

Gut Microbiota and Pediatric Malnutrition: Updates on Mechanisms, Interventions, and Clinical Perspectives

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Abstract

Introduction: Pediatric malnutrition remains a global challenge with repercussions for growth, neurodevelopment, and immunity. The gut microbiota has emerged as a key determinant in the pathophysiology of malnutrition, integrating the axes of nutrition, immunity, and inflammation.

Objective: To synthesize recent advances on the role of the gut microbiota in pediatric malnutrition, highlighting pathophysiological mechanisms, therapeutic interventions, and perspectives in precision nutrition.

Methods: Narrative review of literature indexed in international databases (PubMed, Scopus, Web of Science, Cochrane Library) and institutional sources from 2018 to 2025, including clinical studies, animal models, and intervention trials.

Results: Recent findings demonstrate an immature microbiota in severely malnourished children, characterized by reduced bacterial diversity, depletion of *Faecalibacterium* and *Bifidobacterium*, and predominance of pro-inflammatory species. This dysbiosis correlates with growth faltering (stunting), environmental enteropathy, and systemic endotoxemia. Decreased production of short-chain fatty acids and impaired vitamin biosynthesis have also been documented. Innovative interventions such as microbiota-directed complementary foods (MDCF) and fecal microbiota transplantation show promise, albeit with context-dependent variability.

Conclusion: Integrating nutritional strategies with microbiome modulation is a promising pathway to address childhood malnutrition. The development of microbiome-based biomarkers and precision therapies opens new opportunities for prevention, early diagnosis, and effective treatment.

Keywords: Pediatric Malnutrition; Gut Microbiota; Environmental Enteropathy; Stunting; Short-Chain Fatty Acids; Precision Nutrition, Probiotics

Abbreviations

FMT: Fecal Microbiota Transplantation; LAZ: Length-for-Age Z Score; LPS: Lipopolysaccharides; MDCF: Microbiota-Directed Complementary Foods; MeSH: Medical Subject Headings; WHO: World Health Organization; RCT: Randomized Controlled Trial; SCFAs: Short-Chain Fatty Acids; UNICEF: United Nations International Children's Emergency Fund; WASH: Water, Sanitation and Hygiene

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Introduction

Pediatric malnutrition continues to be a leading cause of childhood morbidity and mortality worldwide, especially in low- and middle-income countries, where it impairs growth, neurodevelopment, and immune maturation in millions of children each year [1]. Although traditionally linked to inadequate intake of macro- and micronutrients, recent evidence has identified a central component in its pathophysiology: disruption of the gut microbiota [2].

The gut microbiota plays a crucial role in nutrient digestion, vitamin and regulatory metabolite synthesis, epithelial barrier development, and immune education [3]. In malnutrition, multiple studies document an immature microbiota characterized by lower bacterial diversity and reductions in beneficial taxa such as *Faecalibacterium*, *Bifidobacterium*, and *Ruminococcus*, together with increased abundance of pathogenic or pro-inflammatory bacteria, including members of the Enterobacteriaceae and *Gammaproteobacteria* [4-6]. This “microbial immaturity” has become a reproducible biomarker of nutritional risk in both acute and chronic malnutrition [7] (See figure 1).

Microbial shifts are not merely observational. Longitudinal studies in Malawi (including work associated with Salk Institute collaborators) show that specific microbiome profiles correlate with the length-for-age Z score (LAZ), and that certain bacterial genomes predict improvement or deterioration in child growth [8,9]. These findings suggest the feasibility of microbiome-based biomarkers to identify at-risk children before malnutrition becomes clinically evident [10].

Malnutrition is also associated with environmental enteropathy, a condition characterized by duodenal dysbiosis, mucosal alterations, and persistent inflammatory signaling that compromise nutrient absorption even when diet is adequate [11]. Increased intestinal permeability facilitates translocation of LPS into the bloodstream, triggering systemic inflammatory responses and chronic endotoxemia, with increased energetic costs for the immune system [12,13].

Metabolically, there is a documented reduction in SCFAs-metabolites with nutritive, anti-inflammatory, and immunomodulatory functions-along with altered biosynthesis of B-vitamins, lipid metabolism, and utilization of non-digestible carbohydrates [14-16]. The loss of these microbial products limits the direct energy supply to the intestinal epithelium, aggravates inflammation, and perpetuates the malnutrition cycle.

Growing understanding of the malnutrition-microbiota interaction has spurred innovative therapies. MDCF are designed to stimulate beneficial bacteria and have yielded promising improvements in intestinal biomarkers and growth parameters [17]. Probiotics, prebiotics, and synbiotics show variable effects depending on geography, strains used, and malnutrition severity [18]. Experimental approaches-such as FMT and engineered microbial strains-are under exploration but face logistical and ethical constraints [19].

