Pediatr Pol 2026; 101 (2): 154-160
DOI: https://doi.org/10.5114/polp.2026.162938
Submitted: 30.11.2025, Accepted: 27.12.2025, Published: 30.06.2026

PEDIATRIA Polska
PISMO POLSKIEGO TOWARZYSTWA PEDIATRYCZNEGO
Polish Journal of PAEDIATRICS
OFFICIAL JOURNAL OF POLISH SOCIETY OF PAEDIATRICS

REVIEW PAPER

Modification of gut microbiota in the treatment of childhood obesity

Kamila Bała, Natalia Staszko

Department of Otolaryngology, Head and Neck Surgery, Wroclaw Medical University, Wroclaw, Poland

ABSTRACT

Childhood obesity is recognized as the most common chronic disease of childhood and adolescence, associated with metabolic disturbances and alterations in the gut microbiome. Research indicates that modulation of the gut microbiota may play a pivotal role in obesity management. This review summarizes current strategies for influencing the pediatric gut microbiome, including probiotics, prebiotics, synbiotics, chitosan, fecal microbiota transplantation, metformin, dietary interventions and physical activity. The applied gut microbiome modifications contribute to an increase in beneficial bacterial populations while simultaneously reducing harmful bacterial populations. These changes enhance the production of short-chain fatty acids and decrease inflammation, ultimately leading to improved metabolic outcomes and weight reduction in pediatric patients. Modulating the gut microbiome represents a promising approach for treating obesity, however, long-term studies involving large populations are needed to confirm the efficacy and safety of these interventions in children.

KEY WORDS:
childhood obesity, gut microbiota, intestinal microbiota, microbiota.

INTRODUCTION

Childhood obesity is a serious health threat and is recognized as the most common chronic disease of childhood and adolescence. According to the 2025 UNICEF report, one in twenty children under the age of 5 (5%) and one in five children and/or adolescents aged 5–19 years (20%) worldwide live with overweight and a significant proportion of them are obese [1]. Among the risk factors for obesity, the role of the gut microbiota is increasingly highlighted in the literature and its modification is considered a promising method for the treatment of pediatric obesity. In modulating the gut microbiome, the most widely studied interventions include probiotics, prebiotics, synbiotics, postbiotics, fecal microbiota transplantation, diet and physical activity. According to the definition of the International Scientific Association for Probiotics and Prebiotics, a probiotic is a live microorganism which, when administered in adequate amounts, confers a health benefit on the host. A prebiotic, on the other hand, is a substrate that is selectively utilized by host microorganisms, conferring a health benefit [2]. Prebiotics include carbohydrate-based products such as oligosaccharides and dietary fiber, as well as other compounds (for example conjugated linoleic acid, polyunsaturated fatty acids, phenolic compounds) [3]. Certain dietary fiber components, such as inulin, fructooligosaccharides and galactooligosaccharides (GOS), possess fermentative capacity and consequently release short-chain fatty acids (SCFAs), which specifically stimulate the growth of beneficial microorganisms, including *Bifidobacterium* and *Lactobacillus* [3]. Another definition concerns synbiotics, which are a mixture of live microorganisms and a substrate selectively utilized by host microorganisms that confer a health benefit on the host. Two types of synbiotics are distinguished: complementary and synergistic synbiotics. A complemen-

ADDRESS FOR CORRESPONDENCE:
Kamila Bała, Department of Otolaryngology, Head and Neck Surgery, Wroclaw Medical University, Wroclaw, Poland, e-mail: kamila.bala@wp.pl

