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The role of the gut microbiota in childhood obesity

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Abstract

The gut microbiota has emerged as an important factor influencing metabolic health of people. Recent research indicates that children with obesity present distinct microbial compositions compared with their normal-weight peers, suggesting a potential role of intestinal microorganisms in the development adiposity. This paper discusses current findings on how dietary patterns, perinatal exposures and inherited microbial traits shape the gut microbiome in childhood. Particular attention is given to taxa associated with metabolic alterations, as well as to

microbial metabolites that may influence energy balance and inflammation. Studies linking the gut microbiota to anthropometric measures and early markers of cardiovascular dysfunction highlight its potential value as a diagnostic tool. Understanding these complex host–microbe interactions may contribute to the development of targeted microbiota-based interventions and personalized therapeutic strategies aimed at preventing or treating childhood obesity.

Key words Childhood obesity, Gut microbiota, Intestinal microbiota, Microbiota

Introduction

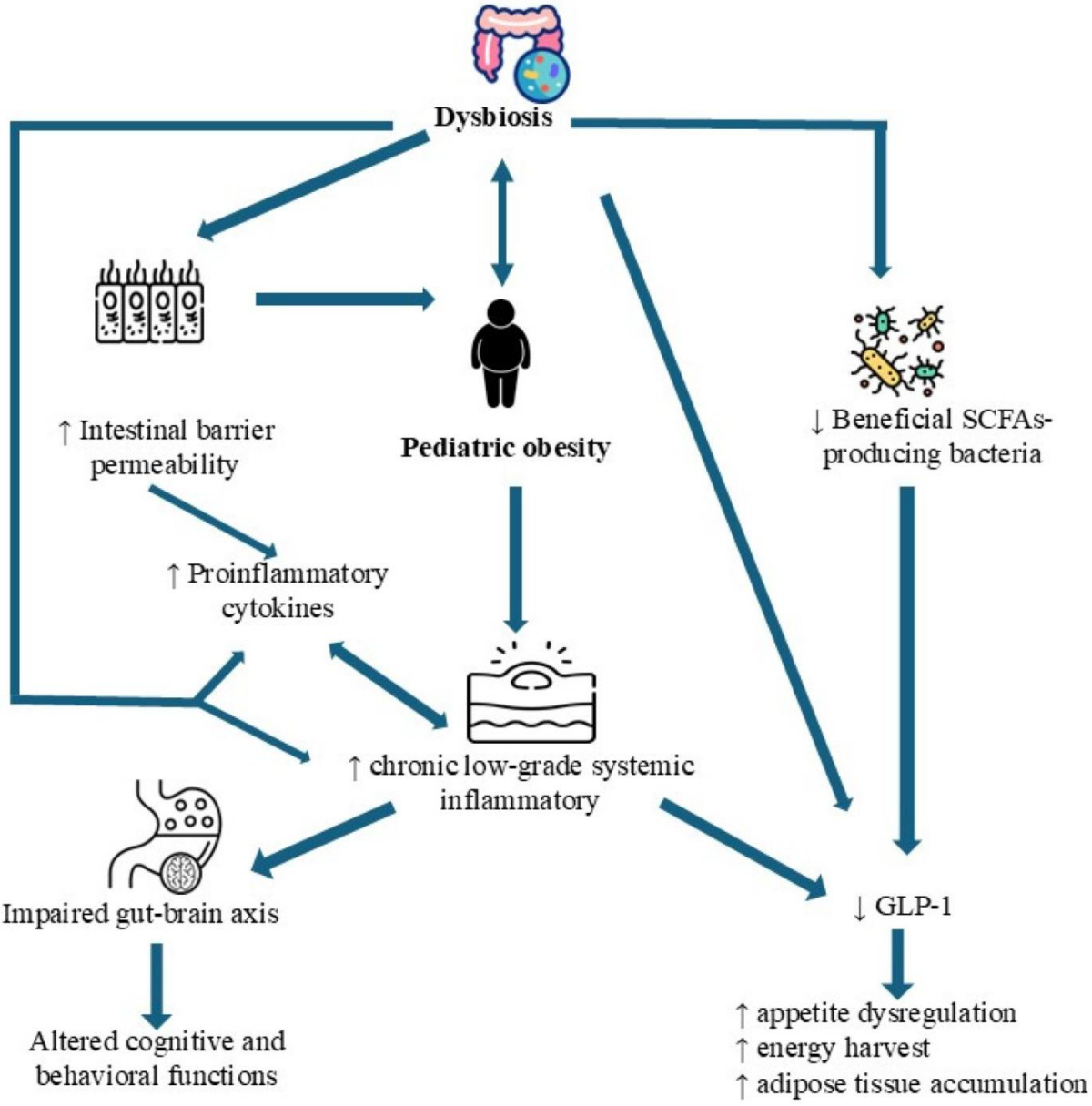
The literature shows growing interest in the topic of gut microbiota. It is composed mainly of bacteria, followed by archaea, viruses, fungi, and protozoa [1]. This article focuses on the role of bacteria, as they represent the primary group of microorganisms inhabiting the intestines. The functions of the microbiota include the synthesis and absorption of numerous nutrients and metabolites—such as short-chain fatty acids (SCFAs)—as well as digestion, maturation and development of the immune system, inhibition of pathogenic microorganism adhesion, and gut–brain interaction [2].

