RESEARCH FINAL REPORT

HOUSEHOLD FOOD SECURITY, FEEDING PRACTICES, AND *CHRONONUTRITION* BETWEEN STUNTED AND NON-STUNTED CHILDREN AGED 6–23 MONTHS

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DESCRIPTION

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EXECUTIVE SUMMARY

Stunting remains an important nutritional problem in Indonesia, particularly during the first 1,000 days of life. Stunting reflects impaired linear growth resulting from chronic or recurrent nutritional and health-related constraints and may have consequences for child development, health, productivity, and human-resource quality. The period from 6 to 23 months is particularly critical because children experience rapid physical and neurodevelopmental growth while transitioning from predominantly breast-milk feeding to complementary foods. During this period, inadequate dietary intake, inappropriate feeding practices, recurrent infection, and unfavorable household conditions may contribute to growth faltering.

Household food security and infant and young child feeding practices are important components of the nutritional environment. However, adequate food availability alone does not necessarily guarantee adequate nutritional status because food selection, feeding practices, caregiving, disease, and access to health services also influence child growth. In addition, chrononutrition—the timing, frequency, duration, and regularity of food intake in relation to biological rhythms—has emerged as a potentially relevant dimension of child nutrition. Evidence concerning chrononutrition among children aged 6–23 months remains limited, particularly in relation to stunting. Therefore, examining household food security, feeding practices, nutrient adequacy, morbidity, and chrononutrition together may provide a more comprehensive understanding of the factors associated with linear growth during early childhood.

The general objective of this study was to analyze differences in household food security, child feeding practices, and chrononutrition between stunted and non-stunted children aged 6–23 months. The specific objectives were to compare child and family characteristics; household food security; breastfeeding and complementary feeding practices; energy, macronutrient, and micronutrient adequacy; chrononutrition patterns; infectious-disease morbidity; and nutritional status based on weight-for-age (WAZ) and weight-for-height (WHZ). The study also aimed to examine the relationships of these factors with stunting and height-for-age Z-score (HAZ).

This was a quantitative exploratory study using a comparative cross-sectional design. The research was conducted in Sayang Village, Cianjur Subdistrict, Cianjur Regency, West Java, Indonesia, a designated stunting locus. Mother-child pairs were recruited purposively according to predefined inclusion and exclusion criteria. The final analytical sample consisted of 160 children aged 6–23 months, including 48 stunted children (30.0%) and 112 non-stunted children (70.0%).

Data were collected through structured maternal interviews, child health records, anthropometric measurements, household food-security assessment, feeding-practice questionnaires, dietary assessment, chrononutrition assessment, and morbidity assessment. Household food security was assessed using the eight-item Food Insecurity Experience Scale (FIES). Breastfeeding and complementary feeding practices were assessed using indicators adapted from the 2023 Indonesian Health Survey and WHO/UNICEF infant and young child feeding indicators. Two non-consecutive 24-hour dietary recalls were used to assess energy and nutrient intake. Chrononutrition assessment included meal and snack timing, eating intervals, meal duration, eating-window duration, breastfeeding frequency and timing, and relationships between feeding and sleep. Nutritional status was assessed using standardized measurements of body weight, recumbent

length, head circumference, and mid-upper arm circumference, with WHO growth standards used for classification.

Data were processed using Microsoft Excel and SPSS 27. Independent-samples *t*-tests were used for continuous variables, chi-square tests for categorical variables, and Pearson correlation tests to examine relationships between continuous variables and HAZ. Ethical approval was obtained from the Research Ethics Committee of Universitas Muhammadiyah Semarang (Reference No. 001/KE/04/2026), and written informed consent was obtained from all participating mothers.

The prevalence of stunting in the study population was 30.0%. Stunted children differed significantly from non-stunted children in several individual characteristics. Boys represented a larger proportion of the stunted group than the non-stunted group (60.4% vs. 41.1%, p=0.019). Stunted children were also older, with a mean age of 15.6 ± 5.4 months compared with 12.9 ± 5.0 months among non-stunted children (p=0.003). Children aged 19–23 months constituted a substantially larger proportion of the stunted group. Birth length was significantly shorter among stunted children (48.4 ± 3.5 cm) than among non-stunted children (49.3 ± 3.5 cm; p=0.010). Birth order, mode of delivery, gestational age, and birth weight did not differ significantly between groups.

The correlation analysis further indicated that age was negatively correlated with HAZ (r=-0.268, p=0.001), whereas birth weight (r=0.156, p=0.049), birth length (r=0.186, p=0.019), and age gap with the nearest older sibling (r=0.208, p=0.012) were positively correlated with HAZ. These relationships were weak, emphasizing that linear growth is influenced by multiple factors rather than by any single characteristic.

Family and socioeconomic characteristics did not show statistically significant differences between stunted and non-stunted children. Parental education, parental occupation, parental age, number of children under five, family size, per-capita household income, and economic status all had p>0.05. Per-capita income tended to be lower among households with stunted children, but the difference was not statistically significant. These findings indicate that the socioeconomic indicators measured in this relatively specific study population did not clearly distinguish the two groups.

More than half of the households (53.1%) experienced some level of food insecurity. Nevertheless, household food-security status did not differ significantly between households with stunted and non-stunted children. The mean FIES score also showed no significant difference between the two groups and was not significantly correlated with HAZ (r=-0.057, p=0.476). Thus, food insecurity was widespread but did not statistically differentiate linear growth status in this sample.

Morbidity was relatively common, with acute respiratory infection (ARI) being the most frequently reported condition, averaging 0.81 episodes per month. Stunted children experienced significantly more dysentery (p=0.030) and toothache (p=0.014), while dermatological disorders were more frequently reported among non-stunted children (p=0.034). ARI frequency was also higher among stunted children, although the difference was borderline and did not reach conventional statistical significance (p=0.052). Importantly, ARI (r=-0.179, p=0.024), urinary tract infection (r=-0.172, p=0.030), and toothache (r=-0.185, p=0.019) were weakly but

significantly associated with lower HAZ. Cumulative illness duration, however, did not differ significantly between groups.

The assessment of infant and young child feeding revealed several areas of suboptimal practice. Early initiation of breastfeeding, exclusive breastfeeding, and minimum milk-feeding frequency remained important areas for improvement. Continued breastfeeding was relatively common, with 72.5% of children still breastfed. Most breastfeeding and complementary-feeding indicators did not differ significantly between stunted and non-stunted children. Mixed milk feeding was the only breastfeeding-related indicator showing a significant difference, occurring in 29.2% of stunted children compared with 45.5% of non-stunted children. Approximately 68.1% of children were introduced to complementary foods at six months, while 29.4% were introduced earlier and 2.5% later. Overall, these findings suggest that conventional feeding indicators alone did not adequately explain the differences in linear growth.

Dietary assessment showed that 43.8% of children had inadequate energy intake. Energy inadequacy was more prevalent among stunted children than non-stunted children (52.1% vs. 40.2%), although the difference was not statistically significant. Protein intake was excessive for approximately 63% of children in both groups. Calcium inadequacy was particularly common among stunted children (70.8%) compared with non-stunted children (58.9%). Inadequacy of fat, iron, vitamin C, and zinc also tended to be higher among stunted children. However, no significant differences were found in the categories or mean intakes of energy and the assessed nutrients between groups. Importantly, iron adequacy showed a significant positive correlation with HAZ (r=0.284, p<0.001), whereas energy and macronutrient adequacy were not significantly correlated with HAZ. Calcium showed a positive trend but did not reach statistical significance (r=0.151, p=0.057).

Chrononutrition provided additional evidence of differences in the temporal organization of feeding. Compared with non-stunted children, stunted children more frequently exhibited earlier evening eating, shorter meal durations, shorter peak breastfeeding duration, and a shorter daily eating window. The mean eating window was 10.01 ± 2.02 hours among stunted children compared with 10.79 ± 2.09 hours among non-stunted children (p=0.030). A particularly notable pattern was that 72.9% of stunted children had completed evening eating by 17:00, whereas 12.5% of non-stunted children continued eating after 20:00. Several temporal variables were significantly associated with HAZ: longer peak breastfeeding duration, longer breakfast, lunch, and dinner durations, and a wider eating window were associated with higher HAZ. However, the correlations were generally weak.

Sleep characteristics were generally similar in timing between groups, although total sleep duration differed significantly. Longer first-nap duration, total nap duration, and total sleep duration showed weak negative correlations with HAZ, while nighttime sleep duration was not significantly associated with HAZ. These findings are exploratory and should not be interpreted as evidence that longer sleep causes poorer growth.

Regarding overall nutritional status, stunting was accompanied by other forms of undernutrition in some children. The prevalence of underweight was 15.0%, wasting 3.8%, chronic energy deficiency 2.5%, overweight 1.2%, and obesity 2.5%. Stunted children had a significantly higher

risk of being underweight than non-stunted children, indicating that linear growth retardation may coexist with inadequate body weight in this population.

The findings support an integrated approach to child-growth promotion rather than reliance on a single intervention. Nutrition education and growth monitoring should emphasize diverse, nutrient-dense, safe, and age-appropriate complementary foods, with particular attention to iron-rich foods and other micronutrient sources. Breastfeeding should be appropriately continued alongside complementary feeding, while caregivers should receive practical guidance on responsive feeding and adequate feeding opportunities.

Routine growth monitoring should also incorporate information on dietary intake, feeding practices, morbidity, and household circumstances. Children experiencing recurrent illnesses, particularly respiratory infections, urinary tract infections, or oral-health problems, should receive closer nutrition and growth monitoring and timely referral when needed.

Counseling may also introduce appropriate daily routines for breastfeeding, meals, snacks, and sleep. However, specific recommendations concerning meal timing, eating-window duration, meal duration, or sleep duration should be applied cautiously because the observed associations were generally weak and the cross-sectional design does not establish causality.

Finally, future research should employ longitudinal or prospective designs to clarify the temporal and potentially bidirectional relationships among feeding behavior, chrononutrition, sleep, dietary adequacy, morbidity, household food security, and linear growth. Future studies should also consider multivariable models incorporating maternal nutritional status and height, birth characteristics, sanitation, water quality, healthcare utilization, and other relevant determinants. Such approaches would provide stronger evidence for identifying pathways through which household, dietary, health, and temporal factors influence stunting.

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INTRODUCTION

The Sustainable Development Goals (SDGs) represent a global action plan aimed at addressing poverty, reducing inequalities, and protecting the planet. Improving population nutritional status is one of the important objectives within the SDGs (United Nations, 2023). Adequate nutritional status is also an important indicator of human resource quality. In Indonesia, stunting remains a major nutritional problem requiring particular attention. Stunting is defined as impaired linear growth in children under five years of age resulting from chronic undernutrition, leading to a height that is too short for their age. Although the underlying nutritional deficiencies may begin during pregnancy and the early postnatal period, their effects often become apparent by the age of two years. Stunting may have long-term consequences, including impaired cognitive development, increased susceptibility to illness, reduced productivity, and constraints on economic growth, while also contributing to persistent poverty and inequality.

The 2023 Indonesian Health Survey indicated that stunting remains a major nutritional problem among children under two years of age. Compared with other nutritional problems, such as underweight (13.3%) and wasting (9.2%), stunting had a higher prevalence of 18.3%. This finding indicates that efforts to address stunting continue to face substantial challenges despite the implementation of various intervention programs. The relatively high prevalence of stunting among children in this age group suggests the presence of complex and persistent risk factors originating from early life (Indonesian Ministry of Health, 2023).

The determinants of stunting during the first 1,000 days of life are multidimensional. Household food security and child feeding practices may influence stunting in children under two. A systematic review demonstrated a significant association between household food security and stunting among children under five years of age in Indonesia (Dewi *et al.*, 2024)

Inappropriate feeding practices, including inadequate exclusive breastfeeding and complementary feeding, may increase the risk of nutritional problems such as stunting (Jeyakumar *et al.,* 2022). The coverage of exclusive breastfeeding in Indonesia remains suboptimal, with nearly half of children under two years of age (44.5%) not receiving exclusive breastfeeding. Furthermore, only 60.9% of children under two years of age consume a diverse diet (Indonesian National Food Agency, 2023).

No single food contains all nutrients required by the human body in adequate amounts, except breast milk, which provides the nutritional requirements of infants during the first six months of life (Khomsan *et al.*, 2023). Exclusive breastfeeding refers to feeding infants aged 0–6 months exclusively with breast milk without the introduction of other foods or liquids, as recommended (WHO, 2019). At six months of age, infants should begin receiving complementary foods alongside continued breastfeeding, with the variety, consistency, and texture of complementary foods gradually adjusted according to age and developmental needs. Inappropriate child feeding practices, including inadequate feeding frequency and portion sizes relative to recommendations, may increase the risk of stunting (Wangiyana *et al.*, 2021). The provision of complementary foods as an important source of nutrients for young children is also influenced by various factors, including household food availability.

Low household food security may compromise children's nutritional status by limiting access to sufficient and nutritious foods. Limited access to food that adequately meets children's nutritional requirements is one of the factors that may contribute to stunting. In addition, inappropriate child feeding practices may further increase the risk of stunting. Various programs have been implemented to prevent further increases in stunting prevalence. Policies aimed at accelerating stunting reduction have been implemented across districts and municipalities through the establishment of stunting reduction acceleration teams. However, policy approaches addressing household food security and child feeding practices remain an area that requires further exploration.

In addition to household food security and child feeding practices, chrononutrition has recently gained attention as a potential factor influencing nutritional status. Chrononutrition refers to the relationship between the timing, frequency, and regularity of food intake and the body's biological rhythms (Khomsan *et al.*, 2024). This concept emphasizes not only *what* and *how much* to eat but also *when to eat*, which may influence energy metabolism, nutrient utilization, and hormonal regulation.

Recent studies have suggested that irregular meal timing, frequent meal skipping, and late-night eating may disrupt circadian rhythms and contribute to metabolic disturbances in children and adolescents (UNICEF, 2020). Although most chrononutrition research has been conducted in adult populations, emerging evidence suggests that regularity in meal timing from early childhood may play an important role in supporting optimal growth and development.

Furthermore, inappropriate timing of complementary feeding, whether introduced too early or too late, has been associated with an increased risk of stunting among children aged 6–23 months (Hall *et al.,* 2018). Another study reported that aligning children's feeding patterns with biological rhythms through a chronobiological feeding model may promote weight gain and linear growth, particularly among infants at risk of (McGovern *et al.*, 2017).

Research on chrononutrition among children aged 6–23 months remains limited, particularly regarding its relationship with stunting. This represents an important research gap, given that studies of chrononutrition in adolescent and adult populations have demonstrated associations between meal timing and nutritional or metabolic outcomes. Although the relationship between household food security, child feeding practices, and stunting has been extensively investigated, the findings have not been entirely consistent. Moreover, relatively few studies have simultaneously examined these factors using a comparative cross-sectional design, and evidence regarding the potential interaction between household food security, child feeding practices, and chrononutrition in relation to stunting remains scarce.

Therefore, investigating household food security, child feeding practices, and chrononutrition among children with and without stunting is important to provide a more comprehensive understanding of the multifactorial determinants of stunting during early childhood. Such evidence may contribute to the development of more integrated strategies for stunting prevention that address not only food availability and feeding practices but also the timing and regularity of food intake.

OBJECTIVES

a. General objective

The general objective of this study was to analyze differences in household food security, child feeding practices, and chrononutrition between stunted and non-stunted children aged 6–23 months.

b. Specific objectives

1. Identify and analyze differences in the characteristics of stunted and non-stunted children aged 6–23 months, including age, sex, mode of delivery, prematurity status, and birth order, as well as family characteristics, including parental age, parental education, parental occupation, household per capita income, food and non-food expenditures, and parental assets.
2. Identify and analyze differences in household food security between stunted and non-stunted children aged 6–23 months.
3. Identify and analyze differences in child feeding practices between stunted and non-stunted children aged 6–23 months.
4. Identify and analyze differences in the adequacy of energy and macronutrient intake (protein, fat, and carbohydrates) and micronutrient intake (iron, calcium, zinc, and vitamin C) between stunted and non-stunted children aged 6–23 months.
5. Identify and analyze chrononutrition patterns among stunted and non-stunted children aged 6–23 months.
6. Identify and analyze differences in the morbidity of infectious diseases between stunted and non-stunted children aged 6–23 months.
7. Identify the nutritional status of children aged 6–23 months based on weight-for-age (WAZ) and weight-for-height (WHZ) among stunted and non-stunted children.
8. Analyze the simultaneous associations of household food security, child feeding practices, energy intake, macronutrient intake, micronutrient intake, chrononutrition, and infectious disease morbidity with stunting among children aged 6–23 months.

METHODS

a. Design, location, and time

This study is a quantitative exploratory study of nutritional problems using a comparative cross-sectional design to analyze several variables among stunted and non-stunted children. The study was conducted in a stunting locus in Sayang Village, Cianjur Subdistrict, Cianjur Regency, West Java, Indonesia.

b. Sampling

The study population consisted of mothers and children aged 6–23 months (young children) with stunting and non-stunting nutritional status. Several inclusion and exclusion criteria were applied for subject recruitment. The inclusion criteria were: the mother and child lived in the same household, the mother was aged 18–45 years, and the mother was willing to sign the informed consent form. The exclusion criteria were: the mother or child had a serious illness, the child had a congenital disability, the child was a twin, or the mother was illiterate. Eligible mother-child pairs were recruited purposively. The final analytic sample comprised 160 children: 48 classified as stunted and 112 classified as non-stunted.

c. Data collection

The data collected in this study consisted of primary and secondary data. Secondary data included data on stunted and non-stunted children in the stunting locus of Cianjur Regency, which was used as a reference for sample selection.

Primary data included information on subject characteristics, family characteristics, household food security, child feeding practices, chrononutrition, adequacy of energy and nutrient intake, morbidity due to infectious diseases, and the nutritional status of children aged 6–23 months. Household food security data was obtained through interviews using the Food Insecurity Experience Scale (FIES) questionnaire (Cafiero *et al.* 2018). Data on child feeding practices was collected through interviews using a questionnaire adapted from the instrument used in the 2023 Indonesian Health Survey (Kemenkes RI 2023; WHO and UNICEF 2021). Data on energy and nutrient adequacy was obtained through two non-consecutive 24-hour dietary recalls (Shim *et al.* 2014). Nutritional status was assessed based on measurements of body weight, recumbent length, head circumference, and mid-upper arm circumference (MUAC). The variables collected are detailed as follows:

Table 1. Data collection

No.	Variable Group	Data Collected	Data Collection Method
1	Individual characteristics of the child	Age; sex; mode of delivery; gestational age or preterm birth status; birth order within the family	Structured maternal interview and child health records
2	Parental and family characteristics	Maternal and paternal age; maternal and paternal education; maternal and paternal occupation; household income per capita; birth interval; number of live births; food expenditure; and non-food expenditure	Structured questionnaire
3	Household food security	Household food-insecurity experience and household food-security category	Eight-item Food Insecurity Experience Scale or FIES (Cafiero *et al.* 2018)
4	Child-feeding practices	Early initiation of breastfeeding; colostrum feeding; prelacteal feeding; exclusive breastfeeding; breastfeeding up to 23 months; bottle-feeding; minimum dietary diversity; minimum meal frequency; minimum acceptable diet; egg and/or flesh-food consumption; sweet-beverage consumption; unhealthy-food consumption; zero vegetable or fruit consumption; continued breastfeeding; and introduction of solid, semi-solid, or soft foods at age 6–8 months	Questionnaire adapted from the 2023 Indonesian Health Survey and WHO/UNICEF infant and young child feeding indicators (Kemenkes RI 2023; WHO and UNICEF 2021)
5	Energy and nutrient intake	Energy; protein; fat; carbohydrate; iron; calcium; zinc; and vitamin C intake and adequacy	Two non-consecutive 24-hour dietary recalls; nutrient adequacy was determined using the Indonesian recommended dietary allowances (Gibson 2005; Kemenkes RI 2019; Shim *et al.* 2014)
6	Chrononutrition	Timing of main meals and snacks; intervals between main meals and snacks; meal composition and portion size at each eating occasion; duration of main meals and snacks; sleep patterns in relation to meal timing; breastfeeding frequency per 24 hours; peak breastfeeding time; and interval between breastfeeding and main meals	Structured chrononutrition questionnaire covering meal timing, frequency, and regularity (Almoosawi *et al.* 2019)
7	Morbidity due to infectious diseases	Type of infectious disease; number of illness episodes; and duration of illness during the preceding month	Maternal interview using a structured morbidity questionnaire

Table 2. Data collection (continued)

No.	Variable Group	Data Collected	Data Collection Method
8	Anthropometry and nutritional status	Length-for-age Z-score or LAZ; weight-for-age Z-score or WAZ; weight-for-length Z-score or WLZ; head circumference; and mid-upper arm circumference or MUAC	Standardized anthropometric measurements; nutritional status was determined using the WHO Child Growth Standards and Indonesian child anthropometry standards (WHO Multicentre Growth Reference Study Group 2006; Kemenkes RI 2020)

Data collection was conducted by trained enumerators with an educational background in nutrition. Prior to data collection, the enumerators will undergo training to ensure standardized data collection procedures and measurement techniques.

d. Data analysis

Following data collection, data cleaning was conducted to assess the completeness of questionnaire responses, detect missing values, and identify illogical responses or values outside the expected range. Subsequently, data processing and analysis were performed using Microsoft Excel and *SPSS 27 for Windows*.

Data on the individual and family characteristics of the subjects were presented descriptively. Household food insecurity data were obtained using the Food Insecurity Experience Scale (FIES), which consists of eight questions. The instrument assesses self-reported experiences related to access to adequate food through eight questions covering concerns about running out of food, limitations in food variety, and experiences of skipping meals or going without food for an entire day due to limited resources. Each affirmative ("yes") response was assigned a score of 1.

Dietary diversity was assessed to determine the adequacy of dietary variety in foods consumed during the 24 hours preceding data collection. The food groups assessed were:

1. Breast milk
2. Cereals, roots and tubers, and their products
3. Legumes, nuts, and their products
4. Dairy products
5. Meat, poultry, fish, seafood, and their products
6. Eggs and their products
7. Vitamin A-rich vegetables and fruits, including sweet potatoes, and their products
8. Other vegetables and fruits and their products

Chrononutrition data among the children aged 6–23 months were processed to characterize the temporal patterns of food consumption and breastfeeding that may be associated with nutritional status and stunting. Data processing was based on recorded meal times, types and amounts of foods consumed, as well as sleep and breastfeeding patterns over a 24-hour period.

1. Main meal and snack timing: The timing of main meals (morning, afternoon, and evening) and snacks was recorded in hours and minutes using a 24-hour format. Meal times were categorized according to chronobiological periods: morning (05:00–10:59), afternoon (11:00–16:59), and evening (17:00–22:59). The distribution of meal times was analyzed to assess the regularity and consistency of eating schedules.
2. Intervals between main meals and snacks: The interval between eating occasions was calculated as the difference in time between consecutive eating events, expressed in hours and minutes. The mean and variability of inter-meal intervals were calculated to assess the rhythm of energy intake throughout the day.
3. Meal composition and portion size: Each eating occasion was analyzed based on energy and macronutrient content (carbohydrates, protein, and fat) using a food composition database. Food portions were initially converted into household measures and subsequently into grams. The resulting data were used to assess the distribution of energy intake across eating occasions and the proportion of daily energy requirements provided by each eating occasion.
4. Duration of main meals and snacks: Eating duration was calculated as the difference between the start and end times of each eating occasion. This analysis was conducted to assess the duration of eating and its potential association with total dietary intake and children's eating behavior.
5. Sleep patterns and their relationship with meal timing: Children's sleep and wake times were processed to determine total sleep duration and nighttime sleep timing. Subsequently, sleep and meal times were mapped to identify potential associations between sleep rhythms and eating patterns, including the possibility of eating shortly before bedtime or immediately after waking.
6. Breastfeeding frequency per 24 hours: The number of breastfeeding episodes was recorded over a 24-hour period and used to calculate breastfeeding frequency. This analysis aimed to characterize the intensity of breastfeeding or formula feeding within a daily cycle.
7. Peak breastfeeding time: The time with the highest breastfeeding frequency or duration over the 24-hour period was identified as the peak breastfeeding time. These data were used to characterize the synchronization of breastfeeding rhythms with the child's main meal patterns.
8. Interval between breastfeeding and main meal times: The interval between the last breastfeeding episode and the subsequent main meal was calculated to assess the potential overlap or separation between breastfeeding and complementary feeding activities.

