



Gut microbiota-derived short-chain fatty acids as mediators of nutritional therapy in pediatric obesity

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Abstract

Type 2 diabetes is the most serious metabolic, endocrine, and cardiovascular consequence of childhood obesity, which is quickly becoming recognised as a worldwide health crisis. New research has shown that the gut microbiota plays a key role in the development of childhood obesity. This role is mainly manifested when the microbiome ferments fermentable fibre, leading to the production of short-chain fatty acids (SCFA). Acetate, propionate, and butyrate show a wide range of biological activities within this class of metabolites. These include reducing hyperphagia, affecting substrate utilisation, and changing the immunological milieu. The present review synthesizes the prevailing body of evidence that links SCFA production to dietary intervention strategies directed at the amelioration of childhood adiposity. Emphasis is directed toward elucidating the intricate signaling pathways that frame SCFA activity, with particular attention to hypothalamic, enteric, and hepato-pancreatic interligature that modulated energy homeostasis, augmented insulin responsiveness, and curtailed systemic and adipocyte-directed inflammation. Furthermore, the manuscript synthesizes the predominant dietary approaches that enhance SCFA synthesis, systematically appraising their feasibility for incorporation into obesity treatment protocols. The examination focuses on trials that quantify SCFA and associated alterations in metabolic parameters among pediatric cohorts, allowing for the systematic evaluation of both favorable outcomes and the obstacles that impede the transposition of these insights into usual care. The last section outlines potential directions for future study, highlighting the

need for dietary treatments tailored to individual gut microbiota compositions to optimise SCFA synthesis and improve ways for monitoring and treating paediatric obesity.

Keywords: Gut microbiota. Short-chain fatty acids. Pediatric obesity. Metabolic regulation. Nutritional therapy.

Introduction

Overview of Gut Microbiota and Pediatric Obesity

The gut microbiota, a diverse community of microbes found in the human intestines, plays an important role in regulating host metabolism and other physiological processes [1-6]. The taxonomic variety and functional resilience of this consortium—which includes bacteria, viruses, fungi, and archaea - are constantly being modulated through interactions with innate and adaptive immunological circuits, concurrent metabolic processes, and neuroendocrine signalling [7-12]. An increasing amount of research has shed light on the microbiome's salutogenic and pathogenic pathways that it uses to connect with and impact systemic biology over the last ten years. An increasingly common example of this axis is obesity [13-16].

In particular, the accelerating prevalence of excess adiposity, operationally defined as a sustained surplus of body fat, is a salient, cross-national public health challenge among adolescents and demands urgent multi-sectoral attention. The excess of body adiposity compromises physical health. Subsequently, it enhances vulnerability to diverse metabolic sequelae,

including, but not limited to, insulin resistance, adolescent-onset type 2 diabetes, and early-onset atherosclerotic disease. Dominant shifts in gut microbiota profiles have been documented in the obese pediatric population, typically manifesting as a reduced diversity of protective taxa and an expansion of potentially pathogenic strains [17]. Such dysbiotic states are posited to modulate host energy homeostasis, the deposition and distribution of adipose tissue, and the systemic synthesis of pro-inflammatory mediators, thereby entrenching the pathophysiological trajectories of childhood obesity.

Importance of Short-Chain Fatty Acids in Health

Short-chain fatty acids - namely, acetate, propionate, and butyrate - arise when intestinal microbiota ferment fermentable dietary fibers. Beyond serving as dietary-derived energy substrates, these fatty acids have critical extra-colonic functions and have thus attracted considerable scientific interest [2]. Their production and absorption influence host metabolic homeostasis, chiefly by directing the activities of intestinal epithelia and by dispatching signalling metabolites into the circulatory system. Within the context of obesity, the metabolic advantages that short-chain fatty acids (SCFA) confer extend to the long-range modulation of gut-brain circuits governing appetite, the promotion of lipid oxidation and storage adjustments, as well as virus- and microbe - triggered inflammatory responses. Among the SCFA, butyrate has emerged as a critical regulator of metabolic health, enhancing insulin sensitivity, reinforcing intestinal epithelial integrity, and curbing systemic inflammation. Its combined actions thus render butyrate essential for the both prevention and therapeutic control of obesity. On top of that, these acids act as an intermediary between the digestive tract and the brain, influencing the neuroendocrine systems that regulate satiety. These metabolites, particularly butyrate, are poised to become nutritionally based therapies against excessive adiposity targets due to their effect trajectory [12].

