Gut Microbiota: A Key Player in Childhood Obesity (Postprint)

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Date: 2024-02-19T16:56:38+00:00

Abstract

The incidence of childhood obesity is increasing annually, posing a new challenge in the global public health domain. Research demonstrates that alterations in early-life gut microbiota can promote the development of childhood obesity through mechanisms including modulation of host nutrient absorption and metabolism, induction of inflammatory responses, and regulation of the gut-brain axis. Currently, Bifidobacterium and Akkermansia muciniphila have been identified to reduce body fat content, exhibit anti-inflammatory properties, and enhance intestinal barrier function, while Prevotella is closely associated with dietary fiber-induced improvements in individual glucose metabolism. Translational applications targeting specific gut microbiota that improve host glucose and lipid metabolism will facilitate the early prevention and management of childhood obesity. This review primarily addresses the influence of compositional changes in early-life gut microbiota on childhood obesity and the mechanisms underlying microbiota involvement in obesity pathogenesis, with particular emphasis on recent advances in short-chain fatty acids for modulating gut microbiota and ameliorating obesity, aiming to provide a theoretical foundation for intervening in childhood obesity development from a gut microbiota perspective.

Full Text

Intestinal Flora: An Important Participant in Childhood Obesity

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Abstract

The increasing incidence of childhood obesity has emerged as a novel challenge in global public health. Studies have demonstrated that alterations in intestinal flora composition during early life contribute to obesity development by influencing nutrient absorption and metabolism, triggering inflammatory responses, and regulating gut-brain axis communication. Currently, *Bifidobacterium* and *Akkermansia muciniphila* have been found to reduce body fat content, exhibit anti-inflammatory properties, and enhance intestinal barrier function, whereas *Prevotella* is strongly associated with improvements in glucose metabolism induced by dietary fiber. Translational application of specific intestinal flora that benefits glycolipid metabolism may facilitate early prevention and treatment of pediatric obesity.

This review elucidates the impact of early-life changes in intestinal flora composition on childhood obesity, explores the mechanisms by which intestinal flora contributes to obesity pathogenesis, and specifically focuses on recent advances in utilizing short-chain fatty acids (SCFAs) to regulate intestinal flora and ameliorate obesity. Our aim is to provide a theoretical foundation for intervening in childhood obesity from the perspective of intestinal flora.

Keywords: Intestinal flora; Pediatric obesity; Mechanism; Short-chain fatty acids

Funding: Sichuan Provincial People's Hospital Special Fund (2021ZX04); Open Project of Key Laboratory of Birth Defects and Related Diseases of Women and Children, Ministry of Education, Sichuan University (2020KF04)

1. Literature Search Strategy

We conducted computerized searches of PubMed, Web of Science, and CNKI (China National Knowledge Infrastructure) databases from inception to August 2023. Chinese search terms included "肠道菌群" (intestinal flora), "儿童肥胖" (childhood obesity), "短链脂肪酸" (short-chain fatty acids), and "炎症" (inflammation). English search terms included "Intestinal flora," "Childhood obesity," "Short-chain fatty acids," and "Inflammation." Inclusion criteria comprised literature addressing the relationship between intestinal flora and childhood obesity and the mechanisms by which intestinal flora promotes childhood obesity. Exclusion criteria comprised irrelevant content, poor quality, and unavailable full text. A total of 70 articles were ultimately included.

2. Gut Microecology and Childhood Obesity

Childhood obesity is not merely an independent disease but also a crucial risk factor for type 2 diabetes mellitus (T2DM), metabolic syndrome (MS), cardiovascular disease, nonalcoholic fatty liver disease (NAFLD), and other conditions. Multiple studies have demonstrated that obesity development is closely related to gut microecology. The human gut harbors the most complex and abundant microecosystem, comprising intestinal tissues, cells, approximately 100 trillion bacteria, and their metabolites. Through the Human Gut Metagenome Project and Human Microbiome Project, scientists have isolated 2,172 bacterial species from the human gut, including 386 obligate anaerobes. These bacteria can be classified into 12 phyla, with Proteobacteria, Firmicutes, Actinobacteria, and Bacteroidetes accounting for 93.5% of the total. The gut microbiota maintains various physiological homeostatic functions, including fermenting indigestible dietary compounds, participating in cholesterol and bile acid transformation, and indirectly regulating emotions and social behavior through the gut-brain axis. When the quantity or composition of gut bacteria changes, this homeostasis is disrupted, leading to inflammatory bowel disease, gastrointestinal cancers, and other diseases.