The categorization of the data is presented in Table 2. Nutritional status was assessed using the height-for-age (HAZ), weight-for-age (WAZ), and weight-for-height (WHZ) indicators, which were calculated from the measured body weight and body length of the children aged 6–23 months using the following formulas:

$$Z - score = \frac{Individual\ measurement - Reference\ median}{Reference\ standar\ deviation}$$

Table 3. Operational Definitions, Data Types, and Variable Categorization

Variable	Operational definition	Data type	Categorization
Individual characteristics			
• Child age	The interval between the child's date of birth and the date of anthropometric assessment, expressed in completed months.	Ratio	Continuous value in months; may be grouped into 6–11, 12–18, and 19–23 months for descriptive analysis
• Sex	The biological sex of the child, as reported by the parent or recorded in the child health book.	Ordinal	1. Girl 2. Boy
• Mode of delivery	The method of childbirth, based on parental reports or information recorded in the Maternal and Child Health Book.	Ordinal	1. **Cesarean section:** delivery of the baby through a surgical incision in the abdominal wall and uterus. 2. **Vaginal delivery:** delivery of the baby through the birth canal without a surgical procedure.
• Preterm birth status	Birth occurring before 37 completed weeks of gestation, based on parental reports or information recorded in the Maternal and Child Health Book.	Ordinal	1. Preterm: gestational age <37 weeks 2. Term: gestational age ≥ 37 weeks.
• Birth order	The child's position in the birth order within the nuclear family.	Ratio	Recorded as the first, second, third, or subsequent child.
Parental and household characteristics:			
• Parental age	The interval between the mother's or father's date of birth and the date of data collection, expressed in completed years.	Ratio	Continuous value in years.
• Parental education	The highest level of formal education completed by the child's mother and father.	Ordinal	1. Did not complete primary school; 2. Primary School; 3. Junior Secondary School; 4. Senior High School; 5. Diploma 6. Bachelor's degree 7. Postgraduate degree
• Parental occupation	The main occupation of the child's mother and father at the time of data collection.	Nominal	Categorized according to the occupation groups used in the questionnaire.

Table 4. Operational Definitions, Data Types, and Variable Categorization (continued)

Variable	Operational definition	Data type	Categorization
• Per capita household income	Total monthly household income divided by the number of household members whose living expenses were covered by the household	Ordinal	1. Poor: per capita income ≤ the applicable Cianjur poverty line 2. Non-poor: per capita income > the applicable Cianjur poverty line
• Household assets	Household ownership of a house, land, vehicles, and livestock.	Ordinal	1. Not owned for each asset category 2. Owned
Household food insecurity	The household's experience of constrained access to sufficient, safe, and nutritious food because of limited resources, assessed using the eight-item Food Insecurity Experience Scale.	Ordinal	1. Severely food insecure (score >6) 2. Moderately food insecure (score 4-6) 3. Food secure (score <4)
Child-feeding practices			
• Early initiation of breastfeeding	Initiation of breastfeeding within the first hour after birth, accompanied by early skin-to-skin contact between the mother and child.	Ordinal	1. No 2. Yes
• Colostrum feeding	Provision of the first breast milk produced during the early postpartum period, which is generally yellowish and thicker than mature breast milk.	Ordinal	1. Colostrum not provided 2. Colostrum partially provided 3. Colostrum fully provided
• Prelacteal feeding	Provision of food or liquids other than breast milk to a newborn before breastfeeding was established, generally during the first three days after birth.	Ordinal	1. Received prelacteal feeding 2. DId not receive prelacteal feeding
• Exclusive breastfeeding	Feeding the child only breast milk during the first six months of life, with no other foods or liquids, except oral rehydration solution, vitamins, minerals, or medicines.	Ordinal	1. Nt exclusively breastfed 2. Exclusively breastfed
• Minimum dietary diversity	Dietary-diversity indicator based on the number of defined food groups consumed by children aged 6–23 months during the preceding day.	Ordinal	1. Not met: < 5 food groups 2. Met: ≥5 food groups (WHO, 2023)

Table 5. Operational Definitions, Data Types, and Variable Categorization (continued)

Variable	Operational definition	Data type	Categorization
• *Minimum meal frequency (MMF)*	The number of times the child consumed solid, semi-solid, or soft foods during the preceding day, including milk feeds for non-breastfed children.	Ordinal	1. Not met: children aged 6–8 months were fed fewer than 2 times per day, and children aged 9–23 months were fed fewer than 3 times per day. 2. Met: children aged 6–8 months were fed at least 2 times per day, and children aged 9–23 months were fed at least 3 times per day. (WHO, 2023)
• *Minimum acceptable diet*	A composite indicator combining minimum dietary diversity and minimum meal frequency; non-breastfed children must also meet the minimum milk-feeding frequency criterion.	Ordinal	1. Not met: the child did not meet the Minimum Dietary Diversity (MDD) and/or Minimum Meal Frequency (MMF) criteria. 2. Met: the child met both the Minimum Dietary Diversity (MDD) and Minimum Meal Frequency (MMF) criteria.
Chrononutrition	Eating behavior characterized by the timing, frequency, and regularity of food intake in relation to the body's circadian rhythm.	Rasio	Meal and snack timing; eating frequency; eating-window duration; inter-meal intervals; eating duration; meal composition; sleep–meal intervals; breastfeeding frequency; and peak breastfeeding time **(Almoosawi *et al.* 2019)**
Adequacy of energy and nutrient intake			
Energy adequacy	The percentage ratio of the child's mean daily energy intake to the age-specific recommended energy allowance	Ordinal	1. Severe deficit: <70% RDA 2. Mild deficit: 70-89% RDA 3. Adequate: 90-119% RDA 4. Excessive ≥120% RDA (Kemenkes, 2019)
Macronutrient adequacy	The percentage ratio of the child's mean daily protein, fat, or carbohydrate intake to the corresponding age-specific recommended intake.	Ordinal	1. Severe deficit: <70% RDA 2. Mild deficit: 70-89% RDA 3. Adequate:: 90-119% RDA 4. Excessive ≥120% RDA (Kemenkes, 2019)
Micronutrient adequacy	The percentage ratio of the child's mean daily iron, calcium, zinc, or vitamin C intake to the corresponding age-specific recommended intake.	Ordinal	1. Inadequate: <77% RDA 2. Adequate ≥77% RDA (Gibson, 2005)

Table 6. Operational Definitions, Data Types, and Variable Categorization (continued)

Variable	Operational definition	Data type	Categorization
Infectious-disease morbidity	The occurrence of infectious diseases experienced by the child during the preceding month, measured according to the number of illness episodes and the duration of each episode.	Ratio	Frequency expressed as episodes per month and duration expressed as days per month for each disease.
Anthropometry and nutritional status			
• Length-for-age Z-score or Height-for-age	An anthropometric index describing the child's linear growth relative to age and sex, based on the WHO Child Growth Standards.	Ordinal	1. Severely stunted: Z-score <-3 SD 2. Stunted: -3 SD to <-2 SD 3. Normal: Z-score -2 SD to +3 SD 4. Tall: Z-score >+3 SD
• Weight-for-age Z-score	An anthropometric index comparing the child's body weight with the WHO standard for children of the same age and sex.	Ordinal	1. Severely underweight): Z-score <-3 SD 2. Underweight: Z-score -3 SD to <-2 SD 3. Normal: Z-score -2 SD to +1 SD 4. Risk of overweight: Z-score >+1 SD
• Weight-for-length Z-score	An anthropometric index describing body-weight proportionality relative to recumbent length and indicating acute nutritional status.	Ordinal	1. *Severely wasted:* Z-score <-3 SD 2. U*nderweight*: Z-score -3 SD to <-2 SD 3. Normal: Z-score -2 SD to +1 SD 4. Risk of overweight: Z-score: +1 SD to +2 SD 5. Overweight: Z-score: >+2 SD to +3 SD 6. Obesity: Z-score >+3 SD

Differences in continuous variables between stunted and non-stunted children were analyzed using independent-samples t-tests. Associations between categorical variables and stunting status were examined using chi-square tests. Pearson correlation tests were performed to assess linear relationships between continuous variables and the height-for-age Z-score (HAZ).

e. Ethical approval

This study received ethical approval from the Research Ethics Committee of Universitas Muhammadiyah Semarang under reference number 001/KE/04/2026. Ethical approval was obtained before participant recruitment and data collection commenced. All participating mothers

provided written informed consent after receiving information about the study's objectives, procedures, potential risks, and confidentiality provisions.

RESULTS AND DISCUSSION

a. Individual and Family Characteristics of the Subjects

Individual characteristics of subjects are presented in Table 3. Among the 160 subjects, 30% were classified as stunted and 70% as non-stunted. Several individual characteristics differed significantly between the stunted and non-stunted groups, including sex, age, birth length, and age gap with the nearest older sibling.

Table 7. Individual characteristics of subjects

Variable	Total		Stunting		Non-stunting		p	HAZ
	n	%	n	%	n	%		
Sex							0.019[a]*	
Boy	75	46.9	29	60.4	46	41.1		-1.38 ± 1.5
Girl	85	53.1	19	39.6	66	58.9		-1.1 ± 1.3
Age							0.007[a]*	
≤ 12 months old	76	47.5	16	33.3	60	53.6		-1.0 ± 1.6
13-18 months old	43	26.9	12	25.0	31	27.7		-1.1 ± 1.2
19-23 months old	41	25.6	20	41.7	21	18.8		-1.8 ± 1.1
Mean ± SD	13.7 ± 5.3		15.6 ± 5.4		12.9 ± 5.0		0.003[b]*	
Birth order							0.414[a]	
1st born	44	27.5	10	20.8	34	30.4		-1.3 ± 1.4
2nd born	37	23.1	11	22.9	26	23.2		-1.2 ± 1.4
3rd born or later	79	49.4	27	56.2	52	46.4		-1.2 ± 1.4
Mean ± SD	2.5 ± 1.3		2.6 ± 1.2		2.5 ± 1.4		0.443[b]	
Mode of delivery							0.193[a]	
Vaginal delivery	128	80.0	36	75.0	92	82.1		-1.2 ± 1.5
Assisted/induced vaginal delivery	3	1.9	0	.0	3	2.7		-0.3 ± 0.4
Cesarean section (C-section)	29	18.1	12	25.0	17	15.2		-1.5 ± 1.2
Gestational age at birth							0.450[a]	
Preterm (<37 weeks)	15	9.4	6	12.5	9	8.0		-1.4 ± 0.9
Term (37-41 weeks)	143	89.4	42	87.5	101	90.2		-1.1 ± 0.2
Post-term (≥42 weeks)	2	1.2	0	0	2	1.8		-1.2 ± 1.5
Birth weight							0.071[a]	
Low (<2.5 kg)	19	11.9	9	18.8	10	8.9		-1.7 ± 0.9
Normal (≥2.5 kg)	141	88.1	39	81.2	102	91.1		-1.2 ± 1.5
Mean ± SD	3.1 ± 0.5		3.0 ± 0.5		3.1 ± 0.5		0.07	
Birth length (mean ± SD)	49.1 ± 3.5		48.4 ± 3.5		49.3 ± 3.5		0.01*	
Age gaps with the nearest older sibling							0.039[a]	
0-5 years	31	21.5	31	70.5	54	57		-1.4 ± 1.3
>5 years	56	38.9	13	29.5	43	43.0		-0.9 ± 1.4
Mean ± SD	55.55 ± 46.13		49.87 ± 37.98		58.06 ± 49.26		0.326[b]	

[a] p-value based on the Chi-Square Test
[b] p-value based on the Independent t-Test
*p-value is significant (p<0.05)

The individual characteristics of the children showed several significant differences between the stunted and non-stunted groups, particularly in sex and age distribution. The significantly higher proportion of boys among stunted children (60,4%) compared with non-stunted children (41,1%) is consistent with growing evidence that boys may be more vulnerable to linear growth faltering and undernutrition than girls. A systematic review and meta-analysis found that boys had higher odds of stunting than girls (pooled OR = 1,29; 95% CI: 1,22–1,37), although the magnitude of the difference varied across settings (Thurstans *et al.*, 2020).

Several mechanisms may contribute to this greater vulnerability among boys. Biological differences in growth trajectories and immune function may make male infants more susceptible to infection and nutritional stress, particularly during periods of rapid growth. However, sex differences in stunting are not considered purely biological; caregiving, feeding practices, healthcare utilization, and broader social and environmental conditions may also modify the relationship between sex and linear growth. Therefore, the higher proportion of boys in the stunted group in the present study may reflect a combination of biological susceptibility and context-specific factors rather than an inherent deterministic effect of sex (Thompson, 2021).

Age also differed significantly between the two groups, with stunted children being older on average than non-stunted children (15,6 ± 5,4 vs. 12,9 ± 5,0 months). The greater proportion of children aged 19–23 months in the stunted group and the greater proportion aged ≤12 months in the non-stunted group suggest that growth faltering may become increasingly apparent during the transition through infancy and the complementary feeding period. The age of 6–23 months represents a particularly critical developmental period because breast milk alone is no longer sufficient to meet all nutritional requirements, while the child is simultaneously experiencing rapid physical and neurodevelopmental growth. WHO identifies this period as a peak period for growth faltering and nutrient deficiencies (WHO, 2023).

The higher age of the stunted children should, however, be interpreted cautiously. In a cross-sectional comparison, the finding does not necessarily indicate that increasing age directly causes stunting. Rather, older children have had a longer cumulative exposure to inadequate dietary intake, recurrent infection, suboptimal feeding practices, and other environmental constraints that can influence linear growth. In addition, age differences between groups are particularly relevant to interpreting IYCF and chrononutrition variables because feeding frequency, dietary diversity, meal timing, breastfeeding patterns, and sleep characteristics naturally vary with age. Therefore, age should be considered an important potential confounder when examining associations between IYCF and chrononutrition characteristics and stunting in this study. Evidence from Indonesia also indicates that complementary feeding practices differ according to child age, with younger children being less likely to meet several recommended feeding indicators.

With regard to birth characteristics, no statistically significant differences were observed between the two groups in birth order, mode of delivery, gestational age, or birth weight. Although the proportion of low-birth-weight children was descriptively higher among the stunted group (18,8% vs. 8,9%), the difference in birth weight did not reach statistical significance (p=0,071).

This finding suggests that, within the present sample, these birth characteristics did not clearly distinguish children who were stunted from those who were not. Nevertheless, the relatively higher proportion of low birth weight among stunted children may indicate the importance of prenatal and early-life conditions and warrants consideration, particularly because impaired growth can originate before birth and continue during infancy. Previous evidence has demonstrated that growth faltering may already be present at birth, emphasizing the contribution of prenatal growth and early-life conditions to subsequent linear growth (Victora *et al.,* 2021).

In contrast, birth length was significantly shorter among stunted children than among non-stunted children (48,4 ± 3,5 vs. 49,3 ± 3,5 cm; p=0,010). This finding is biologically plausible because birth length reflects fetal linear growth and may provide an early indication of intrauterine growth conditions. Children who enter postnatal life with shorter body length may have a greater likelihood of remaining short relative to age if subsequent growth is insufficient to compensate for the early deficit. Importantly, this observation also supports the concept that stunting should not be viewed solely as a consequence of postnatal feeding practices. Maternal nutrition, fetal growth, placental function, intrauterine conditions, and subsequent environmental exposures can jointly contribute to the trajectory of linear growth. The Lancet series on maternal and child undernutrition similarly emphasized that stunting may already be evident at birth and that linear growth faltering is a process beginning early in life rather than an isolated outcome of later childhood nutrition (Victora *et al.*, 2021).

The significant difference in the age gap with the nearest older sibling should also be interpreted carefully. The categorical analysis showed a different distribution between groups, particularly a higher proportion of children with an age gap of 0–5 years among the stunted group. However, the continuous measure of sibling age gap did not differ significantly between groups. This discrepancy indicates that the significant categorical result may reflect differences in the distribution across specific age-gap categories rather than a consistent overall difference in birth spacing. Therefore, it would be inappropriate to conclude that a longer or shorter sibling interval was associated with stunting based solely on the categorical analysis.

The absence of a significant difference in the continuous sibling age gap is also broadly compatible with evidence showing that the relationship between birth spacing and child nutritional status is complex. A recent systematic review and meta-analysis of 56 studies from the Asia-Pacific region found only a slightly higher likelihood of stunting among children born after short birth intervals, and the pooled association was not statistically significant (OR = 1.13, 95% CI: 0.97–1.32), although significant effects were observed in some subgroups. Thus, sibling spacing may influence child nutrition through pathways such as maternal nutritional depletion, breastfeeding duration, caregiving resources, and household competition for food and attention, but these pathways are strongly influenced by socioeconomic and family contexts.

Table 4 illustrates the correlation between individual characteristics and height-for-age Z-score (HAZ). Based on the Pearson Correlation Test, age was significantly and negatively correlated with HAZ ($r = -0.268$, $p = 0.001$), indicating that older children tended to have lower HAZ values in the present sample. The magnitude of the correlation was weak to moderate,

suggesting that age accounted for only part of the variation in HAZ. HAZ is an age-standardized indicator, and a lower HAZ among older children may instead reflect the accumulation of growth deficits over time. Children exposed to inadequate dietary intake, recurrent infections, or unfavorable environmental conditions may progressively deviate from the expected growth trajectory, resulting in increasingly negative HAZ values during the first two years of life (Victora *et al.*, 2021).

Table 8. Correlation between individual characteristics and HAZ

Variable	HAZ	
	p	r
Age	0.001*	-0.268
Birth weight	0.049*	0.156
Birth length	0.019*	0.186
Birth order	0.737	0.027
Age gaps with the nearest older sibling	0.012*	0.208

*significant based on Pearson Correlation Test

The inverse association between age and HAZ is particularly relevant to the present study because all participants were children aged 6–23 months, a period marked by rapid growth and major feeding transitions. The first two years of life represent a critical window during which growth faltering can become increasingly evident. During this period, children transition from predominantly milk-based feeding toward increasingly diverse complementary foods, while their nutritional requirements increase substantially (WHO, 2023). Consequently, inadequate dietary intake or persistent exposure to infection during this period may lead to accumulation and be reflected in a lower HAZ. The present finding is therefore consistent with the concept that stunting represents a cumulative process rather than an acute nutritional condition.

This finding is also consistent with the significant age difference observed between the stunted and non-stunted groups, with stunted children being older than non-stunted children. Taken together, the two findings suggest that the lower HAZ among older children may partly reflect the persistence or accumulation of linear growth deficits across the first two years of life. However, because the present study is cross-sectional, the temporal sequence between age-related exposure and growth faltering cannot be established. The observed association should therefore be interpreted as a pattern within the study population rather than evidence that age independently causes stunting.

In contrast to age, birth weight showed a significant positive correlation with HAZ (r = 0,156; p = 0,049), indicating that children with greater birth weight tended to have higher HAZ values. Although the correlation was weak, the direction of the association is biologically plausible because birth weight reflects fetal growth and the intrauterine environment. Children born with lower birth weight may have experienced restricted fetal growth and consequently enter postnatal life with a lower growth reserve. Evidence from Indonesia supports the importance of birth characteristics in subsequent child growth. A systematic review and meta-analysis of Indonesian studies found that low birth weight was associated with substantially greater odds of childhood

stunting (pooled OR approximately 2,39), although the strength of this association varies across populations and study designs (Gusnedi *et al.*, 2023).

The positive correlation between birth weight and HAZ should nevertheless be interpreted together with the finding for birth length. Birth length demonstrated a slightly stronger positive correlation with HAZ ($r = 0.186$, $p = 0.019$). This pattern is particularly meaningful because birth length is a more direct indicator of fetal linear growth than birth weight. A study demonstrated that birth length and birth length-for-age were among the strongest predictors of subsequent linear growth and stunting at 24 months. Children who had lower length-for-age at birth were more likely to remain on a lower linear growth trajectory and develop stunting during the first two years of life (Krebs *et al.*, 2022).

The positive association between birth length and HAZ in the present study therefore suggests that differences in linear growth may already be established during the prenatal period. This interpretation is consistent with a developmental perspective in which postnatal growth is partly constrained by the child's initial size and fetal growth trajectory. However, the relatively small correlation coefficient indicates that birth length alone does not determine subsequent HAZ. Rather, prenatal growth provides an initial condition that interacts with postnatal nutrition, infection, caregiving, environmental conditions, and other factors influencing growth during infancy and early childhood. The relatively weak correlation observed here is therefore expected given the multifactorial nature of stunting.

The finding that birth length was correlated with HAZ also complements the group comparison presented previously. In the comparison between stunted and non-stunted children, stunted children had significantly shorter birth length than non-stunted children. The correlation analysis extends this observation by demonstrating that birth length was positively associated with the continuous HAZ measure across the study population. Taken together, these findings strengthen the interpretation that early linear growth status is related to subsequent linear growth during the first two years of life.

A significant positive correlation was also observed between the age gap with the nearest older sibling and HAZ ($r = 0{,}208$, $p = 0{,}012$). Thus, children with a wider age gap from their nearest older sibling tended to have higher HAZ. One possible explanation is that a larger interval between siblings may provide mothers with greater opportunity for nutritional recovery and may allow more time and resources to be allocated to breastfeeding, complementary feeding, caregiving, and healthcare for the younger child. Longer birth intervals may also reduce competition between young siblings for maternal care and household resources. However, this interpretation should remain cautious because sibling age gap is an indirect marker of several interrelated maternal and household characteristics rather than a direct biological determinant of HAZ.

Evidence concerning birth spacing and childhood stunting remains mixed. A recent systematic review and meta-analysis in the Asia-Pacific region found a slightly higher likelihood of stunting among children born after short birth intervals, although the pooled association was not statistically significant (OR = 1,13; 95% CI: 0,97–1,32) (Khan *et al.*, 2024). This suggests that the

potential relationship between sibling spacing and child growth may depend on contextual factors such as maternal nutritional status, breastfeeding practices, household resources, and socioeconomic conditions. In the present study, the positive correlation between sibling age gap and HAZ may therefore reflect the combined influence of these underlying pathways rather than a direct effect of sibling spacing itself.

The fact that birth order was not significantly correlated with HAZ (r = 0,027; p = 0,737) provides an additional indication that the number of the child within the family does not necessarily determine linear growth status. Birth order may influence child nutrition through differences in maternal experience, resource allocation, and sibling competition, but these effects are likely to be highly context-specific. The absence of an association in the present study suggests that sibling age spacing may be more informative than birth order alone, although this possibility would require further analysis using multivariable models.

b. Socioeconomic Characteristics of the Households

Table 5 presents the distribution of family and socioeconomic characteristics of the children according to stunting status. Overall, no statistically significant differences were observed between stunted and non-stunted children for any of the family characteristics examined, including parental education, parental occupation, parental age, number of children under five years of age, family size, per capita household income, and household economic status (all $p > 0.05$). These findings suggest that, within the population studied, the measured household socioeconomic characteristics were not sufficiently differentiated between stunted and non-stunted children to demonstrate statistically significant associations with stunting. Nevertheless, several descriptive differences were apparent and may still be relevant from a public health perspective.

