maintains metabolic and immune balance.[21] Whereas, in the right pathway, gut bacteria contain genes that code for carbohydrate-active enzymes (CAZymes), which are essential for breaking down complex carbohydrates into simpler, absorbable components. This enzymatic breakdown made it easier for Firmicutes to get energy, which could have led to more calories being taken in and more fat being stored.[17]

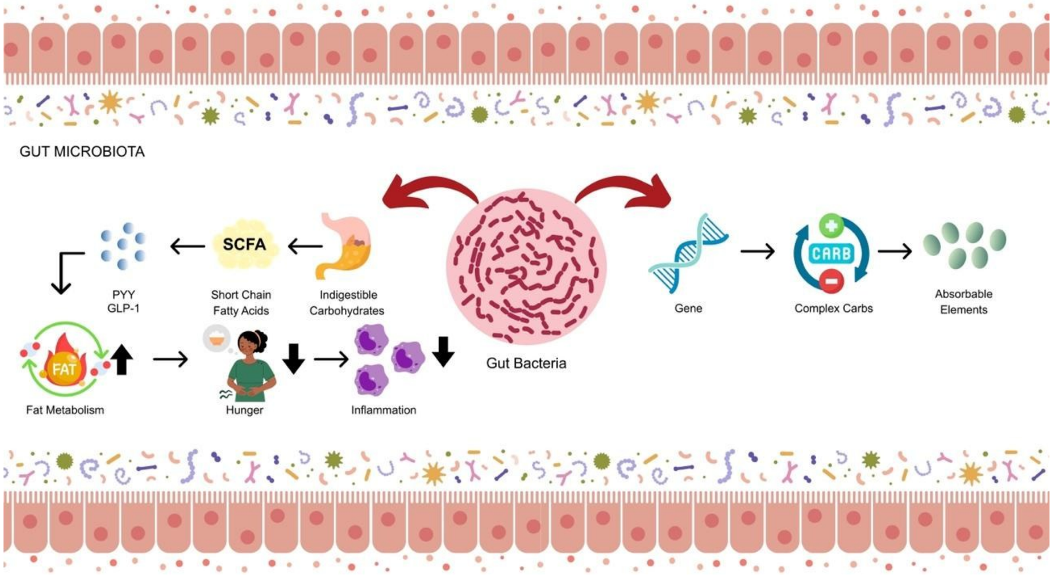


Figure 3. The Role of Gut Microbiota in Energy Metabolism

A decrease in the Bacteroidetes population is linked to continued high-fat food consumption, which may contribute to obesity.[22] In contrast, Bacteroidetes have anti-obesity properties by enhancing insulin sensitivity, raising serum GLP-1, and promoting amino acid catabolism.[23] This finding is consistent with the observed higher F/B ratio in the intestines of obese children. Therefore, the abundance of Bacteroidetes and Firmicutes can be utilized as a biomarker to distinguish between obese children and those of normal weight.

In addition to the phylum-level changes in Firmicutes and Bacteroidetes, some of the studies included found that important beneficial taxa like *Akkermansia muciniphila* and *Faecalibacterium prausnitzii* were less common. *F. prausnitzii* is a crucial butyrate-producing strain vital for gastrointestinal and metabolic health. *A. muciniphila*, on the other hand, is not a butyrate producer, but it is important for keeping the intestinal mucous layer intact, making acetate and propionate, controlling lipid metabolism, and making the intestines less permeable. Both taxa have been identified in markedly reduced abundance in obese or overweight children, indicating their protective functions against metabolic dysregulation.[24]

This review has several limitations that should be acknowledged. First, the majority of the studies included were cross-sectional, limiting the capacity to establish causal inferences or ascertain temporal relationships. It is still not clear if changes in the gut microbiota happen before obesity and growth problems happen, or if they happen because of changes in diet and metabolism. Longitudinal studies examining dietary and physiological factors, alongside comprehensive functional analyses of the metabolic activity of the gut microbiota, are essential to elucidate this directionality.

Since all the studies included came from China, Korea, and Italy, and there were also studies from Poland, Mexico, and the Republic of Trinidad and Tobago, there is a big gap in data from Southeast Asia, such as Indonesia. Future prospective cohort studies in this region are necessary to examine regional dietary patterns, genetic backgrounds, and environmental exposures that may affect gut microbiota composition and its association with pediatric obesity. Such research would augment the applicability and generalizability of findings across varied populations.