Changes in the composition of the gut microbiome directly affect tight junctions, increasing intestinal permeability and consequently promoting inflammation and the development of metabolic diseases [3]. In children, the composition of the gut microbiome has been linked to several conditions, including obesity, malnutrition, gastrointestinal disorders, cardiovascular diseases, allergies, autism and others [3]. An imbalance of the gut

microbiome, known as dysbiosis—characterized by an increased number of pro-inflammatory bacteria in children with obesity compared to those without obesity—leads to increased appetite and exacerbation of metabolic disturbances [4]. Figure 1 illustrates the relationship between dysbiosis and obesity in children [5].

The aim of this article is to analyze the gut microbiome in the childhood obesity, in order to present the occurring changes and their clinical consequences.

Figure 1. The complex pathophysiological interplay between gut dysbiosis and pediatric obesity: inflammatory, endocrine, and neurobehavioral pathways. Abbreviations: GLP-1: Glucagon-like peptide-1; SCFAs: Short-Chain Fatty Acids. [5]



Methods

To prepare this review, a thorough search of scientific publications was conducted, focusing on the most recent and up-to-date data on the topic, with the publication date limited to the last five years. The literature search strategy was carried out using the PubMed database, based on a combination of the following keywords: *childhood obesity*, *intestinal microbiota*, *gut microbiota*, *microbiota*. After analyzing the titles and abstracts, incomplete articles and those not directly related to the gut microbiome in children with obesity or overweight were excluded. The final analysis included 11 publications that met the inclusion criteria.

Results and discussion

Gut microbiome diversity in children with obesity and normal body weight

To compare the composition and diversity of the gut microbiome, as well as microbiological associations with clinical and metabolic parameters in both obese and normal-weight Thai children, a study was conducted including 164 obese children aged 7–15 years [6]. The results were compared with previously published data obtained from 45 non-obese Thai children [6]. Stool samples were collected from all 164 participants and subjected to 16S rRNA sequencing and analysis.

Alpha-diversity indices showed no significant differences between obese and non-obese children, whereas beta-diversity revealed significant differences at the family and genus levels [6]. Table 1 summarizes the differences in bacterial population abundance between obese and normal-weight children.

Table 1. **Composition and diversity of the microbiome.** Based on [6]

Composition and diversity of the microbiome	
in obese children	**in children with normal body weight**
Firmicutes 47,1% ↑	Firmicutes 45,6%
Bacteroidetes 34,4% ↓	Bacteroidetes 45,7%
Proteobacteria 14,0% ↑	Proteobacteria 2,7%
Actinobacteria 2,4% ↓	Actinobacteria 5,2%
Fusobacteria 1,0% ↑	Fusobacteria 0,8%

Among taxa at the genus level, the relative abundance of *Bifidobacterium* was significantly higher, and the abundance of *Blautia* and *Lactobacillus* significantly lower in normal-weight children compared with those with obesity [6]. Univariate analysis showed that the decrease in *Actinobacteria* and *Bifidobacterium* observed in the obese group was associated with lower HDL-C levels, increased body weight, and more screen time, which averaged 6–8 hours daily among obese children [6]. Furthermore, *Actinobacteria* showed a negative association with fasting insulin and the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR).