Table 9. Distribution of the socioeconomic status of households

Variable	Total		Stunting		Non-stunting		p
	n	%	n	%	n	%	
Father's education							0.156[a]
Less than primary school	8	5.1	4	8.3	4	3.7	
Primary school	21	13.5	8	16.7	13	12.0	
Junior secondary school	35	22.4	14	29.2	21	19.4	
Senior secondary	81	51.9	21	43.8	60	55.6	
Higher education	11	7.1	1	2.1	10	9.3	
Mother's education							0.199[a]
Less than primary school	6	3.8	1	2.1	5	4.5	
Primary school	24	15.0	5	10.4	19	17.0	
Junior secondary school	38	23.8	17	35.4	21	18.8	
Senior secondary	78	48.8	22	45.8	56	50.0	
Higher education	14	8.8	3	6.2	11	9.8	
Father's occupation							0.524[a]
Farmer	1	.6	0	.0	1	.9	
Trader/Entrepreneur	39	25.0	9	18.8	30	27.8	

Table 10. Distribution of the socioeconomic status of households (continued)

Variable	Total		Stunting		Non-stunting		p
	n	%	n	%	n	%	
Laborer	64	41.0	25	52.1	39	36.1	
Village government official	1	.6	0	.0	1	.9	
Teacher	2	1.3	0	.0	2	1.9	
Civil servant/military/police officer	2	1.3	0	.0	2	1.9	
Private sector employee	38	24.4	12	25.0	26	24.1	
Unemployed	0	.0	0	.0	0	.0	
Other	9	5.8	2	4.2	7	6.5	
Mother's occupation							0.495[a]
Farmer	0	.0	0	.0	0	.0	
Trader/Entrepreneur	11	6.9	2	4.2	9	8.1	
Laborer	5	3.1	1	2.1	4	3.6	
Village government official	0	.0	0	.0	0	.0	
Teacher	5	3.1	3	6.2	2	1.8	
Civil servant/military/police officer	2	1.3	0	.0	2	1.8	
Private sector employee	9	5.7	2	4.2	7	6.3	
Unemployed	120	75.5	39	81.2	81	73.0	
Other	7	4.4	1	2.1	6	5.4	
Father's age (Mean ± SD)	35.0 ± 7.1		34.7 ± 6.3		35.1 ± 7.6		0.750[b]
Mother's age (Mean ± SD)	31.3 ± 6.0		30.6 ± 4.6		31.6 ± 6.6		0.396[b]
Number of children under five (Mean ± SD)	1.3 ± 0.5		1.3 ± 0.5		1.3 ± 0.5		0.691[b]
Family size (Mean ± SD)	5.0 ± 1.4		5.1 ± 1.3		4.9 ± 1.5		0.530[b]
Per capita household income (Mean ± SD)	899,550.0 ± 148,9440.0		687,742.1 ± 440,350.2		990,331.8 ± 1,751,552.4		0.240[b]
Economic status							0.508[a]
Poor	52	32.9	14	29.2	38	34.5	
Non-poor	106	67.1	34	70.8	72	65.5	

[a] p-value based on the Chi-Square Test
[b] p-value based on Independent T-Test

Regarding parental education, senior secondary education was the most common educational attainment for both fathers and mothers. Among fathers, 51,9% had completed senior secondary school, compared with 43,8% in the stunted group and 55,6% in the non-stunted group (p = 0,156). Similarly, 48,8% of mothers had completed senior secondary education, with relatively comparable proportions among stunted and non-stunted children (45,8% and 50,0%, respectively; p = 0,199). Although the differences were not statistically significant, the somewhat higher proportion of fathers and mothers with senior secondary or higher education in the non-stunted group may indicate a potential socioeconomic gradient that was not sufficiently strong to be detected in this sample. Previous research has consistently identified parental, particularly maternal, education as an important determinant of child nutritional status because education may

improve mothers' knowledge, health-seeking behavior, child-care practices, feeding practices, and ability to utilize available household resources. A review of determinants of stunting in Indonesia identified maternal education as one of the important and consistently reported determinants, together with household socioeconomic status, breastfeeding, birth characteristics, and environmental health conditions (Beal *et al.*, 2018). More recent evidence from a systematic review covering studies published between 2015 and 2024 similarly identified maternal education and socioeconomic status among the most consistent predictors of stunting globally (Ayue *et al.*, 2025). Thus, the lack of statistical significance in the present study should be interpreted as a finding specific to this study population rather than evidence that parental education is generally unrelated to stunting.

The distribution of parental occupation showed a similar pattern. Among fathers, laborer was the most common occupation (41,0%), followed by trader/entrepreneur (25,0%) and private-sector employee (24,4%). The proportion of fathers working as laborers was higher among stunted children than among non-stunted children (52,1% versus 36,1%). Conversely, the proportion of fathers working as traders or entrepreneurs was somewhat higher in the non-stunted group (27,8%) than in the stunted group (18,8%). However, the overall distribution of paternal occupation did not differ significantly between the two groups ($p = 0,524$). Among mothers, 75,5% were classified as unemployed, with similarly high proportions in both the stunted and non-stunted groups (81,2% and 73,0%, respectively; $p = 0,495$). These findings indicate that occupational differences alone may not adequately capture the economic resources available to households. Occupation may influence income, but its effect on child nutrition can be mediated by job stability, household assets, social protection, food prices, access to health services, and intra-household allocation of resources. Therefore, a categorical measure of occupation may have limited ability to distinguish economically vulnerable households in a relatively homogeneous study population.

The mean age of fathers was 35,0 ± 7,1 years, whereas the mean age of mothers was 31,3 ± 6,0 years. Neither paternal age nor maternal age differed significantly according to stunting status. Fathers of stunted children had a mean age of 34,7 ± 6,3 years compared with 35,1 ± 7,6 years among fathers of non-stunted children ($p = 0,750$). Similarly, mothers of stunted children were slightly younger than mothers of non-stunted children (30,6 ± 4,6 versus 31,6 ± 6,6 years), but the difference was not statistically significant ($p = 0,396$). These findings suggest that parental age was relatively comparable between the two groups. Although maternal age has been identified as a potential determinant of child nutritional outcomes in some studies, its relationship with stunting may operate through other factors, such as maternal nutritional status, reproductive history, antenatal care, birth outcomes, and caregiving capacity. A recent systematic review of predictors of stunting found maternal age among the factors reported across studies, but also emphasized that stunting is a multifactorial outcome shaped by interacting biological, socioeconomic, environmental, and caregiving factors (Ayue *et al.*, 2025).

The number of children under five years of age was also similar between the groups, with a mean of 1,3 ± 0,5 children in both the stunted and non-stunted groups ($p = 0,691$). Likewise, mean family size was 5,0 ± 1,4 persons overall, with no significant difference between households

of stunted and non-stunted children (5,1 ± 1,3 versus 4,9 ± 1,5 persons; p = 0,530). Household size and the number of young children may affect the allocation of food, caregiving time, and household expenditure. Larger families can potentially face greater competition for household resources, particularly when income is limited. Indeed, systematic reviews have identified family size and the number of young children in the household as potential determinants of childhood undernutrition. However, the absence of significant differences in the present study may indicate that the variation in household size was relatively small or that other factors, such as food allocation, caregiving practices, and household income, played a more important role in determining children's nutritional status.

Per capita household income was lower among families of stunted children than among families of non-stunted children. The mean per capita household income was IDR 899.550,0 ± 1.489.440,0 overall, compared with IDR 687.742,1 ± 440.350,2 among stunted children and IDR 990.331,8 ± 1.751.552,4 among non-stunted children; however, the difference was not statistically significant (p = 0,240). The relatively large standard deviation, particularly in the non-stunted group, indicates considerable heterogeneity in household income. This variation may have reduced the ability to detect a statistically significant difference. Nevertheless, the descriptive pattern is consistent with the established conceptual understanding that household economic resources can influence children's nutritional status through food access, dietary quality, living conditions, sanitation, healthcare utilization, and other pathways. Evidence from Indonesia has shown that socioeconomic status and household wealth are important determinants of childhood stunting and contribute substantially to socioeconomic inequalities in stunting (Beal *et al.*, 2018; Semba *et al.*, 2020).

Similarly, 32,9% of households were classified as poor, while 67,1% were classified as non-poor. The proportion of poor households was 29,2% among stunted children and 34,5% among non-stunted children, and the difference was not statistically significant (p = 0,508). Interestingly, the slightly higher proportion of poor households in the non-stunted group indicates that economic status, when considered as a simple poor/non-poor classification, did not correspond directly to stunting status in this study. This finding may reflect the complex and multidimensional nature of socioeconomic influences on child growth. Household economic status does not necessarily translate directly into adequate child nutrition because income may be spent on non-food necessities, while nutritional outcomes are also affected by dietary practices, disease burden, sanitation, water quality, breastfeeding and complementary feeding practices, and access to health services. A systematic review and meta-analysis of stunting risk factors in Indonesia identified food insecurity, unimproved drinking water, rural residence, and unimproved sanitation as important household and community-level risk factors, illustrating that socioeconomic conditions influence stunting through multiple pathways rather than through income alone (Sari *et al.*, 2023).

Taken together, the findings from Table 4 indicate that parental education, parental occupation, parental age, number of children under five, family size, per capita household income, and categorical economic status were not significantly associated with stunting in this study. Although some descriptive differences were observed—particularly the higher proportion of

fathers working as laborers and the lower mean per capita household income among households with stunted children—these differences did not reach statistical significance. The findings therefore suggest that socioeconomic characteristics alone may not explain the variation in stunting within this study population. This is consistent with the multifactorial nature of stunting, in which household socioeconomic conditions interact with maternal and child characteristics, dietary intake, feeding practices, morbidity, sanitation, water and hygiene, and access to health and nutrition services.

It is also important to distinguish between the absence of a statistically significant association in this study and the absence of an underlying relationship. Previous studies in Indonesia have demonstrated associations between stunting and parental education, socioeconomic status, household wealth, and other environmental and behavioral determinants (Beal *et al.*, 2018; Semba *et al.*, 2020; Sari *et al.*, 2023). Consequently, the non-significant findings observed here may reflect the characteristics and relatively homogeneous socioeconomic profile of the study population, sample size, variability of the socioeconomic indicators, or the influence of other proximal determinants that were not examined in Table 4. Future analyses should therefore consider multivariable models incorporating dietary intake, food security, morbidity, sanitation, child feeding practices, birth characteristics, and maternal nutritional factors to determine whether socioeconomic characteristics retain an independent association with stunting after adjustment for potential confounders.

Table 6 presents the results of the bivariate Pearson correlation analysis examining the relationship between selected family characteristics and height-for-age Z-score (HAZ). HAZ is an important indicator of linear growth and reflects the cumulative effects of nutritional adequacy, recurrent illness, and other environmental and biological influences on child growth over time. According to the WHO Child Growth Standards, height-for-age is used to assess linear growth relative to age and sex, with a HAZ below −2 standard deviations indicating stunting. Thus, examining factors associated with HAZ provides a more continuous assessment of growth than using stunting status as a dichotomous outcome alone (WHO, 2006).

Table 11. Correlation between family characteristics and HAZ

Variable	HAZ	
	p	r
Father's age	0.120	0.140
Mother's age	0.067	0.164
Number of children under five	0.772	-0.023
Family size	0.680	0.033
Per capita household income	0.423	0.064

The findings in Table 6 indicate that none of the family characteristics examined was significantly correlated with HAZ ($p > 0,05$). Father's age showed a weak positive correlation with HAZ ($r = 0,140$; $p = 0,120$), while mother's age also showed a weak positive correlation ($r = 0,164$; $p = 0,067$). Family size demonstrated an almost negligible positive correlation with HAZ ($r = 0,033$; $p = 0,680$), whereas the number of children under five years of age showed a negligible inverse correlation ($r = −0,023$; $p = 0,772$). Per capita household income was positively but weakly

correlated with HAZ (r = 0,064; p = 0,423). Therefore, although most of the correlations were in the theoretically expected direction, their magnitudes were small and none reached statistical significance.

The weak positive association between father's age and HAZ suggests that children from households with older fathers tended to have slightly higher HAZ values, although the association was not statistically significant. Father's age may indirectly reflect household stability, accumulated economic resources, employment experience, and decision-making capacity within the household. However, paternal age is unlikely to have a direct biological effect on the child's attained linear growth during early childhood. Rather, any potential relationship would probably operate through socioeconomic and caregiving pathways. The absence of a significant association in this study is therefore plausible, particularly if differences in household resources and living conditions across paternal age groups were relatively small. More broadly, recent evidence indicates that stunting is influenced by a combination of household, maternal, child, and environmental factors rather than by parental demographic characteristics in isolation (Gusnedi *et al.*, 2023; Ayue *et al.*, 2025).

Mother's age exhibited the strongest positive correlation with HAZ among the family characteristics assessed (r = 0,164), although the association remained statistically non-significant (p = 0,067). The p-value is relatively close to the conventional 0,05 threshold and may indicate a weak tendency toward higher HAZ among children whose mothers were older. Nevertheless, this result should not be interpreted as evidence of a statistically established relationship. Maternal age may influence child growth through several pathways, including maternal experience, reproductive history, health-seeking behavior, antenatal care, childcare practices, and economic circumstances. A recent systematic review identified maternal age as one of the factors that has been associated with stunting in some populations, although the magnitude and direction of the association vary across settings (Ayue *et al.*, 2025). Similarly, a systematic review and meta-analysis of Indonesian studies reported maternal age ≥30 years among maternal characteristics associated with childhood stunting.

The finding in the present study should therefore be interpreted cautiously because maternal age itself may be acting as a proxy for other characteristics. For example, older mothers may have greater parenting experience but may also have a higher number of previous pregnancies or children. These competing pathways could attenuate the simple bivariate relationship between maternal age and HAZ. Furthermore, the present analysis does not adjust for potentially important maternal factors such as maternal height, nutritional status, education, parity, or quality of antenatal and postnatal care. Consequently, the weak positive correlation observed for maternal age cannot be interpreted independently of these potential confounding or mediating variables.

The number of children under five years of age showed an almost negligible negative correlation with HAZ (r = −0,023; p = 0,772). Conceptually, having more young children in the household could place greater demands on household food resources, parental time, and caregiving capacity. Greater competition for household resources could potentially compromise the quantity or quality of food available to individual children. However, the correlation observed in this study

was extremely small and essentially absent. This suggests that variation in the number of young children was unlikely to explain meaningful differences in linear growth in the study population. This result may also reflect the relatively limited variation in this variable, as the mean number of children under five was approximately 1,3 in both the stunted and non-stunted groups in the preceding analysis.

Family size similarly showed virtually no relationship with HAZ ($r = 0,033$; $p = 0,680$). Although larger households are sometimes assumed to be at greater risk of child undernutrition because household resources must be distributed among more members, household size alone may be an inadequate indicator of resource availability. A larger household can also provide additional caregivers, income earners, and social support. Consequently, the effect of family size on child nutrition may depend on the ratio between household members, economically active adults, and dependent children, rather than on the absolute number of household members. The absence of a significant correlation in this study may therefore reflect the fact that family size does not directly determine the nutritional environment experienced by the child.

Per capita household income demonstrated a weak positive correlation with HAZ ($r = 0,064$; $p = 0,423$). The positive direction is consistent with the theoretical expectation that greater household economic resources can facilitate access to sufficient and diverse foods, healthcare, safe water, sanitation, and healthier living environments. However, the very small correlation coefficient indicates that income alone explained little of the variation in HAZ in this sample. This finding is particularly important because income is a distal determinant of child growth rather than a direct biological determinant. According to the WHO conceptual framework, socioeconomic and environmental conditions influence stunting through more proximal pathways, including food access, feeding practices, infections, healthcare, sanitation, and caregiving.

The weak income–HAZ correlation observed here may also be explained by differences in how households convert economic resources into nutritional and health outcomes. Two households with similar incomes may have substantially different food purchasing patterns, dietary diversity, sanitation facilities, healthcare utilization, and childcare practices. Conversely, households with relatively limited income may protect children's nutritional status through effective breastfeeding and complementary feeding practices, food allocation, social support, home food production, or access to social protection programs. Therefore, household income should be viewed as an enabling resource rather than a direct determinant of linear growth.

The present findings can be compared with evidence from Indonesia. Semba *et al.* (2020), using data from the South East Asian Nutrition Surveys, found that stunting and HAZ were associated with parental education and socioeconomic status among Indonesian children. In their analysis, higher parental education and higher socioeconomic status were associated with better HAZ among stunted children. Likewise, Beal *et al.* (2018) concluded from their review of Indonesian evidence that low household socioeconomic status and low maternal education were important determinants of stunting, together with other factors such as inadequate breastfeeding, prematurity, short birth length, low maternal height, and poor water and sanitation conditions.

The apparent difference between these findings and the present results may be attributable to several factors. First, the present analysis is based on bivariate Pearson correlations, whereas socioeconomic determinants of stunting often operate through multiple interconnected pathways. Second, the distribution of socioeconomic characteristics in the present sample may have been relatively homogeneous, limiting the variability required to detect a correlation. Third, HAZ is the cumulative outcome of exposures occurring over a prolonged period; current household characteristics may therefore not accurately represent the socioeconomic and nutritional conditions experienced during earlier critical periods of growth. Finally, factors more proximal to child growth—such as dietary intake, breastfeeding and complementary feeding, morbidity, birth size, maternal height, and sanitation—may have stronger associations with HAZ than the relatively distal family characteristics examined in Table 6

This interpretation is consistent with the broader evidence that stunting is a multifactorial outcome. The WHO conceptual framework emphasizes that impaired linear growth results from the combined influence of inadequate nutrition, recurrent infections, inadequate care, and broader social, economic, environmental, and health-system conditions. A recent systematic review of predictors of stunting among children under five similarly identified maternal education, socioeconomic status, sanitation, maternal age, maternal height, birth weight, breastfeeding, and complementary feeding as important predictors, while emphasizing that their importance varies across contexts (Ayue *et al.*, 2025). In Indonesia, a systematic review and meta-analysis also found that food insecurity, unimproved drinking water, rural residence, and unimproved sanitation were important household- and community-level risk factors for stunting (Gusnedi *et al.*, 2023).

An additional consideration is the role of parental biological characteristics. Family socioeconomic variables may not capture genetic and intergenerational influences on linear growth. In particular, parental stature has been shown to be associated with stunting among Indonesian children. A systematic review and meta-analysis found that short maternal stature, short paternal stature, and short stature of both parents were associated with increased odds of stunting among Indonesian toddlers, although the evidence for paternal and combined parental stature showed substantial heterogeneity and should be interpreted cautiously (Amriviana *et al.*, 2023). Thus, the lack of significant correlations between parental age, family size, and income and HAZ does not imply that family-related factors are unimportant; rather, it suggests that the specific demographic and socioeconomic indicators examined in Table 6 were not strong predictors of HAZ in this particular population.

Overall, the findings of Table 6 indicate that family demographic and socioeconomic characteristics had little explanatory value for variation in HAZ in this sample. The strongest observed relationship was between maternal age and HAZ, but even this association was weak and did not achieve statistical significance. The other variables showed either negligible or very weak correlations. These findings suggest that future analyses should move beyond simple bivariate associations and examine the interconnected pathways through which family socioeconomic conditions may affect child growth. Multivariable analysis incorporating maternal height and nutritional status, birth weight and birth length, breastfeeding and complementary feeding

practices, dietary adequacy, household food security, morbidity, sanitation, water quality, and healthcare utilization would provide a more comprehensive assessment of the determinants of HAZ. Such an approach would be more consistent with the multifactorial nature of stunting and would help distinguish distal socioeconomic determinants from the proximal factors that directly influence linear growth.

Household Expenditures. Table 7 presents the differences in family expenditures between households with stunted and non-stunted children under two years old. Overall, the data show that while there are variations in spending patterns across categories, most differences are not statistically significant ($p > 0,05$). This suggests that stunting is not directly associated with total household expenditure levels, but rather with how resources are allocated and utilized.

Table 12. The differences of family expenses between stunted and non-stunted children under two

Expenditure Components	Total	Stunting	Non-stunting	p
Staple foods, vegetables, and side dishes (IDR)	1,221,600 ± 700,427	1,147,300 ± 489,188	1,254,300 ± 775221	0.380
Snacks (IDR)	571,770 ± 540,198	586,140 ± 441,879	565,120 ± 582,191	0.832
Drinking water (IDR)	90,637 ± 92,218	76,047 ± 85,429	97,457 ± 94,903	0.210
Coffee, tea, and sugar (IDR)	87,478 ± 127,128	63,409 ± 76,166	98,626 ± 143,792	0.129
Milk and dairy products (IDR)	136,000 ± 43,5047	93,600 ± 191,969	154,000 ± 503,029	0.425
Healthcare and medical expenses (IDR)	77,700 ± 271,825	37,800 ± 73,367	94,900 ± 320,565	0.230
Cigarettes (IDR)	414,320 ± 342,651	345,110±267,329	443,640 ± 367,463	0.149
Personal care and household sanitation (IDR)	17,8740 ± 13,1124	202,750 ± 147,619	167,970 ± 122,226	0.127
Transportation costs (IDR)	252,830 ± 240,979	272,980 ± 280,994	244,100 ± 222,488	0.518
Children's education (IDR)	98,239 ± 204,753	46,357 ± 49,885	118,460 ± 236,899	0.153
School allowance (IDR)	388,160 ± 285,703	332,280 ± 225,076	415,320 ± 308,736	0.159
Clothing and apparel (IDR)	116,680 ± 137,707	98,807 ± 99,870	124,380 ± 150,927	0.305
Telecommunications (IDR)	138,670 ± 188,365	141,070 ± 215,586	137,600 ± 176,052	0.919
Credit installments (IDR)	564,160 ± 824,458	426,370 ± 762,204	611,010 ± 846,777	0.429
Electricity (IDR)	108,320 ± 115,174	96,067 ± 88,665	113,730 ± 125,116	0.393
Water supply (IDR)	160,000 ± 180,076	125,000 ± 99,977	170,000 ± 197,750	0.452
Housing (IDR)	486,990 ± 187,947	522,920 ± 204,176	468,090 ± 178,735	0.295
Cooking fuel (IDR)	77,072.0 ± 87,645.2	71,792.0 ± 97,818.8	79,510 ± 82,923	0.615

Households with non-stunted children tend to spend slightly more on staple foods, vegetables, and side dishes (IDR 1.254.300 ± 775.221) compared to stunted households (IDR 1.147.300 ± 489.188), although the difference is not significant ($p = 0,380$). Similarly, spending on milk and dairy products is higher in non-stunted households (IDR 154.000 ± 503.029) than in stunted households (IDR 93.600 ± 191.969). This pattern aligns with evidence that dietary diversity and protein-rich foods are critical determinants of child growth (Torlesse *et al.*, 2016; Dewey & Begum, 2011).

Healthcare and medical expenses are notably higher among non-stunted households (IDR 94.900,0 ± 320.565,5) compared to stunted households (IDR 37.800 ± 73.367), though again not

statistically significant (p = 0,230). Investment in children's education also shows a similar trend, with non-stunted households spending more (IDR 118.460 ± 236.899) than stunted households (IDR 46.357 ± 49.885). These findings suggest that households with better child growth outcomes may prioritize health and education expenditures, consistent with studies linking parental investment in human capital to improved child development (Victora *et al.*, 2008).

Interestingly, cigarette expenditures are higher in non-stunted households (IDR 443.640 ± 367.463) compared to stunted households (IDR 345.110 ± 267.329). Although not significant (p = 0,149), this highlights the persistence of spending on non-essential items, which may compete with nutrition-related expenditures. Previous research has shown that household spending on tobacco can reduce resources available for food and healthcare, indirectly contributing to child malnutrition (John *et al.*, 2019).

Expenditures on utilities such as electricity, water supply, and housing do not differ significantly between groups. This indicates that basic living costs are relatively fixed and less influenced by child nutritional status. However, personal care and sanitation costs are higher among stunted households (IDR 202.750 ± 147.619) compared to non-stunted households (IDR 167.970 ± 122.226), which may reflect differences in household priorities or external factors such as environmental sanitation challenges.

