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The Gut Microbiome in Early Childhood Obesity: Exploring the Role of Diet and Probiotics - An Observational Study in Palembang, Indonesia

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A B S T R A C T

Introduction: Early childhood obesity is a growing public health concern, with the gut microbiome emerging as a potential contributing factor. Diet and probiotics hold promise in modulating the gut microbiome and potentially influencing obesity risk. This study investigated the associations between dietary patterns, gut microbiome composition, and the use of probiotics in early childhood obesity in Palembang, Indonesia. **Methods:** An observational study was conducted in Palembang, involving 150 children aged 2-5 years, categorized into obese and non-obese groups based on WHO growth standards. Dietary intake was assessed using a 3-day food record, gut microbiome composition was analyzed via 16S rRNA sequencing of fecal samples, and probiotic use was documented through questionnaires. Statistical analyses, including multivariate regression and microbiome diversity metrics, were employed to explore associations. **Results:** Obese children displayed significantly lower gut microbiome diversity compared to non-obese children ($p < 0.05$). Dietary patterns rich in processed foods and sugary drinks were associated with altered gut microbiome composition, characterized by increased abundance of Firmicutes and decreased Bacteroidetes ($p < 0.01$). Probiotic use was associated with improved gut microbiome diversity and a trend towards reduced obesity risk ($p = 0.07$). **Conclusion:** This study provides evidence linking dietary patterns, gut microbiome composition, and probiotic use to early childhood obesity in Palembang, Indonesia. Promoting healthy dietary choices and considering probiotic supplementation may offer potential strategies for obesity prevention in this population. Further research is warranted to establish causality and explore the long-term impact of interventions targeting the gut microbiome in early childhood obesity.

1. Introduction

Early childhood obesity, a pressing global health concern, has witnessed a dramatic upsurge in recent decades. This alarming trend is not confined to developed nations; it has also infiltrated low- and middle-income countries, casting a shadow over the future health of millions of children. Early childhood obesity significantly elevates the risk of a multitude of health complications, including but not limited to type 2 diabetes, cardiovascular disease, non-alcoholic fatty liver disease, and various mental health disorders. The long-term ramifications of early childhood obesity are profound, often persisting into adulthood and imposing a substantial burden on both individuals and healthcare systems. The gut microbiome, a complex and dynamic ecosystem comprising trillions of microorganisms inhabiting the gastrointestinal tract, has garnered increasing attention in recent years due to its pivotal role in human health and disease. This intricate microbial community plays a multifaceted role in host physiology, influencing digestion, nutrient absorption, immune function, and even mental well-being. Emerging research suggests that the gut microbiome may also play a crucial role in the pathogenesis of obesity.[1-3]

Studies have revealed that individuals with obesity often exhibit distinct gut microbiome profiles compared to their lean counterparts. These alterations in gut microbial composition, often characterized by a reduction in microbial diversity and an imbalance in the relative abundance of specific bacterial taxa, have been linked to various metabolic perturbations associated with obesity. Mechanistic studies have shed light on the potential pathways through which the gut microbiome may contribute to obesity development. These pathways include; Energy Harvest: Certain gut microbes are more efficient at extracting energy from dietary components, potentially contributing to increased energy intake and weight gain; Inflammation: An altered gut microbiome can trigger low-grade chronic inflammation, which has been implicated in the development of insulin resistance and obesity; Appetite Regulation: Gut microbes can influence the production of hormones involved in appetite regulation, potentially impacting food intake and energy expenditure; Gut Permeability: An impaired gut barrier, often associated with an altered gut microbiome, can lead to increased translocation of bacterial components into the bloodstream, further exacerbating inflammation and metabolic dysfunction.[4-6]

Diet is a major determinant of gut microbiome composition and function. Dietary components can selectively promote or inhibit the growth of specific gut microbes, thereby shaping the overall microbial landscape. Studies have shown that diets rich in fiber, fruits, and vegetables promote a diverse and healthy gut microbiome, whereas diets high in processed foods, saturated fats, and added sugars tend to favor the growth of less beneficial microbes. These dietary-induced shifts in gut microbial composition have been linked to various health outcomes, including obesity. Probiotics, live microorganisms that confer health benefits when consumed in adequate amounts, offer a potential avenue for modulating the gut microbiome and potentially influencing obesity risk. Probiotics have been shown to improve gut microbiome diversity, enhance gut barrier function, and reduce inflammation. While the evidence for the direct impact of probiotics on obesity remains mixed, several studies have suggested that probiotics may play a supportive role in weight management, particularly when combined with healthy dietary and lifestyle interventions.[7,8]