Paediatric populations provide compelling evidence for this need for investigation since childhood obesity causes long-term changes to metabolic flexibility and developmental pathways. This study investigates the function of SCFA, synthesized by gut microbiota, as endogenous signaling molecules within dietary regimens designed for the treatment of pediatric obesity [3]. Elucidation of these processes is anticipated to provide a mechanistic framework for leveraging SCFA in the targeted manipulation of metabolic pathways implicated in excessive adiposity. Complementarily, the protocol was to compare the

magnitude and clinical relevance of SCFA synthesis evoked by several evidence-based nutritional regimens, allowing for a systematic appraisal of their role in managing childhood obesity. By synthesizing current clinical evidence and delineating a conceptual algorithm for short-chain fatty acid-oriented nutritional therapy, this investigation aimed to elucidate the mechanisms by which tailored interventions may address pediatric obesity through in situ modulation of the enteric microbiome. The overarching aim is to consolidate the evidence base surrounding the physiological and pathophysiological functions of short-chain fatty acids and, by such means, to refine prognostic and therapeutic benchmarks that inform subsequent clinical and population-level programs in the management of excess weight during childhood.

Literature review

Gut Microbiota and Its Role in Metabolic Health

The gut microbiome is crucial for the control of metabolic diseases in part because of its high diversity and dynamic interactions with host immunity, metabolism, and physiology. The gut microbiota is composed of trillions of microorganisms, including bacteria, viruses, fungi, and archaea, which contribute to various aspects of health [13]. Moreover, one of the primary roles of gut microbiota is to ferment dietary fibers, producing SCFA that are crucial for maintaining energy balance, regulating inflammation, and preserving the gut barrier [11].

Metabolically healthy subjects have balanced microbiota levels that help keep metabolic diseases (e.g., obesity, type 2 diabetes, and cardiovascular disease) at bay. A variety of metabolic perturbations, including obesity, have been correlated with an impaired gut microbiota community, identified as dysbiosis. Within obesity, alterations in microbial structure can consequently influence energy extraction during digestion, the deposition of triglycerides, and perceptions of satiety. A preserved, functionally diverse microbiome is therefore posited as a prerequisite for sustaining metabolic balance; conversely, sustained microbiome disturbance is recognized as a potential driver of obesity and its comorbid conditions [18,19].

Mechanisms of SCFA in Obesity and Metabolic Diseases

Gut microbiota ferment complex dietary fibers to produce SCFA, notably acetate, propionate, and butyrate, whose rapid absorption into the circulation initiates multiple metabolic programs with implications for obesity management [20]. Among these, modulation of energy homeostasis commands

particular attention. SCFA promote the secretion of enteroendocrine peptides, chiefly glucagon-like peptide-1 (GLP-1), thereby amplifying satiety, attenuating appetite, and enhancing postprandial insulin biosynthesis. Sequentially, butyrate exerts a distinctive insulin-sensitizing effect coupled with augmented mitochondrial fatty acid oxidation, thereby optimizing substrate partitioning [19].

Concomitantly, SCFA exert signaling across the gut-brain axis, refining the integrated ensemble of neuroendocrine responses that govern hunger and fullness. SCFA are also known to have anti-inflammatory properties that reduce chronic low-grade inflammation often associated with obesity and metabolic diseases. All of these mechanisms position SCFA as viable therapeutic targets in the prevention and treatment of obesity and other metabolic diseases.

