Original Article

Culture-based breast milk and infant gut microbiota profiles among stunted infants: A case-control study from Indonesia

Aslinar Aslinar[1], Herlina Dimiati[2*], Nur Indrawati[3] and Sofia Sofia[4]

[1]Doctoral Program in Medical Science, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia; [2]Department of Pediatrics, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia; [3]Department of Nutrition, Faculty of Medicine, Universitas Andalas, Padang, Indonesia; [4]Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia

*Corresponding author: herlinadimiati@usk.ac.id

Abstract

Stunting remains a major public health problem in Indonesia. Early-life microbiota may contribute to infant growth through their roles in microbial colonization, immune maturation, intestinal function, and nutrient utilization, but evidence from high-stunting settings in Indonesia remains limited. The aim of this study was to compare culture-based breast milk and infant fecal microbiota profiles between stunted and normal infants in Aceh, Indonesia, and to examine their associations with maternal, postnatal, environmental, and socioeconomic factors. A total of 54 breastfed infants aged 6–11 months were included, consisting of 27 stunted and 27 normal infants matched by age and sex. Breast milk and fecal samples were analyzed using culture-based microbiological methods for bacterial identification and colony-forming unit counts. In breast milk, *Streptococcus* sp. and *Staphylococcus* sp. were the predominant bacteria, with higher detection frequencies among mothers of stunted infants. In fecal samples, *Escherichia coli* was the most frequently detected bacterium in both groups, while *Staphylococcus aureus* and *Clostridium innocuum* were significantly more common among stunted infants. Breast milk bacterial colony counts were significantly higher in the stunted group, whereas fecal colony counts were numerically higher but not statistically significant. Poor household sanitation was more frequent among stunted infants. Exploratory analyses among stunted infants showed that maternal height was associated with *Anaerococcus prevotii*, while maternal education was associated with selected fecal bacteria, including *Salmonella choleraesuis*, *Proteus mirabilis*, and *Eggerthella lenta*. These findings suggest culture-detectable differences in breast milk and infant fecal microbiota between stunted and normal infants. Given the small sample size, confirmation using longitudinal and sequencing-based studies is warranted.

Keywords: Stunting, microbiota, breast milk, infant gut, Indonesia

Introduction

Stunting, defined as impaired linear growth with length-for-age below two standard deviations of the World Health Organization (WHO) Child Growth Standards, remains a major child health challenge in low- and middle-income countries. Globally, approximately 149 million children under five years of age were stunted in 2020, with the highest burden reported in South Asia and Sub-Saharan Africa [1]. In Indonesia, stunting continues to require substantial public health attention. The 2022 Indonesian Nutritional Status Survey reported a national stunting prevalence of 21.6%, while Aceh Province recorded a markedly higher prevalence of 31.2%, placing it among

Received: April 30, 2026 - Revised: June 26, 2026 - Accepted: July 6, 2026 - Published online: July 10, 2026

the provinces with the highest burden in the country [2]. According to the 2023 Indonesian Health Survey, the national prevalence decreased only marginally to 21.5%, while Aceh remained above the national average at 29.4% [3].

Stunting is a chronic condition arising from complex interactions among biological, nutritional, household, environmental, socioeconomic, and cultural determinants. In Indonesia, important determinants include preterm birth, lack of exclusive breastfeeding, low socioeconomic status, short birth length, maternal short stature, and educational factors [4]. Environmental conditions may further increase the risk of stunting by shaping exposure to enteric pathogens, affecting child health, and influencing nutrient intake and absorption [5]. Malnutrition remains a major global contributor to impaired growth and development, and children with malnutrition and growth failure are more susceptible to infections. Inadequate nutrition during early life may also interfere with immune maturation, dietary adaptation, and microbial development [6]. If persistent, these processes may contribute to chronic undernutrition and impaired linear growth, ultimately resulting in stunting [7].

Increasing evidence indicates that early-life microbial colonization may be involved in the pathway between undernutrition, infection, and impaired child growth. Alterations in the microbiota, both in breast milk and in the gastrointestinal tract, have been associated with nutritional disorders and growth impairment [8]. A previous study reported differences in gut microbiota composition between stunted and non-stunted children, including differences in beneficial and potentially pathogenic taxa [9]. In non-stunted children, fecal microbiota has been reported to be dominated by beneficial bacteria, such as *Bifidobacterium longum* and *Lactobacillus mucosae*, whereas stunted children showed a higher abundance of pro-inflammatory taxa, including *Desulfovibrio* and *Campylobacterales* [9]. However, evidence from Indonesian settings remains limited, particularly regarding studies that examine breast milk and infant fecal bacteria within the same mother–infant context.

Breast milk is a key biological source of early microbial exposure because it contains not only nutrients, bioactive compounds, and immunological factors, but also commensal bacteria that may be transferred from the mother to the infant's gastrointestinal tract. These milk-associated bacteria contribute to the initial establishment of the infant gut microbiota and may influence gastrointestinal colonization, intestinal barrier maturation, immune development, and overall infant health [8,9]. The composition of breast milk microbiota is highly individualized, suggesting that maternal microbial profiles may contribute to variation in early infant gut colonization. Because the microbiota established during early life may influence health outcomes in both the short and long term, breast milk microbiota may represent an important biological factor in infant growth and development [10].

In breastfed infants, human milk represents a major source of bacterial exposure to the gastrointestinal tract. An estimated daily intake of 800 mL of breast milk may provide approximately 10^5 to 10^7 bacterial cells [11]. This supports the concept that the gut microbiota of breastfed infants is partly shaped by bacterial communities present in maternal milk. Accordingly, the long-standing view that human milk is a sterile biological fluid has been substantially revised over the past decade [11]. Through the transfer of milk-associated bacteria, breast milk may contribute to microbial seeding of the infant gastrointestinal tract, with potential effects on immune maturation, intestinal barrier development, and nutrient processing [8,9].

In settings characterized by repeated pathogen exposure, altered gut colonization may interact with intestinal inflammation and impaired nutrient absorption, pathways that have been linked to environmental enteric dysfunction and linear growth restriction [12]. Pathogenic and opportunistic bacteria, including *Escherichia coli* and *Salmonella*, may be overrepresented in the gut of malnourished children and may contribute to intestinal inflammation and environmental enteric dysfunction [12]. Environmental enteric dysfunction, also referred to as environmental enteropathy, is a chronic subclinical disorder of the small intestine characterized by villous injury, impaired nutrient assimilation, and increased microbial translocation. These changes may promote persistent systemic inflammation and contribute to the pathogenesis of childhood stunting [13].

Indonesia, with its persistently high stunting burden and diverse socio-environmental exposures, represents an important setting for investigating the relationship between early-life

microbiota and infant growth. Aceh Province is particularly relevant because its stunting prevalence remains substantially higher than the national average [2]. Understanding whether breast milk and infant fecal bacterial profiles differ between stunted and normal infants may provide insight into potential microbial pathways involved in growth impairment in this setting. Although sequencing-based approaches, including 16S rRNA sequencing and metagenomics, provide broader microbiome characterization, culture-based methods remain useful for identifying viable and clinically relevant culturable bacteria, particularly in resource-limited settings [11]. Therefore, the aim of this study was to investigate differences in the microbiota composition of breast milk and infant feces between stunted and normal infants in Aceh, Indonesia. This study was designed as an exploratory culture-based assessment and it was hypothesized that stunted infants would show different culture-detectable breast milk and fecal bacterial profiles compared with normal infants, and that these profiles would be associated with selected maternal, environmental, and socioeconomic characteristics.