Significant challenges persist: population and environmental heterogeneity, difficulties establishing causality in humans, and the need to define what constitutes a “healthy microbiota” across diverse cultural and epidemiologic contexts [20,21]. Implementation also requires resources, cultural acceptance, and public policies that integrate sanitation, safe water, infection control, and adequate nutrition [22].

Within this framework, incorporating the microbiota into the study and management of pediatric malnutrition offers a transformative perspective for mechanistic understanding and for preventive and therapeutic strategies that combine conventional nutrition with microbial modulation.

Objectives of the Study

General objective: To synthesize recent advances on the role of the gut microbiota in pediatric malnutrition, emphasizing pathophysiological mechanisms, therapeutic interventions, and perspectives within precision nutrition.

Specific objectives:

1. Analyze evidence on microbial immaturity and its relationship with growth and stunting in malnourished children.
2. Describe pathophysiological mechanisms linking intestinal dysbiosis with inflammation, increased permeability, metabolic alterations, and associated complications such as hemoconcentration.
3. Review major microbiota-based therapeutic interventions (MDCF, probiotics, synbiotics, and FMT) and their clinical outcomes.
4. Identify contextual factors and public-health determinants shaping the interaction between microbiota and pediatric malnutrition.
5. Explore clinical and public-health implications of integrating microbiome modulation into prevention, diagnosis, and treatment strategies for childhood malnutrition.

Materials and Methods

A narrative review of the scientific literature published from January 2018 to August 2025 was conducted to synthesize current knowledge on the relationship between pediatric malnutrition and the gut microbiota.

Search strategy

Sources included international biomedical databases (PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library), grey literature from specialized organizations (WHO, UNICEF, Biocodex Microbiota Institute), and open-access academic repositories. Combinations of keywords and MeSH terms in English and Spanish were used: “pediatric malnutrition”, “gut microbiota”, “stunting”, “environmental enteropathy”, “short-chain fatty acids”, “microbiota-directed complementary foods”, “probiotics”, “fecal microbiota transplantation”, and equivalents in Spanish.

Inclusion criteria

1. **Population:** Studies in children <18 years with documented acute or chronic malnutrition or nutritional risk.
2. **Design:** Clinical trials, longitudinal cohort studies, systematic reviews and meta-analyses, and animal studies with translational relevance.
3. **Content:** Investigations of microbiota composition/function, mechanisms linking microbiota and malnutrition, or microbiome-based interventions.
4. **Language:** English or Spanish.

Exclusion criteria

1. Adult studies or mixed-population studies without stratified pediatric analysis.
2. Studies on malnutrition secondary to chronic non-infectious diseases (e.g. cancer, cystic fibrosis) without linkage to the gut microbiota.
3. Narrative reviews lacking primary references or methodological rigor.
4. Publications with outcomes unrelated to nutrition, growth, or the microbiota (e.g. purely *in-vitro* studies).

Study selection

The initial search identified ~1,230 records. After deduplication, 642 titles/abstracts were screened; 128 full texts were reviewed; 74 studies met inclusion criteria.

Data synthesis

Selected studies were grouped into four thematic domains:

- Microbial immaturity and linear growth (stunting markers, bacterial diversity).
- Pathophysiological mechanisms (dysbiosis, barrier function, inflammation, microbial metabolites).
- Therapeutic interventions (MDCF, probiotics, synbiotics, fecal microbiota transplantation).
- Contextual and public-health determinants (breastfeeding, infections, sanitation, genetic/epigenetic factors).

Data extraction was performed independently by two reviewers; discrepancies were resolved by consensus. No meta-analysis was undertaken due to heterogeneity; findings are presented descriptively and critically, emphasizing recent contributions and clinical/public-health implications.