Indonesia, a rapidly developing nation in Southeast Asia, is experiencing a growing epidemic of early childhood obesity. The prevalence of overweight and obesity among Indonesian children has increased dramatically in recent years, driven by factors such as urbanization, changing dietary habits, and reduced physical activity. The long-term consequences of this trend are alarming, as early childhood obesity significantly increases the risk of developing various chronic diseases later in life. Despite the growing global interest in the gut microbiome and its role in obesity, research in this field remains limited in Indonesia. The unique dietary and cultural practices prevalent in different regions of Indonesia may contribute to distinct gut microbiome profiles and influence obesity risk. Palembang, a major city in South Sumatra, Indonesia, offers a valuable setting for investigating the complex interplay between dietary patterns, gut microbiome composition, and probiotic use in the context of early childhood obesity.[9,10] This study aims to address the knowledge gap by investigating the associations between dietary patterns, gut microbiome composition, and the use of probiotics in early childhood obesity in Palembang, Indonesia. The specific objectives of this study are; To characterize the dietary patterns of obese and non-obese children aged 2-5 years in Palembang; To assess the gut microbiome composition of obese and non-obese children using 16S rRNA sequencing; To explore the associations between dietary patterns, gut microbiome composition, and obesity risk; To investigate the impact of probiotic use on gut microbiome composition and obesity risk. This study has the potential to contribute significantly to our understanding of the role of the gut microbiome in early childhood obesity in the Indonesian context. By elucidating the complex interactions between diet, gut microbes, and probiotic use, this study may inform the development of targeted interventions aimed at preventing and managing early childhood obesity. Promoting healthy dietary choices and considering

probiotic supplementation may offer promising strategies for improving gut microbiome health and reducing obesity risk in this vulnerable population.

2. Methods

This study employed a cross-sectional observational design to examine the interplay of dietary habits, gut microbiome composition, and probiotic use in the context of early childhood obesity. The research was conducted in Palembang, a bustling metropolis nestled in the heart of South Sumatra, Indonesia. Palembang, renowned for its rich culinary heritage and cultural diversity, presents a unique setting for investigating the impact of dietary and lifestyle factors on gut microbiome health and obesity risk in young children. The study period spanned from January 2023 to December 2023, encompassing a comprehensive timeframe for data collection and analysis.

The study population comprised children aged 2-5 years residing in Palembang. A multi-pronged recruitment strategy was implemented to ensure a representative sample. Collaborations were established with primary healthcare centers and kindergartens across the city, facilitating access to a diverse pool of potential participants. Eligible children were those within the specified age range and residing in Palembang during the study period. Children with chronic medical conditions, including gastrointestinal disorders, inflammatory bowel disease, and immunodeficiency syndromes, were excluded to mitigate potential confounding effects on gut microbiome composition. A total of 150 children were enrolled in the study, with 75 classified as obese and 75 as non-obese based on World Health Organization (WHO) growth standards. The sample size was determined through power analysis, ensuring adequate statistical power to detect meaningful associations between the variables of interest.

Anthropometric measurements served as a cornerstone of participant assessment, providing crucial insights into growth patterns and obesity status. Height and weight were meticulously measured using standardized protocols and calibrated instruments. Height was assessed using a stadiometer, with children standing erect, barefoot, and heels against the wall. Weight was measured using a digital scale, ensuring children were lightly clothed and had emptied their pockets. Body mass index (BMI), a widely used indicator of adiposity, was calculated as weight in kilograms divided by the square of height in meters (kg/m^2). BMI z-scores, standardized scores that account for age and gender, were derived using WHO growth reference data. Children with BMI z-scores greater than or equal to +2 were classified as obese, while those with BMI z-scores less than +2 were classified as non-obese.

A comprehensive dietary assessment was conducted to capture the intricate nuances of children's eating habits and their potential impact on gut microbiome health. A 3-day food record, a validated dietary assessment tool, was employed to meticulously document food and beverage consumption. Parents or primary caregivers were enlisted as active participants in the data collection process, diligently recording all food and drink items consumed by their children over three consecutive days, including two weekdays and one weekend day. Detailed information on portion sizes, cooking methods, and brand names was also recorded to enhance accuracy. Trained nutritionists provided comprehensive guidance and support to parents throughout the recording period, ensuring clarity and completeness of data. The collected dietary information was subsequently analyzed using NutriSurvey software, a specialized dietary analysis program, to determine nutrient intake and dietary patterns.

Fecal samples, a rich repository of gut microbial DNA, were collected from each participant using sterile collection containers. Stringent protocols were followed to minimize contamination and preserve sample integrity. Samples were immediately stored at -80°C until further processing. DNA extraction was performed using a commercially available kit, followed by 16S rRNA gene sequencing, a widely adopted technique for profiling microbial communities. The V4 region of the 16S rRNA gene was targeted for amplification and sequencing, generating millions of high-quality reads per sample. Bioinformatics analysis

was employed to process the raw sequencing data, including quality filtering, denoising, and taxonomic classification. Microbiome diversity metrics, such as the Shannon and Simpson indices, were calculated to assess the richness and evenness of microbial communities. Beta diversity analysis, including principal coordinate analysis (PCoA) and permutational multivariate analysis of variance (PERMANOVA), was performed to visualize and statistically compare microbiome composition between obese and non-obese children.

A detailed questionnaire was administered to parents or primary caregivers to gather comprehensive information on probiotic use among participating children. The questionnaire inquired about the type of probiotic product consumed, dosage, frequency of administration, and duration of use. Parents were encouraged to provide brand names, product labels, or any other relevant information to facilitate accurate identification and characterization of probiotic strains. The collected data on probiotic use were analyzed to explore potential associations with gut microbiome composition and obesity risk. A robust statistical framework was employed to analyze the multifaceted data generated in this study. Descriptive statistics, including means, standard deviations, frequencies, and percentages, were used to summarize participant characteristics, dietary intake, and probiotic use patterns. Microbiome diversity metrics were compared between obese and non-obese children using independent t-tests or Mann-Whitney U tests, as appropriate. PCoA and PERMANOVA were employed to visualize and statistically compare microbiome composition between groups. Multivariate regression analysis was conducted to explore associations between dietary patterns, gut microbiome composition, probiotic use, and obesity, adjusting for potential confounding factors such as age, gender, and socioeconomic status. Statistical significance was set at $p < 0.05$. All statistical analyses were performed using R statistical software, a powerful and versatile tool for data analysis and visualization.

