JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i2.53268
JKD (DMJ), Volume 15, Number 2, March 2026 : 81-96

FECAL PH AND DECODING THE HIDDEN ROLE OF GUT DYSBIOSIS FOR STUNTING AMONG TODDLERS IN INDONESIA: A SYSTEMATIC REVIEW

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Keywords:
Fecal pH,
Gut dysbiosis,
Stunting,
Child health,
Indonesia.

Received: 10 August 2025
Revised: 13 September 2025
Accepted: 13 September 2025
Available online: 01 March 2026

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ABSTRAK

Background: Stunting in children is a persistent public health issue in Indonesia, partly linked to gut microbiota dysbiosis. This imbalance alters short-chain fatty acid (SCFA) production, affecting intestinal function and fecal pH. Fecal pH may serve as an indirect marker of microbial disturbance. **Objective**: To examine the association between fecal pH as an indicator of intestinal dysbiosis and stunting incidence among children in Indonesia. **Methods**: This review systematically searched Google Scholar, PubMed, and Scopus. Of 1,777 screened articles, 14 met inclusion criteria and were analyzed qualitatively. **Results**: Most studies reported higher fecal pH and microbial imbalance in stunted children. Common findings included elevated Firmicutes/Bacteroidetes ratios, higher pathogenic species, and reduced SCFA-producing bacteria. Synbiotic interventions improved microbial composition, lowered fecal pH, and supported growth. **Conclusion**: Elevated fecal pH is associated with gut dysbiosis and stunting in children. Its role as a low-cost biomarker could support early screening and targeted stunting prevention strategies in resource-limited settings.

INTRODUCTION

Improving human capital through improved child nutrition and health remains a cornerstone of Indonesia's national development strategy. Despite progress, stunting remains a major obstacle which, in 2018, the World Health Organization (WHO) classified the prevalence of stunting in Indonesia as a high problem[1]. Adequate nutrition during the first 1,000 days of life is essential to maintain linear growth and prevent chronic diseases later in life[2]. Stunting is the consequence of chronic undernutrition and recurrent infections-now demands urgent public health attention in Indonesia[3,4].

Defined as a height-for-age (Z-score) that is more than two standard deviations below the mean, stunting reflects not only malnutrition, but also exposure to chronic infections and inadequate sanitation. The condition usually starts in the womb and continues until the age of two[5]. Effective prevention requires comprehensive interventions, including maternal education, balanced nutrition, and WASH (water, sanitation, and hygiene) programs tailored to the local socio-cultural context[5,6].

Gut health is critical for early childhood development, as the gastrointestinal tract is the primary site of nutrient absorption. During the first 1,000 days, the gut microbiota plays an important role in immune function, endocrine activity and metabolism[7]. When these processes are disrupted, one of the most visible long-term consequences is impaired linear growth, or stunting. Globally, stunting remains the most prevalent form of malnutrition, affecting an estimated 149 million children under five years (22%) and limiting the developmental potential of millions. Although low socioeconomic status, infections, and inadequate diet are well-known contributors, nutrition-specific interventions alone have shown limited impact, suggesting that other

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mechanisms—such as environmental enteric dysfunction (EED) and gut dysbiosis—also play an important role[8].

Research on the association between gut microbiota, fecal pH, and childhood stunting has been widely carried out in different parts of the world, yielding a variety of findings that reflect regional dietary patterns, sanitation levels, and infectious disease exposures. A study in Bangladesh involving 100 children aged 12–18 months reported a mean fecal pH of 5.84±1.11 and a mean LAZ of −2.12±0.80. Regression analysis demonstrated a significant negative association between fecal pH and LAZ scores, indicating that higher fecal pH was correlated with poorer linear growth. These findings suggest that fecal pH may serve as an indirect marker of gut microbiota status and a potential contributing factor to childhood stunting[9].

The study from Kashmir, India, involving 100 children also showed a significant relationship between fecal pH and stunting in children. Children with severe stunting mostly had fecal pH values above 6.5, while those with mild stunting generally had values below this threshold. These findings suggest that elevated stool pH is not only associated with the presence of stunting but also reflects its severity, further strengthening the role of stool pH as a potential indicator of nutritional outcomes in children[8].

Emerging research links gut dysbiosis to impaired nutrient absorption, and one practical marker of microbial balance is fecal pH-a simple and inexpensive measure. A study in Bangladesh found that increased fecal pH was inversely related to linear growth in children[9], reflecting the depletion of acid-producing commensal bacteria such as *Bifidobacterium* and *Clostridia*. Research in Indonesia further showed that children with fecal pH > 6.4 had a 28.5-fold risk of stunting, with a sensitivity of 60% and specificity of 95%[5]. Therefore, this systematic review aims to examine the association between fecal pH - an indicator of gut microbial dysbiosis - and stunting among Indonesian children. By exploring the available evidence from 2020-2024, this study seeks to inform more appropriate and evidence-based strategies for early stunting screening and intervention.

METHODS

This review was conducted following the guidelines of the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA). The studies reviewed focused on fecal pH as an indicator of gut dysbiosis associated with stunting in Indonesia. Studies were searched using three electronic databases which are Scopus, MEDLINE (PubMed), and Google Scholar. The article search strategy was developed using the Population, Concept, and Context (PCC) framework. The target population included children under five years of age; the concepts involved fecal pH and gut microbiota dysbiosis.

The literature search was conducted using open access databases. This decision was taken to ensure that the studies found could be accessed and replicated by readers without any paywall restrictions, thus increasing the transparency and reproducibility of the review process. In addition, the use of open access databases was expected to reduce the risk of selection bias, as the use of paid-access journals may exclude relevant studies that are freely available. By prioritizing open access, the search strategy also increases the likelihood of capturing publications from low- and middle-income countries, including Indonesia, which are highly relevant to the study context.

The keywords used in the search included three primary combinations:

- ("fecal pH" OR "stool pH" OR "pH feses" OR "pH tinja") AND ("dysbiosis" OR "gut microbiota" OR "mikrobiota usus" OR "disbiosis usus") AND ("stunting" OR "growth faltering" OR "linear growth") AND (Indonesia OR Indonesian);
- ("fecal pH" OR "stool pH" OR "pH feses" OR "pH tinja") AND ("stunting" OR "growth faltering" OR "linear growth") AND (Indonesia OR Indonesian);
- ("gut microbiota" OR "dysbiosis" OR "intestinal flora" OR "mikrobiota usus" OR "disbiosis usus") AND ("stunting" OR "growth faltering" OR "linear growth") AND (Indonesia OR Indonesian).

The use of terms in English and Indonesian aims to minimize the risk of missing relevant studies published in journals that only use Indonesian. Boolean operators are used to expand the search by combining synonyms (OR) and limiting the search

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results to studies that discuss all the main concepts (AND).

Keywords are organized to reflect the PICO framework, with the population (children with stunting), exposure (gut dysbiosis or fecal pH), and outcome (growth impairment/linear growth) represented. This approach was chosen to maximize the retrieval of journals needed for this systematic review. The final combination of keywords was considered sufficient to capture all available and relevant literature.