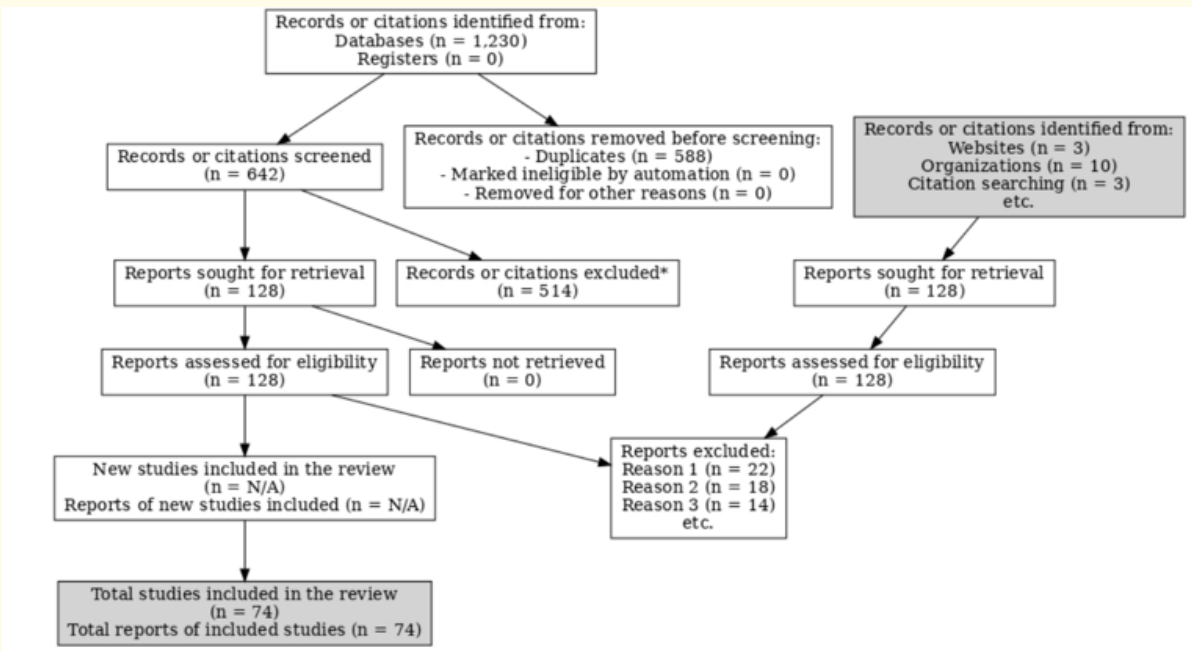


Figure 1: PRISMA flow diagram of the study selection process.

Source: Authors.

Results

Seventy-four studies were included: clinical trials, longitudinal cohorts, translational animal models, and recent systematic reviews. Findings are presented across four thematic areas: immature microbiota and growth, pathophysiological mechanisms, microbiota-based therapeutic interventions, and contextual modulatory factors.

Microbial immaturity and child growth (stunting/low LAZ)

Children with severe malnutrition exhibit an “immature” gut microbiota with low diversity and depletion of beneficial taxa (*Faecalibacterium*, *Bifidobacterium*, *Ruminococcus*), alongside increased pro-inflammatory species (*Enterobacteriaceae*, *Gammaproteobacteria*) [1,2]. This microbial immaturity is a reproducible biomarker of risk in both acute and chronic malnutrition [3].

In longitudinal cohorts from Malawi, the microbial profile was associated with length-for-age Z scores (LAZ), with certain bacterial genomes predicting either improvement or deterioration in growth [4]. These findings suggest that the microbiome could serve as an early marker to identify children at risk of stunting before clinical manifestations become irreversible [5].

Intestinal dysbiosis in malnutrition

The literature documents that pediatric malnutrition is associated with a profound alteration of the intestinal microbiome, characterized by decreased levels of beneficial commensal bacteria (*Bifidobacterium*, *Lactobacillus*) and increased abundance of potentially pathogenic bacteria (*Enterobacteriaceae*) [1,2,5]. This dysbiosis promotes low-grade chronic mucosal inflammation, increased intestinal permeability (“leaky gut”) with translocation of endotoxins (LPS) into the circulation, and impaired nutrient absorption [6-8].

Pathophysiological mechanisms

The included studies documented several key mechanisms linking malnutrition and intestinal dysbiosis:

- **Environmental enteropathy:** In children with chronic malnutrition and growth retardation, duodenal dysbiosis and alterations of the intestinal mucosa have been detected, including a compromised epithelial barrier, increased antimicrobial peptides, and persistent inflammatory signals. This enteropathy may impair nutrient absorption even when the diet improves [6].
- **Inflammation, intestinal permeability, and endotoxemia:** Animal models (and some human data) indicate that malnutrition-particularly acute or moderate-leads to intestinal barrier dysfunction, proliferation of pro-inflammatory bacteria (e.g. *Gammaproteobacteria*), and translocation of lipopolysaccharides (LPS) into the bloodstream, triggering systemic inflammatory responses that exacerbate malnutrition [7].
- **Deficit of microbial metabolites:** A reduction in short-chain fatty acids (SCFAs)-essential for epithelial nutrition, immune regulation, and energy metabolism-has been documented [8].
- **Alterations in vitamin and lipid metabolism:** Loss of the microbiota’s capacity to synthesize B-complex vitamins, ferment fibers, support lipid absorption, and utilize non-digestible carbohydrates has been observed [9].

Malnutrition and hemoconcentration

In malnourished children, elevated hematocrit is often explained by hemoconcentration secondary to dehydration (due to diarrhea, vomiting, or insufficient intake) [17]. During recovery phases, iron and folate repletion reactivate erythropoiesis, leading to a transiently elevated hematocrit while plasma volume remains low [17]. In cases of kwashiorkor, the resolution of edema through diuresis may also produce a falsely elevated hematocrit as a result of plasma volume contraction [12,13].