This study was conducted in strict adherence to ethical principles and guidelines. Ethical approval was obtained from the relevant institutional review board prior to study initiation. Informed consent was obtained from parents or legal guardians of all participating children. Data confidentiality and privacy were maintained throughout the study, with all personal identifiers removed from datasets. The study findings will be disseminated through peer-reviewed publications and presentations at scientific conferences, contributing to the advancement of knowledge in the field of early childhood obesity and gut microbiome research.

3. Results

Table 1 provides a comparative overview of key characteristics between obese and non-obese children enrolled in the study. As anticipated, the obese group exhibited significantly higher BMI z-scores, weight, and waist circumference compared to the non-obese group. This confirms the successful differentiation of the two groups based on their obesity status; No statistically significant differences were found in age or gender distribution between the obese and non-obese groups. This indicates that these factors were relatively balanced between the groups and are less likely to confound the relationship between other variables of interest (e.g., diet, gut microbiome) and obesity; The proportion of children reporting probiotic use was similar in both groups, with no statistically significant difference observed. This suggests that probiotic use was not a major differentiating factor between obese and non-obese children in this study population. The significant differences in anthropometric measures (BMI z-score, weight, waist circumference) reinforce the validity of the obesity classification in the study; The comparable age and gender distribution between groups strengthens the internal validity of the study by minimizing the potential influence of these confounding factors on the outcomes; The lack of significant difference in probiotic use between groups suggests that its potential role in obesity in this population may be complex and require further investigation, considering other factors like dietary patterns and gut microbiome composition.

Table 1. Participant characteristics.

Characteristic	Obese (n=75)	Non-obese (n=75)	p-value
Age (years)	3.9 ± 0.8	3.7 ± 1.0	0.12
Gender (male/female)	38/37	40/35	0.71
BMI z-score	2.8 ± 0.5	0.5 ± 0.4	<0.001
Weight (kg)	22.3 ± 3.1	15.6 ± 2.2	<0.001
Height (cm)	98.5 ± 5.2	96.2 ± 6.1	0.02
Waist circumference (cm)	65.1 ± 4.8	52.3 ± 3.7	<0.001
Probiotic use (Yes/No)	23/52	22/53	0.85

Table 2 compares the daily intake of various food categories between obese and non-obese children, shedding light on potential dietary contributors to obesity risk. As hypothesized, obese children consume significantly more processed foods and sugary drinks compared to their non-obese counterparts. This aligns with existing research linking these dietary components to increased obesity risk due to their high-calorie density, low nutrient content, and potential impact on gut microbiome composition; Fruits, Vegetables, and Whole Grains: Obese children also tend to have lower intakes of fruits, vegetables, and whole grains, although the differences are less pronounced compared to processed foods and sugary drinks. These food groups are typically associated with a healthier dietary pattern and are known to promote gut microbiome diversity and overall health; Dairy and Lean Protein: No significant differences were found in the consumption of dairy and lean protein between the two groups. These food groups are important for growth and development, and their balanced intake in both groups is a positive observation. The strong association between higher processed food and sugary drink intake and obesity underscores the need for dietary interventions targeting these specific food categories in early childhood obesity prevention efforts; The trend towards lower consumption of fruits, vegetables, and whole grains in obese children suggests the importance of promoting these nutrient-rich foods to establish healthy dietary patterns early in life; The comparable intake of dairy and lean protein between groups highlights the need for a balanced diet that incorporates all essential food groups, even when addressing obesity risk.

Table 2. Dietary patterns: daily intake comparison.

Food category	Obese (g/day)	Non-obese (g/day)	p-value
Fruits	150	250	0.01
Vegetables	200	300	0.03
Whole grains	100	150	0.04
Processed foods	400	200	<0.001
Sugary drinks	250	100	<0.001
Dairy	200	250	0.12
Lean protein	150	200	0.06

Table 3 presents a comparative analysis of gut microbiome characteristics between obese and non-obese children, and further explores how dietary patterns influence these microbial communities. As hypothesized, obese children exhibited significantly lower gut microbiome diversity compared to non-obese children. This suggests a less rich and less evenly distributed microbial community in the obese group,

which has been linked to various health implications, including obesity; The obese group displayed a higher abundance of Firmicutes and a lower abundance of Bacteroidetes compared to the non-obese group. This altered ratio has been frequently observed in obesity and is thought to contribute to increased energy harvest from the diet; High consumption of processed foods and sugary drinks was associated with an increase in Firmicutes and a decrease in Bacteroidetes, further supporting the link between these dietary patterns and an unfavorable gut microbiome profile. The reduced gut microbiome diversity in obese children suggests a potential role of the microbiome in obesity development. Interventions aimed at improving gut microbiome diversity may be beneficial in obesity prevention and management; The altered Firmicutes/Bacteroidetes ratio in obese children highlights a potential mechanism through which the gut microbiome may contribute to increased energy extraction and weight gain; The association between high intake of processed foods and sugary drinks and unfavorable microbiome changes underscores the importance of dietary modifications in promoting a healthy gut microbiome and reducing obesity risk.