The inclusion criteria for this review encompassed the following: studies targeting children under five years old living in Indonesia; exposure variables including fecal pH as a marker of intestinal fermentation and/or studies examining gut microbiota dysbiosis; and outcome variables presenting data or discussion related to stunting, linear growth impairment, or growth faltering. Eligible study designs included prospective or retrospective cohort studies, case-control studies, and analytical cross-sectional studies. Experimental animal studies and narrative reviews were excluded. Studies must have been conducted in Indonesia, by Indonesian researchers or within the context of the Indonesian population. Only full-access articles published between 2015 and 2025 were included, either in PDF or full-text format.

The publication year is limited to the period 2015–2024. This time frame was chosen to ensure that the studies included represent the latest and most relevant evidence, given that research on gut microbiota, fecal pH, and stunting has developed significantly over the past decade. Previous studies were considered less comparable due to methodological differences and the limited availability of standardized approaches. Furthermore, this period aligns with global and national agendas, including the Sustainable Development Goals (SDGs), which emphasize reducing stunting in children by 2025. Limiting the search to the last ten years is considered sufficient to capture both the scientific progress and policy relevance of this topic.

Furthermore, eligible articles were required to address at least two of the three main variables: fecal pH, gut microbiota dysbiosis, and stunting or linear growth impairment. In addition, eligible articles must discuss at least two of the three key variables: stool pH, gut microbiota imbalance, and stunting or linear growth disorders. These criteria are applied because the presence of two variables is considered sufficient to produce findings that are relevant to the study objectives. Requiring all three variables simultaneously would limit the number of eligible studies, given the limited number of publications that examine these factors together, and could therefore reduce the comprehensiveness of the review.

Studies involving children with congenital abnormalities or other comorbidities, non-scientific articles, and studies involving children born to parents with chronic conditions were excluded from review. These criteria were applied to minimize potential confounding factors, as the purpose of this review was to focus on stunting caused by nutritional and environmental factors, rather than genetic disorders or underlying medical conditions. By excluding these studies, the findings more accurately represent the relationship between fecal pH, gut dysbiosis, and stunting in children.

All included articles were grouped according to their main research subtopics, then extracted for deeper analysis regarding their outcomes and interrelations, in order to develop a comprehensive synthesis.

A total of 1,777 articles were initially identified, consisting of 1,571 from Google Scholar, 167 from Scopus, and 39 from PubMed. After removing 40 duplicates, title and abstract screening excluded 1,721 articles that did not meet the inclusion criteria. All remaining articles were retrieved in full-text form and met the eligibility criteria. Ultimately, 14 articles were included in the final synthesis of this systematic, which explores the association between fecal pH as an indicator of gut microbiota dysbiosis and stunting in Indonesian children.

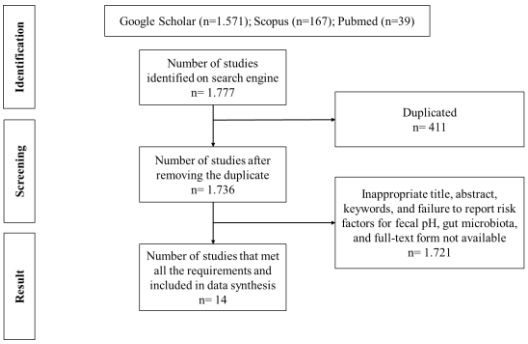

Figure 1. PRISMA flowchart of study identification, screening and selection process

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Table 1. List of Included Studies

Author	Method	Findings
Ratnayani et al. (2024)"Association of Gut Microbiota Composition with Stunting Incidence in Children under Five in Jakarta Slums"	Cross-sectional study involving 42 children aged 2–5 years in Jakarta slums (21 stunted, 21 non-stunted). Gut microbiota was analyzed using 16S rRNA sequencing with NGS. Dietary intake was assessed using SQ-FFQ and analyzed with NutriSurvey.	Significant differences in gut microbiota composition between stunted and non-stunted children. Stunted children had higher proportions of *Mitsuokella*, *Alloprevotella*, and *Providencia alcalifaciens* (pathogenic), while non-stunted children had more *Blautia*, *Lachnospiraceae*, *Akkermansia muciniphila*, and *Odoribacter splanchnicus* (beneficial). Environmental factors, dietary habits, and hand hygiene influenced microbial composition.
Rinanda et al. (2023)"Correlation between Gut Microbiota Composition, Enteric Infections and Linear Growth Impairment"	Case-control study on 42 children (21 stunted, 21 non-stunted) in Pidie, Aceh. Gut microbiota analyzed via 16S rDNA sequencing; qPCR used to detect enteropathogen virulence genes. IGF-1 measured using ELISA.	Stunted children showed lower IGF-1 levels, *Firmicutes* > *Bacteroidetes* dominance, increased pathogens like *Aeromonas*, and high expression of eaeA, estA, ipaH, ompC genes. Dysbiosis was negatively correlated with height and IGF-1 levels.
Surono et al. (2021) "Differences in Immune Status and Fecal SCFA between Indonesian Stunted Children and Children with Normal Nutritional Status"	Observational study comparing immune status and fecal SCFA between stunted and normal children. SCFA measured via GC; immune markers (TGF-β, TNF-α, IL-10, sIgA) analyzed from serum/feces.	Stunted children had higher SCFA (acetate, valerate) excretion indicating energy loss. TGF-β differed significantly. No consistent SCFA-bacteria pattern due to complex microbial cross-feeding.
Surono et al. (2025) "Effect of a 6-Month Functional Food Intervention on the Microbiota of Stunted Children in East Nusa Tenggara"	Parallel RCT over 6 months involving 200 children (probiotic, postbiotic, placebo). Interventions included chocolate milk, biscuits, sanitation, and well water. Gut microbiota analyzed via 16S sequencing.	All groups showed increased height and reduced *Escherichia/Shigella* levels, while Faecalibacterium increased. Microbiota changes associated with improved linear growth.
Judijanto et al. (2024) "Exploring the Role of Gut Microbiota Diversity in Early Childhood Stunting"	Longitudinal multi-center study evaluating early childhood gut microbiota diversity periodically.	Lower gut microbial diversity in stunted children. Microbial richness positively correlated with increased height-for-age z-score. Environmental exposure and hygiene influenced microbial communities.
Surono et al. (2024) "Exploring the Role of Gut Microbiota Diversity in Early Childhood Stunting"	Cross-sectional study; 200 fecal samples from children aged 36–45 months in East Nusa Tenggara analyzed using 16S rRNA sequencing.	Higher *Bacteroidetes* & *Cyanobacteria* in stunted children; *Veillonella*, *Faecalibacterium, Lachnoclostridium* also increased. SCFA levels were lower in stunted children.
Surono et al. (2021) "Gut Microbiota Profile of Indonesian Stunted Children and Children with Normal Nutritional Status"	Cross-sectional study analyzing gut microbiota in children aged 3–5 years from Banten & West Java.	Lower *Prevotella* 9 in stunted children; higher *Firmicutes* and lower *Bacteroidetes.*
Zuhriyah et al. (2024) "Human Milk Oligosaccharide Associated with the Firmicutes-to-Bacteroidetes Ratio among Stunted Infants in Malang"	Observational study analyzing *Firmicutes/Bacteroidetes* ratio and human milk oligosaccharides in Malang.	Higher *Firmicutes/Bacteroidetes* ratio in stunted children, associated with lower levels of milk oligosaccharides.
Masrul et al. (2020) "Microbiota Profile with Stunting Children in West Sumatera Province, Indonesia"	Case-control study; 96 children (48 stunted vs 48 non-stunted); 16S rRNA sequencing.	Dominance of *Firmicutes, Proteobacteria*, and *Bacteroidetes*; correlated with high carbohydrate intake.
Gunawan et al. (2022) "Pengaruh Konsumsi Sinbiotik Powder terhadap Profil Gut Mikrobiota pada Balita Stunting"	Double-blind RCT; 90-day synbiotic intervention with L. plantarum Dad-13 + FOS.	Increased *L. plantarum* & *Bifidobacterium*; decreased *Enterobacteriaceae;* increased SCFA; reduced fecal pH; increased bowel movement frequency.
Susanti et al. (2024) "Profiling of the Intestinal Microbiota of Stunted Children in Semarang, Indonesia"	Cross-sectional; children aged 3–3.5 years; analysis of fecal pH, AAT, and 16S rRNA.	Very high *Firmicutes/Bacteroidetes* ratio in stunted children; higher fecal pH and AAT → signs of dysbiosis & inflammation.
Hasnawati et al. (2022) "Profiling the Total Number of Bacteria in the Digestive Tract of Children with Stunting Conditions"	Cross-sectional study; qPCR used to quantify total gut bacteria in stunted vs normal children.	Lower total bacteria in stunted children (2.28 vs 5.95 log DNA copies/g); indicates impaired bacterial colonization.
Gunawan et al. (2022, CRNFSJ) "Synbiotic (*L. plantarum* Dad-13 and FOS) Powder on Gut Microbiota on Stunting Children in Yogyakarta"	Double-blind 90-day RCT; synbiotic (*L. plantarum* Dad-13 + FOS).	Increased *L. plantarum* & *Bifidobacterium*; reduced *Enterobacteriaceae*; significant increases in weight & height; no side effects.
Firdaus et al. (2024)"The comparison of carbohydrates, fibers, and immunoglobulin-A levels in feces against stunting children in Tuban Regency"	Quantitative cross-sectional study. Sample: 62 stunted and 31 non-stunted children in Tuban. Fecal carbohydrate, fiber, and IgA levels were measured.	Significant differences found between groups ($p < 0.05$). Stunted children had higher fecal carbohydrate, and lower fiber and IgA, indicating impaired digestion and gut immunity.