Nutritional Therapy Approaches in Pediatric Obesity

Nutritional therapy is fundamental in managing obesity in children, as emphasized by dietary modification, which leads to weight management and a decreased risk of metabolic disease in children. A balanced eating pattern that includes whole foods, fruits, vegetables, whole grains, and fiber is essential to stimulate healthy gut microbiota and reduce weight gain in the youth population. In fact, fiber is a necessary substrate for gut bacteria to stimulate the mechanical production of beneficial SCFA [19].

Different dietary patterns examined for the management of pediatric obesity include low-fat, low-carbohydrate, and Mediterranean diets, with evidence gradually supporting the benefits of a high-fiber, low-processed diet for modulating gut microbiota and weight management [4]. Besides changes in food intake, nutritional therapy may include individualized interventions, in which plans are tailored to each individual's metabolic profile and gut microbiota composition [14]. The growing interest in supporting the growth of beneficial bacteria and the production of SCFAs has drawn attention to the potential inclusion of prebiotics and probiotics as nutritional therapies [7,8]. In general, pediatric obesity nutritional therapy approaches promote not only the reduction of excess body weight and apparent concern of weight gain, but also long-term metabolic health by optimizing SCFA production and creating a healthier microbiome [5].

Proposed Model

Conceptual Framework for SCFAs in Pediatric Obesity

The theoretical construct linking SCFA to pediatric obesity foregrounds the reciprocal influences of the

intestinal microbiota, habitual dietary intake, and overall metabolic status. Key to the construct is the observation that intestinal fermentative microbiota process dietary fiber, thereby generating significant quantities of SCFA, notably acetate, propionate, and butyrate. Although these metabolites primarily furnish energy to colonic epithelial cells, they further exert widespread modulation of metabolic processes beyond the gastrointestinal tract. Within the context of pediatric adiposity, the framework posits that microbiota dysbiosis—most commonly characterized by a reduced relative abundance of SCFA-secreting taxa—may engender a secondary perturbation of host metabolism, manifesting as augmented adipose deposition and perturbed neuroendocrine circuitry governing ingestive behavior. It also postulates that diet interventions enhancing the growth of SCFA-producing bacteria, such as high-fiber diets, can restore metabolic balance, improve insulin sensitivity, and decrease inflammation. In addition, SCFA are postulated to impact the gut-brain axis, modulating appetite signals, which are essential for weight management. Therefore, this framework provides the basis for testing SCFA as mediators of nutritional therapy in pediatric obesity.

Hypotheses on the Role of SCFAs in Metabolic Regulation

Deferred from the conceptual framework, several reasoned suppositions regarding the influence of SCFA upon metabolism, and their possible contribution to the etiology of pediatric obesity, become evident. Foremost, and with a particular emphasis upon butyrate, a mediating action upon gene transcription targeting glucose homeostasis in both hepatic and myocytic tissues is postulated, such transcriptional modulation yielding increased glucose uptake in the aforementioned tissues and a concomitant mitigation of the insulin-resistant state that has long characterized the pediatric obese population. Secondly, a modulatory action upon lipid compartmentalization is suggested, the inference grounded upon clinical evidence that fatty acid oxidation and the volumetric redistribution of adipose depot size may both positively respond to SCFA exposure.

First, SCFA, especially butyrate, are hypothesized to have a significant role in improving insulin sensitivity, through altering gene expression involved in glucose metabolism in liver and muscle cells, resulting in improved glucose uptake and reduced insulin resistance, both of which are reported occurrences in children who are obese. Second, SCFA also support adiposity, reportedly through increasing fatty acid oxidation and lowering adipose tissue.

Proposed Nutritional Interventions Based on SCFA Production

The suggested nutritional strategies for childhood obesity are directed toward maximization SCFA production through adjustments to diet. A high-fiber intake diet that contains fruits, vegetables, legumes, and whole grains is considered an important strategy for enhancing SCFA through dietary adjustments because these foods are the substrates for gut bacteria to produce SCFA. In addition to dietary strategies to enhance SCFA production, the introduction of prebiotics and probiotics could also support the growth of beneficial bacteria producing SCFA for therapeutical purposes.