Methods

Study design and setting

A case-control design was used to compare breast milk and infant fecal microbiota profiles between stunted and normal infants. The study was conducted in Aceh Besar District, Aceh Province, Indonesia, a setting with a persistently high burden of childhood stunting. Data were collected from eleven subdistricts, including Indrapuri, Suka Makmur, Kuta Malaka, Kuta Cot Glie, Ingin Jaya, Baitussalam, Montasik, Peukan Bada, Krueng Barona Jaya, Kutabaro, and Blang Bintang. The study was conducted between 2021 and 2025, with field activities supported by local health workers to facilitate participant recruitment and sample collection at the community level.

Study population and sample size

The study population comprised infants aged 6–11 months who resided in the study area. A total of 54 infants were recruited, consisting of 27 stunted infants as cases and 27 infants with normal growth status as controls. Controls were matched to cases by age in months and sex to minimize potential confounding. A formal a priori sample size calculation was not performed because of the absence of local baseline data needed to estimate expected differences in culture-detectable bacterial profiles between groups. Therefore, the statistical analyses were considered exploratory and hypothesis-generating rather than confirmatory. Participants were recruited at the community level with assistance from integrated community health posts (*Posyandu*) and village midwives.

Eligibility criteria

Mothers were eligible if they were currently breastfeeding an infant aged 6–11 months, had exclusively breastfed during the first six months, and had no obstetric complications during pregnancy. Exclusion criteria for mothers comprised chronic gastrointestinal disease, genetic disorders, cardiac, renal, or hepatic disease, immunodeficiency, active mastitis, or use of antibiotics, probiotics, anti-diarrheals, antidepressants, or laxatives within 30 days prior to sample collection. Infants were included if they were born at term (37–42 weeks of gestation) and were otherwise healthy. Infants were excluded if they had severe acute illness such as pneumonia, tuberculosis, or diarrhea, were receiving antibiotic therapy, had been hospitalized within the previous six months, or had congenital gastrointestinal anomalies.

Case definition

Stunting was defined using the WHO Child Growth Standards. An infant was categorized as stunted if the length-for-age Z-score was below -2 standard deviations. Controls were selected from the same communities as the cases and were required to have normal growth status, defined as a length-for-age Z-score between −2 and +2 standard deviations. Matching by age and sex was performed to improve comparability between the stunted and normal infant groups.

Data and sample collection procedures

Demographic, clinical, maternal, environmental, and socioeconomic data were collected using a structured questionnaire administered to mothers or primary caregivers. Infant-related information included age, sex, birth weight, birth length, mode of delivery, and history of infection. Maternal, household, and environmental information, including maternal height, mode of delivery, maternal weight gain, history of infection during pregnancy, maternal personal hygiene, maternal education, household income, household sanitation, and access to clean water, was also assessed and documented. Anthropometric measurements were performed according to WHO standard procedures. Infant weight was measured using a digital scale, and recumbent length was measured using an infant length board. Length-for-age Z-scores were calculated based on the WHO Child Growth Standards.

Breast milk samples were collected in the morning under aseptic conditions. After cleansing the nipple and areolar area with soap, sterile water, and 1% chlorhexidine, samples were obtained by manual expression using sterile gloves. The first 200 µL–2 mL was discarded, and 5–15 mL of milk was collected into sterile containers. Specimens were stored at −20°C, transported to the Microbiology Laboratory, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia, and preserved at −80°C until analysis.

Infant stool samples (2–5 g) were collected in sterile containers, with care taken to avoid contamination with urine, toilet water, or other foreign material. Samples were transported in a cool box with ice gel packs to the Microbiology Laboratory, Faculty of Medicine, Universitas Syiah Kuala, and processed promptly upon arrival.

Microbiological analysis

Microbiota analysis was conducted using standard culture-based microbiological procedures for microbial enumeration and identification. Total viable counts were determined using the Total Plate Count (TPC) method with the pour plate technique. Serial ten-fold dilutions were prepared, and plates yielding 30–300 colonies were selected for enumeration. Results were expressed as colony-forming units (CFU) per mL for breast milk and CFU/g for fecal samples.

Breast milk samples (2 mL) and fecal samples (2 g suspended in 2 mL phosphate-buffered saline) were processed under controlled laboratory conditions in a Biosafety Cabinet (Monmouth Scientific, Bridgwater, United Kingdom) at 35–37°C. Colony morphology was assessed macroscopically based on shape, size, color, elevation, margin, and texture, followed by Gram staining to differentiate Gram-positive and Gram-negative bacteria.

Samples were inoculated onto three selective and differential media, MacConkey Agar, Blood Agar, and De Man, Rogosa, and Sharpe Agar (all media from Oxoid Ltd., Basingstoke, United Kingdom) using the quadrant streak method. Inoculated plates were incubated at 35–37°C for 18–24 h in an incubator (Memmert GmbH & Co. KG, Schwabach, Germany).

Further identification was performed using biochemical assays. Catalase and coagulase tests were used to identify aerobic Gram-positive bacteria, particularly *Staphylococcus* spp., and to differentiate them from *Streptococcus* spp. Anaerobic Gram-positive and Gram-negative bacteria were identified using the RapID ANA II System, while aerobic Gram-negative bacteria were identified using the RapID ONE System (Remel Inc., Lenexa, Kansas, USA). Both systems were used according to the manufacturer's instructions, with results interpreted using ERIC software after 4 h of incubation at 35–37°C (Remel Inc., Lenexa, Kansas, USA).

Statistical analysis

The primary analysis compared breast milk and infant fecal microbiota profiles between stunted and normal infants in the overall study population (n=54). Additional subgroup analyses were performed among stunted infants only (n=27) to explore the associations of maternal, environmental, and socioeconomic factors with the detection of specific fecal bacteria. Continuous variables were compared using the Mann–Whitney test or Wilcoxon test, as appropriate, while categorical variables were analyzed using the Chi-square test or Fisher's exact test according to cell distribution. Statistical significance was set at $p<0.05$. Statistical analyses were conducted using IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA).

Results

Characteristics of infants in stunted and normal groups

A total of 54 infants were included, comprising 27 stunted infants and 27 normal infants matched by age and sex; their characteristics are presented in **Table 1**. Among 54 infants, sex and age distributions were similar between the stunted and normal groups, with no significant differences observed (p=1.000 and p=0.963, respectively). Low birth weight was found in both groups equally (3.7%), while high birth weight was found only in the normal group; however, the difference was not significant (p=0.111). Birth length was also comparable between groups (p=1.000). Infant infection was more frequent in the stunted group than in the normal group (25.9% vs 11.1%), although this difference was not statistically significant (p=0.293). Overall, none of the infant characteristics differed significantly between groups (**Table 1**).

Table 1. Comparisons of infants' characteristics between stunted and normal groups (n=54)

Characteristics	Study group		p-value [a]
	Stunted (n=27)	Normal (n=27)	
	n (%)	n (%)	
Sex			1.000
Male	14 (51.9)	14 (51.9)	
Female	13 (48.1)	13 (48.1)	
Age (months)			0.963
6–7	11 (40.7)	10 (37.0)	
8–9	7 (25.9)	8 (29.6)	
10–11	9 (33.3)	9 (33.3)	
Birth weight			0.111
Low birth weight (LBW)	1 (3.7)	1 (3.7)	
Normal	26 (96.3)	22 (81.5)	
High	0 (0.0)	4 (14.8)	
Birth length			1.000
Short	0 (0.0)	1 (3.7)	
Normal	27 (100.0)	26 (96.3)	
Infant infection			0.293
Yes	7 (25.9)	3 (11.1)	
No	20 (74.1)	24 (88.9)	

[a] Differences between groups were analyzed using Chi-square or Fisher's exact test as appropriate.