A similar study conducted in Chinese children demonstrated that alpha-diversity was significantly higher in normal-weight children than in those who were overweight or obese [7]. Both groups showed significant differences in the relative abundance of *Megamonas*, *Bifidobacterium*, and *Alistipes* [7]. Another Chinese study found that *Prevotella* and *Firmicutes* were more abundant in children with obesity, while *Bacteroides* and *Sanguibacteroides* predominated in normal-weight children [8]. Yet another study reported that the class *Deltaproteobacteria,* family *Bacteroidaceae,* family *Desulfovibrionaceae*, genus *Bacteroides* and genus

Butyricoccus were more abundant in obese children than in those without obesity [9]. These studies collectively reveal notable differences in the composition of gut bacteria between children with obesity and their normal-weight peers.

Squillario and colleagues analyzed the gut microbiome of 55 children and adolescents with obesity and 25 age- and sex-matched individuals with normal body weight [10]. The authors found that *Streptococcus*, *Acidaminococcus, Sutterella, Prevotella, Sutterella wadsworthensis, Streptococcus thermophilus*, and *Prevotella copri* were positively correlated with obesity [10]. Additionally, *Alistipes indistinctus*, *Clostridium innocuum, Desulfovibrio piger, Prevotella ruminicola* and *Prevotella* were significantly associated with elevated CRP levels.

Acidaminococcus fermentans, *Clostridium cocleatum*, and *Clostridium ramosum* showed a negative correlation with blood glucose [10]. Moreover, the researchers discovered that the family *Oxalobacteraceae* and two of its genera—*Herbaspirillum* and *Oxalobacter*—were present in the group negatively correlated with glycemia, and these taxa were already known to be associated with reduced insulin resistance and lower glucose levels [10].

Metabolic pathways strongly associated with obesity included the biosynthesis pathways of tyrosine, phenylalanine, tryptophan, and methionine [10]. In addition, various pathways responsible for polyamine synthesis—compounds involved in bacterial pathogenicity and biofilm formation—and the pro-inflammatory lipopolysaccharide biosynthesis pathway were more abundant in samples from individuals with obesity. Another finding in obese patients was reduced biosynthesis of butanediol, a leptin-sensitizing compound that suppresses hunger [10].

Most authors point to the Firmicutes/Bacteroidetes (F/B) ratio as a biomarker of obesity. However, some publications report that samples from obese patients consistently showed a lower F/B ratio than those from normal-weight individuals, which would exclude this parameter as a reliable indicator of obesity [10].

Gut microbiota in early childhood

Researchers analyzed the relationship between early-life gut microbiota and the development of body mass index (BMI) and body composition throughout childhood [11]. Gut microbiota samples were collected from stool at five time points: at 1 week, 1 month, 1 year, 4 years, and 6 years of age, from a cohort of 700 children.

No consistent associations were found between early-life gut microbiota diversity, its overall composition, or the abundance of individual taxa—across both supervised and unsupervised models—and current or later BMI z-scores, overweight, obesity, obesity relapse, or body composition in childhood [11].

Although the researchers did not observe any consistent associations, this does not definitively exclude the possibility of such a relationship. Instead, it suggests that if these connections exist, they may be more complex and potentially dependent on factors emerging later in life, such as lifestyle changes [11].

Gut microbiota and SCFAs

A study conducted in China examined the relationship between gut microbiota and short-chain fatty acids (SCFAs) in children with obesity [12]. The findings showed that childhood obesity may be associated with an increased abundance of Gram-negative bacteria such as *Escherichia coli* and *Haemophilus*, leading to increased lipopolysaccharide biosynthesis and abnormal metabolism [12].

A decrease in butyrate and isobutyrate levels, along with an increase in caproate levels, was observed in children with obesity. These changes disrupted the colonic epithelial barrier, affected host metabolism, and resulted in increased energy expenditure. Analyses also demonstrated a positive correlation between caproate levels and triglyceride concentration, which may promote adipose tissue development and subsequently childhood obesity [12].

Gut microbiota as a marker of cardiovascular damage

Recent studies suggest that the gut microbiota may serve as a marker of obesity and obesity-related cardiovascular damage in children [13]. Researchers found that gut microbiota dysbiosis was associated with obesity accompanied by damage to the carotid artery intima–media complex in children [13].

The genera *Christensenellaceae_R-7_group*, *UBA1819*, *Iy_XIII_AD3011_group*, and *Family_unclassified_o_Bacteroidales* showed high efficacy in identifying obesity and the associated carotid intima–media complex damage in children [13]. These biomarkers may represent a non-invasive diagnostic tool for detecting obesity-related cardiovascular impairment.