The findings from Table 7 suggest that while overall expenditure levels are similar, households with non-stunted children tend to allocate more resources toward food diversity, healthcare, and education. In contrast, households with stunted children may face constraints in prioritizing these investments. This supports the notion that stunting is not merely a function of poverty, but also of household decision-making and resource allocation (Black *et al.*, 2013). Strengthening nutrition-sensitive interventions and promoting better household spending priorities could help reduce stunting prevalence.

Table 8 presents the distribution of household asset ownership, categorizing the assets into three main groups: electronic and domestic appliances, vehicles, and property and land assets. In epidemiological and public health research, assessing household asset ownership is a robust and widely accepted proxy for evaluating a family's socioeconomic status (SES) (Vyas & Kumaranayake, 2006). Socioeconomic status, in turn, is a critical underlying determinant of child health and nutritional outcomes, including the prevalence of stunting (Black *et al.*, 2013).

Table 13. Distribution of household assets ownership

Asset Category	Total		Stunting		Non-stunting	
	n	%	n	%	n	%
Electronic & Domestic Appliances						
Laptop/computer	25	15.60	6	12.50	19	17.00
Television	118	73.80	35	72.90	83	74.10
Refrigerator	126	78.80	36	75.00	90	80.40
Washing machine	108	67.50	32	66.70	76	67.90
Air conditioner (AC)	33	20.60	9	18.80	24	21.40
Vehicles						
Motorcycle	118	73.80	34	70.80	84	75.00

Asset Category	Total		Stunting		Non-stunting	
	n	%	n	%	n	%
Car	8	5.00	3	6.20	5	4.50

Table 14. Distribution of household assets ownership (continued)

Asset Category	Total		Stunting		Non-stunting	
	n	%	n	%	n	%
Property & Land Assets						
House	54	33.80	13	27.10	41	36.60
Land	33	20.60	8	16.70	25	22.30
Agricultural land/farmland	10	6.20	5	10.40	5	4.50

The data reveals a general trend where households with non-stunted children possess a higher proportion of modern electronic appliances and general property compared to households with stunted children. For instance, refrigerator ownership is noticeably higher in the non-stunting group (80,40%) than in the stunting group (75,00%). This finding aligns with existing literature suggesting that access to a refrigerator allows for better food preservation, which increases household food security and dietary diversity—key factors in preventing chronic undernutrition (Torlesse *et al.*, 2016). Furthermore, ownership of other domestic conveniences such as laptops/computers (17,00% vs. 12,50%), televisions (74,10% vs. 72,90%), washing machines (67,90% vs. 66,70%), and air conditioners (21,40% vs. 18,80%) consistently shows higher prevalence among non-stunting households.

Regarding vehicle ownership, motorcycles are the most common mode of transportation for both groups, but ownership remains higher in non-stunting households (75,00%) compared to stunting households (70,80%). Interestingly, car ownership is slightly higher in the stunting group (6,20%) than the non-stunting group (4,50%). However, because the absolute number of households owning cars is very small (n=3 for stunting and n=5 for non-stunting), this specific metric may not serve as a statistically definitive socioeconomic indicator for this population.

Property and land asset distribution provides compelling insights into the demographic characteristics of the respondents. Homeownership (36,60%) and general land ownership (22,30%) are substantially higher among non-stunting households compared to stunting households, which report 27,10% and 16,70%, respectively. Conversely, a notable divergence is observed in the ownership of agricultural land or farmland; it is more than twice as common in stunting households (10,40%) than in non-stunting households (4,50%). This phenomenon suggests that households experiencing stunting may be more concentrated in rural, agrarian communities. In many developing regions, reliance on small-scale farming—if not supported by adequate modern infrastructure and income—can correlate with rural poverty, poor sanitation (WASH facilities), and limited access to primary healthcare, all of which heavily exacerbate the risk of child stunting (Torlesse *et al.*, 2016; UNICEF, 2021).

The distribution of household assets in this study indicates that modern wealth indicators (such as electronics and residential property) are protective factors associated with non-stunting,

whereas the higher prevalence of agricultural land ownership among the stunting cohort points to specific vulnerabilities often found in rural or farming-dependent households.

c. Nutritional Status of Subjects

The pictures below represent nutritional status based on the height-for-age Z-score (HAZ), weight-for-age Z-score (WAZ), and weight-for-height Z-score (WHZ). Childhood nutritional status is comprehensively evaluated using three fundamental anthropometric indices: Height-for-Age Z-score (HAZ), Weight-for-Age Z-score (WAZ), and Weight-for-Height Z-score (WHZ). While WAZ reflects general undernutrition and WHZ captures acute body mass deficits (wasting), the Height-for-Age Z-score (HAZ) serves as the primary standard indicator for measuring chronic undernutrition and cumulative, long-term linear growth faltering (WHO, 2006; de Onis & Branca, 2016).

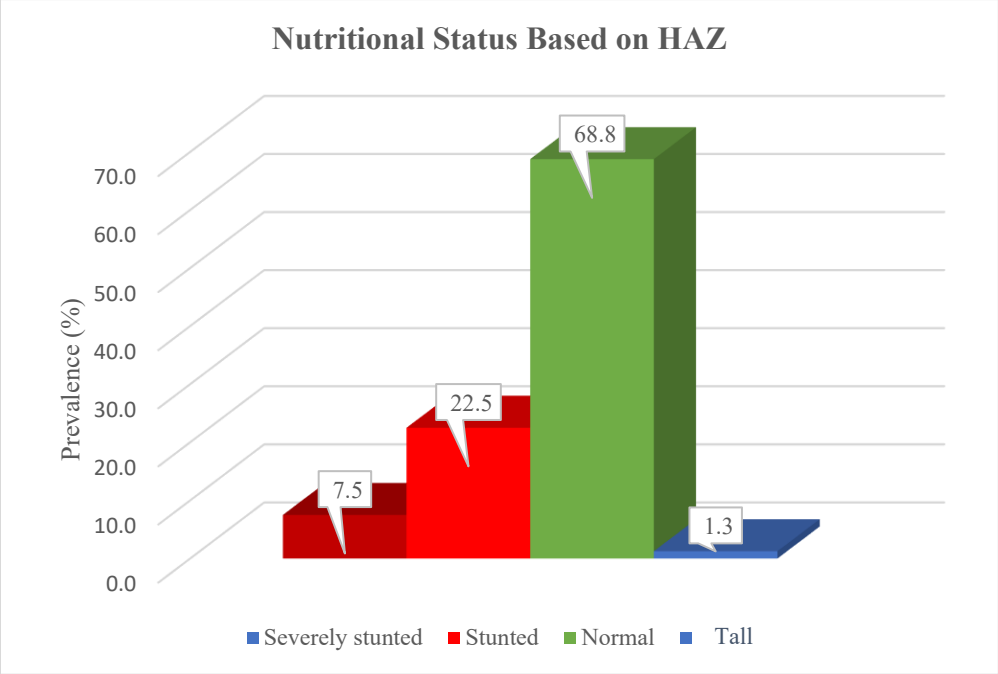

Figure 1. Distribution of nutritional status of subjects based on Height-for-Age Z-scores (HAZ)

As depicted in the chart, nutritional assessment using HAZ revealed that while the majority of children (68,8%) presented with normal stature and a minor proportion (1,3%) were classified as tall, a significant proportion of the population suffered from growth deficits. Specifically, the overall prevalence of stunting reached 30,0%, comprising 22,5% with moderate stunting (-3 SD ≤ HAZ < -2 SD) and 7,5% with severe stunting (HAZ < -3 SD).

According to public health benchmarks established by the World Health Organization (WHO) and UNICEF, a stunting prevalence threshold of 30,0% categorizes this population as facing a "high" to "very high" public health risk (de Onis *et al.*, 2019). This substantial prevalence indicates prolonged nutritional deprivation, recurrent childhood infectious diseases, and

inadequate maternal-child health practices during the crucial first 1.000 days of life (Black *et al.*, 2013).

Furthermore, the overall mean HAZ was observed at -1,2 ± 1,4. In a well-nourished, healthy reference population, the mean HAZ typically centers around 0,0 with a standard deviation of 1.0 (WHO, 2006). A population mean of -1,2 indicates a clear leftward shift in the entire growth distribution curve. This shift suggests that linear growth impairment is not isolated solely to those meeting the stunting threshold (-2 SD); rather, the whole cohort experiences widespread growth constraints. Such systemic faltering is frequently driven by shared socio-environmental determinants, including inadequate infant and young child feeding (IYCF) practices, poor sanitation, and environmental enteric dysfunction (Prendergast & Humphrey, 2014; Danaei *et al.*, 2016).

Unaddressed chronic growth failure of this magnitude bears severe physiological and socio-economic consequences. Stunting in early childhood is strongly linked to irreversible cognitive deficits, lower educational achievement, reduced labor productivity in adulthood, and an increased risk of chronic metabolic disorders later in life (Victora *et al.*, 2008; Dewey & Begum, 2011). Consequently, these findings highlight an urgent need for targeted, multi-sectoral interventions focused on optimizing maternal nutrition, enhancing dietary diversity, and strengthening community hygiene infrastructure.

Weight-for-Age Z-score (WAZ) serves as a key composite anthropometric index that evaluates overall undernutrition by reflecting both acute body mass loss (wasting) and chronic linear growth failure (stunting) (WHO, 2006; de Onis & Branca, 2016). While WAZ does not distinguish between acute and chronic malnutrition on its own, evaluating it alongside stunting provides critical insights into the compound nutritional deficits experienced by children (McDonald *et al.*, 2013).

As illustrated in Figure 2, the overall prevalence of underweight in the study population was 15,0%. According to public health severity thresholds established by the World Health Organization (WHO), an underweight prevalence between 10,0% and 19,9% represents a moderate-to-high public health concern requiring systematic monitoring and targeted nutritional interventions (de Onis *et al.*, 2019).

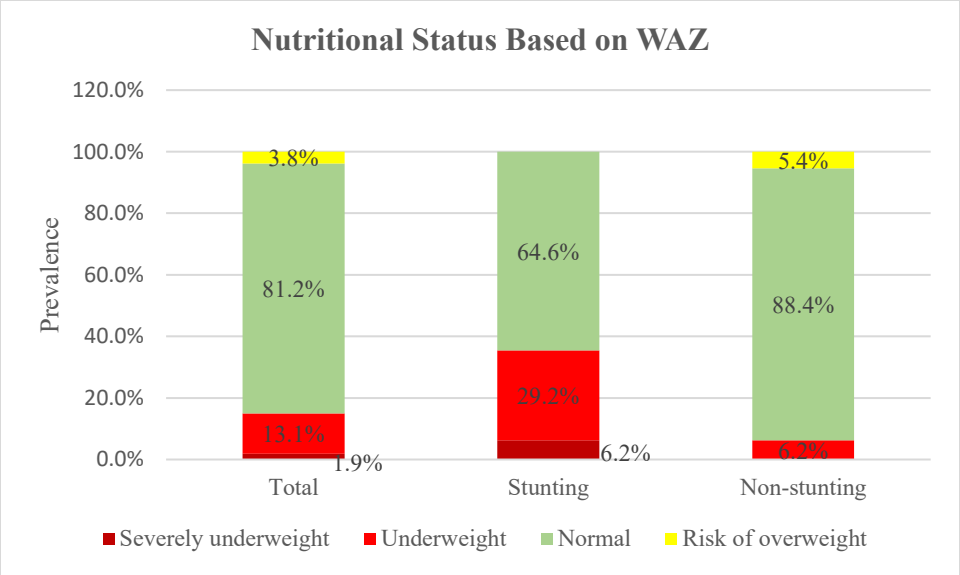

Figure 2. Distribution of nutritional status of subjects based on Weight-for-Age Z-score (WAZ)

A disaggregated analysis reveals a striking disparity in nutritional vulnerability between stunted and non-stunted cohorts. Underweight was disproportionately concentrated among stunted children, reaching a prevalence of 35,4%, compared to only 6,2% among non-stunted children. This pronounced overlap highlights the frequent co-occurrence of multiple anthropometric deficits within the same child. Longitudinal studies indicate that chronic nutrient restriction and recurrent infections do not merely impair linear bone growth; they simultaneously restrict soft tissue development and muscle mass accumulation (Schoenbuchner *et al.*, 2019; Khara *et al.*, 2023). Children presenting with multiple anthropometric deficits (such as concurrent stunting and underweight) are known to suffer from compounded immune dysfunction and face an exponentially elevated risk of mortality compared to those with single deficits (McDonald *et al.*, 2013).

This structural disparity is further demonstrated by the comparison of mean Z-scores. The mean WAZ among stunted subjects (-1,70 ± 0,85) was significantly lower ($p < 0,001$) than that of non-stunted subjects (-0,48 ± 1,00). In a well-nourished population, the mean WAZ centers around 0,0 (WHO, 2006). The substantial negative shift in mean WAZ among stunted children reflects severe, systemic body mass deficits that extend across the entire stunted cohort, reinforcing the understanding that stunting is a manifestation of broader, chronic physiological deprivation (Black *et al.*, 2013).

Conversely, an intriguing divergence was observed regarding overnutrition: the risk of overweight was identified exclusively among non-stunted children (5,4%), with zero prevalence recorded in the stunted group. This finding illustrates a bifurcated nutritional landscape within the community, characteristic of the double burden of malnutrition (DBM) at the population level (Popkin *et al.*, 2020; Wells *et al.*, 2020). As communities undergo socio-economic and dietary transitions, access to energy-dense, nutrient-poor processed foods often increases. While some children continue to suffer from chronic growth faltering, others may accumulate excess weight,

underscoring the need for nuanced public health strategies that simultaneously combat undernutrition while preventing early-onset childhood overweight and obesity (Popkin *et al.*, 2020).

Weight-for-Height Z-score (WHZ) is the standard anthropometric index used to evaluate acute nutritional status, reflecting current body weight relative to linear length or height (WHO, 2006). While Height-for-Age (HAZ) captures chronic, cumulative linear growth faltering (stunting), WHZ is sensitive to short-term changes in energy balance, making it the primary metric for identifying both acute undernutrition (wasting) and acute overnutrition (overweight and obesity) (de Onis & Branca, 2016).

As illustrated in Figure 3, the primary nutritional challenge identified in this population is a prominent risk of overnutrition—encompassing at-risk of overweight, overweight, and obesity—with a combined prevalence of 9,6%. This figure substantially exceeds the 3,8% prevalence of acute wasting. This shift illustrates an ongoing nutrition transition within the community, where the burden of acute undernutrition is increasingly overshadowed by emerging overnutrition, giving rise to a double burden of malnutrition (DBM) at the population level (Popkin *et al.*, 2020).

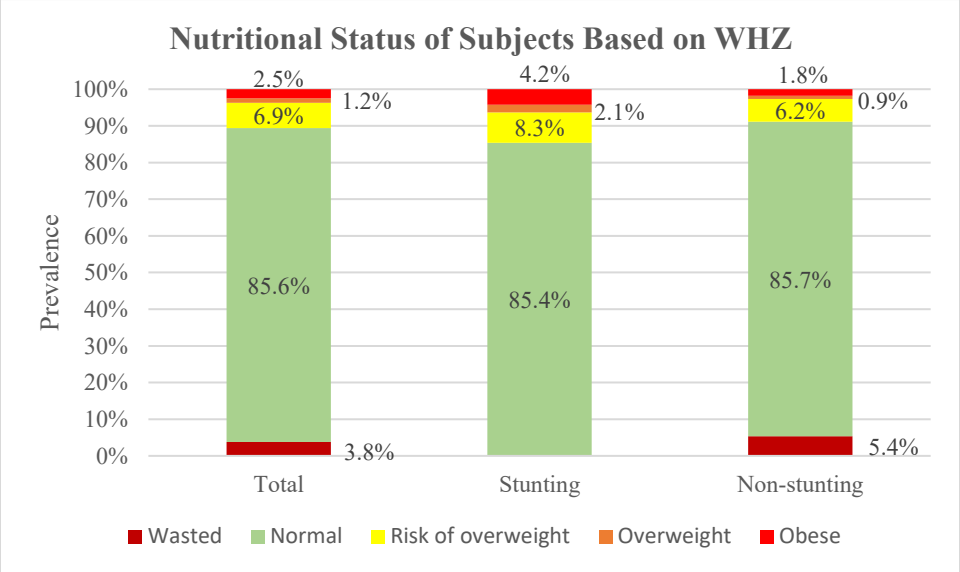

Figure 3. Distribution of nutritional status of subjects based on Weight-for-Height Z-score (WHZ)

A disaggregated evaluation of WHZ across stunting categories reveals a striking and paradoxical trend. Contrary to the conventional expectation that chronic undernutrition co-occurs with acute wasting, wasting in this study was present exclusively among non-stunted children (3,8%), with zero cases recorded among stunted children. This finding indicates that the stunted children in this cohort were not experiencing acute caloric starvation or severe thinness at the time of assessment; rather, their growth restriction was localized to linear skeletal deficits accumulated over time (Prendergast & Humphrey, 2014).

Conversely, the prevalence of overnutrition was noticeably higher among stunted children (14,6%) compared to non-stunted children (8,9%). The coexistence of stunting and elevated

overnutrition risk in the same individuals is a well-documented physiological paradox in public health nutrition (Popkin *et al.*, 1996; Tzioumis & Adair, 2014). Early-life nutritional deprivation induces profound metabolic adaptations, often conceptualized under the "thrifty phenotype hypothesis" (Hales & Barker, 2001). When a child experiences chronic undernutrition during critical developmental windows, the body adjusts its physiology to conserve energy by lowering basal metabolic rate, impairing fat oxidation, and reducing lean muscle mass accumulation (Hoffmann *et al.*, 2000; Sawaya *et al.*, 2003). When these stunted children are subsequently exposed to energy-dense, nutrient-poor diets or reduced physical activity, their altered metabolic capacity promotes preferential fat storage relative to their truncated stature, thereby significantly elevating their susceptibility to overweight and obesity (Hoffmann *et al.*, 2000; Wells *et al.*, 2020).

Despite these divergent categorical distributions, the mean WHZ of stunted subjects (-0,32 ± 1,19) was only slightly lower than that of non-stunted subjects (-0,23 ± 1,10), showing no statistically significant difference (p = 0,631). This lack of statistical significance suggests that, on average, the proportion of weight relative to height remains balanced across both groups. However, the broader variance in the stunted cohort reflects a skewed distribution toward the upper tail of WHZ, where stunted children gain weight out of proportion to their linear growth (WHO, 2006; Wells *et al.*, 2020).

These findings carry critical public health implications. Interventions that rely solely on unmitigated caloric supplementation to treat growth faltering risk exacerbating pediatric obesity and metabolic dysfunction among stunted children (Wells *et al.*, 2020). Public health strategies must therefore move beyond simple calorie delivery toward multi-sectoral programs that prioritize micronutrient-dense diets, promote physical activity, and optimize early maternal-child health to foster true linear growth rather than excess adiposity.

The assessment of Chronic Energy Deficiency (CED) provides crucial insight into the adequacy of subcutaneous fat and muscle mass reserves, serving as a vital indicator of ongoing energy depletion. In this study, the overall prevalence of CED across the entire subject population was relatively low at 2,5%. However, a disaggregated analysis reveals a dramatic disparity between nutritional cohorts, with CED being overwhelmingly concentrated among stunted children. Specifically, children in the stunting group exhibited a CED prevalence of 6,2%, which is nearly seven times higher than the 0,9% observed in their non-stunted counterparts. This pronounced imbalance indicates that children suffering from chronic linear growth faltering are simultaneously far more vulnerable to acute energy deficits and peripheral tissue wasting.

This discrepancy in body composition is further corroborated by the quantitative comparison of Mid-Upper Arm Circumference Z-scores (MUACZ) (Figure 4). Stunted subjects presented with a mean MUACZ of -0,38 ± 0,98, whereas non-stunted subjects maintained a positive mean MUACZ of 0,22 ± 1,00, a difference that reached strong statistical significance (p = 0,001). Because MUACZ evaluates peripheral body composition relative to standard growth norms, the negative mean score in the stunted cohort reflects a generalized leftward shift in muscle and fat accretion across the group. In contrast, the positive average in the non-stunted cohort demonstrates adequate soft-tissue development. This statistically significant divergence

underscores that linear growth deficits in stunted children are closely linked to a broader deficit in body mass reserves rather than being isolated strictly to skeletal stature.

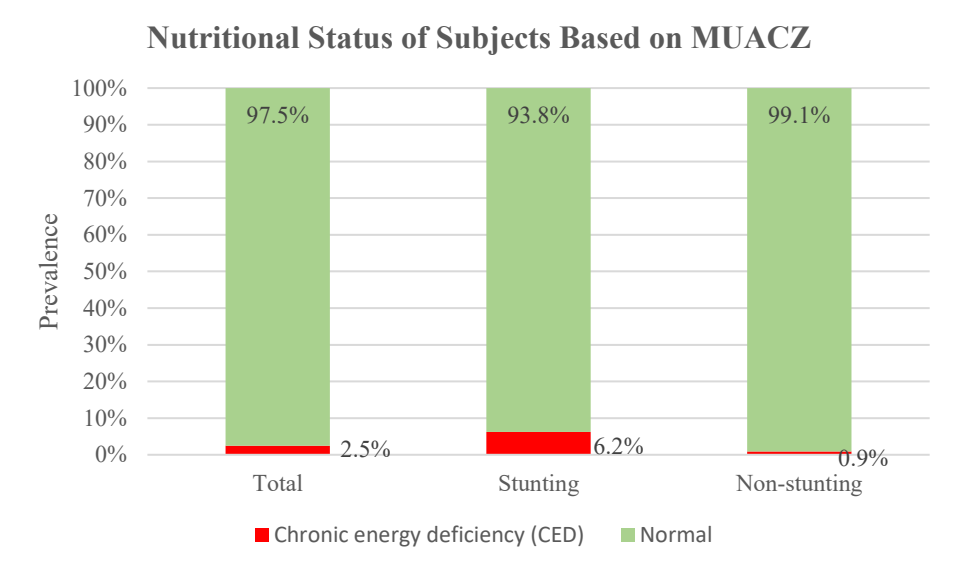

Figure 4. Distribution of nutritional status of subjects based on Mid Upper Arm Circumference Z-score (MUACZ)

From a clinical and public health perspective, the convergence of chronic growth stunting, elevated CED risk, and significantly lower MUAC Z-scores highlights a state of compounding nutritional vulnerability. Reduced peripheral muscle mass and subcutaneous fat stores compromise a child's physiological resilience, impairing immune defense mechanisms and increasing susceptibility to recurrent infections. These findings demonstrate that public health interventions targeting stunted populations must look beyond linear height recovery alone. Comprehensive strategies need to incorporate targeted nutritional support aimed at restoring functional tissue mass and correcting ongoing energy imbalances to ensure complete biological recovery and prevent further nutritional degradation.

d. Household Food Security

As illustrated in Figure 5, household food insecurity represents a pervasive public health challenge within the study population, with more than half of all households (53,10%) classified as food insecure, comprising 32,50% experiencing moderate food insecurity and 20,60% facing severe food insecurity. When disaggregated by child nutritional status, the burden of food insecurity was noticeably higher among households with stunted children (58,30%) compared to those with non-stunted children (50,90%). An item-level analysis of the Food Insecurity Experience Scale (FIES) further highlights that families of stunted children were disproportionately impacted by acute and severe manifestations of dietary disruption. Driven primarily by severe financial constraints and inadequate economic resources to acquire food, these households frequently reported an inability to access healthy and nutritious food, severe restriction

of dietary variety, skipping meals, consuming reduced portion sizes, running out of food entirely, and enduring hunger without eating.