Second, the studies that were included had problems with the way they collected samples. Stool samples, although frequently utilized, may not adequately reflect the microbial environment along the intestinal wall where host–microbiome interactions take place. This method provides limited information on microbial dynamics in the small intestine, the primary site of nutrient absorption. Third, the outcome parameters across studies were heterogeneous. The F/B ratio was the most consistently reported and widely used measure of gut health, but other results were very different, making it hard to compare and combine them. Only two studies (Riva et al. and Shin et al.) were suitable for quantitative synthesis regarding the F/B ratio, involving 124 participants (64 obese and 60 of normal weight), with one assessed as good quality and the other as fair quality. Although the pooled analysis demonstrated high homogeneity ($I^2 = 0\%$), the exclusion of the fair-quality study would leave insufficient data for meaningful pooling, a sensitivity analysis could not be performed.[11, 14] This represents the limitations of the present review, and future meta-analysis including a greater number of high quality studies are needed to confirm the robustness of these findings.

The literature search in this review was limited to four databases and English-language studies that the reviewer could fully access, which also created limitations. While major databases such as Scopus and Web of Science, as well as grey literature sources, were not included, this approach was chosen to maintain methodological consistency. Consequently, some relevant studies may have been missed. Furthermore, the review predominantly contrasted obese or overweight children with their healthy peers, lacking a comprehensive examination of underlying biological mechanisms or broader determinants, such as metabolic parameters. These limitations highlight the necessity for forthcoming studies utilizing longitudinal designs and integrative multi-omics methodologies to more effectively clarify the intricate interactions among gut microbiota, nutrition, and child growth across various populations.

The existing evidence highlights the necessity for longitudinal and mechanistic studies to determine whether alterations in gut microbiota are a cause or a consequence of pediatric obesity. Standardized methodologies and the integration of multi-omics approaches are crucial for enhancing comparability and yielding more profound insights into host-microbe interactions. Investigating contextual factors such as diet, antibiotic exposure, genetics, and regional variations will enhance the applicability of findings across diverse populations. Along with their therapeutic potential, the persistent patterns of microbial imbalance also create possibilities for gut microbiota profiles to function as screening instruments for the early detection of children predisposed to obesity.

From a clinical standpoint, these insights underscore the potential to explore microbiota-targeted approaches, including dietary interventions, prebiotics, probiotics, or synbiotics, as viable instruments for prevention and treatment. *Lactobacillus rhamnosus*, *Lactobacillus gasseri SBT2055*, and *Bifidobacterium breve B-3* are some of the probiotic strains that have shown promise in changing gut dysbiosis related to obesity. *L. rhamnosus* demonstrates anti-obesogenic properties, in part by generating SCFAs that can influence hunger and satiety pathways at both peripheral and central levels. While the majority of studies did not directly isolate SCFAs from this species, it possesses the ability to produce them under diverse conditions, thereby contributing to anti-inflammatory, antimicrobial, and immunomodulatory effects that maintain intestinal integrity and permeability.[25] Randomized trials have shown that *L. gasseri* SBT2055 can help people lose weight by lowering their body mass index, waist circumference, abdominal visceral fat, and hip circumference after 12 weeks of supplementation. These effects are probably due to changes in lipid metabolism and the body's energy balance.[26] Likewise, supplementation with Bifidobacterium breve B-3 (BB-3) in double-blind, placebo-controlled trials resulted in substantial reductions in body weight, waist circumference, and hip circumference, demonstrating its safety and effectiveness in decreasing body fat.[27] Moreover, *A. muciniphila* supplementation has shown potential in enhancing metabolic parameters and preserving intestinal barrier integrity, whereas *F. prausnitzii* exhibits anti-inflammatory properties that promote metabolic health.[28, 29]

To put these ideas into action, we need to do strict tests to see how well they work and how safe they are for kids. Advancing this line of research may ultimately contribute to precision medicine, in which interventions can be customized based on individual microbial and metabolic profiles, thereby improving the efficacy of obesity management in children.

Conclusion

In conclusion, our findings indicate that an imbalance in gut microbiota may contribute to pediatric obesity. The analysis shows that obese children have less microbial diversity overall and a higher F/B ratio. The increase in Firmicutes may lead to greater production of short-chain fatty acids, which in excess provide more energy. Meanwhile, the decrease in Bacteroidetes is linked to lower microbial diversity, which continued high-fat food consumption, and may contribute to obesity. Together, these mechanism may explain how microbial imbalance contribute to metabolic disruption and fat accumulation in pediatric obesity. Nevertheless, further research is needed to clarify causal mechanisms and explore microbiota-based interventions in children

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Conflict of Interest

There is no conflict of interest.

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