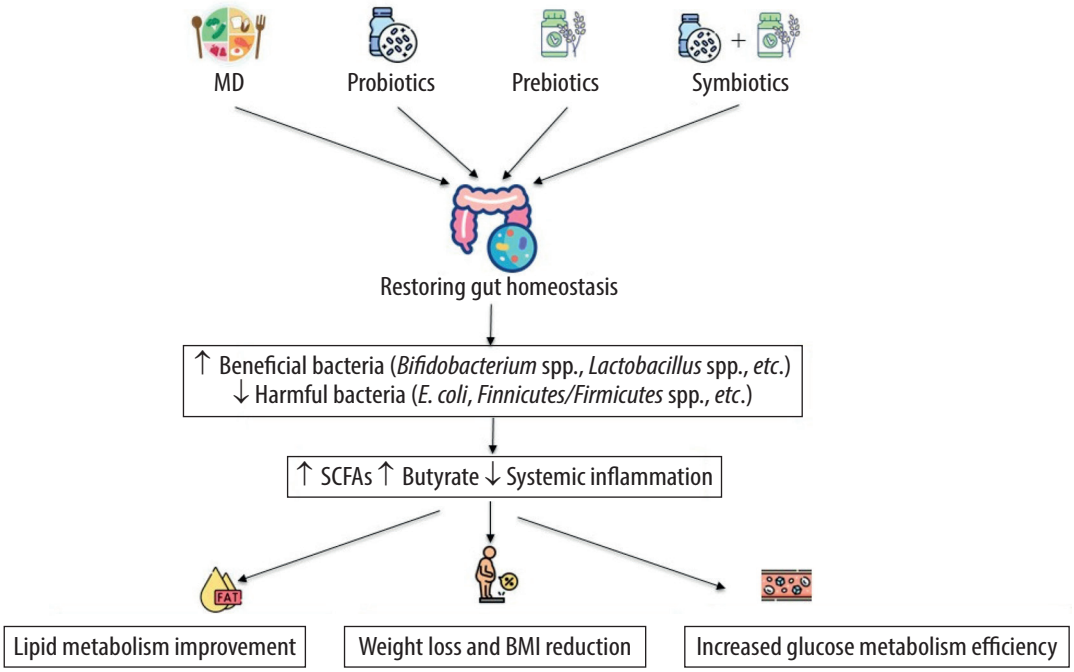

FIGURE 1. Potential therapeutic role of gut microbiota modulation in pediatric obesity [6]

BMI – body mass index, MD – Mediterranean diet, SCFAs – short-chain fatty acids

tary synbiotic consists of a combination of a probiotic and a prebiotic. A synergistic synbiotic is one in which the substrate is specifically designed to be selectively utilized by the co-administered microorganisms [4]. The International Scientific Association for Probiotics and Prebiotics, in another document, further defined a postbiotic as a preparation of inanimate microorganisms and/or their components that confers a health benefit on the host. According to *in vivo* studies, molecules present in postbiotics, such as lactic acid and bacteriocins, may exert direct antimicrobial effects. Postbiotics may also indirectly modulate the microbiota, for example through the transport of lactic acid, which can be absorbed by certain microbiota components, leading to the production of molecules with beneficial functions, including SCFAs and butyrate [5]. Figure 1 presents a simplified overview of gut microbiota modification and its effects in childhood obesity [6].

MATERIAL AND METHODS

To prepare this review, a comprehensive search of scientific publications was conducted, including the most recent and up-to-date data on the topic, with the publication date limited to the past five years. The literature search strategy was carried out using the PubMed database based on a combination of the following key words: *childhood obesity*, *gut microbiota, intestinal microbiota*, and *microbiota*. After screening the titles and abstracts, incomplete articles and those not directly related to the modification of the gut microbiome in children with obesity or overweight were excluded. The final analysis included 21 publications that met the inclusion criteria.

MICROBIOME MODIFICATION

BUTYRATE AND OTHER SHORT-CHAIN FATTY ACIDS

Plant-based foods, particularly indigestible carbohydrates, are fermented by the gut microbiota to produce SCFAs, including butyrate, which exert anti-inflammatory effects and protect against obesity. Therefore, low intake of dietary substrates for butyrate production and a reduced number of butyrate-producing bacteria may contribute to obesity. A study involving 54 children aged 5–17 years demonstrated that a 6-month supplementation with sodium butyrate may reduce body mass index (BMI) and improve glucose metabolism and inflammatory status in children with obesity. Additionally, butyrate reduced homeostatic model assessment – insulin resistance (HOMA-IR), fasting insulin levels and significantly lowered serum ghrelin – a hormone that stimulates appetite – in children with obesity [7]. On the other hand, some studies indicate that SCFAs serve as an important energy source and may promote weight gain by increasing gluconeogenesis and lipogenesis [8, 9]. An example is a study examining the association between the abundance of specific taxa in the gut microbiome and SCFA levels with total and regional body fat distribution in children. The results showed that concentrations of simple/short-chain fatty acids (acetate, propionate and butyrate) were significantly positively correlated with BMI, total and regional adiposity and the fat-to-lean mass ratio. Moreover, no correlation was observed between the firmicutes/bacteroidetes ratio and