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RESULTS

General Characteristics of the Included Studies

This systematic review included 14 eligible studies conducted in various provinces across Indonesia, such as Jakarta, Aceh, East Nusa Tenggara, Yogyakarta, Malang, West Sumatra, West Java, and Banten (10-23). The summary of included studies and main findings are summarized in Table 2. The diversity of these settings reflects variations in socio-demographic and environmental conditions, dietary patterns, and public health infrastructure factors that influence the gut microbiota and child growth outcomes. All included studies focused on children under five years old, comparing those diagnosed with stunting to non-stunted outcomes. This comparison provided a foundation for analyzing gut microbial profiles and related metabolic indicators associated with linear growth impairment.

From the screening studies, the researcher had 7 studied for cross-sectional research method[10,12,14-16,19-23], 2 studies for case-control research method[11,18], 3 studies for randomized controlled trials research method (RCTs)[14,17,21], and 2 studies for longitudinal and correlational research method[13,10].

Table 2. Summary of included studies on gut microbiota, fecal pH, and stunting in Indonesian children.

Author (Year)	Location	Design	Sample	Main Findings
Ratnayani et al. (2024)	Jakarta, Indonesia	Cross-sectional	Children <5 y in slums	Gut microbiota composition significantly associated with stunting incidence.
Rinanda et al. (2023)	Indonesia	Cross-sectional	Children with linear growth impairment	Gut microbiota correlated with enteric infections and impaired growth.
Surono et al. (2021)	Indonesia	Cross-sectional	Stunted vs normal children	Stunted children showed altered immune status and lower fecal SCFA.
Surono et al. (2025)	East Nusa Tenggara, Indonesia	Intervention (6 months)	Stunted children	Functional food intervention modified gut microbiota in stunted children.
Judijanto et al. (2024)	Indonesia	Cross-sectional	Early childhood	Gut microbiota diversity linked to stunting risk.
Surono et al. (2024)	East Nusa Tenggara, Indonesia	Cross-sectional	Children aged 36–45 mo	Gut microbiota differences between stunted and normal children identified.
Surono et al. (2021)	Indonesia	Cross-sectional	Stunted vs normal children	Gut microbiota profile differed between groups.
Zuhriyah et al. (2024)	Malang, Indonesia	Cross-sectional	Infants	Firmicutes-to-Bacteroidetes ratio linked with human milk oligosaccharides.
Masrul et al. (2020)	West Sumatera, Indonesia	Case-control	96 children (48 stunted, 48 non-stunted)	Stunted children showed dominance of Firmicutes and Proteobacteria.
Gunawan et al. (2022)	Indonesia	Intervention	Stunted children	Synbiotic powder improved gut microbiota profile.
Susanti et al. (2024)	Semarang, Indonesia	Cross-sectional	Stunted children	Stunted children had higher Firmicutes/Bacteroidetes ratio and altered profile.
Hasnawati et al. (2022)	Indonesia	Cross-sectional	Stunted children	Total number of gut bacteria lower in stunted children.
Gunawan et al. (2022)	Yogyakarta, Indonesia	RCT (double-blind)	Stunted children	Synbiotic increased Lactobacillus & Bifidobacterium; reduced Enterobacteriaceae.
Firdaus et al. (2024)	Tuban, Indonesia	Cross-sectional	Stunted children	Differences in fecal carbohydrates, fibers, and IgA between groups.

The majority of studies were conducted in Java Island, including those based in Jakarta, Malang, Yogyakarta, Semarang, Tuban, Banten, and West Java[10,13-15,19-23], while the remaining were conducted in non-Java regions such as Aceh, East Nusa Tenggara, and West Sumatra[11,12,16-18]. One study involved multiple regions and may cover both Java and non-Java regions

In terms of laboratory methods, nine studies used 16S rRNA gene sequencing using next-generation sequencing (NGS) to assess the composition of the gut microbiota[10,11,14-18,20]. Two studies used quantitative PCR (qPCR) techniques to measure specific virulence genes or total bacterial populations[11,22]. In addition, some studies included biochemical measurements such as fecal pH, short-chain fatty acids (SCFAs) and alpha-1 antitrypsin (AAT) as indirect markers of gut fermentation, dysbiosis and inflammation[12,14,15,17,20]. Three studies further explored immune-related markers such as TGF-β and fecal immunoglobulin A (IgA), while others examined dietary intake and environmental sanitation practices as contributing factors to stunting[12,19,23].