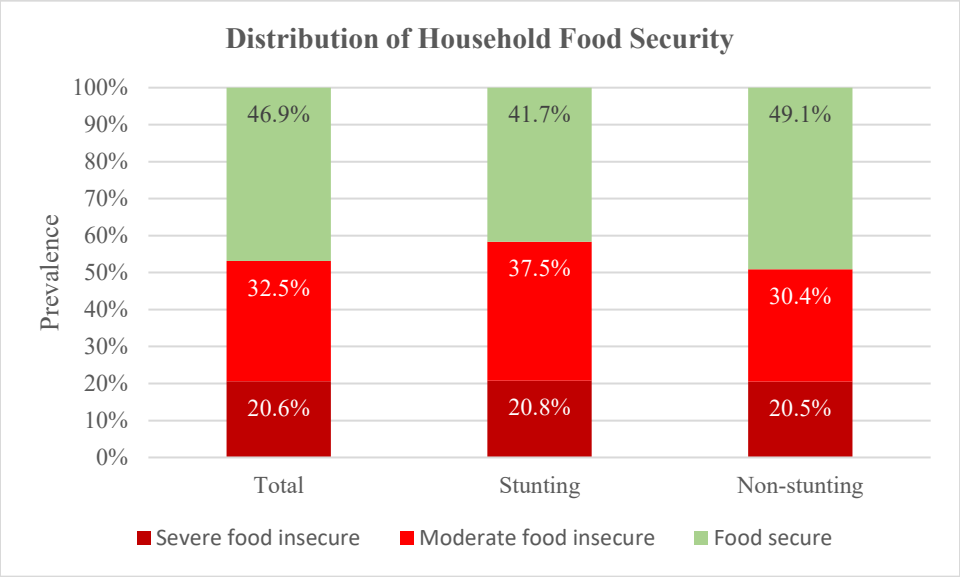

Figure 5. Distribution of household food security

Despite these descriptive divergences in extreme food insecurity experiences, inferential statistical analyses revealed that overall household food insecurity scores did not directly correlate with child linear growth outcomes in a statistically significant manner. The mean FIES score showed no significant difference between the stunted and non-stunted cohorts ($p = 0,522$), and a Pearson correlation test confirmed the absence of a linear association between the FIES score and child Height-for-Age Z-scores (HAZ) ($r = -0,057$, $p = 0,476$). This statistical outcome suggests that while subjective household food insecurity and economic hardship are widespread across the entire population, chronic childhood stunting is a multi-factorial phenomenon influenced by a broader array of underlying determinants—such as intra-household food allocation, maternal caregiving practices, persistent childhood infections, and environmental sanitation—that extend beyond household-level perceptions of food access alone.

e. Morbidity

Table 9 presents the mean monthly frequency of reported morbidity episodes during the preceding month among stunted and non-stunted children. Acute respiratory infection (ARI) was the most frequently reported condition in the overall study population, with a mean of $0,81 \pm 0,71$ episodes per month. Dermatological disorders and diarrhea were the next most frequently reported conditions, with mean frequencies of $0,24 \pm 0,44$ and $0,17 \pm 0,41$ episodes per month, respectively.

Several differences were observed between the stunted and non-stunted groups. The stunted group had a significantly higher mean monthly frequency of dysentery than the non-stunted group ($0,04 \pm 0,20$ vs. $0,00 \pm 0,00$ episodes/month; $p = 0,030$). Toothache was also significantly more frequent among stunted children ($0,10 \pm 0,31$ vs. $0,02 \pm 0,13$ episodes/month; $p = 0,014$). In

contrast, dermatological disorders were significantly more frequent among non-stunted children than among stunted children (0,29 ± 0,47 vs. 0,13 ± 0,33 episodes/month; p = 0,034).

The frequency of ARI was higher among stunted children than among non-stunted children (0,98 ± 0,84 vs. 0,74 ± 0,64 episodes/month), although the difference did not reach conventional statistical significance (p = 0,052). No episodes of typhoid, dengue fever, or filariasis were reported during the observation period; therefore, statistical comparisons for these conditions were not applicable.

Table 15. Mean monthly episode frequency of subjects

Disease	Total	Stunting	Non-stunting	p
Acute Respiratory Infection (ARI)	0.81 ± 0.71	0.98 ± 0.84	0.74 ± 0.64	0.052
Tuberculosis	0.01 ± 0.11	0.02 ± 0.14	0.01 ± 0.09	0.537
Typhoid	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	-
Dengue fever	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	-
Filariasis	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	-
Urinary Tract Infection (UTI)	0.01 ± 0.08	0.02 ± 0.14	0.00 ± 0.00	0.127
Diarrhea	0.17 ± 0.41	0.19 ± 0.45	0.16 ± 0.39	0.705
Dysentery	0.01 ± 0.11	0.04 ± 0.20	0.00 ± 0.00	0.030*
Varicella	0.02 ± 0.14	0.04 ± 0.20	0.01 ± 0.09	0.164
Measles	0.11 ± 0.31	0.13 ± 0.33	0.10 ± 0.30	0.617
Dermatological disorder	0.24 ± 0.44	0.13 ± 0.33	0.29 ± 0.47	0.034*
Toothache	0.04 ± 0.21	0.10 ± 0.31	0.02 ± 0.13	0.014*
Eye infections	0.04 ± 0.21	0.06 ± 0.24	0.04 ± 0.19	0.451

*significant based on Independent t-Test

The predominance of ARI in this study is consistent with the substantial burden of respiratory morbidity among young children, particularly in settings where repeated infections, environmental exposures, and nutritional vulnerability coexist. Importantly, the higher frequency of ARI among stunted children, although not statistically significant, is directionally consistent with the biological relationship between infection, inflammation, and impaired linear growth. DeBoer *et al.* (2017) demonstrated that recent symptoms of diarrhea, cough, and fever in young children were associated with systemic inflammation, while higher inflammatory activity was related to lower IGF-1 and IGFBP-3 concentrations and evidence of growth hormone resistance. These findings provide a plausible biological pathway through which recurrent morbidity may coexist with impaired linear growth, although the present study cannot determine the direction of the relationship.

The significantly greater frequency of dysentery among stunted children is also noteworthy. Enteric infections can affect growth not only through overt gastrointestinal symptoms but also through intestinal inflammation, impaired nutrient absorption, and systemic inflammatory responses. Rogawski *et al.* (2018), using longitudinal data from the MAL-ED cohort, reported that several enteropathogens, particularly Shigella, enteroaggregative Escherichia coli, Campylobacter, and Giardia, were associated with impaired linear growth. More recent evidence

from Malawi similarly indicates that asymptomatic enteric infections may influence subsequent linear growth through systemic inflammation and alterations in IGF-1-related pathways.

The higher frequency of toothache among stunted children should, however, be interpreted separately from the infectious-disease findings. Toothache is an oral-health symptom rather than a specific infectious-disease diagnosis and may reflect dental caries, gingival disease, or other oral conditions. Setijanto *et al.* (2025) reported evidence of an association between dental caries and stunting, with several studies suggesting that severe untreated dental disease may interfere with eating, appetite, and nutrient intake. Nevertheless, because the present study recorded toothache rather than clinically confirmed dental caries, the observed association should not be interpreted as evidence that dental disease caused stunting.

An unexpected finding was the significantly higher frequency of dermatological disorders among non-stunted children. This result is difficult to interpret biologically because the category "dermatological disorder" may encompass heterogeneous conditions with different etiologies and nutritional implications. The finding may reflect differences in exposure, health-seeking behavior, diagnosis, reporting, or the composition of the dermatological category rather than a protective effect of stunting. Millward (2017) emphasized that the relationship between infection, inflammation, nutrient adequacy, and linear growth is multifactorial, making isolated morbidity categories difficult to interpret without information on disease severity, inflammatory status, dietary adequacy, and socioeconomic conditions.

Overall, Table 9 suggests that morbidity patterns differed for selected conditions between stunted and non-stunted children, particularly dysentery and toothache, while ARI showed a borderline difference. However, these findings should be interpreted cautiously because the analysis involved multiple morbidity outcomes, several of which had very low event frequencies. Consequently, isolated statistically significant findings may represent true associations, but chance findings and residual confounding cannot be excluded without adjustment for multiple comparisons and potential confounders.

Table 10 presents the Pearson correlation between the monthly frequency of morbidity episodes and height-for-age Z-score (HAZ). A significant weak inverse correlation was observed between ARI episode frequency and HAZ ($r = -0{,}179$, $p = 0{,}024$). Similarly, UTI frequency was weakly and inversely correlated with HAZ ($r = -0{,}172$, $p = 0{,}030$), as was toothache frequency ($r = -0{,}185$, $p = 0{,}019$).

Dysentery showed a weak inverse correlation with HAZ ($r = -0{,}155$), although the association did not reach statistical significance ($p = 0{,}051$). In contrast, dermatological disorder frequency showed a weak positive correlation with HAZ ($r = 0{,}163$, $p = 0{,}040$). No significant correlations were observed for tuberculosis, diarrhea, varicella, measles, or eye infections. Typhoid, dengue fever, and filariasis could not be analyzed because no episodes were reported for these conditions.

Table 16. Correlation between monthly episode frequency and HAZ

Variable	HAZ	
	p	r
Acute Respiratory Infection (ARI)	0.024*	-0.179
Tuberculosis	0.803	-0.020
Typhoid	-	-
Dengue fever	-	-
Filariasis	-	-
Urinary Tract Infection (UTI)	0.030*	-0.172
Diarrhea	0.165	0.110
Dysentery	0.051	-0.155
Varicella	0.208	-0.100
Measles	0.129	-0.121
Dermatological disorder	0.040*	0.163
Toothache	0.019*	-0.185
Eye infections	0.431	-0.063

*significant based on Pearson Correlation Test ($p<0.05$)

The inverse correlations between ARI frequency, UTI frequency, toothache frequency, and HAZ indicate that children with more frequent episodes of these conditions tended to have lower HAZ values. However, the correlation coefficients were small (approximately r = 0,17–0,19), indicating weak associations rather than strong relationships. These findings therefore should not be interpreted as demonstrating that recurrent illness directly causes stunting. DeBoer *et al.* (2017) provided biological evidence that infection-related inflammation can interfere with the GH–IGF-1 axis, offering a plausible mechanism through which repeated inflammatory insults may be associated with impaired linear growth.

The finding for ARI is particularly relevant because respiratory infections can contribute to inflammatory burden and may coincide with reduced appetite, altered dietary intake, and increased metabolic demands during illness. Millward (2017) proposed that infection-related inflammation can inhibit linear growth through effects on endochondral bone growth and nutrient utilization, while nutritional deficiencies may further increase susceptibility to infection. Thus, the inverse association observed between ARI frequency and HAZ is biologically plausible, although the cross-sectional nature of the analysis prevents determination of whether recurrent illness contributed to low HAZ, whether children with poor growth were more susceptible to illness, or whether both were influenced by common underlying factors.

The inverse association between toothache frequency and HAZ is also potentially important from a nutritional perspective. Oral pain may reduce food intake, interfere with feeding, and alter dietary choices, particularly when children experience painful chewing. Setijanto *et al.* (2025) summarized evidence suggesting a relationship between severe dental caries and childhood undernutrition, although the evidence is heterogeneous. Because the present study assessed toothache rather than clinically diagnosed dental caries, this finding should be regarded as an indicator of potential oral-health vulnerability rather than definitive evidence of a dental-caries–stunting pathway.

The positive correlation between dermatological disorder frequency and HAZ was unexpected and should not be interpreted as indicating that dermatological morbidity is beneficial for growth. The effect size was small (r = 0,163), and the direction of association is difficult to reconcile with a general biological model linking morbidity and growth faltering. Differences in the types of dermatological conditions, exposure patterns, health-care utilization, reporting, or residual confounding may contribute to this finding. Millward (2017) emphasized that linear growth is determined by interacting nutritional, inflammatory, infectious, environmental, and socioeconomic factors rather than by individual morbidity categories in isolation.

Taken together, the results suggest that the frequency of selected morbidity episodes may be more closely related to HAZ than some other morbidity indicators, but the magnitude of these relationships is weak. Furthermore, because several correlations were tested simultaneously, the statistically significant findings should be considered exploratory unless a prespecified correction for multiple testing was applied. Future analyses should consider multivariable models incorporating age, sex, dietary intake, household socioeconomic status, sanitation, birth characteristics, and other potential determinants of linear growth.

Table 11 presents the mean duration of reported illness during the preceding month. ARI contributed the greatest mean duration of illness in the overall study population, at 3,98 ± 5,13 days per month, followed by dermatological disorders at 1,38 ± 3,27 days per month. The overall mean cumulative duration of reported illness was 7,19 ± 7,14 days per month.

The mean cumulative duration of illness was 6,10 ± 7,67 days among stunted children and 7,66 ± 6,88 days among non-stunted children. This difference was not statistically significant (p = 0,207). Similarly, no significant differences between stunted and non-stunted children were observed for the duration of any individual morbidity category, including ARI, tuberculosis, UTI, diarrhea, dysentery, varicella, measles, dermatological disorders, toothache, and eye infections (all p > 0,05). Typhoid, dengue fever, and filariasis were not included in statistical comparisons because no episodes were reported.

Table 17. Mean monthly illness duration (days)

Disease	Total	Stunting	Non-stunting	p
Acute Respiratory Infection (ARI)	3.98 ± 5.13	2.81 ± 3.73	4.47 ± 5.57	0.060
Tuberculosis	0.19 ± 2.37	0.63 ± 4.33	0.00 ± 0.00	0.127
Typhoid	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	-
Dengue fever	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	-
Filariasis	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	-
Urinary Tract Infection (UTI)	0.03 ± 0.32	0.08 ± 0.58	0.00 ± 0.00	0.127
Diarrhea	0.59 ± 1.74	0.29 ± 0.90	0.72 ± 1.98	0.151
Dysentery	0.05 ± 0.56	0.02 ± 0.14	0.06 ± 0.66	0.667
Varicella	0.11 ± 0.81	0.00 ± 0.00	0.15 ± 0.97	0.281
Measles	0.59 ± 2.58	0.35 ± 1.47	0.69 ± 2.93	0.456
Dermatological disorder	1.38 ± 3.27	1.73 ± 3.90	1.22 ± 2.96	0.371
Toothache	0.14 ± 0.76	0.13 ± 0.61	0.14 ± 0.81	0.892
Eye infections	0.16 ± 0.81	0.06 ± 0.43	0.20 ± 0.92	0.337
Total days of illness	7.19 ± 7.14	6.10 ± 7.67	7.66 ± 6.88	0.207

The absence of significant differences in illness duration between stunted and non-stunted children indicates that, within the one-month recall period, the cumulative duration of reported illness was not clearly differentiated by nutritional status. Although non-stunted children had a numerically longer total illness duration than stunted children, the difference was not statistically significant. This finding should not be interpreted as evidence that illness duration has no relationship with stunting. Rather, it indicates that the available one-month morbidity measure did not demonstrate a detectable difference between the two groups.

The distinction between illness frequency and illness duration is important when interpreting Tables 8–10. In the present study, several morbidity frequencies were associated with HAZ, whereas cumulative illness duration did not differ significantly according to stunting status. DeBoer *et al.* (2017) demonstrated that relatively recent symptoms of infection may be accompanied by systemic inflammation and alterations in the GH–IGF-1 axis, suggesting that the biological consequences of infection may depend not only on the number of days of symptoms but also on the type, intensity, recurrence, and inflammatory consequences of infection.

Similarly, Millward (2017) emphasized that growth faltering is the result of cumulative interactions between infection, inflammation, nutrient availability, and other environmental conditions. Therefore, a one-month estimate of illness duration may not adequately capture the cumulative morbidity burden relevant to HAZ, which reflects linear growth accumulated over a much longer period. This temporal mismatch is an important consideration when interpreting the absence of an association between total illness duration and stunting in this study.

The relatively long mean duration attributed to ARI and dermatological disorders may partly explain their contribution to the overall illness burden. Nevertheless, duration alone does not capture disease severity, treatment received, functional consequences, appetite changes, or inflammatory activity. Luoma *et al.* (2023) demonstrated that asymptomatic infections may also be associated with linear growth through inflammatory and endocrine pathways, emphasizing that clinically reported illness duration may underestimate the biological exposure relevant to growth.

Thus, the findings from Table 10 should be interpreted as evidence that cumulative reported illness days during the preceding month did not significantly differ between the two nutritional-status groups. Longitudinal surveillance with repeated morbidity measurements would provide a more appropriate assessment of whether persistent or recurrent illness contributes to subsequent linear growth faltering.

Table 12 presents the Pearson correlation between the duration of reported morbidity episodes and HAZ. The duration of dermatological disorders was significantly and weakly inversely correlated with HAZ ($r = -0{,}202$, $p = 0{,}010$). In contrast, the duration of varicella was positively correlated with HAZ ($r = 0{,}187$, $p = 0{,}018$).

No significant correlations were observed between HAZ and the duration of ARI ($r = 0{,}032$, $p = 0{,}690$), tuberculosis ($r = -0{,}058$, $p = 0{,}464$), UTI ($r = -0{,}048$, $p = 0{,}545$), diarrhea ($r = 0{,}000$, $p = 1{,}000$), dysentery ($r = 0{,}066$, $p = 0{,}404$), measles ($r = 0{,}105$, $p = 0{,}185$), toothache ($r = -0{,}028$, $p = 0{,}722$), or eye infections ($r = 0{,}001$, $p = 0{,}990$). The duration of typhoid, dengue fever, and filariasis could not be analyzed because no cases were reported.

Table 18. Correlation between illness duration and HAZ

Variable	HAZ	
	p	r
Acute Respiratory Infection (ARI)	0.690	0.032
Tuberculosis	0.464	-0.058
Typhoid	-	-
Dengue fever	-	-
Filariasis	-	-
Urinary Tract Infection (UTI)	0.545	-0.048
Diarrhea	1.000	0.000
Dysentery	0.404	0.066
Varicella	0.018*	0.187
Measles	0.185	0.105
Dermatological disorder	0.010*	-0.202
Toothache	0.722	-0.028
Eye infections	0.990	0.001

*significant based on Pearson Correlation Test ($p<0.05$)

The inverse association between the duration of dermatological disorders and HAZ suggests that children with longer reported periods of dermatological morbidity tended to have lower linear-growth status. However, the correlation was weak ($r = -0,202$), and the analysis does not establish a causal relationship. The biological interpretation may depend strongly on the underlying dermatological conditions because the category includes potentially heterogeneous disorders with different nutritional, inflammatory, and infectious characteristics. Millward (2017) emphasized that inflammation and nutrient availability can interact to influence growth-plate activity and linear growth, but the present dataset does not provide sufficient information to determine whether inflammation associated with skin disease contributed to lower HAZ.

The positive association between varicella duration and HAZ is biologically unexpected and should therefore be interpreted with considerable caution. A positive correlation does not imply that longer varicella illness promotes linear growth. Given the low frequency of varicella episodes, the weak effect size, the cross-sectional design, and the large number of correlations examined, this result may reflect sampling variability, influential observations, residual confounding, or a chance finding. DeBoer *et al.* (2017) described the general relationship between infection, inflammation, and impaired growth, but their findings do not provide a biological basis for interpreting prolonged varicella illness as beneficial to growth.

The absence of significant associations for the duration of ARI, diarrhea, UTI, dysentery, measles, toothache, and eye infections further illustrates that illness duration during a short recall period may not be an adequate proxy for cumulative infectious or inflammatory exposure. Luoma *et al.* (2023) showed that apparently asymptomatic infections can be associated with subsequent linear growth through inflammatory pathways and reduced IGF-1, indicating that clinically observable illness duration captures only part of the infection–growth relationship.

An important consideration is the temporal relationship between morbidity and HAZ. HAZ represents accumulated linear-growth performance over time, whereas the morbidity variables in this study were based on a relatively short observation or recall period. Consequently, children

with low HAZ may have experienced substantial morbidity earlier in life that was not captured by the current morbidity assessment. Conversely, recent illness may not yet have had sufficient time to influence measurable linear growth. DeBoer *et al.* (2017) and Millward (2017) both support a model in which repeated infection and inflammation can influence growth over time, reinforcing the value of longitudinal rather than single-period morbidity measurements.

Overall, Table 12 provides evidence of weak associations between illness duration and HAZ for two morbidity categories, but these findings should be regarded as exploratory. In particular, the positive varicella association should not be given a biological interpretation without replication in a larger longitudinal dataset. The results also reinforce the importance of considering cumulative morbidity, disease severity, inflammatory biomarkers, dietary intake, sanitation, and socioeconomic conditions when investigating determinants of childhood stunting.

f. Infant and Young Child Feeding

Table 13 presents the distribution of breastfeeding and early infant-feeding practices according to stunting status. Overall, 13,3% of children had a history of early initiation of breastfeeding (EIBF), while 86,7% had not experienced EIBF. Exclusive breastfeeding was reported for 50,0% of children. Regarding colostrum management, 76,1% of children were reported to have been fully fed colostrum, whereas 10,3% received colostrum partially and 4,5% had colostrum completely discarded. In addition, 32,5% of children received prelacteal feeding, while 67,5% did not. Continued breastfeeding was reported for 72,5% of children. Complementary feeding was initiated at six months in 68,1% of children, whereas 29,4% were introduced to complementary foods before six months and 2,5% after six months. No statistically significant difference was observed in the timing of complementary feeding initiation according to stunting status (p = 0,885).

Table 19. Distribution of breastfeeding practices

Variables	Total		Stunting		Non-Stunting		p
	n	%	n	%	n	%	
Early Initiation of Breastfeeding (EIBF) History							0.534
No	137	86.70	42	87.50	95	86.40	
Yes	21	13.30	6	12.50	15	13.60	
Exclusive Breastfeeding							0.194
No	80	50.00	21	43.80	59	52.70	
Yes	80	50.00	27	56.20	53	47.30	
Management of Colostrum							0.526
Completely discarded	7	4.50	3	6.50	4	3.70	
Partially fed	16	10.30	5	10.90	11	10.10	
Fully fed / Given completely	118	76.10	36	78.30	82	75.20	
Don't know / Forgotten	14	9.00	2	4.30	12	11.00	
Prelacteal Feeding							0.126
Yes	52	32.50	12	25.00	40	35.70	
No	108	67.50	36	75.00	72	64.30	

Table 20. Distribution of breastfeeding practices (continued)

Variables	Total		Stunting		Non-Stunting		p
	n	%	n	%	n	%	
Continued Breastfeeding							0.258
No	44	27.50	11	22.90	33	29.50	
Yes	116	72.50	37	77.10	79	70.50	
Timing of Complementary Feeding Initiation							0.885
Early (<6 months)	47	29.40	13	27.10	34	30.40	
Delayed (>6 months)	4	2.50	1	2.10	3	2.70	
Timely (at 6 months)	109	68.10	34	70.80	75	67.00	
Mixed milk feeding							0.038*
Yes	65	40.60	14	29.20	51	45.50	
No	95	59.40	34	70.80	61	54.50	
Milk Feeding Media							0.628
Feeding bottle / Nipple	59	85.50	11	78.60	48	87.30	
Cup	2	2.90	1	7.10	1	1.80	
Spoon	7	10.10	2	14.30	5	9.10	
Straw	1	1.40	0	0.00	1	1.80	

*significant based on Chi-Square Test

Mixed milk feeding was reported in 40,6% of children. This practice was significantly more common among non-stunted children than stunted children (45,5% vs. 29,2%; p = 0,038). In contrast, EIBF, exclusive breastfeeding, colostrum management, prelacteal feeding, continued breastfeeding, complementary feeding initiation, and milk-feeding media did not differ significantly according to stunting status (all p > 0,05). Among children receiving milk through a feeding device, the bottle/nipple was the predominant feeding medium (85,5%).