The link between dysbiosis and hemoconcentration (See figure 2).

Intestinal dysbiosis contributes to the persistence of diarrhea and environmental enteropathy, conditions that promote digestive losses of water and electrolytes [5,7]. These losses lead to dehydration and, consequently, hemoconcentration. In addition, dysbiosis-mediated intestinal inflammation alters sodium and water transport, further exacerbating fluid loss [6-8]. As a result, malnourished children with intestinal dysbiosis are at increased risk of chronic diarrhea and hemoconcentration with elevated hematocrit, while simultaneously presenting deficiencies of iron, folate, and proteins that induce true anemia [11,12,17].

Malnutrition impairs the microbiota (dysbiosis), perpetuating diarrhea and intestinal inflammation. This promotes fluid loss and hemoconcentration, which may lead to a misleading elevation of hematocrit. At the same time, chronic micronutrient deficiencies often induce anemia, so in these patients hematocrit reflects a dynamic interaction between anemia and hemoconcentration [5,7,13,17].

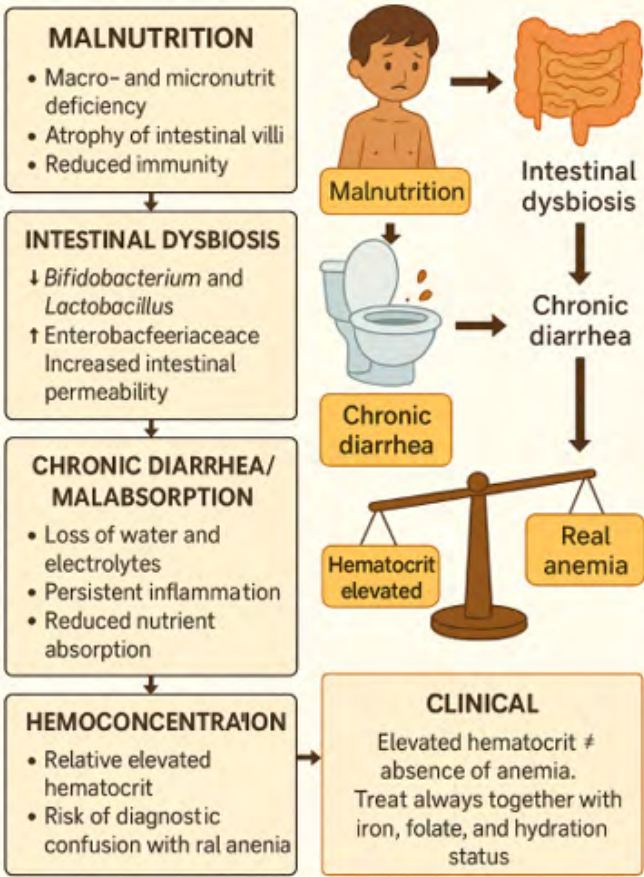


Figure 2: Relationship between malnutrition, intestinal dysbiosis, and hemoconcentration in pediatrics. Source: Authors. The scheme depicts how malnutrition drives dysbiosis, low-grade inflammation, increased permeability, and impaired nutrient absorption, favoring persistent diarrhea and fluid loss that culminate in hemoconcentration with elevated hematocrit. Concurrent micronutrient deficiencies typically cause true anemia, creating a dynamic interplay between hemoconcentration and anemia.

Microbiota-based interventions

Three major strategies have been identified in the literature: (See table 1)

1. **Microbiota-directed complementary foods (MDCF):** Randomized controlled trials published in the *New England Journal of Medicine* demonstrated that these foods, designed to stimulate beneficial bacteria, improve intestinal and growth biomarkers [10].
2. **Probiotics, prebiotics, and synbiotics:** Used in various contexts to restore bacterial diversity, strengthen the intestinal barrier, and reduce inflammation. However, the magnitude of effect varied depending on the strains employed, dosage, and study population [11].

3. **Experimental treatments:** Approaches such as fecal microbiota transplantation (FMT) and microbial strain engineering have shown encouraging results in animal models and pilot studies, although they still lack large-scale clinical validation [12].

Strategy	Examples	Recent observations
Microbiota-directed complementary foods (MDCF)	Studies in the <i>New England Journal of Medicine</i> have designed formulas that promote the growth of <i>Bifidobacterium</i> and <i>Faecalibacterium</i> .	Associated with improvement in biomarkers of intestinal integrity, increased short-chain fatty acids (SCFAs), and partial recovery of linear growth. Their effect depends on cultural context and local diet.
Probiotics / Prebiotics / Synbiotics	Strains such as <i>Lactobacillus rhamnosus</i> GG, <i>Bifidobacterium longum</i> ; fermentable fibers (inulin, FOS); synbiotic combinations.	Have shown benefits in reducing persistent diarrhea, partial restoration of the intestinal barrier, and modulation of the inflammatory response. Results are heterogeneous depending on strain, dose, and study population.
Experimental treatments	Fecal microbiota transplantation (FMT); genetic engineering of microbial strains; adaptation of traditional diets.	Pilot studies and animal models suggest restoration of bacterial diversity and metabolic improvement. However, ethical, logistical, and safety limitations prevent their routine clinical application.