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Regarding publication characteristics, most studies (11 out of 14) were published in SINTA-indexed journals, including studies by Ratnayani et al.[10], Rinanda et al.[11], Surono et al.[12,14,15], Gunawan et al.[17,21], Susanti et al.[19], Hasnawati et al.[22], and Firdaus et al.[23]. The other three studies-Masrul et al.[18], Zuhriyah et al.[16], and Judijanto et al.[13] come from non-indexed sources such as student theses or institutional reports. None of the studies in the current review were indexed in Scopus. Collectively, the 14 studies included a total of 1,061 children as participants, providing a robust dataset to synthesize the relationship between gut microbiota, gut health indicators and stunting risk. A summary of these demographic and methodological characteristics is presented in Table 3.

Table 3. General Characteristicx of the Included Studies

Characteristic	Category	Number of Studies
Total Sample Size		**1,061**
Year of Publication	< 2018	0
	≥ 2018	14
Type of Study	Cross-Sectional	7
	Case-Control	2
	Randomized Controlled Trial (RCT)	3
	Other Observational	2
Research Setting	Java Island	9
	Outside Java Island	5
Index Publication	Indexed in SINTA	11
	Non-Indexed (e.g., Theses)	3
	SCOPUS	0

Key Findings and Study Results

The key findings of the 14 included studies are summarized in Table 4. This table outlines the main variables studied, measurement indicators, direction of association and supporting studies on the association between gut microbiota dysbiosis and stunting in Indonesian children.

Table 4. Summary of key findings across included studies

Variable	Indicators Used	Main Findings	Supporting Studies
Fecal pH	Fecal pH value	Higher fecal pH observed in stunted children, suggesting lower SCFA production and gut dysbiosis	Susanti et al. (2024), Gunawan et al. (2022)
SCFA	Acetate, Valerate, Butyrate (GC analysis)	SCFA levels were lower or less diverse in stunted children, reflecting impaired fermentation and energy absorption	Surono et al. (2021, 2025), Gunawan et al. (2022)
Gut Microbiota Composition	*Firmicutes/Bacteroidetes* ratio, presence of pathogenic vs. beneficial strains	Stunted children had higher *Firmicutes*/Bacteroidetes ratio, more pathogens (e.g., *Providencia*, *Aeromonas*), fewer beneficial strains (e.g., *Akkermansia*, *Blautia*)	Ratnayani et al. (2024), Rinanda et al. (2023), Zuhriyah et al. (2024), Surono et al. (2021, 2024)
Microbial Diversity	Richness, α-diversity from 16S rRNA profiles	Reduced microbial diversity found in stunted children; positively correlated with linear growth	Judijanto et al. (2024)
Intestinal Inflammation	AAT level, qPCR, pathogenic genes (eaeA, estA, ipaH), SCFA imbalance	High AAT, low microbial diversity, increased virulence gene expression linked to inflammation and poor growth	Rinanda et al. (2023), Susanti et al. (2024)
Immune Biomarkers	TGF-β, sIgA, IL-10, TNF-α	Stunted children had lower sIgA, altered cytokines; possibly affecting mucosal immunity	Surono et al. (2021), Firdaus et al. (2024)
Diet & Environmental Factors	Dietary intake (SQ-FFQ), sanitation data, breastfeeding components (oligosaccharides)	Poor diet, low fiber intake, inadequate sanitation, and low HMOs associated with altered microbiota and stunting	Ratnayani et al. (2024), Zuhriyah et al. (2024), Firdaus et al. (2024)
Growth Outcomes	Height-for-age Z-score, IGF-1 level, anthropometric measurement	Gut dysbiosis associated with lower IGF-1 and height; synbiotic/probiotic interventions improved growth	Rinanda et al. (2023), Gunawan et al. (2022), Surono et al. (2025)

One finding that was consistent across the included studies was the change in fecal pH levels among stunted children. An increase in fecal pH was observed in the stunted group, which may show

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reduced short-chain fatty acid (SCFA) production, which is an indicator of impaired gut fermentation. This points out a shift in microbial metabolism and potential dysbiosis, making fecal pH a useful indirect marker of microbial imbalance[19,20].

Short-chain fatty acids, especially acetate, valerate and butyrate, play an important role in maintaining gut health and energy homeostasis. Several studies have found that stunted children have significantly lower SCFA concentrations or an unbalanced profile, indicating a decreased microbial fermentation capacity. This loss of energy through feces may contribute to stunted growth in malnourished children[12, 13, 19].

The majority of studies used 16S rRNA sequencing to identify differences in microbial composition. Stunted children were found to have a higher *Firmicutes-to-Bacteroidetes* ratio, increased prevalence of pathogenic bacteria such as *Aeromonas* and *Providencia alcalifaciens*, and lower presence of beneficial bacteria such as *Akkermansia* and *Blautia*. These compositional changes emphasize the pathogenic pattern of dysbiosis in these populations[10, 11, 15-17].

In addition to taxonomic differences, microbial diversity also appears to be a key factor. Reduced alpha richness and diversity was reported in stunted children and positively correlated with linear growth outcomes. These findings suggest that a more diverse microbiome supports better nutrient absorption and immune modulation[14]. The gut environment in stunted children also presented signs of inflammation, as indicated by elevated alpha-1 antitrypsin (AAT) levels and gene expression of enteric pathogens. These findings, together with low secretory IgA and altered cytokine levels, support the hypothesis that gut inflammation and immune dysregulation may play a role in linear growth failure[11, 12, 20, 23].

Environmental factors such as inadequate sanitation, low-quality food intake, and insufficient breast milk components (e.g., oligosaccharides) have also been found to affect the gut microbiota profile. In addition, several intervention studies revealed that improving microbial health through synbiotic or probiotic supplementation led to better linear growth, increased IGF-1, and improved anthropometric outcomes[10, 13, 19, 23].

Quality Appraisal of the Included Studies

The methodological quality of the 14 included studies was assessed using Joanna Briggs Institute (JBI) tools tailored to each study design[24-27]. Most cross-sectional studies fulfilled core criteria such as clear inclusion, valid outcomes, and proper analysis, though some lacked clarity in confounding control and sample size justification. Both case-control studies met nearly all JBI indicators, including appropriate matching and exposure assessment, enhancing credibility. All three RCTs showed strong internal validity through proper randomization, complete outcome data, and intention-to-treat analysis. However, none reported procedures for monitoring adherence or intervention fidelity, which may limit interpretation of true exposure effects.

The remaining two were observational studies: one longitudinal cohort by Judijanto et al.[21] and one ecological study by Zuhriyah et al.[17]. The longitudinal study assessed gut microbial diversity over time and its association with linear growth, while the ecological study examined the relationship between human milk oligosaccharides and the Firmicutes-to-Bacteroidetes ratio in stunted infants. Both studies clearly defined exposure and outcome variables and applied appropriate temporal frameworks. However, residual confounding could not be completely eliminated due to their non-experimental nature[17,21].

Overall, most included studies demonstrated acceptable methodological validity, although some—particularly cross-sectional designs—had limitations in controlling for confounding and reporting transparency[11, 15, 18].