The relatively low prevalence of EIBF (13,3%) and exclusive breastfeeding (50,0%) indicates that several recommended breastfeeding practices were not consistently achieved in this population. Simbolon *et al.* (2024) similarly reported substantial gaps in IYCF practices in Indonesia, including early breastfeeding initiation, exclusive breastfeeding, and complementary feeding. Their analysis showed that IYCF practices were influenced by maternal education, socioeconomic conditions, antenatal and postnatal care, and access to health information, emphasizing that breastfeeding behavior is shaped by a broader social and health-system context rather than maternal knowledge alone.

The finding that 76,1% of children were reportedly fully fed colostrum is relatively favorable compared with the low prevalence of EIBF and exclusive breastfeeding. These indicators represent different dimensions of breastfeeding practice and therefore do not necessarily move in parallel. Resvick *et al.* (2025), in a systematic review and meta-analysis, found that breastfeeding was associated with improved growth outcomes among infants born small for gestational age, supporting the importance of sustained human-milk feeding for child growth. However, the

magnitude and direction of associations can vary according to maternal, birth, socioeconomic, and complementary-feeding characteristics.

The prevalence of prelacteal feeding in this study (32,5%) is another important finding because prelacteal feeding may interfere with the establishment of breastfeeding and may expose newborns to potentially unsafe substances depending on the type and preparation of the feed. Simbolon *et al.* (2024) demonstrated that suboptimal IYCF practices remain common in Indonesia and are associated with maternal and household characteristics. This suggests that interventions aimed at improving breastfeeding should begin during pregnancy and continue through the early postnatal period rather than focusing only on mothers after delivery.

Continued breastfeeding was relatively common, with 72,5% of children still being breastfed. This is an important positive feature of the feeding pattern because breast milk continues to contribute energy, protein, essential fatty acids, and bioactive components during the complementary-feeding period. Resvick *et al.* (2025) found that breastfeeding can support growth, although breastfeeding should be considered as part of an overall feeding pattern rather than as an isolated determinant of linear growth.

The only breastfeeding-related variable that differed significantly between groups was mixed milk feeding, which was more common among non-stunted children (45,5%) than stunted children (29,2%). This finding should be interpreted cautiously because the direction is not necessarily consistent with a simple model in which additional milk exposure protects against stunting. Pasqualino *et al.* (2025) demonstrated that energy and protein inadequacy during complementary feeding can remain substantial even when children receive additional nutritional products, highlighting the importance of evaluating the total dietary pattern and nutrient adequacy rather than interpreting a single feeding behavior in isolation.

The lack of significant differences in EIBF, exclusive breastfeeding, colostrum management, prelacteal feeding, continued breastfeeding, and complementary feeding initiation between stunted and non-stunted children should also be interpreted carefully. Beatty *et al.* (2024) demonstrated in a large cluster-randomized trial in Indonesia that an integrated intervention improved exclusive breastfeeding and recommended meal frequency but did not produce a significant reduction in stunting. This finding is highly relevant to the present study because it illustrates that improvements in proximal feeding indicators do not necessarily translate directly into differences in stunting prevalence over a relatively short period.

The predominance of bottle/nipple feeding among children receiving milk through a feeding medium (85,5%) also deserves attention. Although this study did not demonstrate a significant association between feeding medium and stunting, feeding method may have implications for responsive feeding, hygiene, and exposure to contamination, depending on how bottles are cleaned and used. Because the present study did not assess bottle-cleaning practices, milk type, volume consumed, or feeding responsiveness, no causal interpretation can be made from this variable alone.

Table 14 presents complementary feeding practices and WHO/UNICEF core feeding indicators according to stunting status. Overall, 68,1% of children were introduced to

complementary foods at six months, while 29,4% were introduced before six months and 2,5 after six months. Almost all children (97,5%) had consumed solid foods during the preceding 24 hours. The mean feeding frequency was 3,50 ± 1,16 times per day, with means of 3,31 ± 1,37 among stunted children and 3,58 ± 1,05 among non-stunted children.

Table 21. Distribution of complementary feeding practices

Variable	Total		Stunting		Non-Stunting		p
	n	%	n	%	n	%	
Timing of Complementary Feeding Initiation							0.885[a]
Early (<6 months)	47	29.40	13	27.10	34	30.40	
Delayed (>6 months)	4	2.50	1	2.10	3	2.70	
Timely (at 6 months)	109	68.10	34	70.80	75	67.00	
Solid food given in the last 24 hours							0.081[a]
No	4	2.50	3	6.20	1	0.90	
Yes	156	97.50	45	93.80	111	99.10	
Frequency (mean ± SD)	3.50 ± 1.16		3.31 ± 1.37		3.58 ± 1.05		
Minimum Meal Frequency							0.482[a]
Not achieved	22	13.80	6	12.50	16	14.40	
Achieved	137	86.20	42	87.50	95	85.60	
Minimum Milk Feeding Frequency							0.300[a]
Not achieved	4	2.50	0	0.00	4	3.60	
Achieved	39	24.40	10	20.80	29	25.90	
Not applicable	117	73.10	38	79.20	79	70.50	
Frequency (mean ± SD)	2.06 ± 2.91		1.85 ± 3.05		2.15 ± 2.87		0.556[b]
Minimum Acceptable Diet							0.374[a]
Not achieved	71	44.70	20	41.70	51	45.90	
Achieved	88	55.30	28	58.30	60	54.10	
Minimum Dietary Diversity							0.460[a]
Not achieved	56	35.00	16	33.30	40	35.70	
Achieved	104	65.00	32	66.70	72	64.30	
Dietary diversity score (mean ± SD)	4.81 ± 1.56		4.71 ± 1.57		4.85 ± 1.57		0.606[b]
Egg & flesh food in the last 24 hours							0.300[a]
No	36	22.5	9	18.8	27	24.1	
Yes	124	77.5	39	81.2	85	75.9	
Sugar-sweetened beverage consumption in the last 24 hours							0.191[a]
Yes	41	25.6	15	31.2	26	23.2	
No	119	74.4	33	68.8	86	76.8	

[a] p-value based on the Chi-Square Test
[b] p-value based on Independent t-Test

Minimum Meal Frequency (MMF) was achieved by 86,2% of children, whereas 13,8% did not meet the criterion. Minimum Dietary Diversity (MDD) was achieved by 65,0% of children, while 35,0% did not meet the criterion. The mean dietary diversity score was 4,81 ± 1,56 food

groups overall, with no significant difference between stunted and non-stunted children (4,71 ± 1,57 vs. 4,85 ± 1,57; p = 0,606).

Minimum Milk Feeding Frequency was achieved by 24,4% of children for whom the indicator was applicable, while 73,1% were classified as not applicable. Minimum Acceptable Diet (MAD) was achieved by 55,3% of children, and 44,7% did not meet the criterion. Consumption of eggs and/or flesh foods during the preceding 24 hours was reported for 77,5% of children. Sugar-sweetened beverage (SSB) consumption was reported for 25,6% of children. None of these complementary feeding indicators differed significantly according to stunting status (all p > 0,05).

The finding that 68,1% of children initiated complementary feeding at six months indicates that timely introduction was achieved by approximately two-thirds of the sample, although almost one-third were introduced to complementary foods earlier than six months. Simbolon *et al.* (2024) similarly identified inappropriate timing and quality of complementary feeding as persistent challenges in Indonesia. Importantly, the absence of a significant difference in timing between stunted and non-stunted children does not imply that timing is irrelevant to child growth; rather, it indicates that this cross-sectional comparison did not detect a difference between the two groups.

The high prevalence of solid-food consumption in the preceding 24 hours (97,5%) and the mean feeding frequency of 3,50 meals per day indicate that food provision was relatively frequent. However, Pasqualino *et al.* (2025) showed that children may continue to experience substantial energy and protein gaps despite receiving complementary foods. Therefore, meal frequency should not be interpreted as synonymous with dietary adequacy. The nutrient density, portion size, food-group composition, and preparation of complementary foods remain critical components of feeding quality.

MMF was achieved by 86,2% of children, representing one of the strongest IYCF indicators in this population. Nevertheless, MDD was achieved by only 65,0%, and MAD by 55,3%. Khura *et al.* (2024) demonstrated that higher dietary diversity was associated with lower risk of childhood underweight, while consumption of dairy, eggs, fruits, and vegetables contributed to more favorable nutritional outcomes. These findings support the concept that frequency of feeding alone is insufficient and that dietary quality and diversity are important components of complementary feeding.

The difference between the relatively high MMF and lower MDD and MAD is particularly informative. Recent intervention evidence indicates that complementary-feeding interventions can improve some IYCF indicators, particularly minimum dietary diversity (MDD) and minimum acceptable diet (MAD), although effects on minimum meal frequency (MMF) may be limited and vary according to the intervention strategy and local context (Beatty *et al.*, 2024). This suggests that increasing the number of meals may be easier than improving the diversity and nutritional quality of those meals. In the present population, therefore, future interventions should not focus solely on meal frequency but should emphasize nutrient-dense, diverse, and locally accessible foods.

The mean dietary diversity score of 4,81 ± 1,56 indicates that children consumed approximately five food groups on average, although one-third did not achieve MDD. Khura *et al.*

(2024) reported an association between MDD and a lower risk of underweight, supporting the nutritional importance of diversified complementary diets. However, the lack of a significant difference in dietary diversity score between stunted and non-stunted children in the present study should not be interpreted as evidence that dietary diversity has no relevance to linear growth. HAZ reflects cumulative growth, while the dietary assessment represents a short reference period and therefore may not capture long-term dietary exposure.

The relatively high consumption of eggs and/or flesh foods (77,5%) is a favorable feature because animal-source foods provide highly bioavailable protein and several micronutrients relevant to growth. Pasqualino *et al.* (2025) showed that complementary food supplementation can help fill energy and protein gaps among children with dietary inadequacy, while previous multi-country longitudinal evidence has linked higher energy and zinc intake with lower risk of some forms of undernutrition. Thus, the presence of animal-source foods should be interpreted together with portion size, frequency, and total dietary intake rather than as a binary indicator of dietary adequacy.

The consumption of sugar-sweetened beverages by 25,6% of children is another important finding. Although the difference between stunted and non-stunted children was not statistically significant, frequent consumption of SSBs may displace nutrient-dense foods and contribute to an overall poor dietary pattern. Because the current analysis captured consumption during only the preceding 24 hours, it cannot determine habitual SSB intake or its relationship with long-term growth.

Overall, the absence of statistically significant differences in complementary feeding indicators between stunted and non-stunted children should be interpreted as evidence that these indicators did not distinguish the two groups in this sample, rather than evidence that complementary feeding is unrelated to stunting. Beatty *et al.* (2024) provide an important Indonesian example showing that interventions can improve proximal IYCF behaviors without necessarily producing measurable reductions in stunting. This reinforces the multifactorial nature of stunting and the importance of considering household food security, maternal characteristics, infection, sanitation, and environmental exposures alongside feeding practices.

Table 15 presents the proportion of children consuming each of the eight food groups used to assess minimum dietary diversity. Grain and starchy roots were the most commonly consumed food group, reported by 91,2% of children. This was followed by vitamin A-rich fruits and vegetables (74,4%) and breast milk (70,6%). Flesh foods and dairy products were consumed by 60,0% and 51,9% of children, respectively, while eggs were consumed by 53,1%. Other fruits and vegetables were consumed by 56,2%, whereas beans were the least frequently consumed food group (23,1%).

Consumption patterns were broadly similar between stunted and non-stunted children. Breast milk, grains and starchy roots, and other fruits and vegetables were numerically more frequently consumed by stunted children, whereas dairy products, flesh foods, eggs, and vitamin A-rich fruits and vegetables were somewhat more frequently consumed by non-stunted children.

However, none of the eight food groups showed a statistically significant difference between stunted and non-stunted children (all p > 0,05).

Table 22. Proportion of children consuming 8 food groups for MDD

Food Group	Total		Stunting		Non-Stunting		p
	n	%	n	%	n	%	
ASI	113	70.60	36	75.00	77	68.80	0.275[a]
Grain and starchy roots	146	91.20	45	93.80	101	90.20	0.346[a]
Bean	37	23.10	8	16.70	29	25.90	0.143[a]
Dairy products	83	51.90	22	45.80	61	54.50	0.204[a]
Flesh foods	96	60.00	28	58.30	68	60.70	0.456[a]
Egg	85	53.10	24	50.00	61	54.50	0.364[a]
Vit.A rich fruit & vegetable	119	74.40	33	68.80	86	76.80	0.191[a]
Other fruit & vegetable	90	56.20	30	62.50	60	53.60	0.193[a]

[a] p-value based on the Chi-Square Test

The dominance of grains and starchy roots (91,2%) indicates that complementary feeding in this population was strongly centred on staple carbohydrate-based foods. Simbolon *et al.* (2024) reported that IYCF practices in Indonesia remain constrained by differences in socioeconomic conditions, maternal education, and access to nutritious foods. Although staple foods are important sources of energy, a diet dominated by grains and starchy roots may provide insufficient amounts of several micronutrients and high-quality protein if not complemented by diverse animal- and plant-source foods.

Vitamin A-rich fruits and vegetables were consumed by 74,4% of children, while other fruits and vegetables were consumed by 56,2%. This indicates relatively good inclusion of plant-source foods compared with beans, which were consumed by only 23,1% of children. Khura *et al.* (2024) found that dietary diversity, including consumption of fruits, vegetables, eggs, and dairy products, was associated with more favourable nutritional outcomes. These findings support the importance of increasing not only the number of food groups but also the regularity and quantity of nutrient-dense foods within complementary diets.

The relatively low consumption of beans (23,1%) is notable because legumes can contribute plant protein, iron, zinc, folate, and other nutrients. Pasqualino *et al.* (2025) emphasized that energy and protein gaps remain important nutritional constraints among children receiving complementary foods. Increasing access to affordable protein-rich foods, including legumes and animal-source foods, may therefore be particularly relevant in populations where complementary diets rely heavily on staple foods.

The consumption of flesh foods (60,0%) and eggs (53,1%) was relatively substantial, although the proportions did not differ significantly between stunted and non-stunted children. Maciel *et al.* (2021) reported that higher energy and zinc intakes from complementary foods were associated with lower risk of several forms of undernutrition across children from multiple low- and middle-income countries. These findings reinforce the importance of considering the nutrient

contribution of complementary foods rather than evaluating food groups solely by their presence or absence.

The absence of statistically significant differences in any of the eight food groups between stunted and non-stunted children should be interpreted in the context of the cross-sectional design and the 24-hour dietary recall. Khura *et al.* (2024) showed that MDD can be associated with nutritional outcomes at the population level, but a single-day dietary measure may not adequately represent usual dietary intake. In the present study, children with similar food-group consumption on the previous day could nevertheless have had substantially different dietary patterns during preceding months, which may be more relevant to accumulated linear growth.

The pattern observed in Table 14, therefore, suggests that the principal nutritional challenge may not simply be whether children consume food, but whether they receive a consistently diverse, nutrient-dense, and adequate diet over time. Pasqualino *et al.* (2025) demonstrated that complementary food supplementation can fill energy and protein gaps in children with dietary inadequacy, while Beatty *et al.* (2024) showed that improving IYCF practices in Indonesia does not necessarily result in immediate reductions in stunting. These findings support an integrated strategy combining dietary diversification, improved nutrient density, household food access, infection prevention, and sustained caregiver support.

Taken together, Tables 13–15 indicate that the study population demonstrated a mixture of favourable and suboptimal IYCF practices. Continued breastfeeding, timely complementary feeding, consumption of eggs and/or flesh foods, and minimum meal frequency were relatively common. However, important gaps remained in early initiation of breastfeeding, exclusive breastfeeding, prelacteal feeding, minimum dietary diversity, minimum acceptable diet, and the consumption of several nutrient-dense food groups. The predominance of grains and starchy roots and the relatively low consumption of beans further suggest that the overall dietary pattern remained strongly dependent on staple foods. Simbolon *et al.* (2024) reported that inappropriate IYCF practices remain common in Indonesia and are influenced by maternal education, socioeconomic conditions, antenatal and postnatal care, and access to health information. The pattern observed in the present study is therefore consistent with the broader Indonesian context, in which achieving recommended IYCF practices remains challenging despite substantial improvements in awareness and health-service coverage.

Pasqualino *et al.* (2025) further demonstrated that children receiving complementary foods may continue to have energy and protein inadequacies, emphasizing that the presence of meals does not necessarily indicate nutritional adequacy. This distinction is important in the present study because MMF was relatively high at 86,2%, whereas MDD and MAD were lower at 65,0% and 55,3%, respectively. Thus, the quality and nutrient density of the diet may represent a more important remaining challenge than meal frequency alone.

Khura *et al.* (2024) found that MDD was associated with a lower risk of childhood underweight, while consumption of several nutrient-rich food groups was associated with more favorable nutritional outcomes. This supports the importance of dietary diversity, but it also highlights why the absence of a significant association between MDD or individual food groups

and stunting in the present study should not be interpreted as evidence that diet quality is unimportant. The cross-sectional design and short dietary recall period may not adequately represent cumulative dietary exposure relevant to HAZ.

Beatty *et al.* (2024) provide an especially relevant interpretation for the Indonesian setting. Their large cluster-randomized trial demonstrated improvements in exclusive breastfeeding and recommended meal frequency following a multifaceted intervention, but no significant reduction in stunting. This finding indicates that stunting is unlikely to respond to improvements in a single proximal feeding indicator alone and that household food security, sanitation, maternal nutrition, infection, healthcare access, and other environmental determinants need to be addressed concurrently.

Overall, the IYCF findings suggest that the principal opportunity for improvement is not simply increasing the frequency of feeding, but ensuring that children receive a diverse, nutrient-dense, safe, and age-appropriate diet while breastfeeding is appropriately continued. The results also suggest that differences in individual IYCF practices may not be sufficient to explain stunting status in this population. A more comprehensive model integrating dietary quality, household food security, morbidity, sanitation, maternal characteristics, and socioeconomic conditions would therefore provide a stronger explanation of differences in linear growth.

g. Energy and Nutrient Adequacy

Energy and nutrient adequacy are important components in assessing the nutritional status and growth of children. In this study, the adequacy of energy and selected macronutrients and micronutrients was assessed and compared between children with stunting and those without stunting. The distribution of energy and nutrient adequacy was categorized as inadequate, adequate, or excessive based on the applicable nutritional requirements, while mean intake was also compared between the two groups. The distribution and comparison of energy and nutrient adequacy among the study subjects are presented in Table 16.

Table 23. Distribution of energy and nutrient adequacy of subjects

Energy and Nutrient Adequacy	Total		Stunting		Non-Stunting		p
	n	%	n	%	n	%	
Energy							0.249[a]
Inadequate	70	43.80	25	52.10	45	40.20	
Adequate	35	21.90	7	14.60	28	25.00	
Excessive	55	34.40	16	33.30	39	34.80	
Mean ± SD	93.45 ± 49.86 kcal		87.88 ± 54.01 kcal		95.83 ± 48.03 kcal		0.357[b]
Protein							0.860[a]
Inadequate	39	24.40	11	22.90	28	25.00	
Adequate	20	12.50	7	14.60	13	11.60	
Excessive	101	63.10	30	62.50	71	63.40	
Mean ± SD	173.45 ± 116.49 g		171.44 ± 124.05 g		174.31 ± 113.67 g		0.887[b]

Table 24. Distribution of energy and nutrient adequacy of subjects (continued)

Energy and Nutrient Adequacy	Total		Stunting		Non-Stunting		p
	n	%	n	%	n	%	
Fat							0.501[a]
Inadequate	79	49.40	26	54.20	53	47.30	
Adequate	36	22.50	8	16.70	28	25.00	
Excessive	45	28.10	14	29.20	31	27.70	
Mean ± SD	89.49 ± 57.56 g		90.39 ± 61.64 g		89.10 ± 56.01 g		0.897[b]
Carbohydrate							0.505[a]
Inadequate	69	43.10	21	43.80	48	42.90	
Adequate	42	26.20	15	31.20	27	24.10	
Excessive	49	30.60	12	25.00	37	33.00	
Mean ± SD	96.32 ± 53.60 g		88.77 ± 57.23 g		99.55 ± 51.90 g		0.245[b]
Calcium							0.105[a]
Inadequate	100	62.50	34	70.80	66	58.90	
Adequate	60	37.50	14	29.20	46	41.10	
Mean ± SD	126.06 ± 156.29 mg		97.06 ± 118.96 mg		138.49 ± 168.75 mg		0.125[b]
Phosphorus							0.541[a]
Inadequate	76	47.50	23	47.90	53	47.30	
Adequate	84	52.50	25	52.10	59	52.70	
Mean ± SD	128.35 ± 120.35 mg		121.77 ± 119.13 mg		131.18 ± 121.30 mg		0.652[b]
Iron							0.378[a]
Inadequate	82	51.20	26	54.20	56	50.00	
Adequate	78	48.80	22	45.80	56	50.00	
Mean ± SD	132.83 ± 269.68 mg		119.25 ± 137.65 mg		138.65 ± 309.91 mg		0.678[b]
Vitamin A							0.272[a]
Inadequate	40	25.00	14	29.20	26	23.20	
Adequate	120	75.00	34	70.80	86	76.80	
Mean ± SD	325.22 ± 329.85 mcg		327.52 ± 353.96 mcg		324.23 ± 320.62 mcg		0.954[b]
Vitamin C							0.142[a]
Inadequate	78	48.80	27	56.20	51	45.50	
Adequate	82	51.20	21	43.80	61	54.50	
Mean ± SD	166.30 ± 184.03 mg		162.14 ± 199.21 mg		168.08 ± 178.04 mg		0.852[b]
Zinc							0.241[a]
Inadequate	65	40.60	22	45.80	43	38.40	
Adequate	95	59.40	26	54.20	69	61.60	
Mean ± SD	199.53 ± 235.19 mg		190.10 ± 245.92 mg		203.57 ± 231.46 mg		0.741[b]

[a] p-value based on the Chi-Square Test
[b] p-value based on Independent t-Test

The analysis of daily energy and nutrient intake adequacy between stunted and non-stunted children is presented in Table 15. Across the total sample, inadequate energy intake was found in 43,8% of subjects, with stunted children showing a higher prevalence of energy deficiency (52,1%)

compared to non-stunted children (40,2%), although the mean energy intake did not significantly differ (87,88 ± 54,01 kcal vs. 95,83 ± 48,03 kcal; p = 0,357). Protein intake was excessive for the majority of subjects in both groups (62,5% in stunted vs 63,4% in non-stunted). Regarding micronutrients, calcium deficiency was particularly evident in the stunted cohort (70,8%) compared to the non-stunted cohort (58,9%), corresponding to lower mean calcium levels (97,06 ± 118,96 mg vs. 138,49 ± 168,75 mg), though this trend did not reach statistical significance (p = 0,105 for proportions; p = 0,125 for means). Similar non-significant patterns of higher inadequacy in stunted children were observed for fat (54,2%), iron (54,2%), vitamin C (56,2%), and zinc (45,8%). Ultimately, statistical evaluation using Chi-Square and Independent Sample t-tests revealed no significant associations between stunting status and either nutrient adequacy categories or absolute mean intakes (p > 0,05 for all metrics).

These patterns are broadly consistent with other studies conducted among Indonesian children aged 6–23 months. Rahmaniah *et al.* (2014), studying the same age range, similarly found that low energy and protein intake were not significant risk factors for stunting, while Limardi *et al.* (2022) reported that although total protein intake was significantly lower among stunted children in bivariate analysis in rural Sumba, this association was not retained after multivariate adjustment. A plausible explanation is that once minimum energy needs are approximately met, further variation in macronutrient adequacy contributes relatively little to explaining short-term differences in linear growth in this age group, which instead appears to be more strongly associated with micronutrient status. The linear associations between energy and nutrient adequacy levels and children's growth outcomes (HAZ) are summarized in Table 17.