Comparisons of maternal, household, and environmental characteristics between infants in the stunted and normal groups

Most maternal characteristics were comparable between the stunted and normal groups (**Table 2**). Short maternal height was more frequent among mothers of stunted infants than normal infants (22.2% vs 7.4%), but the difference was not statistically significant (p=0.250). Cesarean delivery was also more common in the stunted group (66.7% vs 51.9%; p=0.268). Maternal weight gain did not differ significantly between groups (p=0.588), and no maternal infection during pregnancy or poor maternal personal hygiene was reported in either group.

Table 2. Comparisons of maternal, household, and environmental characteristics of infants in the stunted and normal groups (n=54)

Characteristics	Study group		p-value [a]
	Stunted (n=27)	Normal (n=27)	
	n (%)	n (%)	
Maternal height (cm)			0.250
Short	6 (22.2)	2 (7.4)	
Normal	21 (77.8)	25 (92.6)	
Mode of delivery			0.268
Cesarean section	18 (66.7)	14 (51.9)	
Vaginal delivery	9 (33.3)	13 (48.1)	
Maternal weight gain			0.588
Inadequate	11 (40.7)	9 (33.3)	
Adequate	12 (44.5)	11 (40.7)	
Excessive	4 (14.8)	7 (26.0)	
Maternal infection during pregnancy			—
Yes	0 (0.0)	0 (0.0)	

Characteristics	Study group		*p*-value [a]
	Stunted (n=27)	Normal (n=27)	
	n (%)	n (%)	
No	27 (100.0)	27 (100.0)	
Maternal personal hygiene			
Poor	0 (0.0)	0 (0.0)	—
Good	27 (100.0)	27 (100.0)	
Maternal education			0.386
Primary	8 (29.6)	4 (14.8)	
Secondary	7 (25.9)	10 (37.0)	
Higher	12 (44.4)	13 (48.1)	
Household income			0.785
Low	14 (51.9)	13 (48.1)	
Adequate	13 (48.1)	14 (51.9)	
Household sanitation			0.039*
Poor	8 (29.6)	2 (7.4)	
Adequate	19 (70.4)	25 (92.6)	
Access to clean water			0.584
Poor	11 (40.7)	13 (48.1)	
Adequate	16 (59.3)	14 (51.9)	

[a] Differences between groups were analyzed using Chi-square or Fisher's exact test as appropriate.

Maternal education was also similar between groups, with higher education reported in 44.4% of mothers in the stunted group and 48.1% in the normal group (p=0.386) (**Table 2**). Household income and access to clean water did not differ significantly between groups (p=0.785 and p=0.584, respectively). However, household sanitation showed a significant difference, with poor sanitation being more common among stunted infants than normal infants (29.6% vs 7.4%; p=0.039). Overall, household sanitation was the only maternal, household, or environmental characteristic that differed significantly between the stunted and normal groups (**Table 2**).

Breast milk microbiota profiles

Bacterial growth was detected in breast milk samples from all 54 enrolled mothers, and their profiles are presented in **Figure 1**. Across both groups, *Streptococcus* and *Staphylococcus* were the predominant genera. In the stunted group, *Streptococcus* sp. was the most prevalent species (48.1%), followed by *Staphylococcus* sp. (40.7%) and *Staphylococcus aureus* (33.3%). In the normal group, a similar pattern was observed but at lower proportions, with *Streptococcus* sp. (33.3%), *Staphylococcus* sp. (25.9%), and *S. aureus* (14.8%).

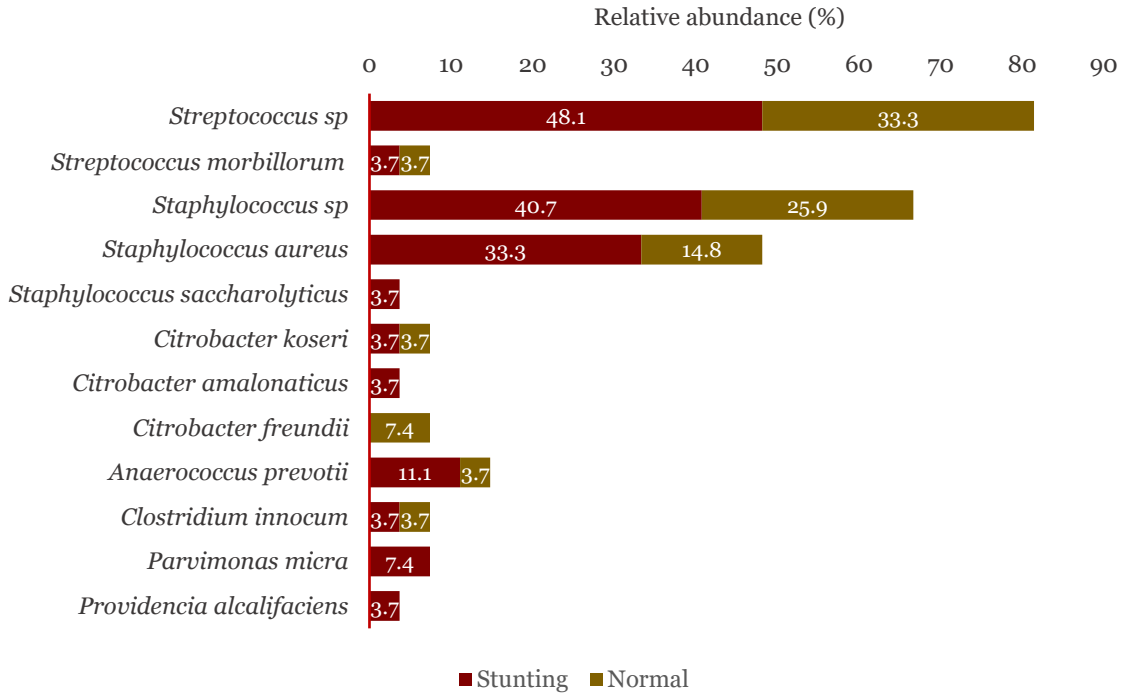

Figure 1. Distribution of breast milk microbiota composition profiles in stunted and normal infants.

Breast milk from mothers of normal infants showed greater microbial diversity, including the presence of *Citrobacter freundii* (7.4%), which was absent in the stunted group. *Anaerococcus prevotii* was detected in both groups, but at a higher proportion among stunted infants (11.1% vs 3.7%) (**Figure 1**). Overall, the culture-based approach identified differences in the detection frequency of selected culturable bacteria in breast milk between groups. *Streptococcus* sp. (48.1%), *Staphylococcus* sp. (40.7%), and *S. aureus* (33.3%) were more frequently detected among mothers of stunted infants (**Figure 1**). However, microbial diversity was not formally assessed and therefore cannot be inferred from these findings.