DISCUSSION

Interpretation of The Association Between Fecal pH, Gut Microbiota Dysbiosis, and Stuting

Most of the studies included in this review show a consistent association between elevated faecal pH levels and the presence of gut microbiota dysbiosis in stunted children. High faecal pH often represents decreased production of short-chain fatty acids (SCFAs), especially acetate, butyrate and propionate, which are mainly produced by beneficial bacteria such as *Faecalibacterium prausnitzii*, *Bifidobacterium* and *Akkermansia muciniphila*[10, 14, 20]. The reduction of these beneficial microbes is a frequent finding in stunted children, indicating

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DOI : 10.14710/dmj.v15i2.53268
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impaired fermentation capacity and intestinal inflammation[10, 11, 14].

Several cross-sectional and case-control researches have also reported significant increases in *Firmicutes/Bacteroidetes* ratio and richness of potential pathogenic microbes such as *Escherichia coli*, *Shigella*, *Clostridium*, and *Aeromonas* in stunted children[11,12,15,22]. These findings support the hypothesis that mucosal inflammation triggered by dysbiosis and impaired nutrient absorption may contribute to chronic malnutrition and impaired linear growth.

In addition, several studies have shown that stunted children not only exhibit dysbiotic microbiota patterns but also have significantly higher fecal pH values, with some studies reporting mean pH higher than 7.0 [15, 21, 22]. These data confirm the potential of fecal pH as an alternate biomarker for detecting early gut microbiota imbalances in community settings, especially in areas where advanced microbiome testing remains difficult to access.

The relationship between microbiota ecology, inflammatory pathways and mucosal immunity appears to be central in explaining how dysbiosis contributes to stunting. High faecal pH is not only a by-product of microbiota shifts, but also a marker of impaired fermentation and compromised gut barrier integrity. These mechanisms are in line with the concept of environmental enteric dysfunction (EED), a subclinical condition that is common in resource-constrained environments and underlies chronic malabsorption and inflammation [12, 14, 23].

Therefore, interpreting fecal pH alongside microbiota profiles may offer an accessible and informative method to monitor gut health status in children and support early stunting prevention strategies.

Alterations in Gut Microbiota Composition and Functional Impact on Growth

Several studies have reported a significant decrease in gut microbiota diversity in stunted children, characterized by a lower alpha diversity index. This decrease in microbiota diversity is associated with impaired nutrient metabolism and reduced growth potential, where higher microbiota diversity is positively correlated with height-for-age z scores (HAZ) [19, 20]. A decrease in microbiota diversity, especially in beneficial commensal microbiota, may inhibit fiber fermentation, vitamin synthesis and mucosal immunity.

One common feature observed in the included studies was a decrease in *Prevotella* 9, a genus known for its ability to break down complex dietary fibers into short-chain fatty acids (SCFAs)[15,16]. This decrease may reflect a reduced fermentation capacity in the gut ecosystem of stunted children, which is especially concerning in the high-fiber diets common in Indonesian communities. *Prevotella* deficiency may result in decreased energy extraction and impaired linear growth.

Pro-inflammatory bacteria are defined as microbial taxa that induce or exacerbate inflammation within the host gut environment. Remarkably, *Veillonella* and *Lachnoclostridium* are consistently found in higher numbers in stunted children[17, 20]. These bacteria have been associated with activation of mucosal immune responses and impaired epithelial barrier integrity, which contribute to increased intestinal permeability and chronic low-grade inflammation[28]. These conditions impair nutrient absorption and disrupt metabolic signaling pathways. Importantly, high *Veillonella* levels are associated with increased inflammatory cytokine production, while *Lachnoclostridium* is known to modulate immune responses induced by lipopolysaccharide (LPS)[29]. These immunological disturbances correlate with decreased circulating levels of insulin-like growth factor 1 (IGF-1), a key hormonal regulator of linear growth in early childhood[11]. Therefore, the presence of pro-inflammatory bacteria not only reflects dysbiosis but may also mediate the biological mechanisms underlying growth failure in stunting.

At the phylum level, several studies reported a consistent dominance of *Firmicutes* over *Bacteroidetes* in the gut microbiota of stunted children[21,22]. Under normal physiological conditions, a balanced *Firmicutes/Bacteroidetes* (F/B) ratio supports optimal nutrient metabolism, immune modulation and microbiota diversity. *Bacteroidetes* are generally associated with dietary fiber degradation and production of beneficial metabolites, while *Firmicutes* contribute to energy extraction through fermentation of carbohydrates into SCFAs[30]. However, abnormally high F/B ratios-especially when *Firmicutes* predominate-have been associated with impaired gut homeostasis, inefficient microbiota colonization, and systemic inflammation[31]. In the

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(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i2.53268
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context of early childhood, this imbalance may signal a maladaptive microbiota response to environmental stressors such as inadequate diet and frequent infections, thus exacerbating the risk of stunting.

Although Firmicutes are the dominant producers of SCFAs, paradoxical findings were observed in some studies where children with stunting showed high levels of fecal acetate and valerate[15]. Instead of signaling microbiota productivity, these excessive SCFA concentrations may reflect impaired colonic absorption and mucosal dysfunction, where energy-rich metabolites are excreted instead of absorbed[32]. This phenomenon indicates enteric nutrient loss and compromised gut integrity, often referred to as enteric environmental dysfunction (EED)[33]. In addition, no consistent relationship was found between individual SCFA profiles and specific microbiota taxa across different studies. The absence of this pattern highlights the complexity of the microbiota feeding network, where metabolic output is the result of cooperative or competitive interactions between different genera[34].

Immunologically, low concentrations of transforming growth factor beta (TGF-β) were reported in stunted children, suggesting impaired mucosal immune regulation[15]. TGF-β plays an important role in maintaining intestinal homeostasis by promoting immune tolerance and supporting epithelial integrity. Impaired function of this cytokine can disrupt the gut environment, increase susceptibility to inflammation, and exacerbate dysbiosis.

As a response to this dysbiosis, synbiotic interventions show potential. Clinical trials using a combination of *Lactobacillus plantarum* Dad-13, *Bifidobacterium*, and the prebiotic fructooligosaccharides (FOS) showed improvements in gut microbiota composition and host health. These interventions were associated with increased number of beneficial commensal microbes, decreased *Enterobacteriaceae*, lower fecal pH, and measurable improvements in linear growth indicators, with no reported adverse effects[23]. These findings suggest that modulating gut microbiota through targeted supplementation could be an effective adjuvant strategy in the prevention of stunting.

In addition, high fecal pH and elevated fecal alpha-1 antitrypsin (AAT) levels were observed in stunted children, providing additional evidence of intestinal barrier dysfunction and subclinical inflammation[20]. AAT, as a marker of protein leakage, reflects impaired gut permeability, while high faecal pH indicates impaired microbiota fermentation processes. Together, these markers reinforce the diagnostic value of non-invasive gut biomarkers in assessing dysbiosis and directing early intervention.