Table 25. Correlation between energy and nutrient adequacy with HAZ

Variable	HAZ	
	p	r
Energy	0.568	0.045
Protein	0.820	0.018
Fat	0.606	-0.041
Carbohydrate	0.272	0.087
Calcium	0.057	0.151
Phosphorus	0.301	0.082
Iron	0.000*	0.284
Vitamin A	0.234	-0.095
Vitamin C	0.682	-0.033
Zinc	0.589	0.043

*significant based on Pearson Correlation Test (p<0.05)

Pearson correlation testing revealed that micronutrient intake—specifically iron—plays a prominent role in linear growth in this sample. Iron adequacy demonstrated a statistically significant positive correlation with HAZ (r = 0,284; p = 0,000). Calcium adequacy also displayed a positive trend toward significance (r = 0,151; p = 0,057).

In contrast, macronutrient adequacy levels—including energy (r = 0,045; p = 0,568), protein (r = 0,018; p = 0,820), fat (r = -0,041; p = 0,606), and carbohydrates (r = 0,087; p = 0,272)—were not significantly correlated with HAZ. Similarly, other micronutrient adequacy parameters,

such as phosphorus (r = 0,082; p = 0,301), vitamin A (r = -0,095; p = 0,234), vitamin C (r = -0,033; p = 0,682), and zinc (r = 0,043; p = 0,589), did not yield statistically significant relationships with HAZ.

The finding that iron adequacy was significantly and positively correlated with HAZ provides further evidence that iron plays an important role in children's linear growth, not only through its role in hemoglobin synthesis and hematological status, but also as a cofactor for enzymes involved in collagen synthesis and bone metabolism. This finding is consistent with the study by Sirajuddin *et al.* (2020) in Maros Baru District, which found a significant positive correlation between iron intake and height-for-age (HAZ) scores among children aged 12–36 months (p = 0.036), as well as the study by Jufri *et al.* (2023), which demonstrated that iron adequacy was one of the most significant predictors of stunting status among young children in coastal areas of Kendari City.

The positive association between calcium adequacy and HAZ observed in this study (r = 0,151; p = 0,057), although not statistically significant, is consistent with stronger evidence from other Indonesian populations. Jufri *et al.* (2023) found that calcium adequacy was significantly correlated with HAZ among children aged 48–59 months, while Solechah *et al.* (2026) reported a significant positive association between calcium intake and calcium adequacy and nutritional status based on the HAZ index among young children in Banjarbaru. Physiologically, calcium plays an important role in bone matrix formation and mineralization during growth. Therefore, inadequate calcium intake during the first 1,000 days of life may impair linear growth, although its effects may become more apparent in studies with larger sample sizes.

Overall, although most indicators of energy and nutrient adequacy in this study did not show statistically significant differences between the stunting and non-stunting groups, the observed trends for energy, calcium, and iron were consistent with broader epidemiological evidence from Indonesia. These findings reinforce that adequate nutrient intake remains an important determinant of children's linear growth, although its effects may not be statistically detectable in populations with limited sample sizes.

In contrast, macronutrient adequacy levels—including energy (r = 0,045; p = 0,568), protein (r = 0,018; p = 0,820), fat (r = -0,041; p = 0,606), and carbohydrates (r = 0,087; p = 0,272)—were not significantly correlated with HAZ. Similarly, other micronutrient adequacy parameters, such as phosphorus (r = 0,082; p = 0,301), vitamin A (r = -0,095; p = 0,234), vitamin C (r = -0,033; p = 0,682), and zinc (r = 0,043; p = 0,589), did not yield statistically significant relationships with HAZ.

h. Chrononutrition

While conventional infant and young child feeding indicators primarily assess the type, diversity, and frequency of food consumption, chrononutrition offers an additional perspective by considering the timing and temporal organization of eating relative to the daily sleep–wake cycle. In early childhood, feeding patterns are closely intertwined with behavioral and circadian rhythms, and the timing of food intake may provide additional information beyond the conventional

assessment of dietary adequacy. Therefore, to further characterize the feeding patterns of the children in this study, chrononutrition-related variables were assessed, including meal timing, eating window, duration of eating occasions, intervals between eating and sleep, and other temporal characteristics of food intake. The distribution of these chrononutrition characteristics among the study subjects is presented in Table 18.

Table 26. Distribution of chrononutrition among subjects

Variable	Total		Stunting		Non-Stunting		p
	n	%	n	%	n	%	
The largest meal time							0.560
Breakfast	66	45.80	20	44.40	46	46.50	
Lunch	53	36.80	19	42.20	34	34.30	
Dinner	25	17.40	6	13.30	19	19.20	
Equivalent across all meals	0	0.00	0	0.00	0	0.00	
Peak lactation time							0.000*
No peak	10	7.00	10	20.80	0	0.00	
01.00 to <05.00	2	1.40	0	0.00	2	2.10	
05.00 to <21.00	88	61.50	30	62.50	58	61.10	
21.00 to <01.00	43	30.10	8	16.70	35	36.80	
Breakfast time							0.237
< 6:00	4	2.50	0	0.00	4	3.60	
6:00 s/d 9:00	153	97.50	47	100.00	106	96.40	
Morning snack time							0.390
No morning snack consumption	29	18.10	7	14.50	22	19.60	
Prior to main breakfast	126	78.80	41	85.40	85	75.90	
30-180 minutes post-breakfast	5	3.10	0	0.00	5	4.50	
>180 minutes post-breakfast	0	0.00	0	0.00	0	0.00	
Lunch time							0.176
<12:00	21	14.00	3	6.40	18	17.50	
12:00 to 13:00	106	70.70	37	78.70	69	67.00	
>13:00	23	15.30	7	14.90	16	15.50	
Afternoon snack time							0.929
<13:00	8	6.50	3	7.30	5	6.00	
13:00-16:00	105	84.70	34	82.90	71	85.50	
>16:00	11	8.90	4	9.80	7	8.40	
Dinner time							0.000*
No dinner consumption	14	8.80	0	0.00	14	12.50	
< 17:00	54	33.80	32	66.70	22	19.60	
> 17:00	92	57.50	16	33.30	76	67.90	
Evening snack time							0.038*
No snack consumption	100	62.50	28	58.30	72	64.30	
< 19:00	42	26.20	18	37.50	24	21.40	
19:00 s/d 20:00	18	11.20	2	4.20	16	14.30	
> 20:00	0	0.00	0	0.00	0	0.00	

Table 27. Distribution of chrononutrition among subjects (continued)

Variable	Total		Stunting		Non-Stunting		p
	n	%	n	%	n	%	
Eating window							0.017*
<=10 hours	90	56.20	35	72.90	55	49.10	
> 10 to 14 hours	60	37.50	12	25.00	48	42.90	
> 14 hours	10	6.20	1	2.10	9	8.00	
Evening eating							0.006*
<=17:00	91	56.90	35	72.90	56	50.00	
>17:00 to 20:00	55	34.40	13	27.10	42	37.50	
>20:00	14	8.80	0	0.00	14	12.50	

*significant based on Chi-Square Test

As presented in Table 18, the majority of subjects reported breakfast as their largest meal of the day (45,80%), followed by lunch (36,80%) and dinner (17,40%). There was no statistically significant difference in the largest meal timing between the stunting and non-stunting groups (p=0,560). The absence of a significant difference in the timing of the largest meal between the stunting and non-stunting groups (p=0,560) may indicate that meal-size hierarchy alone is insufficient to distinguish children according to their growth status. Although the timing of food intake may influence peripheral circadian rhythms, the physiological consequences of meal timing are likely to depend on the overall temporal pattern of eating, including meal frequency and distribution across the day. In a systematic review and meta-analysis of children and adolescents, chrononutrition was described as encompassing meal regularity, frequency, and timing, with evidence suggesting that these dimensions may be relevant to metabolic health (Fiore *et al.*, 2024). In the present study, breakfast was the largest meal for 45,80% of participants, followed by lunch (36,80%) and dinner (17,40%), and this distribution did not differ significantly according to stunting status.

Regarding peak lactation timing, most subjects experienced their peak between 05:00 and <21:00 (61,50%). A statistically significant association was observed between peak lactation timing and stunting status (p = 0,000). Notably, 20,80% of subjects in the stunting group had no defined peak lactation timing, whereas all subjects (100,00%) in the non-stunting group exhibited a distinct peak time. A particularly notable finding was the significant association between peak lactation timing and stunting status (p < 0,001). Among children with stunting, 20,80% did not demonstrate a defined peak lactation timing, whereas all children in the non-stunting group exhibited a distinct peak. This finding should be interpreted cautiously because the presence or absence of a discernible breastfeeding peak may reflect the temporal regularity and organization of breastfeeding rather than an independent determinant of linear growth. Nevertheless, human milk has biological characteristics that support its consideration within the framework of chrononutrition. A systematic review identified circadian variation in several human milk components, including tryptophan, fats, triacylglycerol, cholesterol, iron, melatonin, cortisol, and cortisone, suggesting that milk may provide time-dependent biological signals to the infant (Italianer *et al.*, 2020).

The vast majority of subjects had breakfast between 06:00 and 09:00 (97,50%) and consumed a morning snack prior to their main breakfast (78,80%). Similarly, lunch was predominantly consumed between 12:00 and 13:00 (70,70%), and afternoon snacks were mostly taken between 13:00 and 16:00 (84,70%). None of these three timing variables showed a statistically significant association with stunting ($p > 0,05$).

The timing of breakfast, morning snacks, and lunch did not differ significantly between the two groups. Breakfast was consumed between 06:00 and 09:00 by 97,50% of participants, while lunch was predominantly consumed between 12:00 and 13:00 (70,70%). The relatively narrow distribution of these meal times may have reduced variability between groups and consequently limited the ability to detect an association with stunting. Moreover, the timing of these meals may represent relatively stable household feeding routines in this population. Importantly, feeding timing in children aged 6–23 months should be considered together with meal frequency, dietary diversity, dietary adequacy, and responsive feeding. The WHO complementary feeding guideline emphasizes that feeding during this period should provide adequate energy and nutrients through age-appropriate frequency, diversity, and responsive feeding practices (WHO, 2023).

In contrast, dinner timing differed significantly between groups ($p < 0,001$). A greater proportion of children with stunting consumed dinner before 17:00 (66,70%), whereas most non-stunted children consumed dinner after 17:00 (67,90%). Evening snack timing showed a similar pattern, with a higher proportion of children with stunting consuming an evening snack before 19:00 than non-stunted children (37,50% vs. 21,40%; $p = 0,038$). These findings indicate that the temporal distribution of food intake during the latter part of the day differed according to stunting status. However, an earlier dinner or evening snack should not automatically be interpreted as an adverse chrononutrition pattern. Rather, earlier cessation of food intake may reflect a shorter overall eating period, fewer opportunities for complementary food consumption, earlier bedtime, or differences in household feeding routines. In young children, these factors may also be influenced by breastfeeding patterns, appetite, caregiver practices, and socioeconomic circumstances.

Finally, both eating window duration and evening eating timing were significantly associated with stunting status ($p = 0,017$ and $p = 0,006$, respectively). The stunting group was dominated by subjects with an eating window of ≤ 10 hours (72,90%), compared to 49,10% in the non-stunting group. Correspondingly, 72,90% of stunted subjects concluded their evening eating by ≤ 17:00, whereas 12,50% of non-stunted subjects continued eating past 20:00.

The significant association between eating-window duration and stunting status ($p = 0,017$) further emphasizes the importance of considering the duration of daily food exposure. A substantially higher proportion of children with stunting had an eating window of ≤10 hours compared with non-stunted children (72,90% vs. 49,10%). A shorter eating window could potentially reduce opportunities for food intake, particularly in young children who have relatively high nutrient requirements in relation to their body size and limited gastric capacity. Nevertheless, eating-window duration should not be interpreted as equivalent to dietary adequacy. A short eating

window can still contain an adequate number of nutrient-dense meals and snacks, whereas a longer eating window does not necessarily ensure adequate nutrient intake.

The observed eating-window pattern is also relevant to the emerging pediatric chrononutrition literature. Townley *et al.* (2024), in a systematic review and meta-analysis of children and adolescents, reported substantial variation in daily eating windows, ranging from 9,7 to 16,4 hours, with a pooled mean of approximately 11,3 hours. Importantly, the authors concluded that further high-quality research is required to establish associations between eating-window duration and health outcomes in pediatric populations. Thus, the present finding that a shorter eating window was more prevalent among children with stunting contributes preliminary evidence regarding eating-window patterns in relation to early childhood growth, but it should not be interpreted as evidence that a specific eating-window duration causes or prevents stunting.

The significant association between evening eating timing and stunting status ($p = 0,006$) should likewise be interpreted within the broader temporal organization of feeding. In the present study, 72,90% of children with stunting had completed their evening eating by ≤17:00, whereas 12,50 of non-stunted children continued eating after 20:00. One possible explanation is that an earlier final eating occasion resulted in a shorter period during which complementary foods were offered. However, reverse causation is also possible: children experiencing growth faltering may have different appetites, feeding behaviors, sleep patterns, or caregiver feeding practices that subsequently influence meal timing. Therefore, the association between evening eating timing and stunting should not be interpreted independently of sleep-wake patterns, breastfeeding, feeding frequency, dietary intake, and household routines.

From a chronobiological perspective, the temporal organization of feeding may interact with circadian physiology through peripheral biological clocks and metabolic signaling. In breastfed infants, this temporal signaling may be complemented by circadian variation in human milk composition. Italianer *et al.* (2020) found circadian variation in multiple bioactive components of human milk, while Suwaydi *et al.* (2023) demonstrated 24-hour variation in several hormones, macronutrients, and milk volume. More recently, Aksu and Arslan (2026) summarized evidence that day and night milk differ in several neuroendocrine, nutritional, and immunological components, supporting the concept of human milk as a form of chrononutrition. These mechanisms provide biological plausibility for the potential importance of feeding timing during early life, although direct evidence connecting specific feeding-time patterns with linear growth remains insufficient.

Taken together, the present findings indicate that stunting was associated with a distinct temporal pattern of food intake, characterized particularly by earlier dinner timing, earlier evening snack timing, earlier cessation of evening eating, and a shorter eating window. The significant difference in peak lactation timing further suggests that the temporal organization of breastfeeding may also vary with growth status. However, these associations should be interpreted cautiously because chrononutrition variables are highly interrelated and may reflect broader feeding, sleep, behavioral, and socioeconomic patterns. In addition, the cross-sectional design prevents the determination of temporal sequence or causality. Longitudinal studies integrating meal timing,

breastfeeding patterns, dietary adequacy, sleep-wake rhythms, and circadian biomarkers are therefore needed to determine whether these temporal feeding characteristics precede and contribute to impaired linear growth or instead represent behavioral patterns associated with existing growth faltering.

The findings in Table 19 indicate that several dimensions of breastfeeding and eating behavior differed significantly between stunted and non-stunted children, particularly the duration of individual breastfeeding episodes, peak breastfeeding duration, the interval between breastfeeding and main meals, the duration of main meals, and the daily eating window. These findings suggest that feeding behavior in children aged 6–23 months may involve not only the frequency of feeding episodes but also the temporal characteristics and duration of feeding opportunities. This is relevant to the concept of chrononutrition, which considers the timing, frequency, and regularity of food intake as important dimensions of feeding behavior, although most existing chrononutrition research in children has focused on metabolic outcomes rather than linear growth (Fiore *et al.*, 2024).

Table 28. The mean of breastfeeding and eating duration, eating window, and evening latency

Variable	Total	Stunting	Non-stunting	p
Breastfeeding frequency (times/day)	7.34 ± 4.61	6.94 ± 5.32	7.52 ± 4.28	0.465
Breastfeeding duration per session (min)	14.20 ± 15.38	10.21 ± 9.56	15.99 ± 17.11	0.008*
Interval between breastfeeding and meal timing (min)	58.00 ± 66.28	41.32 ± 27.21	64.89 ± 75.90	0.010*
Peak breastfeeding duration (min)	39.40 ± 28.46	30.26 ± 16.26	43.16 ± 31.46	0.002*
Evening latency: interval between the last meal and nighttime sleep onset (min)	151.58 ± 89.68	166.88 ± 68.33	145.03 ± 96.95	0.159
Breakfast duration (min)	19.96 ± 13.22	15.73 ± 10.74	21.77 ± 13.79	0.004*
Morning snack duration (min)	9.22 ± 8.83	9.49 ± 9.44	9.10 ± 8.60	0.807
Lunch duration (min)	19.92 ± 13.18	15.94 ± 9.57	21.71 ± 14.19	0.004*
Afternoon snack duration (min)	8.04 ± 7.12	7.51 ± 5.98	8.28 ± 7.59	0.536
Dinner duration (min)	18.59 ± 12.61	13.94 ± 10.38	20.60 ± 12.99	0.001*
Evening snack duration (min)	4.44 ± 6.50	4.03 ± 5.72	4.65 ± 6.87	0.597
Eating window (min)	10.55 ± 2.09	10.01 ± 2.02	10.79 ± 2.09	0.030*
Wake to 1st eating interval (min)	108.38 ± 64.72	108.12 ± 58.62	108.49 ± 67.42	0.974

*significant based on Independent t-Test

The significantly longer breastfeeding duration per session among non-stunted children compared with stunted children (15,99 ± 17,11 vs. 10,21 ± 9,56 minutes; p = 0,008) may indicate differences in milk intake patterns between the two groups. Breastfeeding duration itself, however, should not be interpreted as a direct proxy for the amount of milk consumed, because milk transfer depends on factors such as infant sucking effectiveness, breast milk availability, feeding technique, and the stage of lactation. In addition, breastfeeding among children aged 6–23 months occurs alongside complementary feeding rather than serving as the sole source of nutrition. The 2023 WHO guideline emphasizes that breastfeeding should continue during the complementary-feeding

period, while complementary foods progressively become necessary to meet increasing energy and nutrient requirements. Therefore, the longer breastfeeding duration observed among non-stunted children may reflect a more sustained feeding episode or more effective feeding interaction rather than necessarily indicating greater total breast milk intake.

A similar pattern was observed for peak breastfeeding duration, which was significantly longer among non-stunted children (43,16 ± 31,46 vs. 30,26 ± 16,26 minutes; p = 0,002). This finding is particularly interesting in the context of the present chrononutrition analysis because the previous results also showed differences in the timing of peak breastfeeding between the two groups. Human milk is known to exhibit circadian variation in several components, including melatonin, cortisol, tryptophan, lipids, and other bioactive constituents (Italianer *et al.*, 2020). More recent evidence also demonstrates circadian variation in milk hormones and macronutrients, including leptin, adiponectin, insulin, glucose, fat concentration, and milk volume (Suwaydi *et al.*, 2023). However, evidence directly linking the duration or timing of individual breastfeeding episodes with linear growth or stunting remains limited. Therefore, the present finding should be considered hypothesis-generating, suggesting that both the timing and duration of breastfeeding may warrant further investigation in relation to growth during the complementary-feeding period.

The interval between breastfeeding and the subsequent main meal was also significantly longer among non-stunted children (64,89 ± 75,90 vs. 41,32 ± 27,21 minutes; p = 0,010). A shorter interval may indicate that breastfeeding and complementary feeding episodes occurred relatively close together, potentially influencing the child's appetite for the subsequent meal. Nevertheless, the direction of this relationship should be interpreted cautiously. Breastfeeding and complementary feeding are not necessarily competing behaviors, particularly in younger children, and WHO recommends continued breastfeeding together with age-appropriate complementary foods. The observed shorter interval among stunted children may instead reflect differences in appetite, feeding routines, caregiver responses to hunger cues, or the child's existing nutritional status. Thus, it would be inappropriate to conclude that a shorter breastfeeding-to-meal interval itself increases the risk of stunting.

An important finding was the significantly longer duration of the main meals among non-stunted children for breakfast, lunch, and dinner. Breakfast duration was 21,77 ± 13,79 minutes compared with 15,73 ± 10,74 minutes among stunted children (p = 0,004), lunch duration was 21,71 ± 14,19 versus 15,94 ± 9,57 minutes (p = 0,004), and dinner duration was 20,60 ± 12,99 versus 13,94 ± 10,38 minutes (p = 0,001). One possible interpretation is that a longer meal duration may provide greater opportunities for children to consume an adequate amount of complementary food, particularly when feeding is responsive to the child's appetite and developmental capacity. The 2023 WHO guideline recommends responsive feeding, in which caregivers recognize and respond appropriately to children's hunger and satiety cues. Evidence reviewed by WHO suggests that responsive-feeding interventions may improve dietary diversity, frequency of consumption of some healthy foods, and energy and nutrient intake, particularly in interventions aimed at preventing undernutrition.

The shorter meal durations among stunted children may therefore reflect differences in eating pace, appetite, food acceptance, caregiver–child interaction, or feeding difficulties. Importantly, meal duration should not be interpreted independently of the amount and nutritional quality of food consumed. A longer meal is not necessarily a better meal if it reflects prolonged feeding attempts, distraction, or coercive feeding. Conversely, a short meal may be nutritionally adequate if the child consumes an appropriate quantity and density of food. Consequently, the present results are better interpreted as evidence of differences in the temporal characteristics of feeding, rather than evidence that extending meal duration alone would prevent stunting. This interpretation is consistent with the broader evidence that feeding behavior is multidimensional and includes the interaction between caregiver behavior, child cues, feeding environment, and the foods offered (WHO, 2023).

The daily eating window was also significantly longer among non-stunted children (10,79 ± 2,09 vs. 10,01 ± 2,02 hours; p = 0,030). Although the absolute difference was relatively small, this finding is consistent with the concept that the temporal distribution of food intake may influence the number and spacing of opportunities for energy and nutrient consumption. A systematic review and meta-analysis by Townley *et al.* (2024) reported substantial variation in daily eating duration among children and adolescents, with a pooled mean eating duration of approximately 11,3 hours, while emphasizing that evidence linking eating-window duration to health outcomes in pediatric populations remains limited. Importantly, the concept of a shorter eating window in young children should not be interpreted in the same way as time-restricted eating in adults. For children aged 6–23 months, the primary nutritional concern is ensuring adequate energy and nutrient intake through age-appropriate meal frequency, dietary diversity, and responsive feeding. WHO recommends 2–3 complementary meals per day for breastfed children aged 6–8 months and 3–4 meals per day for those aged 9–23 months, with nutritious snacks when appropriate. Insufficient feeding frequency can compromise energy and micronutrient intake and contribute to growth faltering and stunting.

Interestingly, breastfeeding frequency and snack duration did not differ significantly between the two groups. This suggests that the distinction between stunted and non-stunted children may not simply be explained by the number of breastfeeding episodes or snack opportunities, but rather by how long feeding episodes lasted and how they were distributed across the day. Similarly, the absence of a significant difference in evening latency suggests that the interval between the last meal and nighttime sleep may not be the principal differentiating factor in this particular sample. These findings reinforce the multidimensional nature of feeding behavior: frequency, duration, timing, meal composition, and feeding responsiveness may interact, and no single temporal variable should be interpreted as an isolated determinant of growth.

Overall, the findings from Table 18 suggest that non-stunted children exhibited a more prolonged feeding pattern, characterized by longer breastfeeding episodes, longer peak breastfeeding duration, a longer interval between breastfeeding and main meals, longer main-meal durations, and a slightly wider daily eating window. These patterns may provide more sustained opportunities for breastfeeding and complementary food consumption during a period when

nutritional requirements increase rapidly. Nevertheless, because the study was cross-sectional, the observed differences cannot establish whether these feeding patterns preceded the development of stunting or were partly influenced by the child's existing nutritional status. For example, stunted children may have altered appetite, feeding behavior, or feeding tolerance as a consequence of underlying nutritional or health conditions. Therefore, these results should be interpreted as associations rather than causal relationships, and longitudinal studies are needed to determine whether temporal and duration-related feeding characteristics predict subsequent linear growth.