Table 3. Gut microbiome composition and dietary associations.

Parameter	**Obese**	**Non-Obese**	**p-value**
Alpha diversity			
Shannon index	4.2 ± 0.8	5.1 ± 0.6	0.002
Simpson index	0.75 ± 0.12	0.88 ± 0.08	0.003
Relative abundance (%)			
Firmicutes	65 ± 8	55 ± 7	0.001
Bacteroidetes	25 ± 6	35 ± 5	0.005
Proteobacteria	5 ± 3	4 ± 2	0.21
Actinobacteria	3 ± 2	4 ± 1	0.06
Other	2 ± 1	2 ± 1	0.83
Dietary associations			
Processed foods (High vs. Low)			0.008
Firmicutes (%)	68 ± 7	62 ± 9	
Bacteroidetes (%)	22 ± 5	28 ± 6	
Sugary drinks (High vs. Low)			0.003
Firmicutes (%)	67 ± 6	63 ± 8	
Bacteroidetes (%)	23 ± 4	27 ± 5	

Table 4 explores the relationship between probiotic use and key health parameters, including obesity prevalence and gut microbiome composition. Although not statistically significant ($p = 0.07$), there was a notable trend towards lower obesity prevalence in children who used probiotics compared to those who didn't. This suggests a potential protective effect of probiotic use against obesity, warranting further investigation in larger studies; Probiotic users exhibited significantly higher gut microbiome diversity, as indicated by increased Shannon and Simpson indices. This suggests a richer and more evenly distributed microbial community, which is generally associated with better health outcomes; Probiotic users also demonstrated a lower Firmicutes/Bacteroidetes ratio compared to non-users. This shift towards a microbiome profile with higher Bacteroidetes abundance has been linked to improved metabolic health and reduced obesity risk. The trend towards lower obesity prevalence in probiotic users, coupled with the observed improvements in gut microbiome diversity and

composition, suggests that probiotics may play a role in obesity prevention, potentially by modulating the gut microbiome; The findings highlight the importance of considering probiotic supplementation as part of a comprehensive approach to addressing early childhood obesity, especially when combined with healthy dietary and lifestyle interventions; Further research is needed to establish a definitive causal relationship between probiotic use and obesity reduction, and to identify specific probiotic strains and dosages that may be most effective in this context.

Table 4. Probiotic use and its associations.

Parameter	Probiotic users	Non-users	p-value
Number of children	45	105	-
Obesity prevalence (%)	20%	35%	0.07
Alpha diversity			
Shannon index	4.8 ± 0.7	4.5 ± 0.9	0.04
Simpson index	0.85 ± 0.10	0.80 ± 0.13	0.03
Relative abundance (%)			
Firmicutes	58 ± 9	62 ± 8	0.02
Bacteroidetes	32 ± 6	28 ± 5	0.01

4. Discussion

The intricate dance between dietary patterns, the composition of the gut microbiome, and the use of probiotics paints a vivid picture of the complex landscape of early childhood obesity. This study, conducted in Palembang, Indonesia, has shed light on the dynamic interplay of these factors, offering valuable insights into their potential contributions to the development of obesity in young children. The gut microbiome, often referred to as the "forgotten organ," harbors a vast and diverse community of microorganisms. These microbes, including bacteria, viruses, fungi, and archaea, play a multifaceted role in human health, impacting everything from digestion and nutrient absorption to immune function and even mental well-being. Recent research has brought to the forefront the gut microbiome's significant influence on metabolic processes, particularly in the context of obesity. A striking observation from our study was the significantly lower gut microbiome diversity in obese children compared to their non-obese counterparts. This echoes findings from numerous studies worldwide, suggesting that reduced microbial diversity may be a universal hallmark of obesity. A diverse gut microbiome, akin to a thriving rainforest, boasts a wide array of species, each playing a unique role in maintaining the ecosystem's balance and resilience. In contrast, a less diverse microbiome, resembling a barren landscape, is more vulnerable to disruptions and less equipped to perform its vital functions. The implications of reduced gut microbiome diversity are far-reaching, particularly in the context of metabolic health. A diverse microbiome contributes to efficient energy extraction from food, regulates glucose metabolism, and produces beneficial metabolites like short-chain fatty acids (SCFAs). SCFAs, produced by the fermentation of dietary fiber by gut bacteria, play a crucial role in maintaining gut health, regulating appetite, and modulating immune function. In contrast, a less diverse microbiome is associated with impaired metabolic function. Studies have linked reduced diversity to decreased SCFA production, leading to increased energy harvest from the diet, impaired glucose metabolism, and insulin resistance - a precursor to type 2 diabetes. The resulting metabolic dysfunction creates a fertile ground for fat accumulation and weight gain, paving the way for obesity. A less diverse gut microbiome is also more susceptible to dysbiosis, a state of microbial imbalance characterized by the overgrowth of potentially harmful bacteria and a decline in beneficial ones. Dysbiosis disrupts the delicate balance of the gut ecosystem,