Fecal microbiota profiles

Fecal bacterial growth was detected in all 54 infant samples and the distribution of fecal microbiota based on genus and species in the stunted and normal infant groups is presented in **Figure 2**. Overall, the fecal microbiota of both groups was dominated by Gram-negative facultative anaerobes, with *E. coli* being the most prevalent species in both the stunted (81.5%) and normal (81.5%) groups. *Streptococcus* sp. was found more frequently in the stunted group (55.6%) compared with the normal group (33.3%). *S. aureus* was detected at a significantly higher proportion in the stunted group (33.3%) than in the normal group (7.4%) (p=0.018). *C. innocuum* was identified in 29.6% of stunted infants and 11.1% of normal infants, with a statistically significant difference (p=0.011). Overall, while *E. coli* predominated in both groups, the stunted group showed a consistently higher prevalence of opportunistic and potentially pathogenic species across multiple taxa, with statistically significant differences observed for *S. aureus* and *C. innocuum* (**Figure 2**).

Bacterial colony counts of breast milk and feces

A comparison of microbial colony counts between stunted and normal infants in both breast milk and fecal samples is presented in **Table 3**. The mean microbial colony count in breast milk among the stunted group was approximately 3.06×10^4 CFU/mL, significantly higher than that of the normal infant group (8.1×10^3 CFU/mL), p=0.001. For fecal samples, the mean colony count was also higher in the stunted group (1.87×10^9 CFU/mL) compared with the normal group (8.75×10^7 CFU/mL), although this difference did not reach statistical significance (p=0.56) (**Table 3**).

Table 3. Comparison of microbial colony counts between stunted and normal infants in both breast milk and fecal samples

Variable	Total (n=54)	Stunted infants (n=27)	Normal infants (n=27)	p-value [a]
Microbial colony count in breast milk (CFU/mL)	1.94×10^4	3.06×10^4	8.1×10^3	0.001*
Microbial colony count in feces (CFU/g)	9.79×10^8	1.87×10^9	8.75×10^7	0.560

CFU: colony-forming units
[a] Analyzed using the Mann–Whitney test
*Statistically significant at p<0.05

Factors associated with breast milk microbiota profiles

Among the factors examined, most infant, maternal, and environmental variables were not significantly associated with breast milk microbiota species composition (p>0.05). Significant associations were observed only for maternal education level (**Table 4**) and household income level (**Table 5**), each of which showed at least one statistically significant association with a specific microbiota species. Fisher's exact test showed that maternal education level was significantly associated only with the presence of *Streptococcus* sp. (p=0.038), whereas no significant associations were observed for the other identified breast milk microbiota (p>0.05) (**Table 4**).

Aslinar et al. Narra X 2026; 4 (2): e294 - http://doi.org/10.52225/narrax.v4i2.294

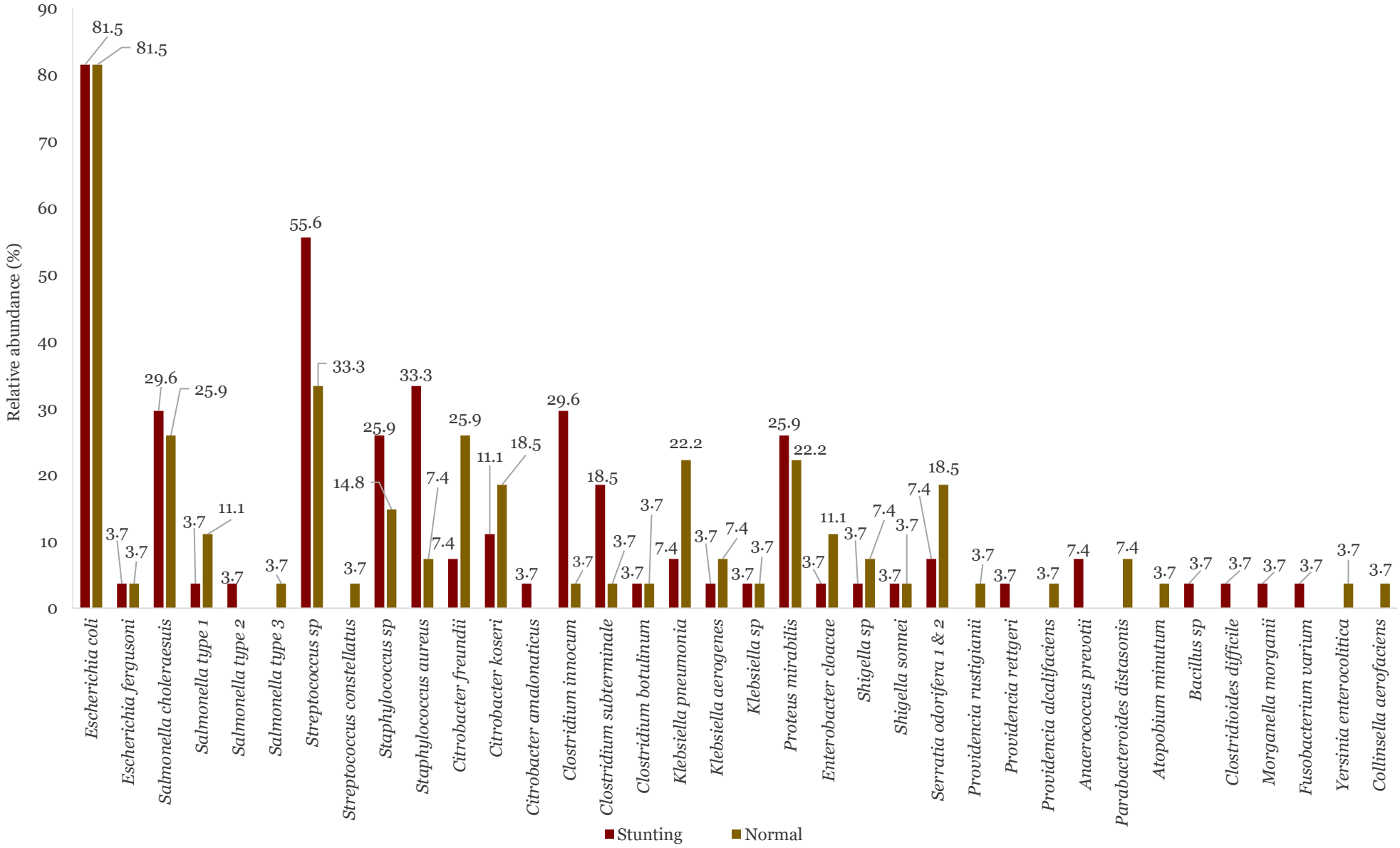

Figure 2. Distribution of infant fecal microbiota composition in stunted and normal groups.

The distribution of breast milk microbiota according to household income level is presented in **Table 5**. The only statistically significant finding was observed for *Streptococcus morbillorum* (p=0.041), which was detected in one participant from the adequate-income group and none from the low-income group. However, because this species was rare and the analysis involved sparse cell counts, this finding should be interpreted cautiously as an exploratory observation.