Prebiotics, like inulin and fructooligosaccharides, can be added to the diet to preferentially stimulate SCFA-producing microbes, while probiotics can be used to replace beneficial strains of bacteria. Additionally, fermented foods have been proposed as an intervention because live bacteria are present in fermented foods and contribute to a healthy gut microbiome and SCFA production. The potential benefit of increasing metabolic health outcomes may be possible through the three dietary interventions, personalized to the individuals gut microbiota composition, including insulin sensitivity, fat storage, and appetitive behavior.

Mathematical Model

A simple mathematical model to describe the relationship between dietary fiber intake (F), SCFA production (S), and metabolic health (M) in pediatric obesity could be represented in equation 1:

$$M = \alpha \cdot S - \beta \cdot F + \gamma \quad (1)$$

Where:

- M represents the metabolic health indicator (e.g., insulin sensitivity, fat oxidation rate),
- S is the SCFA production, which is influenced by dietary fiber intake F,
- α is a constant representing the positive impact of SCFAs on metabolic health,
- β represents the negative impact of low fiber intake on metabolic health, and
- γ is a baseline metabolic health constant, which accounts for other genetic and environmental factors.

This model assumes a direct relationship between fiber intake and SCFA production, which in turn influences metabolic health. As dietary fiber increases, SCFA production rises, leading to improved metabolic health.

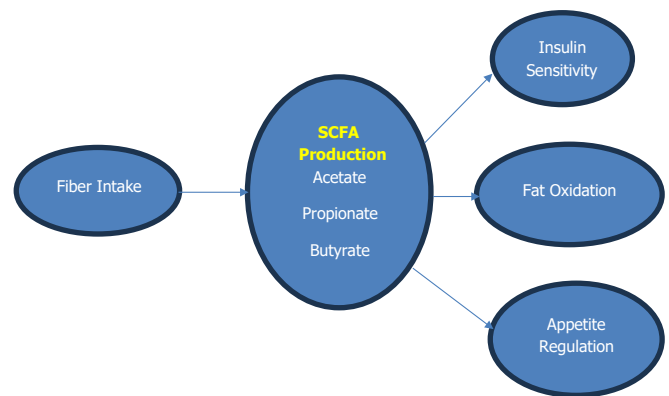


Figure 1. Impact of SCFA Production on Pediatric Obesity Metabolic Health. Source: Own authorship.

In children who are overweight, there is a suggested connection between the ingestion of dietary fibre, the synthesis of colonic SCFA, and metabolic health indices (Figure 1). Metabolic health outcomes, such as improved insulin sensitivity, increased fatty acid oxidation, and better hunger management, are thought to be improved by increasing SCFA generation, which is predicted to occur with an increase in fibre consumption. The model will also show directional arrows and visual nodes connecting the composition of the gut microbiota, concentrations of SCFA, and metabolic health markers to capture the direction and reciprocity of these processes.

Summary of Key Findings from Clinical Studies

This study's sample size was determined by reviewing previous research that examined the correlation between SCFA and obesity indices in similar paediatric groups. To guarantee that the chosen sample had sufficient statistical power to identify significant differences in SCFA levels among groups, a power analysis was carried out. The computation followed the accepted biomedical practice of using a 0.05 significance criterion and an 80% statistical power. Although this strategy did not eliminate the risk of Type II error, it did increase the likelihood that the sample size would be sufficient to generate reliable and statistically significant results.

Researchers have uncovered processes related to metabolic regulation by studying the role of SCFA in childhood obesity. Observational studies show that decreased SCFA synthesis is a common trait in obese children, which is associated with changes in the makeup of gut microbes. Results from intervention studies show that increasing dietary fibre intake significantly increases SCFA pools, with butyrate, propionate, and acetate concentrations being the most important metabolites. Increasing data suggests a potential treatment pathway in paediatric obesity

originating from the microbiome, which is associated with these SCFA elevations and better insulin sensitivity in the liver and peripheral areas, less visceral adipose accumulation, and better regulation of caloric intake.