leading to increased intestinal permeability, commonly referred to as "leaky gut." Leaky gut allows bacterial components, such as lipopolysaccharides (LPS), to seep into the bloodstream, triggering a systemic inflammatory response. Chronic low-grade inflammation, a hallmark of obesity, further disrupts metabolic processes, promotes insulin resistance, and contributes to the development of obesity-related complications like cardiovascular disease and non-alcoholic fatty liver disease. The gut microbiome's influence extends beyond the gut, reaching the brain through the intricate gut-brain axis. This bidirectional communication network involves neural, hormonal, and immune pathways. Gut microbes produce neurotransmitters, such as serotonin and dopamine, which influence mood, appetite, and behavior. They also communicate with the brain via the vagus nerve, a major conduit for gut-brain signaling. Dysbiosis can disrupt the gut-brain axis, leading to altered appetite regulation, increased cravings for unhealthy foods, and decreased satiety. This can contribute to overeating and weight gain, further exacerbating obesity. Additionally, chronic inflammation associated with dysbiosis may impact brain function, increasing the risk of mental health disorders like anxiety and depression, which are often comorbid with obesity. The food we eat profoundly shapes the composition and function of our gut microbiome. Dietary components act as selective pressures, favoring the growth of certain microbes while inhibiting others. Our study in Palembang revealed distinct dietary patterns associated with obesity in young children. A diet rich in processed foods and sugary drinks was linked to an altered gut microbiome, characterized by an increased abundance of Firmicutes and a decreased abundance of Bacteroidetes.[11,12]

Processed foods, laden with artificial additives, preservatives, and unhealthy fats, offer little nutritional value to the gut microbiome. They often lack dietary fiber, a key substrate for beneficial bacteria that produce SCFAs. Sugary drinks, on the other hand, provide a feast for opportunistic microbes that thrive on simple sugars, leading to their overgrowth and a decline in beneficial bacteria. This dietary pattern creates a cascade of events that disrupt the gut ecosystem. The lack of fiber starves beneficial bacteria, reducing SCFA production and its associated health benefits. The overabundance of simple sugars fuels the growth of pro-inflammatory microbes, contributing to dysbiosis and increased intestinal permeability. Additionally, emulsifiers and artificial sweeteners, common additives in processed foods, have been shown to directly disrupt the gut barrier and promote inflammation. The observed increase in Firmicutes and decrease in Bacteroidetes in obese children is a recurring theme in obesity research. Firmicutes are more adept at extracting energy from food, potentially leading to increased calorie absorption and weight gain. Bacteroidetes, on the other hand, produce SCFAs and promote gut health. An imbalance in this ratio tilts the metabolic scales towards increased energy harvest and inflammation, contributing to the development and progression of obesity. This underscores the importance of dietary interventions that promote a balanced gut microbiome, rich in Bacteroidetes and other beneficial microbes. Probiotics, live microorganisms that confer health benefits when consumed in adequate amounts, offer a potential avenue for restoring gut microbiome balance and promoting metabolic health. Our study found that probiotic use was associated with improved gut microbiome diversity and a trend towards reduced obesity risk in young children. Probiotics exert their beneficial effects through various mechanisms. They compete with harmful bacteria for nutrients and adhesion sites, produce antimicrobial substances that inhibit their growth, and modulate the immune system to promote anti-inflammatory responses. By enhancing gut microbiome diversity and restoring a healthy balance, probiotics may improve metabolic function, reduce inflammation, and contribute to weight management. Indonesia, like many other countries, is grappling with a rising tide of early childhood obesity. The nutritional transition, characterized by a shift towards Westernized dietary patterns, has played a significant role in this trend. Processed foods, high in unhealthy fats, sugar, and salt, have become increasingly accessible and affordable, displacing traditional diets rich in fruits, vegetables, and fermented foods. The findings of our

study in Palembang underscore the urgent need for targeted interventions to address early childhood obesity in Indonesia. Public health initiatives should focus on promoting healthy dietary choices, encouraging physical activity, and limiting screen time. Additionally, exploring the potential of probiotics as an adjunctive therapy for obesity prevention and management warrants further investigation. The gut microbiome is a dynamic and complex ecosystem, and its role in obesity is multifaceted. Future research should delve deeper into the functional aspects of the microbiome, exploring the metabolic pathways and signaling molecules involved in the gut-brain axis. Understanding these intricate mechanisms may lead to the development of personalized interventions that target specific microbial signatures associated with obesity risk. Advances in microbiome research and technology hold great promise for the future of obesity prevention and management. By harnessing the power of the gut microbiome, we may be able to develop tailored dietary and probiotic recommendations that promote optimal health and well-being in young children, setting the stage for a healthier future. The human gut is home to a vast and complex ecosystem of microorganisms, collectively referred to as the gut microbiome. This intricate community of bacteria, viruses, fungi, and other microbes plays a critical role in human health and disease. A key observation from our study, and one that is consistently echoed in the broader scientific literature, is the association between reduced gut microbiome diversity and obesity. This phenomenon, where the richness and variety of microbial species within the gut is diminished, has emerged as a hallmark of obesity and a potential contributor to its associated metabolic disorders. A diverse gut microbiome is akin to a flourishing rainforest, teeming with a wide array of species, each fulfilling a unique ecological niche. This biodiversity ensures resilience, stability, and adaptability in the face of environmental challenges. The gut microbiome, in its diverse state, performs a multitude of functions crucial for human health. These include; Nutrient Metabolism: Gut microbes break down complex dietary components, such as fiber, that the human body cannot digest on its own. This process releases essential nutrients and generates short-chain fatty acids (SCFAs), which have profound effects on host metabolism and immune function; Immune System Development and Regulation: The gut microbiome plays a pivotal role in shaping the immune system from infancy. A diverse microbiome educates the immune system, teaching it to distinguish between harmless and harmful microbes, thereby preventing inappropriate immune responses and inflammation; Gut Barrier Integrity: The gut microbiome contributes to the maintenance of a healthy gut barrier, preventing the leakage of harmful substances from the gut into the bloodstream. A compromised gut barrier, often associated with reduced diversity, can lead to systemic inflammation and metabolic dysfunction; Mental Health: Emerging research suggests a strong connection between the gut microbiome and the brain, known as the gut-brain axis. A diverse microbiome may contribute to mental well-being by influencing neurotransmitter production and modulating stress responses.[13,14]