total or regional fat content. However, several operational taxonomic units (OTUs), such as *Ruminococcus gnavus* and *Flavonifractor plautii*, were negatively associated with obesity-related parameters, while two OTUs assigned to the genera *Blautia* and *Romboutsia* correlated positively with adiposity parameters [9]. Given these divergent findings, further research is necessary to clarify the role of SCFAs in obesity within the pediatric population.

PREBIOTIC – INULIN

Dietary substrates for butyrate production include inulin supplementation, whose effect on the gut microbiome was investigated by Visuthranukul *et al.* [10]. In the group of patients receiving inulin, an increase in butyrate-producing bacteria was observed, specifically *Agathobacter*, *Subdoligranulum* and *Eubacterium coprostanoligenes*. The change in the abundance of *Eubacterium coprostanoligenes* was negatively correlated with high energy intake, and animal model studies additionally demonstrated a negative association with body weight and cholesterol levels. Furthermore, the change in *Subdoligranulum* abundance was negatively correlated with BMI *Z*-score [10]. As a consequence of the increased α diversity of the gut microbiota in patients supplemented with inulin, this prebiotic may serve as a therapeutic strategy aimed at restoring beneficial microorganisms and gut microbiome balance in children with obesity. Another study supporting the role of inulin demonstrated its potential impact on the gut-brain axis and metabolic regulation in obesity through an increase in amino acids and biogenic amines in the inulin-supplemented group. Specifically, concentrations of tyrosine, putrescine and spermine showed significant increases compared with baseline values in the inulin group. Tyrosine levels correlated positively with *Lactobacillus* but negatively with *Alistipes*. Additionally, putrescine levels correlated positively with *Bacteroides* and *Fusobacterium*, while spermine correlated with *Bifidobacterium* and *Blautia*. Putrescine, which demonstrated the greatest increase among the polyamines in the inulin group, activates histamine receptors, participating in the regulation of appetite and satiety. Moreover, putrescine is converted into spermidine and spermine, which exert even stronger biological effects. Another amino acid that increased significantly was tyrosine – a dopamine precursor that plays a key role in appetite regulation and energy balance [11]. Additionally, concentrations of tryptophan, tyrosine, and spermine were negatively correlated with screen time, further highlighting the importance of physical activity [11]. These mechanisms play an important role in obesity management. Another study evaluated the effects of inulin on the gut-muscle axis in children with obesity. The *in vivo* analysis showed a significant increase in IL-15 and the creatinine-to-cystatin C ratio in the inulin-supplemented group compared with the maltodextrin placebo. In an *in vitro* study investigating inflammatory and anti-inflammatory gene expression, significant reductions in TNF, IL-6, IL-1β and iNOS expression were observed, along with significant increases in the anti-inflammatory genes *FIZZ-1* and *TGF-β* in cultures treated with *Bifidobacterium* combined with inulin. Inulin supplementation significantly stimulated muscle-building biomarkers, corresponding to increased lean body mass in children with obesity. This effect results from the anti-inflammatory metabolites produced by *Bifidobacterium* during inulin digestion, which support muscle mass development through the gut-muscle axis [12].