Table 4. Association between maternal education level and breast milk microbiota composition in stunted infants (n=27)

Microbiota	Maternal education level			p-value
	Primary (n=8)	Secondary (n=7)	Higher (n=12)	
	n (%)	n (%)	n (%)	
Streptococcus sp.	0 (0.0)	4 (30.8)	9 (69.2)	0.038*
Citrobacter koseri	0 (0.0)	1 (100.0)	0 (0.0)	0.617
Citrobacter amalonaticus	0 (0.0)	0 (0.0)	1 (100.0)	0.523
Staphylococcus aureus	1 (11.1)	4 (44.4)	4 (44.4)	0.343
Staphylococcus sp.	0 (0.0)	5 (45.5)	6 (54.5)	0.533
Streptococcus morbillorum	0 (0.0)	1 (100.0)	0 (0.0)	0.617
Clostridium innocuum	0 (0.0)	0 (0.0)	1 (100.0)	0.523
Anaerococcus prevotii	0 (0.0)	3 (100.0)	0 (0.0)	0.209
Staphylococcus saccharolyticus	0 (0.0)	0 (0.0)	1 (100.0)	0.523
Providencia alcalifaciens	0 (0.0)	1 (100.0)	0 (0.0)	0.617
Parvimonas micra	0 (0.0)	2 (100.0)	0 (0.0)	0.367

Associations were analyzed using Fisher's exact test because of small cell counts. Given the exploratory nature of the analyses, small sample size, sparse cells, and multiple comparisons, unadjusted p-values should be interpreted cautiously.
*Statistically significant at $p<0.05$

Table 5. Association between household income level and breast milk microbiota composition in stunted infants (n=27)

Microbiota	Household income level		p-value
	Low-income (n=14) n (%)	Adequate income (n=13) n (%)	
Streptococcus sp.	6 (46.2)	7 (53.8)	0.706
Citrobacter koseri	1 (100.0)	0 (0.0)	1.000
Citrobacter amalonaticus	1 (100.0)	0 (0.0)	1.000
Staphylococcus aureus	5 (55.6)	4 (44.4)	1.000
Staphylococcus sp.	3 (27.3)	8 (72.7)	0.054
Streptococcus morbillorum	0 (0.0)	1 (100.0)	0.041*
Clostridium innocuum	1 (100.0)	0 (0.0)	1.000
Anaerococcus prevotii	2 (66.7)	1 (33.3)	1.000
Staphylococcus saccharolyticus	0 (0.0)	1 (100.0)	0.481
Providencia alcalifaciens	1 (100.0)	0 (0.0)	1.000
Parvimonas micra	1 (50.0)	1 (50.0)	1.000

Associations were analyzed using Fisher's exact test because of small cell counts. Given the exploratory nature of the analyses, small sample size, sparse cells, and multiple comparisons, unadjusted p-values should be interpreted cautiously.

Factors associated with breast milk bacterial colony counts

The associations between infant characteristics and breast milk bacterial colony counts are presented in **Table 6**. Since sex and age were used as matching variables in the case-control design, they were excluded from the association analyses. Therefore, only birth weight, birth length, and infant infection status were examined, and none showed statistically significant associations with breast milk colony counts (all p>0.05).

Table 6. Association of infant characteristics with breast milk colony counts in stunted infants (n=27)

Characteristics	n	Breast milk colony count, median (range), CFU/mL	p-value [a]
Birth weight			0.304
Low birth weight	1	—	
Normal	26	5.9×10^3 (0–2.3×10^5)	

Characteristics	n	Breast milk colony count, median (range), CFU/mL	*p*-value [a]
Birth length			0.354
Short	1	—	
Normal	26	5.9×10^3 ($0–2.3\times10^5$)	
Infant infection			0.561
Yes	7	2.9×10^3 ($0–1.8\times10^5$)	
No	20	6.45×10^3 ($0–2.3\times10^5$)	

[a] Analyzed using the Mann–Whitney test

Associations between maternal, household, and environmental factors and breast milk bacterial colony counts among stunted infants are presented in **Table 7**. No statistically significant associations were observed between any of the examined factors, including maternal height, mode of delivery, maternal weight gain, maternal education, household income, household sanitation, and access to clean water, with breast milk colony counts (all $p>0.05$).

Table 7. Association of maternal, household, and environmental factors with breast milk colony counts in stunted infants (n=27)

Characteristics	n	Breast milk colony count, median (range), CFU/mL	*p*-value [a]
Maternal height (cm)			0.785
Short	4	7.1×10^3 ($2.4\times10^2–1.8\times10^5$)	
Normal	23	5.9×10^3 ($0–2.3\times10^5$)	
Mode of delivery			0.625
Cesarean section	18	2.25×10^3 ($0–2.3\times10^5$)	
Vaginal delivery	9	7×10^3 ($0–1.9\times10^4$)	
Maternal weight gain			0.563
Inadequate	11	4.7×10^3 ($4.3\times10^2–1\times10^5$)	
Adequate	12	3.18×10^3 ($0–2.3\times10^5$)	
Excessive	4	9.8×10^3 ($2.3\times10^3–1.8\times10^5$)	
Maternal infection during pregnancy			—
Yes	0	0	
No	27	5.9×10^3 ($0–2.3\times10^5$)	
Maternal personal hygiene			—
Poor	0	0	
Good	27	5.9×10^3 ($0–2.3\times10^5$)	
Maternal education			0.080
Primary	1	—	
Secondary	14	7.95×10^3 ($0–2.3\times10^5$)	
Higher	12	3.5×10^3 ($0–1\times10^5$)	
Household income			0.698
Low	12	4.65×10^3 ($0–2.3\times10^4$)	
Adequate	15	5.9×10^3 ($4.6\times10^2–9.9\times10^4$)	
Household sanitation			0.826
Poor	15	4.7×10^3 ($0–2.3\times10^5$)	
Adequate	12	7.25×10^3 ($0–1.1\times10^5$)	
Access to clean water			0.958
Poor	15	5.9×10^3 ($0–2.3\times10^5$)	
Adequate	12	1.75×10^3 ($0–1.1\times10^5$)	

[a] Analyzed using the Mann–Whitney test

Factors associated with fecal milk microbiota profiles

Among all examined determinant factors, bivariate analysis of fecal microbiota species composition revealed that the majority of variables, including mode of delivery, maternal weight gain, infant characteristics (birth weight, birth length, and infant infection), household income, household sanitation, and access to clean water, showed no statistically significant associations with any specific fecal microbiota species (all $p>0.05$). Therefore, only maternal height (**Table 8**) and maternal education level (**Table 9**), which each yielded at least one statistically significant association with a specific fecal microbiota species, are presented in tabular form.

The association between maternal height and infant fecal microbiota is presented in **Table 8**. Among the identified microorganisms, only *A. prevotii* showed a statistically significant association with maternal height ($p=0.043$). This finding indicates that maternal height was

significantly associated with the presence of *A. prevotii*, which was detected exclusively in infants born to mothers with short stature.

Table 8. Association between maternal height and fecal microbiota composition in stunted infants (n=27)

Microbiota	Maternal height		*p*-value
	Normal (n=21) n (%)	Short (n=6) n (%)	
Serratia odorifera	1 (50)	1 (50)	0.402
Escherichia coli	17 (77.3)	5 (22.7)	1.000
Citrobacter freundii	2 (100.0)	0 (0.0)	1.000
Citrobacter koseri	3 (100.0)	0 (0.0)	0.455
Streptococcus sp.	9 (75)	3 (25)	1.000
Salmonella choleraesuis	6 (75)	2 (25)	1.000
Staphylococcus aureus	6 (66.7)	3 (33.3)	0.367
Clostridium subterminale	4 (80)	1 (20)	1.000
Clostridium innocuum	6 (75)	2 (25)	1.000
Staphylococcus sp.	4 (57.1)	3 (42.9)	0.290
Proteus mirabilis	6 (85.7)	1 (14.3)	1.000
Anaerococcus prevotii	0 (0.0)	2 (100.0)	0.043*
Klebsiella sp.	0 (0.0)	1 (100.0)	0.222
Klebsiella aerogenes	0 (0.0)	1 (100.0)	0.222
Bacillus sp.	0 (0.0)	1 (100.0)	0.222
Morganella morganii	0 (0.0)	1 (100.0)	0.222
Eggerthella lenta	2 (100.0)	0 (0.0)	1.000

Associations were analyzed using Fisher's exact test because of small cell counts. Given the exploratory nature of the analyses, small sample size, sparse cells, and multiple comparisons, unadjusted *p*-values should be interpreted cautiously.
*Statistically significant at $p<0.05$

The association between maternal education level and infant fecal microbiota is presented in **Table 9**. *S. choleraesuis* was predominantly detected in the secondary education group (87.5%) and was absent in the higher education group ($p=0.006$), while *P. mirabilis* was detected exclusively in the secondary education group (100%) ($p=0.012$). In addition, *E. lenta* was identified in the primary (50%) and secondary education groups (50%), but not in the higher education group ($p=0.001$).