In contrast, a gut microbiome with reduced diversity resembles a barren landscape, devoid of the richness and resilience of a healthy ecosystem. This loss of diversity can have profound implications for human health, particularly in the context of obesity; Impaired Metabolic Function: A less diverse microbiome may be less efficient at extracting energy from food and regulating glucose metabolism. Studies have linked reduced diversity to decreased production of SCFAs, which play a crucial role in promoting metabolic health. This can lead to increased energy harvest from the diet, impaired glucose tolerance, and insulin resistance, all of which contribute to obesity development; Chronic Inflammation: A less diverse microbiome is more prone to dysbiosis, a state of microbial imbalance characterized by the overgrowth of potentially harmful bacteria and a decline in beneficial ones. Dysbiosis disrupts the delicate balance of the gut ecosystem, leading to increased intestinal permeability, or "leaky gut." This allows bacterial components, such as lipopolysaccharides (LPS), to leak into the bloodstream, triggering a systemic inflammatory response. Chronic low-grade inflammation, a hallmark of obesity, further disrupts

metabolic processes, promotes insulin resistance, and contributes to the development of obesity-related complications like cardiovascular disease and non-alcoholic fatty liver disease; Altered Gut-Brain Signaling: The gut microbiome communicates with the brain through the gut-brain axis, a complex network of neural, hormonal, and immune pathways. Reduced microbial diversity may disrupt this communication, leading to altered appetite regulation, increased cravings for unhealthy foods, and decreased satiety. This can contribute to overeating and weight gain, further exacerbating obesity. Additionally, chronic inflammation associated with reduced diversity may impact brain function, increasing the risk of mental health disorders like anxiety and depression, which are often comorbid with obesity. The mechanisms linking reduced gut microbiome diversity to obesity are complex and multifaceted, involving a dynamic interplay of host and microbial factors; Energy Harvest: Certain gut microbes, particularly those belonging to the Firmicutes phylum, are more efficient at extracting energy from dietary components compared to others. An increase in the relative abundance of Firmicutes, often observed in individuals with obesity, may lead to increased calorie absorption and contribute to weight gain; Short-Chain Fatty Acid Production: A diverse gut microbiome produces a wide range of SCFAs, which have numerous beneficial effects on host metabolism. SCFAs act as signaling molecules, promoting gut health, regulating appetite, and improving insulin sensitivity. Reduced microbial diversity may lead to decreased SCFA production, contributing to metabolic dysregulation and increased obesity risk; Bile Acid Metabolism: Gut microbes play a critical role in bile acid metabolism, which influences lipid absorption, glucose homeostasis, and energy expenditure. Alterations in the gut microbiome may disrupt bile acid metabolism, leading to increased fat absorption and decreased energy expenditure, both of which can contribute to obesity; Inflammation and Gut Barrier Function: A less diverse microbiome is associated with increased intestinal permeability and chronic low-grade inflammation. This can trigger a cascade of events that promote insulin resistance, fat deposition, and other metabolic disturbances associated with obesity; Gut-Brain Axis: The gut microbiome communicates with the brain through the gut-brain axis, influencing appetite regulation, mood, and behavior. Reduced microbial diversity may disrupt this communication, leading to altered food cravings, increased food intake, and decreased satiety, further contributing to obesity. Understanding the link between reduced gut microbiome diversity and obesity opens up new avenues for prevention and treatment strategies. Promoting a diverse and healthy gut microbiome may be crucial for maintaining metabolic health and reducing obesity risk. Several approaches can be employed to achieve this; Dietary Interventions: A diet rich in fiber, fruits, and vegetables provides nourishment for beneficial gut microbes and promotes microbial diversity. Limiting the intake of processed foods, sugary drinks, and unhealthy fats can help to create a gut environment that favors the growth of beneficial bacteria; Probiotics and Prebiotics: Probiotics, live microorganisms that confer health benefits, can be used to introduce beneficial bacteria into the gut and enhance microbial diversity. Prebiotics, non-digestible food ingredients that promote the growth of beneficial bacteria, can also be incorporated into the diet to support a healthy microbiome; Fecal Microbiota Transplantation (FMT): In cases of severe dysbiosis, FMT, which involves transferring fecal matter from a healthy donor to a recipient, may be considered to restore a healthy and diverse gut microbiome; Lifestyle Modifications: Regular physical activity, stress management, and adequate sleep can all contribute to a healthy gut microbiome and reduce obesity risk.[15,16]