Table 1: Microbiota-based therapeutic strategies in pediatric malnutrition: examples and recent observations.

Source: Authors.

Recently supported interventions (See table 2)

Recent studies have reinforced the potential of microbiota-targeted strategies in nutritional recovery. Microbiome-directed nutrition, including complementary foods such as MDCF and local combinations of legumes and cereals, has been shown to improve intestinal biomarkers and growth in low- and middle-income countries, underscoring the importance of adapting these matrices to Latin American diets [1,12]. Continued breastfeeding during the introduction of complementary feeding was associated with a more favorable microbial succession in Peruvian cohorts [13]. The use of prebiotics, probiotics, and synbiotics has produced heterogeneous results, but with more consistent benefits when integrated into appropriate dietary guidelines and in contexts with improved sanitation [14-16,18]. In parallel, the reduction of ultra-processed foods in early childhood has been linked to unfavorable microbial profiles in regional birth cohort studies [20,21]. Finally, water, sanitation, and hygiene (WASH)-centered interventions have been shown to reduce environmental enteropathy, representing a central modulator of the microbiota-nutrition axis [5-7,22].

Intervention	Age group	Objective	Practical strategy	Recent evidence
Exclusive breastfeeding	0-6 mo	Immune maturation and microbiome	Promote exclusive breastfeeding, support for mothers	Peru cohorts, 2024
Breastfeeding + complementary feeding	6-24 mo	Microbial diversification	Introduce foods rich in fiber and local protein sources	<i>Nature</i> 2023
Adapted MDCF	6-24 mo	Correct microbial immaturity	Use of legumes + whole grains	<i>NEJM</i> 2020, synthesis 2024
Micronutrient supplements	6-59 mo	Reduce Fe/Zn deficiency	Iron + zinc in moderate malnutrition	Mexico guidelines, 2023

Probiotics / Synbiotics	>6 mo	Modulate dysbiosis and diarrhea	<i>Lactobacillus</i> GG, <i>Bifidobacterium</i>	Review 2023
WASH (water, sanitation, hygiene)	All	Prevent environmental enteropathy	Safe water, latrines, hygiene	EED review 2022
Family education (diet)	All	Prevent dysbiosis from ultra-processed foods	Household counseling	Save the Children Mexico, 2025

Table 2: Integrated interventions in pediatric malnutrition by age group, objectives, and recent evidence.

Source: Authors.

Modulating factors and contextual determinants (See figure 3)

The interaction between the microbiota and nutritional status was influenced by multiple external determinants:

- **Diet:** Exclusive breastfeeding was associated with a more diverse and protective microbial profile [8,13].
- **Infections and recurrent diarrhea:** Contributed to dysbiosis and increased the risk of malnutrition [14,18].
- **Environmental conditions:** Lack of sanitation, access to safe water, and hygiene promoted environmental enteropathy [6,7,15,22].
- **Genetic and epigenetic factors:** Some studies indicated that differences in host immune response and metabolism influence the interaction with the microbiota [9,16].

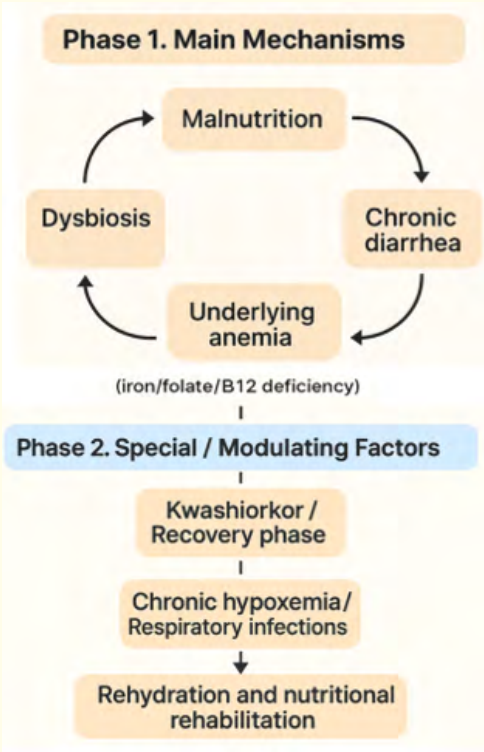


Figure 3: Core mechanisms and modulating factors linking malnutrition, intestinal dysbiosis, and anemia.

Source: Authors.