In the summary, the current evidence highlights the role of gut dysbiosis - characterized by decreased microbiota diversity, predominance of pro-inflammatory taxa, altered SCFA fermentation and impaired immune regulation - in the pathophysiology of childhood stunting. Monitoring gut-related biomarkers such as fecal pH, SCFAs, TGF-β, and AAT may offer a practical and low-cost strategy to identify at-risk children and tailor targeted interventions, especially in resource-limited settings.

Variability of Findings

In the studies included in this review, there was significant variation in findings regarding gut microbiota composition and its association with stunting. Although most studies reported higher fecal pH and decreased microbiota diversity in stunted children, other studies showed less consistent patterns in bacterial richness and specific taxon shifts[15, 20, 22]. For example, *Prevotella* 9 was consistently reduced in several studies, yet the prevalence of *Faecalibacterium* and *Veillonella* varied depending on geographic region, dietary intake, and sanitation practices[14,17,18]. These inconsistencies may reflect the complex interactions between environmental exposures, host genetics, and cultural feeding practices that form the gut microbiota differently among the Indonesian population.

The variability in reported bacterial findings across the included studies is also strongly influenced by the methods used to assess gut microbiota. Studies that applied 16S rRNA sequencing were able to provide a broad overview of the microbial community. This approach captures the relative abundance of bacteria across major phyla and genera, making it possible to detect patterns such as the Firmicutes-to-Bacteroidetes ratio or the presence of specific genera like *Mitsuokella*, *Veillonella*, and *Akkermansia*. For example, children with stunting in East Nusa Tenggara and Semarang were reported to have higher proportions of Firmicutes and distinct taxa compared to their healthy peers[10,15,16,20]. The

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strength of this method is its ability to show the "big picture," but it often lacks resolution at the species or strain level.

In contrast, studies using quantitative PCR (qPCR) focused on specific bacterial groups or genes, which allowed for more sensitive and accurate detection of certain targets. This method helped reveal increased levels of pathogens such as *Aeromonas* and *Escherichia coli*, along with the presence of virulence genes like *eaeA* and *ipaH*, which were linked to poorer growth outcomes and lower IGF-1 concentrations in stunted children[11,21]. Because qPCR only detects what is being specifically looked for, its findings may not overlap with broader sequencing studies, but they provide important evidence for pathogenic disruptions that sequencing may overlook.

Other studies assessed the gut environment by analyzing short-chain fatty acids (SCFAs), which are key metabolites produced by bacterial fermentation. Measurement of SCFAs in stool samples showed that stunted children had higher excretion of acetate and valerate, suggesting inefficient energy harvest and loss of valuable nutrients through feces[12]. This approach highlights the functional consequences of microbiota imbalance rather than identifying the exact bacteria involved. By focusing on metabolic output, these studies added an important dimension to understanding how dysbiosis can impair child growth.

Finally, some studies used fecal pH as a simple yet informative indicator of microbial activity. Elevated stool pH was consistently found among stunted children, pointing to a reduction in beneficial acid-producing bacteria such as *Bifidobacterium* and *Lactobacillus*[20]. Although this method cannot identify specific microbial taxa, it provides a practical and low-cost proxy for the overall balance of gut microbial metabolism. When considered together, the use of different methodological approaches—16S rRNA sequencing, qPCR, SCFA measurement, and fecal pH—helps explain why the bacterial findings appear varied across studies. Each method highlights a different aspect of the gut ecosystem, and when integrated, they collectively reinforce the evidence that stunting is associated with both structural and functional disruptions of the gut microbiota in Indonesian children.

Another factor that contributes to the variability of findings is the study design applied in the included articles. Many studies used a cross-sectional design, which provides a snapshot of gut microbiota composition at a single point in time. This design is useful for identifying associations between microbial patterns and stunting but cannot determine causality. For example, studies conducted in East Nusa Tenggara, Jakarta, and Semarang reported differences in microbial communities between stunted and non-stunted children, such as higher levels of Firmicutes or pathogenic taxa, but these results only show correlations rather than cause-and-effect relationships[10,15,16,20].

Other studies used a case–control design, directly comparing stunted and non-stunted children to highlight distinct bacterial features. For instance, research in Aceh and West Sumatera demonstrated higher dominance of Firmicutes and increased detection of enteropathogens in stunted groups[11,18]. By using non-stunted children as a comparator, these studies strengthened the evidence that specific microbial profiles are consistently linked with growth impairment, although they remain limited by potential recall bias in dietary or environmental data collection.

In contrast, randomized controlled trials (RCTs) offered a more robust framework for testing causal relationships by introducing specific interventions. Studies using synbiotic supplementation with *Lactobacillus plantarum* Dad-13 and fructo-oligosaccharide (FOS), or functional food interventions such as fortified chocolate milk and biscuits, demonstrated that altering the gut microbiota composition could improve both microbial balance and growth outcomes[13,19,22]. These interventional designs provided stronger support for the role of gut microbiota in influencing stunting, as they were able to measure changes before and after intervention under controlled conditions.

In General, differences in study design explain part of the variability observed across findings. Cross-sectional and case–control studies primarily identify associations and patterns of dysbiosis, while RCTs provide experimental evidence that manipulating the gut microbiota can directly contribute to improved child growth. This range of study designs enriches the evidence base, but it also underscores the need for careful interpretation when comparing results across heterogeneous methodologies.

The demographic characteristics of the study populations also played an important role in shaping the variability of findings. Differences in age groups

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DOI : 10.14710/dmj.v15i2.53268
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contributed to distinct microbial profiles, as infants and preschool children are known to harbor different stages of gut microbial development. For instance, studies focusing on infants demonstrated that breastfeeding practices and exposure to human milk oligosaccharides were strongly associated with gut microbial balance. In Malang, stunted infants exhibited a higher Firmicutes-to-Bacteroidetes ratio, which was linked with lower levels of milk oligosaccharides, suggesting that inadequate breastfeeding support could alter microbial composition at a critical developmental window[17]. In contrast, studies involving older preschool children emphasized dietary diversity and infection exposure as the main factors influencing microbial communities. Research conducted in Semarang and East Nusa Tenggara, for example, found that stunted children aged 3–4 years had higher fecal pH, elevated Firmicutes, and increased presence of *Veillonella* and *Faecalibacterium*, reflecting environmental and dietary drivers rather than early-life milk composition[15,20].

Geographical and environmental factors further explain the observed differences across studies. Children living in urban slums, such as those in Jakarta, showed enrichment of pathogenic taxa like *Mitsuokella* and *Providencia alcalifaciens*, which may reflect the combined impact of poor sanitation, crowding, and limited access to clean water[10]. Meanwhile, in more rural settings such as East Nusa Tenggara and West Sumatera, the microbial composition was influenced by high-carbohydrate diets and limited dietary protein, leading to dominance of Firmicutes and Proteobacteria[15,18]. These findings highlight that local diets, sanitation infrastructure, and antibiotic exposure vary greatly between regions and can markedly alter microbial colonization patterns.

In general, the variability in study populations—whether by age group, breastfeeding practices, or environmental context—illustrates that gut microbiota findings are not uniform across Indonesia. Instead, they reflect the complex interplay between biological development and local living conditions. Recognizing these differences is crucial, as it not only explains the heterogeneity in results but also emphasizes the need for region-specific strategies to address stunting through microbiota-targeted interventions.