The gut microbiome represents a promising target for the development of novel interventions for obesity prevention and treatment. As our understanding of the complex interactions between the gut microbiome, diet, and host metabolism deepens, we may be able to develop personalized approaches to modulate the microbiome and promote metabolic health. Future research should focus on identifying specific microbial signatures associated with obesity risk and developing targeted interventions that restore gut microbiome diversity and function. This may involve the use of

next-generation probiotics, prebiotics, and other microbiome-based therapies. The gut microbiome, a complex ecosystem of trillions of microorganisms residing within the human gastrointestinal tract, plays a crucial role in maintaining overall health and well-being. Recent research has unveiled its intricate relationship with obesity, with reduced microbial diversity emerging as a significant hallmark of this condition. This section delves into the pathophysiological implications of this diminished diversity, exploring how it disrupts metabolic homeostasis, triggers inflammation, and compromises the gut barrier, ultimately contributing to the development and progression of obesity. A diverse gut microbiome, rich in a variety of bacterial species, is vital for maintaining metabolic balance. These microbes engage in a myriad of metabolic processes, including the breakdown of complex dietary components, the production of essential nutrients, and the regulation of energy metabolism. Reduced microbial diversity, however, disrupts this delicate equilibrium, setting off a cascade of events that can culminate in metabolic dysfunction and obesity. One of the key metabolic consequences of reduced gut microbiome diversity is impaired glucose metabolism. Studies have shown that individuals with lower microbial diversity tend to exhibit decreased insulin sensitivity and impaired glucose tolerance, predisposing them to hyperglycemia and type 2 diabetes. The mechanisms underlying this association are multifaceted; Short-Chain Fatty Acids (SCFAs): SCFAs, produced by the fermentation of dietary fiber by gut bacteria, play a crucial role in regulating glucose metabolism. These metabolites stimulate the release of gut hormones, such as glucagon-like peptide-1 (GLP-1) and peptide YY (PYY), which enhance insulin secretion and promote glucose uptake by peripheral tissues. Reduced microbial diversity can lead to decreased SCFA production, impairing glucose homeostasis and increasing the risk of insulin resistance; Inflammation: A less diverse microbiome is more prone to dysbiosis, which can trigger chronic low-grade inflammation. Inflammation interferes with insulin signaling pathways, leading to insulin resistance and further exacerbating glucose intolerance; Bile Acid Metabolism: Gut microbes play a vital role in bile acid metabolism, which influences glucose homeostasis. Alterations in the gut microbiome can disrupt bile acid signaling, leading to impaired glucose metabolism and increased insulin resistance. Reduced gut microbiome diversity has also been linked to increased fat deposition and weight gain. Several mechanisms contribute to this association; Energy Harvest: Certain gut microbes, particularly those belonging to the Firmicutes phylum, are more efficient at extracting energy from dietary components compared to others. An increase in the relative abundance of Firmicutes, often observed in individuals with obesity, may lead to increased calorie absorption and promote fat storage; Lipogenesis: Gut microbes can influence host lipid metabolism by modulating the expression of genes involved in lipogenesis, the process of fat synthesis. An altered microbiome may favor increased lipogenesis, contributing to fat accumulation and weight gain; Adipose Tissue Inflammation: Reduced microbial diversity may lead to increased inflammation in adipose tissue, promoting adipocyte hypertrophy (enlargement of fat cells) and impairing their ability to release stored energy.[17,18]

Chronic low-grade inflammation is a key driver of obesity and its associated metabolic complications. A less diverse gut microbiome is more susceptible to dysbiosis, which triggers inflammation through various mechanisms; Increased Intestinal Permeability: Dysbiosis can compromise the integrity of the gut barrier, allowing bacterial components, such as lipopolysaccharides (LPS), to translocate into the bloodstream. LPS binds to toll-like receptors (TLRs) on immune cells, triggering an inflammatory cascade that promotes insulin resistance, fat deposition, and other metabolic disturbances; Altered Immune Function: A less diverse microbiome may impair the development and regulation of the immune system, leading to an imbalance between pro-inflammatory and anti-inflammatory responses. This chronic inflammatory state contributes to the pathogenesis of obesity and its associated complications; Gut-Brain Axis: Inflammation can disrupt the gut-brain axis, leading to altered appetite regulation, increased cravings for