Factors associated with fecal bacterial colony counts

The associations between infant characteristics and fecal colony counts are presented in **Table 10**. Since sex and age were used as matching variables in the case-control design, they were excluded from the association analyses. Therefore, only birth weight, birth length, and infant infection status were examined, and none showed statistically significant associations with fecal colony counts (all $p>0.05$).

Table 9. Association between maternal education level and fecal microbiota composition in stunted infants (n=27)

Microbiota	Maternal education level			*p*-value
	Primary (n=8) n (%)	Secondary (n=7) n (%)	Higher (n=12) n (%)	
Serratia odorifera	0 (0.0)	1 (50.0)	1 (50.0)	0.953
Escherichia coli	1 (4.5)	10 (45.5)	11 (50.0)	0.370
Citrobacter freundii	0 (0.0)	0 (0.0)	2 (100.0)	0.259
Citrobacter koseri	0 (0.0)	1 (33.3)	2 (66.7)	0.697
Streptococcus sp.	0 (0.0)	7 (58.3)	5 (41.7)	0.603
Salmonella type I	0 (0.0)	0 (0.0)	1 (100.0)	0.227
Shigella sp.	0 (0.0)	1 (100.0)	0 (0.0)	0.617
Salmonella choleraesuis	1 (12.5)	7 (87.5)	0 (0.0)	0.006*
Klebsiella sp.	0 (0.0)	0 (0.0)	1 (100.0)	0.523
Staphylococcus aureus	0 (0.0)	5 (55.6)	4 (44.4)	0.765
Clostridium subterminale	0 (0.0)	4 (80.0)	1 (20.0)	0.370
Klebsiella pneumoniae	0 (0.0)	0 (0.0)	2 (100.0)	0.259
Enterobacter cloacae	0 (0.0)	1 (100.0)	0 (0.0)	0.617
Streptococcus constellatus	0 (0.0)	0 (0.0)	1 (100.0)	0.523

Microbiota	Maternal education level			*p*-value
	Primary (n=8) n (%)	Secondary (n=7) n (%)	Higher (n=12) n (%)	
Clostridium innocuum	0 (0.0)	2 (25.0)	6 (75.0)	0.111
Proteus mirabilis	0 (0.0)	7 (100.0)	0 (0.0)	0.012*
Staphylococcus sp.	0 (0.0)	4 (57.1)	3 (42.9)	0.816
Anaerococcus prevotii	0 (0.0)	1 (50.0)	1 (50.0)	0.953
Clostridioides difficile	0 (0.0)	1 (100.0)	0 (0.0)	0.617
Morganella morganii	0 (0.0)	0 (0.0)	1 (100.0)	0.523
Eggerthella lenta	1 (50.0)	1 (50.0)	0 (0.0)	0.001*

Associations were analyzed using Fisher's exact test because of small cell counts. Given the exploratory nature of the analyses, small sample size, sparse cells, and multiple comparisons, unadjusted *p*-values should be interpreted cautiously.
*Statistically significant at $p<0.05$

Table 10. Association of infant characteristics with fecal colony counts in stunted infants (n=27)

Characteristics	n	Fecal colony count, median (range), CFU/g	*p*-value [a]
Birth weight			0.248
Low birth weight	1	—	
Normal birth weight	26	2.3×10^4 (5×10^1–3×10^{10})	
Birth length			—
Short birth length	0	—	
Normal birth length	27	2.3×10^4 (5×10^1–3×10^{10})	
Infant infection			0.376
Present	7	9.7×10^5 (1.5×10^2–3×10^{10})	
Absent	20	2.3×10^4 (5×10^1–2×10^{10})	

[a] Analyzed using the Mann–Whitney test

Associations between maternal, household, and environmental factors and fecal bacterial colony counts among stunted infants are presented in **Table 11**. No statistically significant associations were observed between fecal colony counts and any of the examined factors, including maternal height, mode of delivery, maternal weight gain, maternal education, household income, household sanitation, and access to clean water (all $p>0.05$).

Table 11. Association of maternal, household, and environmental factors with fecal colony counts in stunted infants (n=27)

Characteristics	n	Fecal colony count, median (range), CFU/g	*p*-value [a]
Maternal height (cm)			0.243
Short	6	9.2×10^3 (1.8×10^2–2×10^8)	
Normal	21	1.2×10^5 (5×10^1–3×10^{10})	
Mode of delivery			0.520
Cesarean section	18	1.95×10^4 (5×10^1–3×10^{10})	
Vaginal delivery	9	1.2×10^5 (6.3×10^3–2×10^9)	
Maternal weight gain			0.434
Inadequate	11	6.3×10^6 (5×10^1–3×10^{10})	
Adequate	12	1.95×10^4 (1.5×10^2–2×10^9)	
Excessive	4	4.93×10^5 (1.3×10^3–2×10^8)	
Maternal infection during pregnancy			—
Yes	0	—	
No	27	2.3×10^4 (5×10^1–3×10^{10})	
Maternal personal hygiene			—
Poor	0	—	
Good	27	2.3×10^4 (5×10^1–3×10^{10})	
Maternal education			0.092
Primary	1	—	
Secondary	14	3.6×10^6 (1.8×10^2–3×10^{10})	
Higher	12	1.45×10^4 (5×10^1–2×10^{10})	
Household income			0.357
Low	13	2.2×10^4 (3.5×10^2–2×10^9)	
Adequate	14	1.2×10^5 (5×10^1–3×10^{10})	
Household sanitation			0.884
Poor	15	1.2×10^5 (5×10^1–3×10^{10})	
Adequate	12	2.2×10^4 (1.5×10^2–2×10^{10})	
Access to clean water			0.494

Characteristics	n	Fecal colony count, median (range), CFU/g	*p*-value [a]
Poor	15	2.3×10^4 (5×10^1–3×10^{10})	
Adequate	12	9.01×10^6 (1.8×10^2–2×10^{10})	

[a] Analyzed using the Mann–Whitney test

Discussion

This study examined culture-detectable differences in breast milk and infant fecal microbiota profiles between stunted and normal infants in Aceh, Indonesia. The findings suggest differences in the detection frequency of selected culturable bacteria and colony counts between groups. However, because this study used culture-based methods, the results reflect only viable culturable bacteria and do not represent comprehensive microbiome composition, microbial diversity, or formal dysbiosis. Therefore, the findings should be interpreted as exploratory and hypothesis-generating.