By age 1, an infant' s gut microbiota diversity begins transitioning toward an adult-like profile, and by 2.5 to 3 years, the composition largely resembles that of adults. Multiple studies have found that obese children and adults exhibit increased Firmicutes and decreased Bacteroidetes. However, some analyses have reported decreased Firmicutes and *Bifidobacterium* with unchanged Bacteroidetes in obese individuals. Consequently, some researchers argue that Firmicutes may serve as a more effective obesity predictor than Bacteroidetes, playing a more important role in promoting energy storage and weight gain. International consensus on the pattern of gut microbiota changes in obese children remains elusive. Previous studies have shown that the Firmicutes/Bacteroidetes ratio correlates closely with obesity in animals and adults, that obese gut microbiota is characterized by low diversity, and that obesity progression is closely associated with reduced *Bifidobacterium* and *Akkermansia* abundance. These findings suggest that identifying characteristic compositional or quantitative changes in gut microbiota could help predict obesity status. Based on this, researchers at the Mayo Clinic proposed the novel concept of the "Gut Microbiome Health Index (GMHI)," a species-level taxonomic profile based on fecal metagenomic samples that compares relative abundances of relevant microbial species between healthy subjects and non-healthy subjects (e.g., those with colon cancer, diabetes, heart disease, or obesity). This method identified 50 specific microbial species that can define healthy microbiome characteristics. Independent of traditional clinical diagnosis, GMHI holds promise as a new concept for defining obesity.

3. Factors Influencing Gut Microbiota in Childhood Obesity

3.1 Early-Life Factors Gut microbiota establishment begins in utero or after birth and continues until stabilization at 3–4 years of age. Disruption during this critical window affects weight gain and obesity development at 12–14 years. The perinatal period and first year of life represent a golden window for microbiota colonization, with numerous factors influencing microbial abundance and diversity, including intrauterine microbial exposure, delivery mode, feeding patterns, and antibiotic use.

3.1.1 Intrauterine Microbial Exposure: While healthy fetuses were previously considered sterile, current evidence suggests bacteria may exist in the placenta, amniotic fluid, and fetal membranes. Das et al. found that maternal gut microbes can transfer to the fetus, with bacteria detectable in meconium, supporting the possibility of a placental microbiome. Some researchers have noted similarities between human placental microbiota and oral microbiota, suggesting placental microbes may originate from translocated oral bacteria. However, Goffau et al., through whole-genome sequencing of 537 placental samples, found no microbial colonization at the maternal-fetal interface during healthy pregnancy, concluding that the placenta cannot serve as a primary channel for infant microbiome development. Another study analyzing 76 placentas from term deliveries also suggested that observed microbes likely represented contamination rather than true placental microbiota. While understanding of the placental microbiome remains limited, premature intrauterine microbial exposure clearly jeopardizes fetal development and pregnancy outcomes, increasing risks of chorioamnionitis, fetal malformations, and even miscarriage.

Maternal gut microbiota may also influence offspring metabolic health. Conventionally raised mouse offspring show rapid weight gain with high-fat diets, accompanied by increased body fat and hyperlipidemia. However, when pregnant mice receive dietary fiber supplementation, their offspring are protected from obesity even when fed high-fat diets, possibly because high-fiber diets promote maternal gut bacteria to produce propionate that enters the embryo via maternal blood. While animal studies suggest maternal fiber supplementation may protect against metabolic diseases, whether this applies to humans requires further validation. Maternal perinatal antibiotic use also affects vaginal lactobacilli growth and colonization of dominant infant microbiota. Not all vaginal lactobacilli benefit pregnancy outcomes; for example, *Lactobacillus iners* is a risk factor for preterm birth, whereas *Lactobacillus crispatus* is protective.

3.1.2 Delivery Mode: Delivery mode is a key factor influencing vertical transmission of gut microbiota. Vaginally delivered infants harbor neonatal gut microbiota resembling maternal vaginal and skin microbiota, dominated by lactobacilli, followed by *Prevotella*. By days 4–7, bifidobacteria become dominant with the highest relative abundance. Cesarean-delivered neonates acquire microbiota from maternal skin and hospital environmental bacteria, dominated by staphylococci, followed by propionibacteria and corynebacteria. Lee et al. found

cesarean-born neonates had lower abundance and diversity of Bacteroidetes but higher Firmicutes diversity. Low Bacteroidetes abundance and diversity have been linked to allergic diseases. Cesarean-related microbiota differences may also relate to pregnancy complications, perinatal antibiotic use, and delivery environment disinfection.