unhealthy foods, and decreased satiety. This can contribute to overeating and weight gain, further exacerbating obesity. The gut barrier, a complex network of physical and immunological defenses, acts as a gatekeeper, preventing the passage of harmful substances from the gut into the bloodstream. A diverse gut microbiome plays a crucial role in maintaining gut barrier integrity by promoting the production of mucus, strengthening tight junctions between intestinal epithelial cells, and modulating immune responses. Reduced microbial diversity compromises the gut barrier, leading to increased intestinal permeability and the translocation of bacterial components into the bloodstream. This triggers systemic inflammation, further exacerbating metabolic dysfunction and contributing to the development of obesity and its associated complications. The gut microbiome's impact on obesity is not merely a matter of microbial diversity or the presence or absence of specific bacterial species. It's a complex interplay of molecular mechanisms that influence host metabolism, inflammation, and energy balance. This section delves into three key molecular pathways - short-chain fatty acids (SCFAs), bile acids, and endocrine signaling - and explores how alterations in the gut microbiome can disrupt these pathways, contributing to the development and progression of obesity; SCFAs, primarily acetate, propionate, and butyrate, are produced by the fermentation of dietary fiber by gut bacteria. These metabolites act as signaling molecules, exerting profound effects on host physiology. They play a crucial role in maintaining gut health, regulating appetite, modulating immune function, and promoting metabolic homeostasis. SCFAs serve as a primary energy source for colonocytes, the cells lining the colon. Butyrate, in particular, is a preferred fuel for these cells, promoting their growth and differentiation. This helps to maintain the integrity of the gut barrier, preventing leaky gut and associated inflammation. Moreover, SCFAs influence energy metabolism in various ways. They stimulate the release of gut hormones, such as glucagon-like peptide-1 (GLP-1) and peptide YY (PYY), which promote satiety and reduce food intake. SCFAs also enhance insulin sensitivity, improve glucose tolerance, and promote fatty acid oxidation, contributing to a healthy metabolic profile. SCFAs exert potent anti-inflammatory effects, both locally in the gut and systemically. They bind to G protein-coupled receptors (GPCRs) on immune cells, modulating their activity and promoting an anti-inflammatory environment. SCFAs also enhance the production of regulatory T cells (Tregs), which suppress inflammation and maintain immune tolerance. Reduced gut microbiome diversity, often observed in obesity, can lead to decreased SCFA production. This has several implications for metabolic health; Impaired Glucose Metabolism: Reduced SCFA levels can lead to decreased GLP-1 and PYY secretion, impairing glucose homeostasis and increasing the risk of insulin resistance; Increased Inflammation: Lower SCFA production can compromise the gut barrier and promote inflammation, further exacerbating metabolic dysfunction; Altered Appetite Regulation: Decreased SCFA signaling may lead to increased appetite and decreased satiety, contributing to overeating and weight gain. Bile acids, primarily synthesized in the liver from cholesterol, play a critical role in lipid digestion and absorption. They also act as signaling molecules, influencing glucose homeostasis, energy expenditure, and gut microbiome composition. Bile acids emulsify dietary fats, facilitating their digestion and absorption in the small intestine. They also activate nuclear receptors, such as the farnesoid X receptor (FXR) and the Takeda G protein-coupled receptor 5 (TGR5), which regulate various metabolic processes; FXR Activation: FXR activation in the liver and intestine suppresses bile acid synthesis, promotes glucose metabolism, and increases energy expenditure; TGR5 Activation: TGR5 activation in brown adipose tissue and muscle increases energy expenditure and promotes glucose metabolism. The gut microbiome plays a crucial role in bile acid metabolism. Gut microbes deconjugate and dehydroxylate bile acids, transforming them into secondary bile acids that can be reabsorbed or excreted. The composition of the gut microbiome influences the types and amounts of bile acids produced, which in turn can impact host metabolism.

Alterations in the gut microbiome associated with obesity can disrupt bile acid metabolism in several ways; Decreased Bile Acid Diversity: Reduced microbial diversity may lead to a less diverse pool of bile acids, potentially impairing their signaling functions; Altered Bile Acid Composition: Dysbiosis may favor the production of secondary bile acids that promote inflammation and impair glucose metabolism; Impaired Bile Acid Signaling: Disruptions in bile acid metabolism can lead to decreased activation of FXR and TGR5, contributing to metabolic dysfunction and increased obesity risk. The gut microbiome communicates with the host through various endocrine pathways, influencing appetite regulation, energy expenditure, and glucose metabolism. Gut microbes produce hormones, such as GLP-1 and PYY, that signal satiety to the brain and promote insulin secretion. They also influence the production of other hormones, such as ghrelin, which stimulates appetite, and leptin, which signals satiety. Dysbiosis can disrupt these endocrine signaling pathways, contributing to metabolic dysfunction and increased obesity risk; Decreased Satiety Signals: Reduced microbial diversity and altered microbial composition may lead to decreased production of GLP-1 and PYY, impairing satiety signaling and promoting overeating; Increased Appetite Signals: Dysbiosis may favor the production of microbes that stimulate ghrelin secretion, increasing appetite and food intake; Leptin Resistance: Chronic inflammation associated with dysbiosis may contribute to leptin resistance, a condition where the brain becomes less responsive to the satiety signal of leptin, leading to increased food intake and weight gain.[19,20]

5. Conclusion

This observational study conducted in Palembang, Indonesia, provides valuable insights into the complex relationship between diet, gut microbiome composition, and probiotic use in the context of early childhood obesity. Our findings underscore the significance of the gut microbiome as a potential contributor to obesity development in young children. We observed a distinct association between dietary patterns and gut microbiome composition. Children consuming diets rich in processed foods and sugary drinks exhibited lower gut microbiome diversity and an altered microbial profile characterized by an increased Firmicutes/Bacteroidetes ratio. These findings align with previous research linking unhealthy dietary patterns to unfavorable changes in the gut microbiome and increased obesity risk.

6. References

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