Although a meta-analysis would have provided stronger quantitative evidence regarding the association between gut microbiota and stunting, it could not be performed in this review due to the substantial heterogeneity among the included studies. Differences in study designs (cross-sectional, case–control, and randomized controlled trials), population demographics (age groups, urban versus rural settings), and methodological approaches for microbiota assessment (16S rRNA sequencing, qPCR, SCFA measurement, fecal pH analysis) resulted in incomparable outcome measures[10–23]. In addition, variability in the bacterial taxa reported and the lack of standardized effect sizes across studies precluded statistical pooling. As such, a narrative synthesis was chosen as the most appropriate approach to summarize the findings and highlight overarching trends while acknowledging study-specific limitations.

Confounding Factors

When interpreting the findings of this review, it is important to recognize that several confounding factors may have influenced the observed associations between gut microbiota composition and childhood stunting. These factors, which include dietary patterns, breastfeeding practices, antibiotic exposure, socioeconomic and environmental conditions, and infection burden, varied across studies and likely contributed to the heterogeneity of results.

Among the potential confounding factors, dietary habits and diet quality represent a major determinant of gut microbiota composition. Several studies highlighted that diets predominantly high in carbohydrates were linked with microbial imbalances. In West Sumatera, stunted children consuming carbohydrate-rich diets showed a dominance of *Firmicutes* and *Proteobacteria*[18]. Similarly, in Tuban Regency, elevated fecal carbohydrate residues and reduced fiber intake were observed in stunted children, along with lower levels of immunoglobulin A, suggesting impaired digestion and weakened mucosal immunity[23]. These findings emphasize that excessive carbohydrate intake may foster overgrowth of opportunistic taxa while simultaneously reducing beneficial microbial activity.

Low protein intake also emerged as an important factor, particularly in rural settings where access to animal-source foods was limited. Diets deficient in

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protein may inhibit the colonization of beneficial bacteria such as *Bifidobacterium*, which are essential for the production of short-chain fatty acids (SCFAs) that support intestinal health and growth[18]. This protein deficiency makes it difficult to determine whether dysbiosis is a contributing factor or simply a reflection of chronic malnutrition.

In addition, dietary fat intake appeared variable but was generally low in populations studied in East Nusa Tenggara and West Sumatera[15,18]. Reduced lipid intake may deprive certain microbial taxa of necessary substrates, while in other contexts, excessive fat consumption has been associated with proliferation of pro-inflammatory bacteria such as *Bilophila wadsworthia*, although such patterns were not explicitly reported in the included studies.

Another important aspect is the role of fiber and dietary diversity, which further contribute to altered microbial ecosystems. Low fiber availability reduces the growth of *Bifidobacterium* and SCFA production, as demonstrated in Tuban children[23], while monotonous diets dominated by staple carbohydrates (e.g., rice and cassava) were linked to poor microbial diversity in rural settings[15,18]. Collectively, these findings suggest that dietary quality—characterized by high carbohydrate dependence, low protein and fiber, reduced lipid intake, and poor diet diversity—confounds the relationship between gut microbiota dysbiosis and stunting in the Indonesian context.

Breastfeeding practices and the availability of human milk oligosaccharides (HMOs) are another important confounding factor in the relationship between gut microbiota and stunting. HMOs are known to selectively promote the growth of beneficial bacteria such as *Bifidobacterium*, resulting in the colonization of the microbiota early in life. In Malang, a study among stunted infants demonstrated that children with higher Firmicutes-to-Bacteroidetes ratios had significantly lower levels of HMOs[17]. This suggests that inadequate breastfeeding or insufficient exposure to breast milk oligosaccharides may predispose infants to dysbiosis, which in turn could impair growth.

The impacts of breastfeeding are further complicated by its interaction with environmental conditions and maternal nutrition. The limitation of maternal dietary diversity or early weaning practices can reduce both the quantity and quality of HMOs available to infants. As a result, the development of gut microbiota during this critical window may deviate from a healthy pathway, making infants more susceptible to infections and growth disorders. These findings highlight the need to interpret the relationship between microbiota and stunting with caution, as differences in breastfeeding exposure may partly explain the inconsistencies observed across studies.

Another confounding factor that may influence the association between gut microbiota and stunting is antibiotic exposure. Antibiotics, while crucial for treating infections, can disrupt the delicate balance of gut microbial communities by reducing microbial diversity and suppressing beneficial taxa such as *Lactobacillus* and *Bifidobacterium*. Although not all included studies explicitly reported patterns of antibiotic usage, this factor is particularly relevant in low-resource settings where antibiotics are frequently prescribed or obtained without strict regulation.

The consequences of repeated or inappropriate antibiotic exposure can resemble dysbiosis observed in stunted children, making it difficult to disentangle whether microbial alterations are primarily driven by nutritional deficits, infections, or medication history. For instance, children in regions with high infection burdens, such as Aceh and East Nusa Tenggara, were frequently exposed to enteric pathogens[11,15]. It is probable that antibiotic treatment in this contexts further compounded microbiota disruption, potentially hiding or enhancing the association with stunting. This emphasizes the necessity for future studies to collect detailed antibiotic histories and consider them in the analysis to avoid overestimating the direct role of gut microbiota alterations.

Socioeconomic status and sanitation conditions also represent critical confounding factors in the microbiota–stunting relationship. Children living in environments with poor hygiene and inadequate access to clean water are more likely to be exposed to pathogenic bacteria, which can significantly alter gut microbial composition. For example, children from Jakarta slums exhibited enrichment of potentially pathogenic taxa such as *Mitsuokella* and *Providencia alcalifaciens*, reflecting the combined influence of overcrowding, poor sanitation, and limited hygiene practices[10]. Similarly, research in Aceh revealed increased detection of enteropathogen virulence genes, which may be partly attributed to

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DOI : 10.14710/dmj.v15i2.53268
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environmental contamination and low household sanitation standards[11].

These findings illustrate that microbiota changes often attributed to stunting may, in part, be triggered by structural determinants of health. Low socioeconomic conditions not only constrain dietary diversity but also increase the likelihood of recurrent infections and frequent antibiotic use, thereby compounding microbial imbalance. Consequently, socioeconomic and environmental contexts should be carefully considered when interpreting gut microbiota patterns, as they may obscure the direct biological pathways linking dysbiosis to impaired linear growth.

Enteric infections are another major confounding factor that complicates the interpretation of gut microbiota findings in stunted children. The presence of pathogens can independently disrupt microbial balance, alter intestinal function, and impair nutrient absorption, making it challenging to distinguish whether observed dysbiosis is a cause or a consequence of infection. In Aceh, stunted children exhibited a high prevalence of enteropathogenic genes such as *eaeA*, *estA*, *ipaH*, and *ompC*, alongside increased abundance of pathogenic taxa including *Aeromonas*[11]. Similarly, in East Nusa Tenggara, the dominance of *Escherichia/Shigella* was observed in stunted children, further supporting the role of recurrent infections in shaping microbial communities[15].