Additionally, some studies have shown that SCFA can reduce the high levels of proinflammatory cytokines in the blood, which is a hallmark of childhood obesity. As a result, our results provide support for the idea that SCFA may have a beneficial effect on metabolic pathways that are dysregulated by extra fat, which could make them a useful supplementary treatment or preventative strategy for the clinical management of childhood obesity. The function of SCFA in improving obesity-related dysbiosis is further supported by research showing that they increase intestinal eubiosis by strengthening the gastrointestinal epithelial barrier, an adaptation that is often reduced in obese populations.

Impact of SCFAs on Pediatric Obesity Outcomes

As researchers look for ways these metabolites control energy homeostasis and weight gain, they have ramped up their studies into the relationship between SCFA and childhood obesity and its consequences. Butyrate is a promising member of this subgroup because it may exert anti-adiposity benefits via regulating insulin sensitivity in the liver and fat oxidation in adipose tissue. Increased circulating SCFA concentrations in children are associated with improved glucose metabolic profiles, decreased visceral adipose tissue, and an adverse phenotype that is strongly associated with the development of type 2 diabetes mellitus, according to meta-analyses and cohort studies.

The function of SCFA in the signalling pathways that regulate hunger and fullness has recently been the subject of research into their potential as modulators of the gut-brain axis. SCFAs stimulate the enteroendocrine secretion of satiety-regulating peptides, notably glucagon-like peptide-1 (GLP-1) and peptide YY (PYY), leading to enhanced fullness, reduced caloric intake, and the consequent attenuation of weight gain. Furthermore, these fatty acids exert anti-inflammatory effects by reducing gut-derived pro-inflammatory mediators, a biomechanical alteration of importance given the elevated systemic inflammation characteristic of childhood obesity, which undermines insulin sensitivity. Coupling appetite regulation with inflammation attenuation, SCFAs emerge as promising agents, influencing not only weight trajectory but also the underlying metabolic dysfunction observed in pediatric populations.

Implications of SCFAs for Nutritional Therapy and Future Treatment Strategies

The corpus of SCFA research within pediatric obesity furnishes persuasive implications for the design of nutritional therapy. Recent investigations demonstrate that dietary interventions expressly aimed at amplifying SCFA synthesis afford both therapeutic and preventive leverage against the development and progression of pediatric obesity. Foods characterized by dense fiber content, concomitant prebiotic substrates, or probiotic microorganisms exert a synergistic influence, thereby elevating SCFA yields and, by extension, modulating key metabolic outcomes. Such interventions may lend themselves to be a more sustainable and non-pharmacological approach to managing obesity in children.

Customized nutrition that considers a person's gut microbiota composition and fiber consumption has the potential to further improve the efficacy of SCFA-based therapies. In addition, future therapies may involve SCFA supplementation, although this remains to be validated clinically. As the field continues to study particular strains of microbes that produce SCFA and the possible genetic effects these strains may have, therapies may be personalized based on the intention to increase beneficial microbes that produce SCFA. In summary, SCFA provide an exciting target for nutritional therapies to mitigate pediatric obesity with the potential for novel microbiome-based interventions.

Table 1 provides a summary of the salient outcomes from clinical studies on the role of SCFAs in pediatric obesity. The summary addresses important information such as study design, sample size, the SCFA (dietary fiber, prebiotics, and probiotics) intervention, and outcomes focusing on metabolic health, insulin sensitivity, fat mass, and inflammation. The table compares the effectiveness of interventions designed to increase SCFA production and improve obesity-related outcomes in children.

Study	Intervention Type	SCFA Levels	Outcome	Sample Size	Duration	Key Findings
Study 1	High-Fiber Diet	Increased acetate, propionate, butyrate	Improved insulin sensitivity, reduced storage	50	12 weeks	Significant reduction in visceral fat and improved glucose metabolism
Study 2	Probiotic Supplementation	Increased butyrate	Enhanced appetite regulation, improved barrier	40	8 weeks	Decreased hunger and better gut health
Study 3	Prebiotic Supplementation	Increased acetate and propionate	Decreased inflammation, improved metabolic health	60	10 weeks	Lower levels of pro-inflammatory cytokines

Table 1 Clinical Studies on SCFAs and Pediatric Obesity. Source: Own authorship.