Synthesis

Overall, pediatric malnutrition is not solely a nutrient-deficit state; it reflects a complex interplay among diet, microbiota, and the immune system. Microbial immaturity, chronic intestinal inflammation, and reduced beneficial metabolites are central drivers that perpetuate malnutrition.

Discussion

The findings of this review confirm that pediatric malnutrition is not solely the result of a caloric or protein intake deficit, but rather reflects a dynamic interaction among diet, the gut microbiota, the immune system, and the environment [1-3]. Recent evidence provides a conceptual framework in which microbial immaturity is established as a reproducible marker of nutritional risk, both in acute and chronic forms of malnutrition [4,5].

Immature microbiota as a clinical biomarker

Longitudinal studies conducted in high-burden malnutrition settings, such as Malawi, suggest that the composition and functionality of the microbiota precede or accompany linear growth faltering [6]. The identification of specific microbial genomes associated with improvement or deterioration in length-for-age scores opens the possibility of developing early diagnostic biomarkers, which could be applied in nutritional screening programs [7].

Key pathophysiological mechanisms

Malnutrition induces intestinal dysbiosis, leading to reduced production of beneficial metabolites, increased intestinal permeability, and chronic endotoxemia [8,9]. This vicious cycle contributes to systemic inflammation and diverts energy resources toward immune responses, thereby reducing the availability of energy for growth and tissue repair [10]. Likewise, alterations in vitamin synthesis and fiber fermentation highlight the importance of the microbiota as an essential metabolic cofactor [11].

Malnutrition and hemoconcentration

A frequently underestimated aspect is the alteration of hematocrit in malnourished children. Elevated hematocrit is often due to hemoconcentration secondary to dehydration, resulting from diarrhea, vomiting, or insufficient intake. During nutritional recovery, iron and folate supplementation can stimulate erythropoiesis, causing a transient rise in hematocrit while plasma volume remains reduced [17]. In kwashiorkor, resolution of edema through diuresis may also produce a falsely elevated hematocrit due to plasma volume contraction [12,13].

The link between dysbiosis and hemoconcentration

Intestinal dysbiosis and environmental enteropathy play a key role in this phenomenon. Both conditions favor persistent diarrhea and digestive losses of water and electrolytes [5-7], leading to dehydration and hemoconcentration [17]. In addition, dysbiosis-mediated intestinal inflammation interferes with sodium and water transport, exacerbating fluid loss [6-8]. In this context, malnourished children with dysbiosis are at increased risk of chronic diarrhea and hemoconcentration with elevated hematocrit, while simultaneously presenting deficiencies of iron, folate, and proteins that lead to true anemia [12,13,17]. Thus, hematocrit in these patients reflects a dynamic and complex interaction between anemia and hemoconcentration, complicating its clinical interpretation and requiring a comprehensive approach that considers both hematologic and hydration status [5,7,13,17].

Therapeutic interventions and translational potential

Microbiota-targeted strategies have shown promising, though heterogeneous, results. Microbiota-directed complementary foods (MDCF) represent an innovative advance, demonstrating improvements in intestinal biomarkers and growth in clinical trials [12].

However, their applicability requires adaptation to diverse cultural and economic contexts. Probiotics, prebiotics, and synbiotics have been tested in different settings, with variable effects depending on strains and dosages [13]. While some trials report benefits in restoring the intestinal barrier and reducing inflammation, the lack of standardization limits their routine use. More experimental interventions, such as fecal microbiota transplantation (FMT) and microbial strain engineering, still face ethical, logistical, and safety challenges [14].

Contextual and public health factors

The evidence reinforces that malnutrition must be addressed within a comprehensive public health framework, which includes:

- Promotion of exclusive breastfeeding, a cornerstone of a healthy microbiota during the first months of life [15].
- Prevention and timely treatment of infections and recurrent diarrhea, which perpetuate dysbiosis [16].
- Improvement of environmental conditions (safe water, sanitation, hygiene), essential to prevent environmental enteropathy [17].
- Consideration of genetic and epigenetic factors, which modulate host-microbiota interactions and may explain individual variability [18].