These infection-driven alterations may mimic or amplify the dysbiotic patterns linked with stunting, particularly by reducing beneficial commensals and enhancing gut inflammation. Consequently, the overlap between infection-related changes and nutritional dysbiosis creates interpretive challenges. Without accounting for infection status, studies may overestimate the contribution of microbiota alterations to growth faltering, when in fact recurrent enteric infections could be a primary driver of both dysbiosis and stunting outcomes.

Age and developmental stage also play a critical role as confounding factors in gut microbiota studies. The composition of the gut microbiome changes rapidly during early life, transitioning from a milk-oriented microbiota dominated by *Bifidobacterium* in infancy to a more diverse, adult-like community by the preschool years. As a result, comparing microbiota profiles across different age groups can yield apparent inconsistencies that are driven more by developmental stage than by nutritional status. For example, in Malang, stunted infants exhibited a higher Firmicutes-to-Bacteroidetes ratio linked with lower exposure to human milk oligosaccharides, highlighting the importance of breastfeeding in shaping microbial development during early infancy[17]. In contrast, studies involving older preschool children in Semarang and East Nusa Tenggara emphasized environmental exposures, dietary diversity, and fecal pH as dominant influences on microbial patterns[15,20].

This variation indicates that the relationship between gut microbiota and stunting cannot be interpreted in isolation from age. Younger children are more affected by breastfeeding practices and maternal factors, whereas older children are increasingly shaped by diet, sanitation, and infection exposure. Therefore, age-related differences must be considered when evaluating microbiota–stunting associations to avoid conflating developmental maturation with pathological dysbiosis.

These confounding factors—ranging from diet quality, breastfeeding practices, antibiotic exposure, socioeconomic conditions, gut infections, to age-related differences—underscore the complexity of interpreting gut microbiota findings related to stunting. Each factor has a different but overlapping influence on microbiota composition, making it difficult to isolate the independent role of dysbiosis. The heterogeneity observed across studies reflects not only methodological or regional differences but also underlying confounding factors. Recognizing their impact is crucial, as it strengthens the credibility of the review and provides a more nuanced understanding of why findings may differ. More importantly, it also highlights the need for future research to control for or stratify based on these variables, so that the relationship between gut microbiota and child growth can be more accurately elucidated.

Recommendations for Future Research

This review highlights several important gaps that need to be addressed by future research in order to strengthen the evidence base linking fecal pH, gut dysbiosis, and stunting in children. First, most of the included studies were cross-sectional or case–control, which limits the ability to conclude causal relationships. Well-designed longitudinal studies and

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cohort studies are needed to clarify the temporal sequence between dysbiosis and growth disorders, determining whether changes in the gut microbiota precede stunting or arise as a consequence of nutritional deficiencies. Such an approach would allow for the identification of more precise causal pathways and provide a stronger basis for intervention strategies.

Second, there is an urgent need to develop and adopt standard protocols for measuring fecal pH. Current studies vary in terms of sample collection (fresh versus stored samples), preparation, and measurement techniques, leading to inconsistencies in findings. Establishing clear methodological guidelines—including collection times, homogenization procedures, and the use of calibrated pH meters—will facilitate comparisons between studies and improve the reliability of fecal pH as a biomarker of gut dysbiosis.

Strengths and Limitations

In interpreting the findings of this review, it is important to consider how the methodological criteria applied may affect the generalizability of the results. Database selection was limited to PubMed, ScienceDirect, Google Scholar, and the Indonesian indexing system (SINTA), which provide comprehensive coverage of international and national research. However, the exclusion of other databases, such as Web of Science, may limit access to studies indexed elsewhere. This reliance on open-access databases could potentially reduce the comprehensiveness of searches by ignoring paid-for journals, some of which may contain high-impact or region-specific studies. Nonetheless, the use of open-access journal platforms is considered appropriate to ensure transparency, reproducibility and inclusivity, especially in capturing literature from low- and middle-income countries such as Indonesia, which is the main focus of this review.

In addition, limiting the search to studies published in English and Indonesian may have resulted in the ignorance of relevant literature available in other languages. This limitation may reduce the diversity of perspectives included in the review. However, these restrictions are considered appropriate given the primary focus on the Indonesian context and the requirement to ensure accurate interpretation of the findings. The use of English is also reasonable, as it remains the dominant language in global scientific communication and is likely to cover the majority of high-quality publications in this field. Overall, the combination of Indonesian and English is considered sufficient to capture the most relevant evidence while maintaining the validity and accuracy of the review process.

The decision to limit the inclusion criteria of the study population to children under five years old was based on the WHO definition of stunting. The WHO definition of stunting is when height-for-age is more than two standard deviations below the WHO Child Growth Standards median, and this is operationally calculated for children under 5 years of age (0-59 months). The inclusion of older children or those >5 years of age would result in heterogeneity and weaken the interpretability of the findings, as stunting above the age of five years would begin to be less responsive to interventions if they were to be targeted. Therefore, this limitation was necessary to ensure that the analysis included stunting in the period when prevention and interventions are most effective.

Publication restrictions for the period 2015–2024 may have excluded earlier studies that discussed gut dysbiosis more broadly. However, these criteria are unlikely to disrupt the completeness of the review, as no studies prior to 2015 specifically examined the relationship between fecal pH and stunting. Focusing on the last decade is considered appropriate to ensure that the evidence presented reflects the latest methodological advances and current understanding in gut microbiota research.

ACKNOWLEDGE, FUNDING & ETHICS POLICIES

The author would like to sincerely thank the Faculty of Medicine, University of Malikussaleh, for the academic support and facilities provided throughout the completion of this systematic review. Special appreciation is also extended to Dr. dr. Cut Khairunnisa, M.Kes, for her invaluable guidance, encouragement, and constructive input during the research process. The author is especially grateful to the author's parents for their unconditional love, prayers, and emotional support throughout this academic journey.

This study was entirely self-funded and received no financial support or sponsorship from any

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(*DIPONEGORO MEDICAL JOURNAL*)
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institution or organization. As this was a systematic literature review, ethical approval was not required.

CONCLUSON AND RECOMMENDATION

Conclusion

This systematic review highlights a consistent association between intestinal microbiota dysbiosis—marked by reduced diversity, altered microbial composition, and increased inflammatory markers—and the incidence of stunting in Indonesian children. Elevated fecal pH, increased Firmicutes-to-Bacteroidetes ratios, and higher prevalence of Veillonella and Lachnoclostridium were commonly observed in stunted children, suggesting a dysfunctional gut environment. These findings support the potential use of fecal pH as a non-invasive, practical biomarker for early detection of gut dysbiosis associated with impaired growth outcomes.

Recommendation

To enhance stunting prevention strategies in Indonesia, future research should focus on developing standardized protocols for microbiota profiling and expanding longitudinal studies to better understand the causal relationships. Integrating fecal pH screening into early child health programs—especially in low-resource settings—may serve as a cost-effective tool for identifying children at risk. Furthermore, synbiotic interventions targeting gut microbial restoration hold promise and should be considered as adjunctive strategies within public health frameworks

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