A 16-year cohort study showed cesarean-delivered offspring had significantly higher obesity risk than vaginally delivered offspring. Women with prior cesarean deliveries who subsequently delivered vaginally had 31% lower obesity risk in offspring compared to those with repeat cesareans. However, some studies find no significant relationship: Riva et al. reported no statistically significant difference in weight between cesarean and vaginally delivered children. Overall, multiple systematic reviews and meta-analyses indicate cesarean-delivered offspring have higher obesity risk from childhood through adulthood. China' s current cesarean rate of 45% far exceeds the global average of 21.1%, making it one of the highest worldwide. Reducing unnecessary cesarean deliveries may help lower childhood obesity incidence.

3.1.3 Feeding Patterns: Human milk oligosaccharides in breast milk selectively shape beneficial microbiota and improve microbial balance. Formula milk lacks bacteriophages, carbohydrates, and immune factors that shape infant gastrointestinal microbiota. Breastfed infants have gut microbiota dominated by bifidobacteria, a hallmark of healthy infant microbiota that improves glucose tolerance and reduces low-grade intestinal inflammation. Formula-fed infants show decreased bifidobacteria and increased proportions of *Escherichia coli*, *Clostridium*, and Bacteroidetes. While formula cannot replace breast milk, selecting formulas fortified with key breast milk components benefits infant health. Before solid food introduction, infants already possess the capacity to digest plant polysaccharides. Laursen et al. found that with weaning and solid food introduction, infants gradually develop adult-like gut microbiota, characterized by increasing colonization of Bacteroidetes, *Clostridium*, and *Akkermansia muciniphila* (Akk) with age. In breastfed infants receiving formula supplementation, increased Firmicutes proportions may represent one mechanism by which early feeding transitions promote obesity.

3.1.4 Antibiotic Use: The 0–6 month period represents a window of heightened sensitivity to antibiotic exposure. European multicenter studies found infant antibiotic use increased early childhood obesity risk, particularly in boys. Gu et al. evaluated short-term effects of fluoroquinolone and β-lactam antibiotics on mouse gut microbiota using 16S rRNA gene sequencing, showing that just 4 days of antibiotic exposure significantly reduced both α- and β-diversity. The relationship between early antibiotic exposure and childhood obesity remains controversial. Some researchers argue early antibiotic exposure has cumulative effects on obesity, depending on frequency and duration. However, a randomized trial of 607 children found no association between early antibiotic exposure and overweight/obesity, suggesting retrospective studies only assessed effects of incidental antibiotic use and existing evidence is limited to observa-

tional studies with low evidence grades. Overall, child development is continuous and irreversible, warranting attention to potential obesity effects following antibiotic exposure during critical early developmental windows. Studying relationships between antibiotics, obesity-related gut microbiota changes, and underlying mechanisms can provide theoretical foundations for more cautious antibiotic use in early life.

3.2 Diet and Behavior Dietary habits and patterns may influence gut microbiota more than genetic factors, promoting overgrowth of bacteria that prefer specific dietary components. High-fat diets represent a primary cause of childhood nutritional obesity. Nakayama et al. found that Western diets characterized by high fat and sugar reduced *Prevotella* and promoted childhood obesity, while carbohydrate-based diets increased *Prevotella* and decreased Bacteroidetes. *Prevotella* is often considered a "probiotic" associated with healthy vegetarian diets, capable of degrading non-cellulosic polysaccharides and pectin. Vegetarians have more *Prevotella* and higher *Prevotella/Bacteroides* ratios than non-vegetarians, associated with improved glucose metabolism induced by dietary fiber. High-fat or high-carbohydrate diets can activate microglia via the gut-brain axis to release interleukin (IL)-1β, IL-6, and tumor necrosis factor-α (TNF-α), triggering inflammatory responses in the hypothalamic base that cause central leptin resistance, promoting insulin resistance and T2DM development. Such diets also activate protease receptors in intestinal epithelial cells, downregulating tight junction proteins ZO-1 and occludin, damaging the intestinal mechanical barrier, increasing intestinal permeability, and causing inflammatory bowel disease.