PREBIOTIC – RESISTANT DEXTRIN

A study investigated the effect of a fruit-vegetable preparation enriched with resistant dextrin (RD) derived from potato starch on selected health indicators in overweight children. The intervention involved administering the fruit-vegetable preparation enriched with RD for 6 months to participants aged 6–10 years. The study group consisted of 42 children who received the RD-enriched preparation (10 g/day), while the control group, comprising 41 children, was given a fruit-vegetable preparation enriched with glucose. Participants were instructed to consume two packages of the preparation daily (100 g each, containing 5 g of RD), one in the morning and one in the evening. Administration of the fruit-vegetable preparation enriched with RD from potato starch increased the concentrations of lactic acid and SCFAs (formic, acetic, propionic, and butyric acids, except for valeric acid), while simultaneously decreasing the levels of branched-chain fatty acids (isobutyric and isovaleric acids) in overweight children. It also positively influenced the durability of the outcomes obtained during supplementation (for at least 3 months after discontinuation). A similar effect on the evaluated health markers was observed with the preparation without RD, although its effects were shorter-lasting and did not persist for 3 months after supplementation in overweight children. In addition, the inclusion of RD from potato starch in the fruit-vegetable preparation had a positive effect on fecal enzyme activity in overweight children and resulted in prolonged enhancement of α-glucosidase and α-galactosidase activity (3 months after the last intake), contributing to carbohydrate digestion [13]. The findings support the widespread use of these preparations in children with overweight or obesity.

PROBIOTICS

A study was conducted to assess the effects of two *Bifidobacterium breve* strains on children and adolescents with obesity over an 8-week period. The selection of *B. breve* strains was based on their anti-inflammatory properties, ability to colonize the human intestine

and protect the integrity of the intestinal epithelium, stimulate immune responses and compete with pathogens, including certain *Escherichia coli* strains. Eligible participants were 6–18 years old, diagnosed with obesity, with a HOMA-IR index > 2.5 or insulin levels > 15 μU/ml, and a pubertal stage ≥ 2. One group received supplementation with probiotic sachets containing *B. breve* B632 and *B. breve* BR03, while the other group received a placebo containing the same excipients but without *Bifidobacterium* strains [14]. Additionally, all participants received training on the Mediterranean diet (MD) and daily physical activity. Following the intervention, improvements in metabolic parameters, weight reduction and a decrease in *Escherichia coli* counts were observed in all participants. The researchers further noted that *B. breve* supplementation was positively associated with fasting insulin sensitivity and insulin response after oral glucose tolerance test. This represents one of the main goals in obesity management to maintain a healthy phenotype and reduce cardiovascular risk. It was shown that the overall abundance of SCFAs in stool remained stable, while levels of acetic acid and pentyl acetate increased in the placebo group, in contrast to the probiotic group [14]. The intervention was safe, well tolerated and effective, indicating the need for further studies on larger populations to confirm these findings.

SYNBIOTICS

In another study, the effects of *Lacticaseibacillus paracasei* K56, xylooligosaccharide (XOS), GOS, polydextrose (PD), and their synbiotic combinations (XOS + K56, GOS + K56, and PD + K56) on the gut microbiota of children with obesity were analyzed using an *in vitro* fermentation model. Stool samples from 14 children with obesity were collected for *in vitro* fermentation, and the effects of different treatments on gas production and short-chain fatty acid synthesis were assessed. The study demonstrated that the prebiotics GOS and XOS promoted the abundance of *Bifidobacterium*, and combining these prebiotics with *L. paracasei* K56 increased the abundance of *Limosilactobacillus* while decreasing *Escherichia/Shigella*, thereby increasing acetic acid production and reducing propionic and butyric acids, as well as CO_2, H_2, CH_4, and H_2S levels in the gut [15]. Similar results were obtained in studies investigating the impact of fructooligosaccharides on gut microbiome composition and metabolite production in children with obesity [16]. Acetic acid produced by the gut microbiota enters the circulation and crosses the blood-brain barrier, thereby influencing appetite and promoting weight loss. Although *in vitro* experiments may not fully replicate *in vivo* conditions, this study exemplifies the potential for personalized synbiotic programs, including K56-based prebiotics, to improve pediatric obesity [15]. Other researchers demonstrated that consumption of various fermented dairy products containing *Lacticaseibacillus casei* Shirota with inulin or fructans by children aged 6–10 modulates gut microbiome diversity. As a result, the lipid profile improved, specifically with an increase in high-density lipoprotein (HDL) cholesterol. Additionally, molecular-level changes were observed, including decreased expression of *FFAR2* and *ANGPTL4*, increased expression of *FFAR3* and elevated levels of propionate and butyrate [17]. These findings represent another promising therapeutic approach for managing overweight and obesity in children.