Parent–child gut microbiome relationship

Begoña de Cuevillas and colleagues investigated the heritability/transmission of the gut microbiome from parents (mothers and fathers) to children as a potential risk factor for obesity [14]. The study included 146 parent–child pairs, divided into four groups:

- **Group 1:** Parent and child with normal body weight
- **Group 2:** Parent and child with overweight or obesity
- **Group 3:** Parent with normal body weight and child with overweight or obesity
- **Group 4:** Parent with overweight or obesity and child with normal body weight

The presence of high abundance of five taxa (*Catenibacterium mitsuokai*, *Prevotella stercorea*, *Desulfovibrio piger*, *Massiliprevotella massiliensis*, and *Phascolarctobacterium succinatutens*) was significantly more common (72.7%) in group 2 than in the other groups (1.3%, 15.3%, and 6.5% in groups 1, 3 and 4, respectively) [14].

A specific association was identified between *P. succinatutens* in the parent and *M. massiliensis* in the child, whose co-occurrence was linked to childhood obesity [14]. The higher the abundance of both bacteria in the stool, the greater the risk of obesity in children, whereas lower bacterial abundance in parents was associated with a reduced risk of overweight in offspring [14]. These findings may be useful for predicting obesity in children, adolescents and later in adults within a given family.

Tobacco smoking and the gut microbiome

A study examining the impact of maternal smoking during pregnancy on the risk of microbiota-related obesity in children included 1 592 infants from the Canadian Healthy Infant Longitudinal Development (CHILD) Cohort [15]. Data on environmental exposures and lifestyle were collected prenatally and during the first three years of life, while body weight outcomes were measured at ages 1 and 3 years.

The study showed that alpha-diversity indices of the gut microbiota in early and late infancy were significantly higher in children of mothers who smoked during pregnancy, particularly for *Firmicutes* and *Actinobacteria* [15]. Moreover, *Firmicutes* was positively associated with BMI z-scores. Elevated levels of butyrate were observed in the stool of children with a history of maternal smoking during pregnancy. Although butyrate generally has anti-obesity effects in adults, several studies have shown that obese individuals have higher levels of short-chain fatty acids in stool compared with lean individuals [15].

One possible mechanism for the positive correlation between butyrate and obesity in children is that excess available butyrate may lead to the production of excessive substrates for lipid biosynthesis, ultimately promoting excessive weight gain [15]. These findings highlight the mediating role of the gut microbiota in the effects of harmful prenatal exposures on childhood outcomes and open possibilities for future microbiome-based weight management strategies in pediatric populations.

Diet and the gut microbiome

Another study examined the gut microbiome and its diet-dependent relationship with obesity in children [16]. The study included 46 children aged 6–12 years, of whom 26 had normal body weight, 11 were overweight, and 9 were obese.

The results showed that children with high protein intake and a higher abundance of *Holdemania* spp. had lower BMI z-scores, waist circumference, and hip circumference [16]. Additionally, *Coprococcus catus* and low protein intake were associated with increased hip circumference, while *Bilophila* spp., *Paraprevotella xylaniphila*, and high intake of saturated fats and simple carbohydrates showed a positive correlation with BMI z-scores, waist circumference, and hip circumference [16]. These findings suggest that the synergy between diet and gut microbiome profile in children may be a contributing factor to the development of obesity [16].

Summary

Childhood obesity is associated with alterations in the gut microbiome compared to normal-weight children, affecting the production of short-chain fatty acids and inflammatory markers. This article highlighted the role of environmental factors, such as children's diet and maternal smoking, as well as the heritability of the microbiome, in shaping gut microbiota in children. Additionally, the gut microbiota was identified as a potential marker of cardiovascular damage. The gut microbiome represents a promising area for future research aimed at leveraging existing differences in obese children to develop targeted therapeutic strategies based on individualized patient approaches.

Disclosure

Author's Contribution

Conceptualization: Kamila Bała, Natalia Staszko

Methodology: Kamila Bała, Natalia Staszko

Formal analysis: Kamila Bała, Natalia Staszko

Investigation: Kamila Bała, Natalia Staszko

Writing rough preparation: Kamila Bała, Natalia Staszko

Writing review and editing: Kamila Bała, Natalia Staszko

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

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