The correlation between SCFA levels (acetate, propionate, and butyrate) and critical metabolic outcomes in childhood obesity, namely insulin sensitivity and fat oxidation, is seen in Figure 2. Low, medium, and high SCFA levels are shown on the x-axis, with insulin sensitivity and fat oxidation percentages shown on the y-axis. The red line represents fat oxidation, while the blue line represents insulin sensitivity. Insulin sensitivity and fat oxidation both rise with increasing SCFA concentrations, as seen in the figure. This pattern lends credence to the idea that SCFA could have a positive impact on metabolism and could be useful in the treatment of childhood obesity.

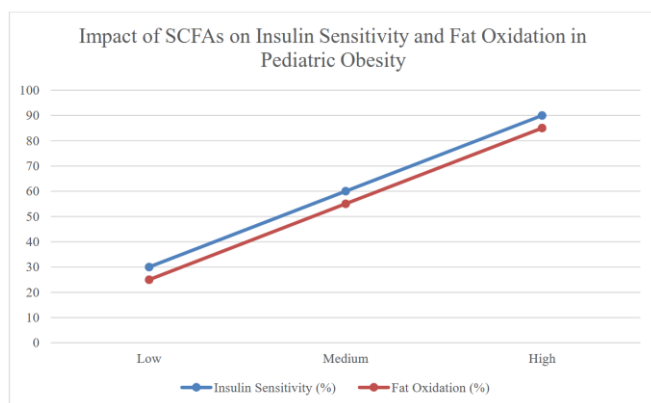


Figure 2. Impact of SCFAs on Insulin Sensitivity and Fat Oxidation in Pediatric Obesity. Source: Own authorship.

Study Limitations

Results may not be comparable or applicable to a broader population due to differences in methodology, sample size, and participant characteristics among the studies included in this analysis. Limited understanding of metabolic consequences over the long term resulted from the majority of studies' reliance on indirect short-chain fatty acids assays and short-term data. Diet, heredity, and antibiotic use are all potential confounding factors that were not adequately controlled for. Studies reporting positive findings may have been skewed by publication bias. To confirm and build upon these findings, future studies should use longitudinal designs and standardized methodologies.

Conclusion

The short-chain fatty acids acetate, propionate, and butyrate, which are produced by the gut microbiota, have a major impact on the metabolic consequences linked to childhood obesity, according to recent research. In children, there is a correlation

between higher dietary fiber intake and enhanced insulin sensitivity, less systemic inflammation, and lower visceral fat storage, according to both clinical and preclinical findings. These results lend credence to the idea that pediatric nutritional therapy could benefit from dietary changes that target short-chain fatty acids, such as those that emphasize high-fiber, prebiotic, and probiotic interventions. However, these results cannot be applied to a broader population due to study design heterogeneity, small sample sizes, and variability in microbiome composition among children. The best way to determine long-term effectiveness, ideal food compositions, and causal pathways is with more controlled longitudinal research. Optimizing short-chain fatty acids synthesis and metabolic benefit by personalized nutritional interventions informed by microbial profile is an area that needs further investigation in the future. Ultimately, it is essential to approach the incorporation of short-chain fatty acids into therapeutic diets with caution, relying on actual evidence instead of theoretical speculation, even though short-chain fatty acids are a biologically reasonable supplement for treating pediatric obesity.

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Data Sharing Statement

Upon reasonable request, the corresponding author can provide all the data that support the conclusions of this study. Publicly available resources like PubMed, ScienceDirect, and Google Scholar house the majority of the research publications that the study relied on. In this investigation, no new datasets were created or examined.

Conflict of Interest

The authors declare no conflict of interest.

Similarity Check

It was applied by Ithenticate®.

Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

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