CHITOSAN

Another substance studied was chitosan, a polysaccharide produced from crustacean shells or fungal cell walls through deacetylation. As a potentially indigestible oligosaccharide for the host, it can be metabolized by the gut microbiota and influence its composition. A study was conducted to evaluate the effect of chitosan on gut microbiome changes in adolescents with overweight or obesity. The 12-week analysis included 61 participants, of whom 31 were in the intervention group receiving 3 g/day of chitosan, and 30 were in the placebo group, receiving maltodextrin. The study demonstrated a significant decrease in BMI *Z*-score in the chitosan group compared with the placebo group. Additionally, a significant reduction in *Firmicutes* populations and a significant increase in *Bacteroidetes* and *Akkermansia* populations were observed in adolescents consuming chitosan [18]. The firmicutes/bacteroidetes ratio also significantly decreased in the intervention group. These findings support the use of chitosan as an adjunct for modulating the gut microbiome and promoting weight loss in adolescents with overweight or obesity.

FECAL MICROBIOTA TRANSPLANTATION

Another method considered in the literature for treating obesity is fecal microbiota transplantation (FMT). Transplantation of gut microbiota from lean donors did not affect body weight in adolescents with obesity. However, a reduction in the android-to-gynoid fat ratio was observed, particularly in females, 6 weeks after FMT, which persisted for at least 26 weeks [19]. After a single FMT procedure, researchers demonstrated a sustained change in gut microbiota composition for up to 12- weeks, accompanied by increased microbial diversity in females 6 weeks post-FMT. Post hoc analyses among participants with metabolic syndrome at baseline revealed transient improvements in glucose metabolism and insulin sensitivity after 6 weeks, as well as resolution of metabolic syndrome in most participants 26 weeks after FMT [19]. Participants from this study were subsequently followed for 4 years, providing additional insights. Similar to the initial findings, no difference in BMI was observed

between groups after accounting for sex, age, diet, and physical activity. Nevertheless, clinical improvements in body composition and metabolic health were noted in FMT recipients compared with the placebo group. These improvements included reduced waist circumference, lower total body fat, decreased severity of metabolic syndrome and systemic inflammation and increased HDL cholesterol levels [20].

METFORMIN

One of the complications associated with obesity is type 2 diabetes. Metformin is well-known as an antihyperglycemic drug and as a medication that supports weight reduction [21]. The effect of metformin on the gut microbiome has been studied in the literature. However, data on children with obesity but without diabetes are limited. The first study analyzing the gut microbiota in children with obesity after metformin treatment was conducted by Pastor-Villaescusa *et al.* [22]. The researchers observed an interesting, although not statistically significant, change in *Actinobacteria* abundance, which increased 4.6–8.1% in the placebo group, compared with metformin, where *Actinobacteria* abundance decreased 3.8–2.6%. Additionally, a significant increase in the abundance of *Bacillus* species of 2.5–5.7% was observed in the placebo group, whereas in the metformin group, *Bacillus* abundance slightly decreased by 1.5–0.8%. In obesity, both *Actinobacteria* and *Bacillus* abundance generally increase [22]. Metformin may slightly attenuate the growth of these bacteria in children with obesity. However, further studies with long-term follow-up and larger sample sizes are required.

ANTIBIOTICS

Very interesting data regarding the protective role of the microbiota against environmental factors promoting obesity in children comes from a study conducted by Shelton *et al.* [23]. The researchers developed a mouse model in which young (3-week-old) mice were exposed to either a low-fat diet or a high-fat diet (HF) with or without low-dose penicillin (LDP) treatment. After 5 weeks, mice exposed to the HF diet and LDP gained significantly more weight and had increased visceral fat compared with mice fed only the HF diet, despite equal food intake. Subsequently, for an additional 5 weeks, mice were fed only the HF diet to determine whether metabolic dysfunction persisted after discontinuation of antibiotic treatment. The results were consistent with the first 5 weeks: mice initially exposed to HF + LDP, even after cessation of LDP for 5 weeks, continued to gain more weight and had higher visceral fat than mice maintained on the HF diet for 10 weeks [23]. This study demonstrated that a metabolite produced by *Lactobacillus*, phenylacetic acid (PLA), protects against metabolic disturbances induced by early exposure to antibiotics (penicillins being the most commonly used) and a HF diet. Phenylacetic acid increases the amount of peroxisome proliferator-activated receptor γ in small intestinal epithelial cells. Notably, despite the recovery of *Lactobacillus* levels in mice where penicillin was discontinued after 5 weeks, their weight gain persisted. This indicates that even short-term microbiota disturbances can lead to long-lasting metabolic changes. Similarly, decreases in *Bifidobacteriaceae* and *Peptostreptococcaceae* were observed, suggesting that multiple bacterial species may contribute to PLA production in the small intestine. Furthermore, PLA is present in the feces of infants, indicating that this metabolite activates protective pathways in the small intestinal epithelium, preventing antibiotic-associated obesity during early life in humans [23].