The baseline characteristics of the infants were matched between the stunted and normal groups in terms of age and sex, in accordance with the sampling criteria of this study. A higher proportion of infants with a history of infection was observed in the stunted group compared with the normal group. Recurrent infections during early life may disrupt gut microbiota stability through increased intestinal permeability, mucosal inflammation, and potential antibiotic exposure [13]. These findings support the hypothesis that postnatal factors, particularly infection exposure, may contribute to alterations in the breast milk and gut microbiota profiles, which may subsequently influence infant growth status.

A significantly higher proportion of poor household sanitation was observed in the stunted group compared with the normal group (29.6% vs 7.4%; p=0.039) (**Table 1**). This finding is consistent with evidence that inadequate household sanitation is among the key environmental determinants of stunting in Indonesia, operating through increased pathogen exposure and the promotion of fecal–oral transmission routes [5,14]. Poor sanitation conditions are known to facilitate colonization of the infant gastrointestinal tract by enteric pathogens, which in turn trigger mucosal inflammation, impair intestinal barrier integrity, and ultimately compromise nutrient absorption, a cascade of events recognized as environmental enteric dysfunction (EED) [12,15]. The present findings reinforce the hypothesis that household sanitation conditions represent a proximal environmental pathway linking socioeconomic disadvantage to impaired linear growth in early childhood.

One of the central findings was the predominance of *Streptococcus* and *S. aureus* in the breast milk of mothers with stunted infants (**Figure 1**). A study has shown that breast milk microbiota plays a critical role in seeding the infant gut [16]. While *Streptococcus* and *Staphylococcus* are commonly detected in human milk, the higher detection frequency of *S. aureus* is notable because this species has been associated with mastitis and may represent a clinically relevant culturable bacterium in breast milk [17]. The culture-based approach identified differences in the detection frequency of selected culturable bacteria in breast milk from mothers of stunted and normal infants. However, microbial diversity was not formally assessed; therefore, these findings should not be interpreted as evidence of reduced breast milk microbial diversity.

E. coli was the most prevalent microbiota species in feces of both groups, accounting for 81.5% in both stunted and normal infants (**Figure 2**). The predominance of *E. coli* among infants aged 6–11 months reflects the early maturation phase of the gut microbiota, which is typically dominated by facultative anaerobic bacteria before transitioning to obligate anaerobes such as *Bifidobacterium* and *Bacteroides* as dietary diversification progresses. A high proportion of Enterobacteriaceae in infants living in environments with limited sanitation is often associated with fecal–oral pathogen exposure and low-grade intestinal mucosal inflammation. These conditions may contribute to intestinal barrier dysfunction and increase the risk of linear growth impairment [12,18,19].

The proportions of *S. aureus* and *C. innocuum* were higher in the stunted group compared with the normal group (**Figure 2**). The presence of *S. aureus* in the infant gut reflects opportunistic colonization, which may be associated with environmental hygiene conditions, household exposure, or antibiotic use. A recent study showed that colonization by opportunistic bacteria in infants is associated with reduced gut microbiota diversity and an increased risk of

intestinal mucosal inflammation, which may indirectly influence nutritional status and growth [20]. *C. innocuum* has been reported as an opportunistic bacterium that may increase under conditions of dysbiosis and impaired mucosal immunity. Emerging evidence suggests that the proliferation of certain non-commensal *Clostridium* species is associated with intestinal inflammation, disruption of short-chain fatty acid metabolism, and reduced energy utilization efficiency, which in infants and young children may contribute to growth faltering [12,19]. Although most differences were not statistically significant, the higher proportions of *S. aureus* and *C. innocuum* in the stunted group remain biologically relevant, as they may indicate gut dysbiosis and mucosal inflammation.

The results demonstrated a significant association between maternal education level and the presence of *Streptococcus* sp. in breast milk (p=0.038) (**Table 4**), as well as an association between household income level and *S. morbillorum* (p=0.041), with a tendency toward differences in *Staphylococcus* sp (**Table 5**). The finding that mothers with higher educational attainment had a greater proportion of *Streptococcus* sp. colonization is consistent with previous literature indicating that education level is associated with diet quality, health literacy, and hygiene practices during breastfeeding, which may influence breast milk microbiota composition through skin microbiota transfer and the entero-mammary pathway. The presence of *Streptococcus* sp. in breast milk, in the context of early infant colonization, generally reflects physiological commensal microbiota that function as early colonizers in the establishment of the infant gut microbiota ecosystem, provided that pathogenic species do not dominate [21,22]. Maternal education is associated with health literacy, diet quality, hygiene practices, and adherence to breastfeeding promotion, which may indirectly influence the composition of breast milk microbiota. Household income reflects access to nutritious food, clean water, and healthcare services, which in turn affects pathogen exposure and microbiota quality. Recent literature suggests that socioeconomic determinants shape microbial ecology through nutritional and environmental pathways, thereby influencing infant gut colonization and the risk of stunting [23,24].

A significant association was observed between maternal height and the presence of *A. prevotii* in infant feces, where this bacterium was found exclusively among infants born to mothers with short stature (**Table 8**). Maternal height reflects chronic nutritional status and long-term environmental exposures since childhood. Maternal chronic nutritional status may influence the intrauterine environment, fetal immune system maturation, and maternal microbiota profile, which subsequently affect the initial colonization of the infant gut microbiota [25].

The colonization of *S. choleraesuis* and *P. mirabilis* in infants suggests exposure to contaminated environmental sources or food (**Table 9**). Enteric pathogens are more frequently identified in populations with limited access to clean water and inadequate sanitation, conditions that are commonly associated with lower socioeconomic status. Repeated colonization by pathogenic bacteria has been linked to chronic intestinal inflammation and impaired linear growth, particularly through mechanisms related to environmental enteric dysfunction [26,27]. The detection of *E. lenta*, an opportunistic anaerobic bacterium associated with intestinal mucosal inflammation, further indicates the potential presence of microbial dysbiosis in specific socioeconomic groups. Dysbiosis, characterized by the presence of opportunistic or pathogenic microorganisms, may disrupt intestinal barrier function, alter host–microbe metabolic interactions, and contribute to subclinical intestinal inflammation, which has been implicated in growth impairment during early childhood [28].

These findings highlight the role of socioeconomic determinants, particularly maternal education, as an important factor influencing the composition of the infant gut microbiota (**Table 9**). Maternal education is closely associated with health literacy, household hygiene practices, food safety behavior, infant feeding practices, and utilization of healthcare services. Collectively, these factors influence the risk of colonization by pathogenic and opportunistic bacteria, thereby shaping the early microbial ecosystem of the infant gut [29].

This study has some limitations that need to be acknowledged. Microbiota characterization was based on conventional culture methods, which detect only culturable bacteria and do not provide comprehensive microbiome profiling compared with 16S rRNA sequencing or

metagenomic approaches. Therefore, the study could not assess alpha diversity, beta diversity, and relative abundance across the full microbial community or microbial functional pathways. Microbial samples were collected at a single time point, preventing assessment of temporal changes in breast milk and infant gut bacteria or their relationship with the onset of stunting. In addition, data on maternal diet, complementary feeding, formula use, probiotic exposure, and detailed antibiotic history were not collected, although these factors may strongly influence infant gut microbiota. Furthermore, multiple Fisher's exact tests were conducted across many bacterial species, increasing the possibility of false-positive findings. Because the study was exploratory and the sample size was small, unadjusted *p*-values should be interpreted cautiously. Although infants were matched by age and sex, the analysis did not fully account for individual matched-pair structure. Finally, functional outcomes such as short-chain fatty acid production, bile acid metabolism, intestinal inflammation, and intestinal barrier function were not measured; therefore, mechanistic conclusions cannot be drawn.