High-fat diets disrupt the brain' s satiety-control clock, reducing neural stem cell rhythmic activity and increasing daytime food intake, ultimately causing overeating and obesity. Some preservatives, emulsifiers, and artificial sweeteners increase childhood obesity risk by altering gut microbiota diversity. Propionic acid and its sodium/calcium salts (organic acid preservatives) may act as "metabolic disruptors," increasing T2DM and obesity risk. Gut microbiota composition before age 3 also correlates with infant behavior. Bacteria on breast skin may directly enter the infant gut during breastfeeding. Pannaraj et al. analyzed breast milk, areolar skin, and infant fecal samples from 107 healthy mother-infant pairs, finding that infants consuming >75% breast milk daily obtained 27.7% of their gastrointestinal bacteria from breast milk and 10.3% from areolar skin. Infant behaviors like thumb-sucking, crawling, walking, and hand contact with floors or objects increase bacterial exposure and infection opportunities compared to adults. Sedentary lifestyles and staying up late also promote obesity. Therefore, reducing high-fat diets, sugary beverages, and increasing physical exercise are effective obesity prevention measures.

3.3 Geographic Factors The Guangdong Gut Microbiome Project in 2018 found through data mining that regional factors influence microbiota more significantly than age, disease, lifestyle, or other factors, representing a major source

of variation in human gut microbiota. Geographic location effects on microbiota composition likely stem from lifestyle and dietary culture differences. European infants show a "geographic gradient," with Nordic infants (Denmark, Sweden, UK) having higher bifidobacteria and *Clostridium* abundance, while Southern European infants (Spain, Portugal) have higher lactobacilli and Bacteroides. Compared to Italian children, 1–6-year-old children in rural Burkina Faso have significantly reduced Firmicutes but abundant *Prevotella* and xylanolytic bacteria, likely shaped by their polysaccharide-rich diet that maximizes energy extraction from plant fiber while protecting against intestinal inflammation and non-communicable colon diseases. Infants in developed countries like the US have lower Firmicutes and Actinobacteria than African infants. Developed regions with better healthcare and facilities provide more favorable environments for beneficial bacterial colonization in neonates.

4. Mechanisms of Gut Microbiota in Childhood Obesity

4.1 Influencing Energy Absorption Gut bacteria and their metabolites play unique and complex roles in regulating glucose-lipid metabolism and energy balance. In healthy states, Bacteroidetes degrade complex carbohydrates and plant fiber, facilitating absorption of monosaccharides and nutrients. Liu et al. demonstrated through mouse gavage experiments that *Bacteroides thetaiotaomicron* reduces serum glutamate concentrations and increases adipocyte lipolysis and fatty acid oxidation, thereby reducing fat accumulation. However, previous animal studies showed that germ-free mice colonized with *B. thetaiotaomicron* from conventional mice exhibited 57% increased body fat after 2 weeks, as the bacterium promoted expression of genes degrading and absorbing dietary lipids, causing excessive intestinal fat absorption. *Bacteroides* thus exhibits both probiotic mechanisms and pathogenic potential, maintaining a complex and delicate relationship with the host like other gut bacteria, requiring a dialectical perspective on their role in children.

Glycoside hydrolases for digesting plant polysaccharides are normally limited in humans, but anaerobes like *Lactobacillus* and *Ruminococcus* synthesize these enzymes abundantly to degrade dietary fiber, producing short-chain fatty acids (SCFAs) including butyrate, acetate, and propionate in the proximal colon. Butyrate-producing bacteria and butyrate itself are considered beneficial. The health effects of SCFAs depend on metabolite concentration and organ location. Butyrate serves as the primary energy source for colonocytes, while other SCFAs enter the portal circulation to the liver, where acetate participates in fatty acid and cholesterol synthesis and propionate serves as a gluconeogenesis substrate. SCFAs stimulate intestinal epithelial growth, promote nutrient absorption, and regulate peptide tyrosine tyrosine (PYY) and glucagon-like peptide-1 (GLP-1) via free fatty acid receptors, forming a negative feedback loop for energy regulation that suppresses appetite. SCFAs also act on G-protein coupled receptors 41 and 43 (GPR41, GPR43) to inhibit hepatic fat accumulation, accelerate catabolism of unbound lipids and glucose in extrahepatic tissues, and

improve insulin sensitivity. Recent research indicates obese individuals have significantly reduced total SCFA production with elevated propionate proportions, and propionate can induce specific DNA methylation (cg26345888 locus) predisposing obese individuals to T2DM. Rapid colonic infusion of acetate or SCFA mixtures in overweight/obese individuals increases fasting lipid oxidation rates and resting energy expenditure. SCFA production is significantly influenced by host commensal bacterial ratios; disrupting commensal bacteria causes SCFA proportion imbalances, accelerating obesity and MS development.