DIET

A study was conducted to evaluate the effects of a 10-week, high-fiber dietary intervention during the summer months, targeting low-income caregiver-child dyads. The intervention resulted in improved overall diet quality, increased consumption of whole-grain products, reduced energy intake, and enhanced fecal excretion of the microbial metabolite lithocholic acid (LCA) in the lifestyle intervention group compared with the control group. Moreover, despite no changes in dietary cholesterol intake, the intervention increased cholesterol excretion in caregivers. Among children in the high-fiber diet group, excretion of total conjugated bile acids was increased. The analyzed microbiomes were highly personalized, strongly correlated within dyads and modified by the lifestyle intervention. Taxa associated with overall diet quality, whole-grain intake, and LCA excretion were identified, including well-known fiber-degrading bacteria such as *Subdoligranulum* and bile acid-metabolizing organisms such as *Bifidobacterium*, through differential microbiome composition modeling. Importantly, associations between the microbiota and blood pressure, a cardiovascular risk marker associated with obesity, were shared within caregiver-child pairs, suggesting similar responses to the lifestyle intervention. A key aspect of this study was the inclusion of family members, who most often shop, prepare meals, and shape and control children's eating behaviors [24]. Other researchers have investigated different types of diets, such as a calorie-restricted diet for treating obesity in children. Results showed that this diet enabled weight loss without affecting muscle nutritional status, which is particularly important for school-aged children with obesity who are still growing and developing. Furthermore, the intervention altered the gut microbiota of obese children by promoting beneficial gut microorganisms, especially *Bifidobacterium* and *Blautia*, which are capable of produc-

ing SCFAs and thereby improving metabolism [25]. The effects of the MD on gut microbiota and the immune system have also been studied. The Mediterranean diet, rich in polyunsaturated fatty acids, dietary fiber, polyphenols, vitamins, and trace elements, can mitigate inflammation and gut microbiome dysbiosis by achieving a proper Th17/Treg balance in the host and ensuring high microbial diversity [26].

PHYSICAL ACTIVITY

Another important factor influencing the gut microbiota, alongside diet, is physical activity. Researchers analyzed the effects of a 6-week training program combined with dietary restriction on central hemodynamics in relation to altered gut microbiota in adolescents with obesity. The study included 24 participants aged 9–16 years, who followed a calorie-restricted but nutritionally complete diet. Additionally, they performed an intensive exercise program lasting 5 hours per day, 6 days a week, with sessions scheduled at 8:00–9:30, 10:00– 11:30 and 15:00–17:00 [27]. The study found that the average number of observed gut bacterial species significantly increased after the intervention. Exercise and diet significantly improved central hemodynamic parameters, including subendocardial viability ratio and resting heart rate, which were correlated with changes in the gut microbiota of adolescents with obesity. Results showed that the abundance of *Lactobacillales*, *Bacilli*, *Streptococcaceae*, *Streptococcus* and *Veillonella*, all belonging to the phylum Firmicutes, was significantly reduced after the intervention. Another study demonstrated an association between the abundance of *Lactobacillus*, *Streptococcus*, and *Veillonella* and coronary artery disease in obese patients [27]. Therefore, exercise and diet can modulate gut microbiota diversity and abundance, which may be utilized in the treatment of patients with cardiovascular disease and obesity.