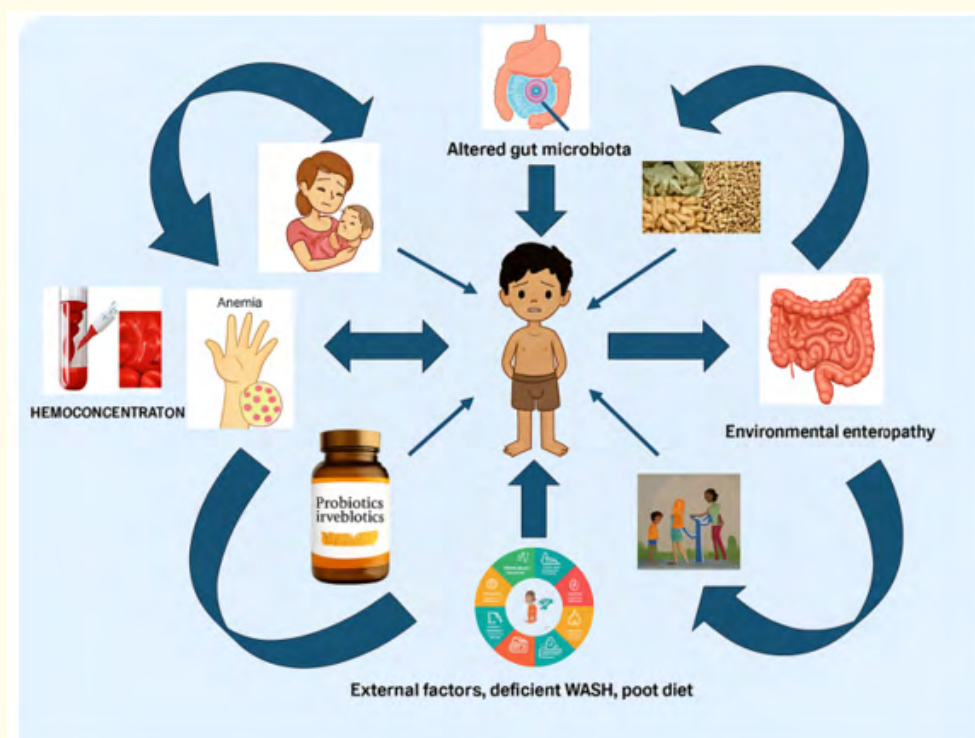


Figure 4: Integrative model of pediatric malnutrition: interaction among intestinal dysbiosis, hemoconcentration, environmental enteropathy, and external factors.

Source: Authors. Pediatric malnutrition is understood as the result of multiple interactions: intestinal dysbiosis, environmental enteropathy, hemoconcentration, and anemia, modulated by external factors such as poor diet and inadequate water, sanitation, and hygiene (WASH). Strategies such as breastfeeding, microbiota-directed complementary foods, and probiotics may help break this vicious cycle.

Remaining challenges

1. Heterogeneity of results according to region, diet, and socioeconomic context.
2. Causality vs. association: although animal models support causality, human data remain limited.
3. Definition of a “healthy” microbiota adapted to different settings, which complicates the standardization of interventions.
4. Scalability of innovative therapies, such as MDCF or FMT, which require resources, cultural acceptability, and risk management.

Clinical and public health implications

Recent evidence underscores the need for early interventions to prevent the progression of childhood malnutrition. Strategies such as exclusive breastfeeding, timely introduction of complementary feeding, and the design of microbiota-directed complementary foods (MDCF) have been shown to improve bacterial diversity, production of beneficial metabolites, and growth biomarkers [11,12,15,16,18]. Likewise, the concept of immature microbiota is being consolidated as a clinical biomarker of risk, opening the possibility of developing microbiome-based diagnostic tools to identify children at risk of stunting before the effects become irreversible [1,2,20].

At the public health level, environmental enteropathy remains a central obstacle to nutritional recovery, as it compromises nutrient absorption even in the presence of an adequate diet [5-7]. Hence the importance of comprehensive policies that include access to safe water, sanitation, and hygiene-elements shown to directly reduce the risk of malnutrition and improve child growth [22].

Finally, population differences in microbiome composition and functionality highlight the need to adapt interventions to each epidemiological and cultural context [21]. In this framework, integrating microbiome modulation with classical strategies for malnutrition prevention (breastfeeding, infection control, WHO guidelines) represents a promising pathway to achieve sustainable impact [13,14].

Future directions

Recent advances reinforce the need to integrate the gut microbiota into the management of pediatric malnutrition. The future perspective points toward precision nutrition, combining microbiome analysis, metabolomics, and genetic factors to design personalized therapies [19,21]. The development of microbial biomarkers will enable the early identification of at-risk children [12,20] while microbiota-targeted foods could become a central tool in nutritional recovery [11,12].

Conclusion

Current evidence demonstrates that pediatric malnutrition must be understood as a multifactorial condition in which the gut microbiota plays a central role in pathophysiology, clinical evolution, and response to nutritional interventions. The presence of an immature microbiota, the reduction of beneficial bacteria and essential metabolites, together with intestinal and systemic inflammation, configure a vicious cycle that perpetuates nutritional deficits and compromises child growth [1-5].

A particularly relevant finding is the link between dysbiosis, chronic diarrhea, and hemoconcentration. In malnourished children, hematocrit may increase due to dehydration and plasma volume contraction, while true anemia often coexists due to deficiencies in iron, folate, and proteins [12,13,17]. This makes hematocrit a dynamic and complex parameter, reflecting the interaction between anemia and hemoconcentration, and requires that its values be interpreted within the broader clinical context [5,7,13,17].