4.2 Altering Metabolic Pathways Gut microbiota metabolites enter systemic circulation through intestinal absorption, enterohepatic circulation, or altered intestinal permeability. As major metabolites, SCFAs significantly affect adipose tissue metabolism. Acetate inhibits β-adrenergic receptor-mediated lipolysis in adipocytes, possibly through G-protein coupled receptor-dependent inhibition of hormone-sensitive lipase phosphorylation. Animal models demonstrate that exogenous SCFA supplementation inhibits cholesterol synthesis and reduces hepatic fat accumulation, potentially via AMPK-acetyl CoA carboxylase pathways that increase hepatic lipid oxidation and decrease fatty acid synthase activity.

During dysbiosis, metabolites like lipopolysaccharide (LPS) and succinate increase, causing metabolic disturbances that promote obesity. LPS, a typical Gram-negative bacterial outer membrane glycolipid, not only triggers inflammation but also promotes insulin resistance, contributing to T2DM and NAFLD. Succinate, an intermediate in bacterial propionate synthesis, reduces lipid catabolism when elevated, causing lipid accumulation and obesity. Vadder et al. found that adding fructooligosaccharides (prebiotics) activated intestinal gluconeogenesis and improved obesity outcomes in high-fat diet-fed mice.

Bile acids are determinants of gut microbiota abundance, diversity, and metabolic activity, while gut microbiota also metabolizes bile acids, affecting lipid metabolism and intestinal health. During development, increased primary bile acid concentrations enrich bacteria expressing bile acid metabolism genes in the neonatal small intestine. Secondary bile acids produced by microbial metabolism have complex functions, including enterohepatic circulation and lipid emulsification. Bile acid synthesis is negatively regulated by the farnesoid X receptor (FXR), which enhances expression of intestinal protection genes and inhibits bacterial overgrowth and mucosal damage. Evans' team found high-fat diet-fed mice had increased total and secondary bile acids accompanied by increased intestinal permeability, related to FXR-mediated ZO-1 protein reduction and altered cecal and plasma bile acid concentrations. Collins et al. noted that in MS patients, reduced Firmicutes and secondary bile acid depletion correlate with decreased insulin sensitivity, while microbiota-dependent secondary bile acid increases associate with NAFLD. Besides metabolites, reduced Akk abundance correlates with T2DM in obese mice; supplementing Akk or its outer membrane protein Amuc_{1100} prevents high-fat diet-induced MS

and improves glucose tolerance. However, excessive Akk supplementation may over-degrade intestinal mucus, increasing leaky gut and allergic disease risks.

4.3 Inducing Inflammatory Responses Persistent low-grade systemic inflammation from immune responses to LPS characterizes obesity. Obese individuals have increased plasma inflammatory factors including TNF-α, IL-6, and adiponectin, confirming subclinical inflammatory states that also relate to NAFLD and T2DM pathogenesis. High-fat diets are a significant contributor to microbiota-induced intestinal inflammation. Schertzer et al. fed high-fat diets to mice lacking NOD1/2 genes and found that diet-induced microbiota dysbiosis activated NOD proteins, promoting NOD-like receptor inflammasome formation and macrophage release of inflammatory factor TNF-α, accelerating insulin resistance in obese mice, though this may also involve innate immunity. A randomized controlled trial of dietary patterns in obese individuals found that Mediterranean diets increased *Roseburia* and *Oscillospira* abundance, while complex carbohydrate-rich diets increased *Prevotella* and *Faecalibacterium*; both diets improved insulin sensitivity. Probiotic supplementation (*Bifidobacterium*, *Lactobacillus*) in high-fat diet-fed rats reduced plasma LPS and IL-1β, decreased inflammatory indices, and improved insulin sensitivity and obesity. Probiotic and prebiotic supplementation to improve inflammatory status and promote intestinal health may be an effective intervention strategy. However, each individual' s microbiome is unique, and believing that mass-produced probiotics will function identically in everyone is incorrect. Given the regional and individualized nature of microbiomes, concepts like microbiome information databases and personalized microbiome therapy have been proposed, with future hopes to build a comprehensive, sensitive microbial marker database to identify microbiome diversity and distribution patterns for early obesity detection and intervention. However, microbiome sequencing is labor-intensive and limited by volunteer numbers and funding, currently remaining in the platform development stage.