CONCLUSIONS

Childhood obesity is associated with changes in the gut microbiome. This article presents various strategies for modulating the gut microbiota. Probiotics, prebiotics, synbiotics, fecal microbiota transplantation, metformin, dietary interventions and physical activity were discussed. These approaches show promising results in the management of obesity in children. However, further long-term studies with larger sample sizes are needed to definitively confirm their role in gut microbiota modulation in pediatric populations.

DISCLOSURES

1. Institutional review board statement: None.
2. Assistance with the article: None.
3. Financial support and sponsorship: None.
4. Conflicts of interest: None.

REFERENCES

1. United Nations Children's Fund (UNICEF), Feeding Profit. How food environments are failing children. Child Nutrition Report 2025, UNICEF, New York 2025.
2. Marco ML, Sanders ME, Gänzle M, Arrieta MC, Cotter PD, de Vuyst L, et al. The International Scientific Association for Probiotics and Prebiotics (ISAPP) consensus statement on fermented foods. Nat Rev Gastroenterol Hepatol 2021; 18: 196-208.
3. Luzzi A, Briata IM, Di Napoli I, Giugliano S, Di Sabatino A, Rescigno M, et al. Prebiotics, probiotics, synbiotics and postbiotics to adolescents in metabolic syndrome. Clin Nutr 2024; 43: 1433-1446.
4. Swanson KS, Gibson GR, Hutkins R, Reimer RA, Reid G, Verbeke K, et al. The International Scientific Association for Probiotics and Prebiotics (ISAPP) consensus statement on the definition and scope of synbiotics. Nat Rev Gastroenterol Hepatol 2020; 17: 687-701.
5. Salminen S, Collado MC, Endo A, Hill C, Lebeer S, Quigley EMM, et al. The International Scientific Association of Probiotics and Prebiotics (ISAPP) consensus statement on the definition and scope of postbiotics. Nat Rev Gastroenterol Hepatol 2021; 18: 649-667.
6. Forcina G, Di Filippo P, De Biasio D, Cesaro FG, Frattolillo V, Massa A, et al. Targeting the gut microbiota in pediatric obesity: a paradigm shift in prevention and treatment? A comprehensive review. Nutrients 2025; 17: 2942.
7. Coppola S, Nocerino R, Paparo L, Bedogni G, Calignano A, Di Scala C, et al. Therapeutic effects of butyrate on pediatric obesity: a randomized clinical trial. JAMA Netw Open 2022; 5: e2244912.
8. Jaimes JD, Slavíčková A, Hurych J, Cinek O, Nichols B, Vodolánová L, et al. Stool metabolome-microbiota evaluation among children and adolescents with obesity, overweight, and normal-weight using 1H NMR and 16S rRNA gene profiling. PLoS One 2021; 16: e0247378.
9. Wei Y, Liang J, Su Y, Wang J, Amakye WK, Pan J, et al. The associations of the gut microbiome composition and short-chain fatty acid concentrations with body fat distribution in children. Clin Nutr 2021; 40: 3379-3390.
10. Visuthranukul C, Sriswasdi S, Tepaamorndech S, Chamni S, Leelahavanichkul A, Joyjinda Y, et al. Enhancing gut microbiota and microbial function with inulin supplementation in children with obesity. Int J Obes (Lond) 2024; 48: 1696-1704.
11. Andriyas T, Sriswasdi S, Tansawat R, Uaariyapanichkul J, Chomtho S, Visuthranukul C. Inulin supplementation modulates gut microbiota derived metabolites related to brain function in children with obesity. Sci Rep 2025; 15: 34843.
12. Visuthranukul C, Leelahavanichkul A, Tepaamorndech S, Chamni S, Mekangkul E, Chomtho S. Inulin supplementation exhibits increased muscle mass via gut-muscle axis in children with obesity: double evidence from clinical and in vitro studies. Sci Rep 2024; 14: 11181.
13. Śliżewska K, Włodarczyk M, Barczyńska R, Kapuśniak J, Socha P, Wierzbicka-Rucińska A, et al. Impact of a fruit-vegetable preparation fortified with potato starch resistant dextrin on selected health indicators in overweight children. Nutrients 2024; 16: 2321.
14. Solito A, Bozzi Cionci N, Calgaro M, Caputo M, Vannini L, Hasballa I, et al. Supplementation with Bifidobacterium breve BR03 and B632 strains improved insulin sensitivity in children and adolescents with obesity in a cross-over, randomized double-blind placebo-controlled trial. Clin Nutr 2021; 40: 4585-4594.
15. Zhang P, Dong X, Zeng Y, Chen J, Yang S, Yu P, et al. Synbiotic effects of Lacticaseibacillus paracasei K56 and prebiotics on the in-