Recent advances-including the development of microbiota-directed complementary foods (MDCF), the use of probiotics and prebiotics, and the exploration of innovative strategies such as fecal microbiota transplantation-offer new therapeutic opportunities, although they require validation in multicenter studies and adaptation to diverse epidemiological contexts [6-10].

From a public health perspective, integrating microbiota modulation with traditional strategies for malnutrition prevention (breastfeeding, infection control, basic sanitation) is essential to achieve sustainable impact [11-13].

In conclusion, addressing childhood malnutrition must evolve toward a comprehensive and precision-based model that recognizes the gut microbiota as an active player in growth and nutritional recovery. This will enable progress toward more effective strategies for prevention, early diagnosis, and treatment, with the potential to transform child nutrition programs worldwide [14-16].

Bibliography

1. Li Y., *et al.* "Global burden of children and adolescents' nutritional deficiencies in 0-19 years: deaths and DALYs from 1990 to 2021". *Frontiers in Pediatrics* 13 (2025): 1583167.
2. Kumar M., *et al.* "Gut microbiota dysbiosis is associated with malnutrition in children". *Metabolic Engineering* 49 (2018): 128-142.
3. Iddrisu I., *et al.* "Malnutrition and the gut microbiota in children: a scoping review". *Nutrients* 13.8 (2021): 2727.
4. Hardjo J., *et al.* "Stunting and gut microbiota: a literature review". *Pediatric Gastroenterology, Hepatology and Nutrition* 27.3 (2024): 137-150.
5. Pesu H., *et al.* "Effects of lipid-based nutrient supplements on gut markers in children with environmental enteric dysfunction: a randomized trial in Malawi". *Journal of Pediatric Gastroenterology and Nutrition* 80.5 (2025): 889-898.
6. Tickell KD and Denno DM. "Environmental enteric dysfunction and child stunting". *Paediatrics and International Child Health* 39.3 (2019): 200-208.
7. Kelly P., *et al.* "Histopathology underlying environmental enteric dysfunction and chronic inflammation in children". *American Journal of Clinical Nutrition* 120.1 (2024): S15-S30.
8. Harper KM., *et al.* "Environmental enteric dysfunction pathways and child stunting: a systematic review". *PLOS Neglected Tropical Diseases* 12.1 (2018): e0006205.
9. Parada Venegas D., *et al.* "Short-chain fatty acids (SCFAs)-mediated gut epithelial and immune regulation". *Frontiers in Immunology* 10 (2019): 277.
10. Chandel N., *et al.* "Characterisation of Indian gut microbiome for B-vitamin production and its comparison with Chinese cohort". *British Journal of Nutrition* 131.4 (2024): 686-697.
11. Chen RY., *et al.* "A microbiota-directed food intervention for moderate acute malnutrition". *New England Journal of Medicine* 384.16 (2021): 1517-1528.
12. Hibberd MC., *et al.* "Bioactive glycans in a microbiome-directed food for malnourished children". *Nature* 625.7993 (2023): 157-165.
13. World Health Organization. "WHO guideline on the prevention and management of wasting and nutritional oedema (acute malnutrition) in infants and children under 5 years". Geneva: World Health Organization (2023).
14. World Health Organization. "Fluid management in severely malnourished children". Geneva: WHO (2023).
15. Nuzhat S., *et al.* "Effects of probiotic and synbiotic supplementation on rate of weight gain and length in young severely acute malnourished (SAM) infants: a randomized clinical trial". *Scientific Reports* 13.1 (2023): 1845.

16. Ghose R, *et al.* "Synbiotic therapy in severe acute malnutrition: a randomized, double-blind, placebo-controlled trial". *Clinical Nutrition* 42.2 (2023): 195-203.
17. Ertaş K, *et al.* "Central hematocrit levels in fetal malnourished term infants". *Journal of Pediatric Hematology/Oncology* 39.8 (2017): e426-e429.
18. Nuzhat S, *et al.* "Effects of probiotic and synbiotic supplementation on growth of young infants with severe acute malnutrition: a randomized clinical trial substudy". *Scientific Reports* 13.1 (2023): 1845.
19. Ko Y, *et al.* "A review of fecal microbiota transplantation in children-past, present, and future". *Frontiers in Pediatrics* 12 (2024): 1410528.
20. Chibuye M, *et al.* "Systematic review of associations between gut microbiome and stunting in low- and middle-income countries". *npj Biofilms and Microbiomes* 10 (2024): 46.
21. Govender P, *et al.* "Population-specific differences in the human microbiome: implications for nutrition and health". *Plant Gene* 36 (2025): 101014.
22. Nguyen AT, *et al.* "Pathways through which water, sanitation, hygiene, and environmental enteric dysfunction affect child growth: evidence from the WASH Benefits EED substudy". *International Journal of Environmental Research and Public Health* 22.3 (2025): 456.

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