4.4 Gut-Brain Axis The gut-brain axis is a complex neurohumoral communication system with bidirectional regulation between the gut and brain, comprising the central nervous system, autonomic nervous system, enteric nervous system, and hypothalamic-pituitary-adrenal (HPA) axis. It regulates immunity, inflammation, and stress responses, serving as a crucial signaling axis for metabolic balance. Various neuroactive factors produced by gut microbiota play key roles in emotional regulation. In 2017, gut microbiota-induced changes in emotion and social behavior were first confirmed. Brain MRI studies in two groups of women revealed that those with *Bacteroides*-rich microbiota had lower hippocampal activity and more negative emotions like anxiety, distress, and irritability compared to those with *Prevotella*-rich microbiota. Reissland et al. found that maternal depression and anxiety during pregnancy led to low birth weight newborns, and combined with recent host-microbe studies in insulin resistance, suggested this relates to reduced secretion of various hormones

and neuromediators including insulin-like growth factors mediated by gut microbiota such as Lachnospiraceae. These findings lay the foundation for further exploring mechanisms by which gut microbiota influences neurological function.

SCFAs can directly regulate the sympathetic nervous system via free fatty acid receptors, participating in glucose homeostasis and hunger-satiety hormone balance. Maternal microbiota-derived SCFAs also affect fetal intestinal, pancreatic, and neural development through GPR43 and GPR41. In the central nervous system, SCFAs promote microglial maturation and differentiation into other glial cell types. Shulman' s team found acetate is a key obesity factor: large amounts of acetate produced by microbial fermentation are absorbed into blood, cross the blood-brain barrier to activate the parasympathetic nervous system, stimulate insulin secretion, and activate cellular energy storage programs. Simultaneously, the parasympathetic system promotes ghrelin release from the stomach, increasing hunger and food intake. Over time, this energy imbalance causes obesity. However, Canfora et al. found that intraperitoneal SCFA injection inhibited energy intake in mice via vagal afferent stimulation.

Gut microbiota produces various neuroactive molecules essential for regulating gut function, including 5-hydroxytryptamine (5-HT), gamma-aminobutyric acid (GABA), acetylcholine, and catecholamines. Multiple studies show 5-HT synthesis, progression, and termination are primarily regulated by gut microbiota. Yano et al. found that SCFA-induced 5-HT synthesis influences host food choices during eating, promoting selection of microbiota-preferred foods that reinforce unhealthy dietary patterns and exacerbate dysbiosis. Bhattarai et al. found that dysbiosis downregulates 5-HT receptor expression and inhibits 5-HT secretion, reducing 5-HT' s regulatory effect on the gut-brain axis tryptophan transduction pathway and causing insulin resistance. GABA, a typical inhibitory neurotransmitter in the central nervous system, stimulates feeding, increases energy accumulation, and induces obesity. Chronic or acute stress also activates the HPA axis, increases intestinal wall permeability, and promotes intestinal inflammation. Additionally, cholinergic anti-inflammatory signaling via the vagus nerve helps alleviate LPS-induced systemic low-grade inflammation. When vagal signaling is dysfunctional, metabolites and inflammatory factors can cause intestinal diseases like inflammatory bowel disease and irritable bowel syndrome through bidirectional gut-brain axis regulation. The brain-gut-microbiome axis concept is established, with evidence showing that obesity, T2DM, chronic inflammation, and related metabolic diseases partly result from imbalanced host-microbe or metabolite interactions. However, pathways and mechanisms among the brain, gut microbiota, and peripheral target organs remain incompletely understood, necessitating accelerated mapping of gut microbiome gene profiles to clarify the gut-brain axis role in childhood obesity.

Conclusion and Future Directions

Gut microbiota clearly plays a crucial role in childhood obesity development and represents a promising therapeutic target. Gut microbiota research will

provide more theoretical foundations for obesity prevention and treatment, while individualized microbiota interventions will offer new strategies for managing childhood obesity. Current research focuses primarily on preschool children and uses cross-sectional designs. Future studies must further identify obesity susceptibility factors, clarify microbiome establishment and influencing factors, and elucidate molecular mechanisms linking microbiota to obesity. Building a sensitive, early obesity-detection microbial marker database will fill current gaps in understanding the gut microbiota-childhood obesity relationship and open new avenues for obesity management.

Author Contributions: LUO Yu conceived and designed the study, organized research materials, and drafted the manuscript; LUO Dan collected research materials and organized literature; TANG Binzhi revised the initial manuscript, controlled article quality, performed final revisions, and took overall responsibility for supervision.

Conflict of Interest: The authors declare no conflict of interest.

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(Received: October 3, 2023; Revised: December 16, 2023)
(Editor: JIA Mengmeng)

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