testinal microecology of children with obesity. Probiotic Antimicrob Protein 2024; 17:4525-4535.
16. Mo C, Zhou S, Du Z, Huang X. Impact of fructooligosaccharides on gut microbiota composition and metabolite production: implications for childhood obesity. Peer J 2025; 13: e19894.
17. Vega-Cárdenas M, Martínez-Gutierrez F, Lara-Ramírez EE, Reynaga-Hernandez E, Yañez-Estrada L, Ratering S, et al. Agave fructans enhance the effects of fermented milk products on obesity biomarkers: a randomised trial. Benef Microbes 2023; 14: 153-164.
18. Fatahi S, Sohouli MH, Vahidshahi K, Rohani P, Safa M, Salehi M, et al. Changes in gut microbiota following supplementation with chitosan in adolescents with overweight or obesity: a randomized, double-blind clinical trial. Diabetol Metab Syndr 2025; 17: 120.
19. Leong KSW, Jayasinghe TN, Wilson BC, Derraik JGB, Albert BB, Chiavaroli V, et al. Effects of fecal microbiome transfer in adolescents with obesity: the gut bugs randomized controlled trial. JAMA Netw Open 2020; 3: e2030415.
20. Wilson BC, Zuppi M, Derraik JGB, Albert BB, Tweedie-Cullen RY, Leong KSW, et al. Long-term health outcomes in adolescents with obesity treated with faecal microbiota transplantation: 4-year follow-up. Nat Commun 2025; 16: 7786.
21. Carreras-Badosa G, Gómez-Vilarrubla A, Mas-Parés B, Martínez-Calcerrada JM, Xargay-Torrent S, Prats-Puig A, et al. A 24-month metformin treatment study of children with obesity: Changes in circulating GDF-15 and associations with changes in body weight and visceral fat. Pediatr Obes 2022; 17: e12845.
22. Pastor-Villaescusa B, Plaza-Díaz J, Egea-Zorrilla A, Leis R, Bueno G, Hoyos R, et al. Evaluation of the gut microbiota after metformin intervention in children with obesity: a metagenomic study of a randomized controlled trial. Biomed Pharmacother 2021; 134: 111117.
23. Shelton CD, Sing E, Mo J, Shealy NG, Yoo W, Thomas J, et al. An early-life microbiota metabolite protects against obesity by regulating intestinal lipid metabolism. Cell Host Microbe 2023; 31: 1604-1619.e10.
24. Hill EB, Chen L, Bailey MT, Singh Khalsa A, Maltz R, Kelleher K, et al. Facilitating a high-quality dietary pattern induces shared microbial responses linking diet quality, blood pressure, and microbial sterol metabolism in caregiver-child dyads. Gut Microbes 2022; 14: 2150502.
25. Zhou M, Peng C, Miao Z, Wang K, Zhou H, Li Y, et al. Improved diet-based nutritional interventions can improve childhood obesity with the synergistic regulation of gut microbiota. Benef Microbes 2024; 15: 495-513.
26. García-Montero C, Fraile-Martínez O, Gómez-Lahoz AM, Pekarek L, Castellanos AJ, Noguerales-Fraguas F, et al. Nutritional components in western diet versus Mediterranean diet at the gut microbiota-immune system interplay. Implications for health and disease. Nutrients 2021; 13: 699.
27. Huang J, Liao J, Fang Y, Deng H, Yin H, Shen B, et al. Six-week exercise training with dietary restriction improves central hemodynamics associated with altered gut microbiota in adolescents with obesity. Front Endocrinol (Lausanne) 2020; 11: 569085.