Conclusion

This exploratory case-control study identified culture-detectable differences in breast milk and infant fecal microbiota profiles between stunted and normal infants in Aceh, Indonesia. Breast milk from mothers of stunted infants showed higher colony counts and higher detection frequencies of selected culturable bacteria, while fecal samples from stunted infants showed higher detection of *S. aureus* and *C. innocuum*. However, fecal colony counts did not differ significantly between groups. Some maternal and socioeconomic characteristics showed potential associations with selected bacterial species, but these findings should be interpreted cautiously because of the small sample size, sparse cell counts, multiple comparisons, and the culture-based cross-sectional design. The results should therefore require confirmation through larger longitudinal studies using sequencing-based microbiome profiling and functional biomarkers.

Ethics approval

Ethical approval was obtained from the Health Research Ethics Committee, Faculty of Medicine, Universitas Syiah Kuala – Dr. Zainoel Abidin General Hospital, Banda Aceh (KEPPKN Reg. No. 1171012P; Approval No. 132/EA/FK-RSUDZA/2021).

Acknowledgments

None.

Competing interests

The authors declare that there are no conflicts of interest.

Funding

This study received no external funding.

Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

Declaration of artificial intelligence use

We hereby confirm that no artificial intelligence (AI) tools or methodologies were utilized at any stage of this study, including during data collection, analysis, visualization, or manuscript preparation. All work presented in this study was conducted manually by the authors without the assistance of AI-based tools or systems.

How to cite

Aslinar A, Dimiati H, Indrawati N, Sofia S. Culture-based breast milk and infant gut microbiota profiles among stunted infants: A case-control study from Indonesia. Narra X 2026; 4 (2): e294 - http://doi.org/10.52225/narrax.v4i2.294.

References

1. UNICEF, World Health Organization, The World Bank. Levels and trends in child malnutrition: UNICEF/WHO/World Bank Group Joint Child Malnutrition Estimates. Geneva: World Health Organization; 2025.
2. Kementerian Kesehatan Republik Indonesia. Buku saku hasil survei status gizi Indonesia (SSGI). Jakarta: Kementerian Kesehatan RI; 2022.
3. Kementerian Kesehatan Republik Indonesia. Survei kesehatan Indonesia tahun 2023. Jakarta: Kementerian Kesehatan RI; 2023.
4. Beal T, Tumilowicz A, Sutrisna A, *et al.* A review of child stunting determinants in Indonesia. Matern Child Nutr 2018;14(4):e12617.
5. Wicaksono F, Harsanti T. Determinants of stunted children in Indonesia: A multilevel analysis at the individual, household, and community levels. Kesmas 2020;15(1):48-53.
6. Neu J. Fueling the optimal microbiome: interventions for severe acute malnutrition. Cell Host Microbe 2019;26(3):307–308.
7. de Onis M, Branca F. Childhood stunting: A global perspective. Matern Child Nutr 2016;12:12-26.
8. Lyons KE, Ryan CA, Dempsey EM, *et al.* Breast milk, a source of beneficial microbes and associated benefits for infant health. Nutrients 2020;12(4):1-30.
9. Xu D, Wan F. Breastfeeding and infant gut microbiota: influence of bioactive components. Gut Microbes 2025;17(1):2446403.
10. Beghetti I, Biagi E, Martini S, *et al.* Human milk's hidden gift: Implications of the milk microbiome for preterm infants' health. Nutrients 2019;11(12):1-13.
11. Gomez-Gallego C, Garcia-Mantrana I, Salminen S, *et al.* The human milk microbiome and factors influencing its composition and activity. Semin Fetal Neonatal Med 2016;21(6):400-405.
12. Vonaesch P, Morien E, Andrianonimiadana L, *et al.* Stunted childhood growth is associated with decompartmentalization of the gastrointestinal tract and overgrowth of oropharyngeal taxa. Proc Natl Acad Sci USA 2018;115(36):E8489-E8498.
13. Clara M, Ribeiro SA, Sousa LS De, *et al.* Undernutrition and intestinal infections in children: A narrative review. Nutrients 2025;17(9):1479.
14. Vilcins D, Sly PD, Jagals P. Environmental risk factors associated with child stunting: A systematic review of the literature. Ann Glob Health 2018;84(4):551-562.
15. Collado MC, Cernada M, Baüerl C, *et al.* Microbial ecology and host-microbiota interactions during early life stages. Gut Microbes 2012;3(4).
16. Notarbartolo V, Giuffre M, Montante C, *et al.* Composition of human breast milk microbiota and its role in children's health. Pediatr Gastroenterol Hepatol Nutr 2022;25(3):194-210.
17. Liu A, Garrett S, Hong W, *et al.* Staphylococcus aureus Infections and Human Intestinal Microbiota 2024:1-16.
18. Cowardin CA, Syed S, Iqbal N, *et al.* Environmental enteric dysfunction: Gut and microbiota adaptation in pregnancy and infancy. Nat Rev Gastroenterol Hepatol 2023;20(4):223-237.
19. Zhao L, Yang X, Liang Y, *et al.* Temporal development and potential interactions between the gut microbiome and resistome in early childhood. Microbiol Spectr 2024;12(2):e0317723.
20. Dombrowska-Pali A, Wiktorczyk-kapischke N, Chrustek A, *et al.* Human milk microbiome-A review of scientific reports. Nutrients 2024;16(10):1420.
21. Hardjo J, Selene NB, Selene NB. Stunting and gut microbiota: A literature review. Pediatr Gastroenterol Hepatol Nutr 2024;27(3):137-145.
22. Fernández L, Pannaraj PS, Rautava S, Rodríguez JM. The microbiota of the human mammary ecosystem. Front Cell Infect Microbiol 2020;10:586667.
23. Ratcliff JS, Kumari M, Varga-Weisz P, O'Gorman R. Socioeconomic position and the gut microbiota: A narrative synthesis of the association and recommendations. Gut Microbes 2026;18(1):2623356.
24. Tamarelle J, Doria MV, Rambolamanana V, *et al.* Socioeconomic and nutritional determinants outweigh gut microbiota influence on neurodevelopment in young children from Antananarivo, Madagascar. Sci Rep 2026;16:5484.
25. Fajar NA, Liberty I, Ananingsih ES, *et al.* Utilizing maternal height as a predictor for childhood stunting prevention: A health promotion strategy rooted in early risk identification. Media Publ Promosi Kesehatan Indones 2025;8(11).
26. Billah MM, Rahman S. Salmonella in the environment: A review on ecology, antimicrobial resistance, seafood contaminations, and human health implications☆. J Hazard Mater Adv 2024;13(6):100407.

27. Chakkour M, Hammoud Z, Farhat S. Overview of *Proteus mirabilis* pathogenicity and virulence. Insights into the role of metals. Front Microbiol 2024;15:1383618.
28. Buttimer C, Bottacini F, Shkoporov AN, *et al*. Selective isolation of *Eggerthella lenta* from human faeces and characterisation of the species prophage diversity. Microorganisms 2022;10(1):195.
29. Fatema SR, Kabir MH. Maternal education, hygienic practices and children's health status. EPH-Int J Med Health Sci 2015;1(4):24-30.