Table 20 presents the correlations between breastfeeding- and eating-related chrononutrition variables and height-for-age z-score (HAZ). Several feeding-time characteristics were significantly correlated with HAZ, whereas other variables showed no statistically significant association. Overall, significant correlations were observed for peak breastfeeding duration, evening latency, the duration of the three main meals, and the daily eating window. Most of these correlations were weak, indicating that feeding-related temporal characteristics may represent one component of the multifactorial determinants of linear growth rather than independent determinants of HAZ. This is consistent with the broader chrononutrition literature, which recognizes meal timing, frequency, and regularity as important dimensions of eating behavior, although evidence specifically linking chrononutrition to linear growth in young children remains limited (Fiore *et al.*, 2024).

Table 29. Correlation between eating duration, eating window, and evening latency with HAZ

Variable	HAZ	
	p	r
Breastfeeding frequency	0.541	0.049
Breastfeeding duration per session	0.068	0.147
Interval between breastfeeding and meal timing	0.519	0.057
Peak breastfeeding duration	0.010*	0.222
Evening latency: interval between the last meal and nighttime sleep onset	0.031*	-0.171
Breakfast duration	0.004*	0.225
Morning snack duration	0.718	-0.029
Lunch duration	0.002*	0.243
Afternoon snack duration	0.765	-0.024
Dinner duration	0.002*	0.239
Evening snack duration	0.854	-0.016
Eating window	0.023*	0.180
Wake to 1st eating interval	0.737	-0.027

*significant based on Pearson Correlation Test

Among breastfeeding-related variables, breastfeeding frequency was not significantly correlated with HAZ (r = 0,049; p = 0,541). The correlation coefficient was very close to zero, suggesting virtually no linear association between the number of breastfeeding episodes per day and HAZ in this population. Similarly, breastfeeding duration per session was not significantly correlated with HAZ (r = 0,147; p = 0,068). These findings suggest that frequency and duration of individual breastfeeding episodes, when considered separately, were not strongly associated with linear growth in this sample. This does not imply that breastfeeding is unimportant for growth.

Breastfeeding remains an important component of infant and young child feeding during 6–23 months, while complementary foods become increasingly important to meet the child's increasing energy and nutrient requirements. The WHO guideline recommends continued breastfeeding alongside adequate complementary feeding throughout this period.

The absence of a significant correlation between breastfeeding duration per session and HAZ may partly reflect the limitation of using duration as an indicator of milk intake. The amount of breast milk transferred during a feeding episode can vary according to sucking effectiveness, milk availability, feeding technique, and individual child characteristics. Therefore, a longer breastfeeding episode does not necessarily represent a proportionally greater nutrient intake. Likewise, the interval between breastfeeding and meal onset showed no significant correlation with HAZ ($r = 0{,}057$; $p = 0{,}519$). The very small correlation coefficient suggests little evidence of a linear relationship between this interval and linear growth. Thus, the temporal spacing between breastfeeding and complementary meals, considered independently, may be less important than the overall adequacy and distribution of feeding throughout the day.

In contrast, peak breastfeeding duration was significantly and positively correlated with HAZ ($r = 0{,}222$; $p = 0{,}010$), although the association was weak. This finding is particularly interesting from a chrononutrition perspective because human milk exhibits circadian variation in several bioactive components. A systematic review by Italianer *et al.* (2020) documented temporal variation in components including melatonin, cortisol, tryptophan, lipids, and other constituents of human milk. More recent research has also demonstrated circadian variation in milk hormones and macronutrients, including leptin, adiponectin, insulin, glucose, fat concentration, and milk volume. However, the present study measured breastfeeding duration during the peak period, rather than the circadian composition of breast milk. Therefore, the positive correlation observed here cannot establish that exposure to time-specific milk components promotes linear growth. Instead, it suggests that the temporal characteristics of breastfeeding may be relevant and warrant further investigation.

Another significant finding was the negative correlation between evening latency and HAZ ($r = -0{,}171$; $p = 0{,}031$). Thus, children with a longer interval between the last meal and nighttime sleep onset tended to have lower HAZ, although the association was weak. One possible interpretation is that a longer evening latency may reflect earlier cessation of food intake and consequently fewer opportunities for energy and nutrient consumption during the latter part of the day. This possibility is relevant in children aged 6–23 months because their nutritional requirements relative to body size are high, while their gastric capacity remains relatively small. WHO recommends age-appropriate complementary feeding frequency, increasing from 2–3 meals per day at 6–8 months to 3–4 meals per day at 9–23 months, with nutritious snacks when appropriate.

This finding should nevertheless not be interpreted as evidence that children should be encouraged to eat immediately before bedtime. Evening latency is likely to be influenced by dinner timing, bedtime, sleep duration, appetite, and household routines. Therefore, its negative correlation with HAZ may represent the broader organization of food intake across the day rather

than an independent effect of the pre-sleep interval. This interpretation is also consistent with the results presented in Table 17, in which earlier cessation of evening eating was more common among stunted children. Together, these findings suggest a potentially relevant relationship between the end of the daily eating period and linear growth, although longitudinal evidence is needed to clarify the direction of the association.

A consistent pattern was observed for the duration of the three main meals. Breakfast duration was positively correlated with HAZ ($r = 0,225$; $p = 0,004$), as were lunch duration ($r = 0,243$; $p = 0,002$) and dinner duration ($r = 0,239$; $p = 0,002$). Although all three correlations were weak, their consistency across breakfast, lunch, and dinner is noteworthy. Longer meal episodes may provide greater opportunity for children to consume complementary foods, particularly during a developmental period when children are still acquiring oral-motor skills and adapting to different food textures and consistencies. The WHO complementary-feeding guideline emphasizes responsive feeding, whereby caregivers respond appropriately to children's hunger and satiety cues and provide developmentally appropriate support during meals.

However, meal duration should not be interpreted as a direct proxy for food consumption. A longer meal may provide greater opportunity for adequate intake, but it may also reflect slow eating, food refusal, distraction, or prolonged caregiver attempts to feed the child. Conversely, a relatively short meal may still provide adequate nutrition when the food is nutrient-dense and the child consumes an adequate portion. Therefore, the positive correlations observed in this study are better interpreted as associations between meal-time characteristics and HAZ, rather than evidence that prolonging meal duration would improve linear growth. Importantly, these findings are consistent with Table 18, where non-stunted children also demonstrated significantly longer breakfast, lunch, and dinner durations. The convergence of the group-comparison and correlation analyses strengthens the possibility that meal duration represents a relevant characteristic of feeding behavior in this population.

The daily eating window was also significantly and positively correlated with HAZ ($r = 0,180$; $p = 0,023$), although the correlation was weak. Children with a wider daily eating window tended to have higher HAZ. A wider eating window may provide more opportunities to distribute food intake across the day, which could be relevant for young children who have high nutritional requirements but relatively limited capacity to consume large amounts at a single meal. WHO recommendations emphasize adequate meal frequency and dietary diversity during complementary feeding, rather than prolonged fasting periods.

This finding is also consistent with Table 18, where the eating window was significantly wider among non-stunted children. Nevertheless, the concept of an eating window in young children should be distinguished from time-restricted eating in adults. Townley *et al.* (2024), in a systematic review and meta-analysis, reported substantial variation in daily eating duration among children and adolescents and emphasized that evidence regarding the health implications of eating-window duration in pediatric populations remains limited. Thus, the present finding should not be interpreted as suggesting that a longer eating window is inherently beneficial. Rather, in children

aged 6–23 months, a wider eating window may indicate more opportunities for age-appropriate energy and nutrient intake.

In contrast, morning, afternoon, and evening snack durations were not significantly correlated with HAZ, with correlation coefficients close to zero (r = −0,029, −0,024, and −0,016, respectively; all p > 0,05). This may indicate that the duration of individual snack episodes is less relevant to linear growth than the nutritional quality, portion size, frequency, and overall contribution of snacks to daily nutrient intake. Similarly, the wake-to-first-eating interval was not significantly correlated with HAZ (r = −0,027; p = 0,737). Thus, within the range observed in this population, the time elapsed between waking and the first eating episode did not appear to be related to linear growth. Although first-meal timing is an important chrononutrition dimension, its relevance may depend on the broader feeding pattern across the entire day rather than on this interval alone.

Taken together, the findings suggest that HAZ was more consistently associated with the duration and temporal distribution of feeding episodes than with breastfeeding frequency, snack duration, or the wake-to-first-eating interval. Specifically, longer peak breastfeeding duration, longer main-meal durations, and a wider eating window were associated with higher HAZ, whereas longer evening latency was associated with lower HAZ. These findings complement the between-group differences observed in Table 18 and the meal-timing patterns in Table 17, suggesting that the temporal organization of feeding may be an important dimension of feeding behavior in relation to growth status.

Nevertheless, the magnitude of most correlations was weak, and the cross-sectional design prevents conclusions regarding causality or temporal direction. It is possible that existing growth faltering influences appetite, feeding duration, meal behavior, or caregiver feeding practices, rather than feeding behavior being the sole determinant of growth. Therefore, these findings should be considered hypothesis-generating. Longitudinal studies are needed to determine whether feeding duration, eating-window characteristics, and evening feeding patterns predict subsequent changes in HAZ after accounting for dietary intake, nutrient adequacy, illness, socioeconomic conditions, sleep, and other established determinants of child growth.

Table 21 presents the distribution of sleep timing and sleep duration among children with and without stunting. Nighttime sleep time did not differ significantly between the stunting and non-stunting groups (p = 0,247). Most children in both groups initiated nighttime sleep between 19:00 and 21:00, accounting for 87,4% of children with stunting and 80,5% of non-stunted children. Similarly, the timing of the first nap did not differ significantly between the two groups (p = 0,644), with most children having their first nap between 08:00 and 10:00 (64,6% in the stunting group and 70,3% in the non-stunting group).

Table 30. Distribution of sleeping time and duration

Variabel	Total		Stunting		Non-stunting		p
	n	%	n	%	n	%	
Nighttime sleep							0.247
<19:00	12	7.40	3	6.30	9	8.04	
19:00 - 21:00	132	82.60	42	87.40	90	80.50	
>21.00	16	9.90	3	6.30	13	11.60	
1st nap time							0.644
<8.00	13	8.20	3	6.20	10	9.00	
8.00 - 10.00	109	68.60	31	64.60	78	70.30	
> 10.00 - 12.00	25	15.70	10	20.80	15	13.50	
>12.00	12	7.50	4	8.30	8	7.20	
2nd nap time							0.717
No 2nd nap	32	20.00	11	22.90	21	18.80	
<=15:00	120	75.00	34	70.80	86	76.80	
> 15:00	8	5.00	3	6.20	5	4.50	
3rd nap time							0.650
No 3rd nap	4	7.50	1	6.20	3	8.10	
<=16:00	49	92.50	15	93.80	34	91.90	
>16:00	0	0.00	0	0.00	0	0.00	
Wake-up nighttime sleep							0.093
<7:00	136	85.00	44	91.70	92	82.10	
≥7:00	24	15.00	4	8.30	20	17.90	
Total sleep duration							0.035*
<11 jam	38	23.80	5	10.40	33	29.50	
11-14 jam	108	67.50	38	79.20	70	62.50	
>14 jam	14	8.80	5	10.40	9	8.00	

*significant based on Chi-Square Test

The timing of the second nap also showed no significant difference between the groups (p = 0,717). A second nap occurring at or before 15:00 was the most common pattern in both groups, observed in 70,8% of children with stunting and 76,8% of non-stunted children, while 22,9% and 18,8%, respectively, did not have a second nap. Likewise, no significant difference was observed in third nap timing (p = 0,650). Among children with available third-nap data, most had their third nap at or before 16:00, with similar proportions in the stunting (93,8%) and non-stunting (91,9%) groups.

The timing of nighttime wake-up also did not differ significantly between the stunting and non-stunting groups (p = 0,093). Most children woke before 07:00, comprising 91,7% of the stunting group and 82,1% of the non-stunting group. In contrast, total sleep duration differed significantly between the two groups (p = 0,035). Among children with stunting, 79,2% had a total sleep duration of 11–14 hours, compared with 62,5% of non-stunted children. A total sleep duration of less than 11 hours was observed in 10,4% of children with stunting and 29,5% of non-

stunted children, while sleep duration exceeding 14 hours was observed in 10,4% and 8,0% of the respective groups.

Table 31. The mean of sleeping duration

Variable	Total	Stunting	Non-stunting	p
1st nap (min)	80.49 ± 45.62	100.94 ± 49.87	71.72 ± 40.87	0.000*
2nd nap (min)	59.56 ± 45.13	70.21 ± 52.61	55.00 ± 40.94	0.050*
3rd nap (min)	17.72 ± 31.56	13.65 ± 26.05	19.46 ± 33.61	0.287
Night sleep (min)	551.44 ± 85.18	565.00 ± 60.74	545.63 ± 93.36	0.188
Total nap (min)	157.77 ± 73.60	184.79 ± 75.37	146.19 ± 70.03	0.002*
Total sleep (min)	709.21 ± 107.05	749.79 ± 83.47	691.81 ± 111.57	0.001*

*significant based on Independent t-Test

The absence of significant differences in nighttime sleep onset, nap timing, and nighttime wake-up suggests that sleep timing was relatively similar between children with and without stunting in this population. The predominance of nighttime sleep initiation between 19:00 and 21:00 and waking before 07:00 in both groups indicates a relatively consistent sleep–wake schedule. From a chronobiological perspective, regular sleep and wake timing contribute to the organization of the daily circadian rhythm, although sleep timing should be considered together with sleep duration and sleep quality. The WHO similarly emphasizes regular sleep and wake-up times as part of healthy sleep practices in young children (WHO, 2019).

The lack of differences in nap timing may also indicate that daytime sleep was relatively similarly structured in both groups. In infants and toddlers, naps contribute substantially to total 24-hour sleep, and the balance between nighttime and daytime sleep changes with age. Consequently, examining individual nap timing separately may not fully capture the child's overall sleep exposure. The WHO recommends evaluating sleep over a 24-hour period, including naps, rather than considering nighttime sleep alone (WHO, 2019).

In contrast, total sleep duration differed significantly between the two groups (p = 0,035). Interestingly, the direction of this difference does not support a simple interpretation that shorter sleep was associated with stunting in the present population. A greater proportion of stunted children had a total sleep duration of 11–14 hours (79,2%), whereas sleep duration of less than 11 hours was more common among non-stunted children (29,5%). Therefore, the significant p-value should be interpreted as indicating a difference in the distribution of sleep-duration categories rather than evidence that longer sleep is associated with stunting. This interpretation is particularly important because recommended sleep duration varies according to age during the 6–23-month period (WHO, 2019).

According to WHO recommendations, infants aged 4–11 months should obtain 12–16 hours of good-quality sleep per 24 hours, including naps, whereas children aged 12–23 months should obtain 11–14 hours. Thus, the 11–14-hour category used in the present study represents the recommended range for children aged 12–23 months but does not fully correspond to the recommended range for infants aged 6–11 months, for whom 12–16 hours is recommended. This age dependency should be considered when interpreting the significant difference in sleep-duration

categories in the present study. In particular, applying a single cut-off of <11, 11–14, and >14 hours across the entire 6–23-month age range may mask age-specific differences in sleep adequacy (WHO, 2019).

The biological plausibility of a relationship between sleep and linear growth is supported by the role of sleep in growth-related endocrine processes. Growth hormone secretion is closely linked to sleep and predominantly occurs during deep sleep. However, the relationship between sleep and growth hormone is complex, and evidence in children remains limited and heterogeneous. A recent review concluded that the available evidence is insufficient to establish a simple relationship between particular sleep characteristics and growth-related endocrine function in children (Zaffanello *et al.*, 2024).

Previous epidemiological evidence has also suggested a possible relationship between sleep duration and physical growth during early childhood. A systematic review by Chaput *et al.* (2017), which included children aged 0–4 years, reported that shorter sleep duration was associated with impaired growth in the available studies. However, the authors emphasized important limitations in the evidence, including heterogeneity in study designs and sleep measurements, and rated the quality of evidence differently across outcomes. Therefore, although adequate sleep may contribute to healthy growth, the existing literature does not justify assuming a uniform linear relationship between sleep duration and linear growth across all populations (Chaput *et al.*, 2017).

More recent evidence further supports a cautious interpretation. A systematic review of sleep consolidation and growth and development among infants and toddlers under 3 years found no significant association between sleep consolidation and physical growth indicators such as BMI or weight gain. The authors concluded that the evidence was heterogeneous and insufficient to establish sleep consolidation as a strong predictor of growth and development. Although sleep consolidation is conceptually different from total sleep duration, this finding emphasizes that individual sleep dimensions may not have uniform relationships with physical growth (Bemanalizadeh *et al.*, 2024).

The unexpected pattern observed in the present study may therefore reflect the complexity of the relationship between sleep and growth. Total sleep duration does not capture sleep quality, sleep continuity, nighttime awakenings, or sleep consolidation. Two children with the same total sleep duration may have substantially different sleep patterns, particularly if one experiences frequent nighttime awakenings or fragmented sleep. Consequently, total sleep duration alone may not adequately represent the sleep characteristics that are biologically relevant to growth (Bemanalizadeh *et al.*, 2024; Zaffanello *et al.*, 2024).

Furthermore, sleep in children aged 6–23 months occurs within an integrated 24-hour behavioral pattern that includes feeding, breastfeeding, physical activity, and sedentary behavior. Changes in nighttime sleep may be partially compensated for by daytime naps, while feeding schedules and caregiver routines may also influence sleep timing and duration. WHO therefore recommends considering sleep together with physical activity and sedentary behavior within the overall 24-hour pattern rather than interpreting sleep as an isolated (WHO, 2019).

The discrepancy between the present findings and the expectation that insufficient sleep may be related to poorer growth may also reflect the cross-sectional nature of the study. It is not possible to determine whether differences in sleep duration preceded differences in growth or whether children's sleep patterns were influenced by other factors related to their nutritional or health status. Moreover, parental reporting of sleep duration and timing may introduce measurement error, particularly for nighttime awakenings and nap duration. These considerations are important when interpreting the significant difference in total sleep duration observed between the two groups (Chaput *et al.*, 2017; Zaffanello *et al.*, 2024).

Overall, the findings indicate that sleep timing—including nighttime sleep onset, first, second, and third nap timing, and nighttime wake-up—was relatively similar between stunted and non-stunted children, whereas total sleep duration showed a significant difference in its distribution. Importantly, the direction of this difference does not support the interpretation that shorter sleep was associated with stunting in this population. Instead, the findings suggest that sleep-duration patterns differed between the two groups, while the specific relationship between sleep duration and linear growth remains uncertain. Future longitudinal studies should consider age-specific sleep recommendations and assess not only total sleep duration but also sleep quality, consolidation, regularity, and nighttime awakenings to better understand the relationship between sleep and linear growth in children aged 6–23 months (WHO, 2019; Bemanalizadeh *et al.*, 2024).

Table 23 presents the correlations between sleep duration and HAZ. The duration of the first nap was significantly and negatively correlated with HAZ (r = −0,241, p = 0,002), indicating a weak negative correlation, whereby longer first-nap duration was associated with lower HAZ. Similarly, total nap duration was significantly negatively correlated with HAZ (r = −0,183, p = 0,021), also indicating a weak negative correlation. Total sleep duration was likewise significantly and negatively correlated with HAZ (r = −0,226, p = 0,004), suggesting that children with longer total sleep duration tended to have lower HAZ. In contrast, the duration of the second nap (r = −0,126, p = 0,112), third nap (r = 0,061, p = 0,446), and nighttime sleep (r = −0,077, p = 0,334) were not significantly correlated with HAZ. Thus, significant associations were observed primarily for first-nap duration, total nap duration, and total sleep duration, with all significant correlations showing a negative direction.

Table 32. Correlation between sleeping duration and HAZ

Variable	HAZ	
	p	**r**
1st nap	0.002*	-0.241
2nd nap	0.112	-0.126
3rd nap	0.446	0.061
Night sleep	0.334	-0.077
Total nap	0.021*	-0.183
Total sleep	0.004*	-0.226

*significant based on Spearman Correlation Test

The negative correlations observed in the present study are noteworthy because they differ from the direction generally expected from the literature on sleep and child growth. A systematic

review of sleep duration and health indicators among children aged 0–4 years reported that shorter sleep duration was associated with impaired growth in the available studies, although the evidence was based on a limited number of studies and was heterogeneous in design and measurement. Thus, the negative association observed in the present study should not be interpreted as evidence that longer sleep impairs linear growth. Rather, it suggests that the relationship between sleep duration and growth may be more complex in this population and may not follow a simple linear pattern (Chaput *et al.*, 2017).

One possible explanation for the inverse association is that longer sleep may reflect a response to the child's underlying health or nutritional condition rather than a cause of poorer growth. Children experiencing illness, nutritional deficits, or reduced energy and activity may exhibit alterations in sleep behavior, including increased sleep duration. In a cross-sectional study, such a pattern can produce an inverse association because sleep duration and growth status are measured at the same time. Therefore, the observed correlation cannot establish whether longer sleep preceded lower HAZ or whether differences in growth or health status contributed to longer sleep. This possibility is consistent with the broader literature emphasizing the complexity and heterogeneity of the relationship between sleep and growth in young children (Zaffanello *et al.*, 2024).

The negative correlation between first-nap duration and HAZ may also indicate that daytime sleep is compensatory for differences in nighttime sleep or overall sleep regulation. In infants and toddlers, sleep is distributed across nighttime sleep and multiple daytime naps, and changes in one component may be accompanied by changes in another. Therefore, a longer first nap does not necessarily represent greater overall sleep need or better sleep quality. It may instead reflect a shift in the distribution of sleep across the 24-hour period. The WHO consequently recommends assessing sleep as total sleep over 24 hours, including naps, while also emphasizing good-quality sleep and regular sleep and wake times (WHO, 2019).

The significant negative correlation between total nap duration and HAZ is consistent with the finding for first-nap duration, suggesting that the inverse association was partly driven by daytime sleep rather than nighttime sleep. Interestingly, nighttime sleep duration itself was not significantly correlated with HAZ ($r = -0{,}077$, $p = 0{,}334$). This pattern may indicate that the association observed for total sleep duration was not attributable to nighttime sleep alone but was partly related to the amount of daytime sleep. However, because the correlations were weak, the contribution of nap duration to variation in HAZ should not be overstated. The literature on sleep consolidation also suggests that different dimensions of sleep may have different relationships with child growth, and a 2024 systematic review found no consistent association between sleep consolidation and physical growth indicators in children aged 0–3 years (Bemanalizadeh *et al.*, 2024).

Another important consideration is the age range of the participants. Children aged 6–23 months undergo substantial developmental changes in sleep organization, including changes in the number and duration of naps and the progressive consolidation of nighttime sleep. Consequently, the same nap duration may have different meanings for a younger infant compared with an older

toddler. WHO recommends 12–16 hours of total sleep per 24 hours for children aged 4–11 months and 11–14 hours for children aged 12–23 months, including naps. Therefore, age-specific developmental differences should be considered when interpreting the negative association between sleep duration and HAZ in a sample spanning 6–23 months (WHO, 2019).

The biological relationship between sleep and growth also provides an important context for interpreting these findings. Sleep is closely linked to growth hormone secretion, particularly during deep sleep, and growth hormone plays an important role in linear growth. However, the relationship between sleep and growth hormone secretion is not determined solely by total sleep duration. A recent review of pediatric evidence emphasized that studies examining the relationship between sleep and growth hormone are limited and heterogeneous, with inconsistent findings across populations. Thus, a longer observed sleep duration does not necessarily imply greater growth-promoting endocrine activity, particularly when sleep quality, sleep architecture, and underlying health conditions are not assessed (Zaffanello *et al.*, 2024).

It is also important to distinguish between sleep duration and sleep quality or consolidation. Two children may have the same total sleep duration but substantially different sleep patterns, including differences in nighttime awakenings, sleep continuity, and distribution of naps. Conversely, a child with a longer total sleep duration may have fragmented or irregular sleep. A systematic review of sleep consolidation among children aged 0–3 years found heterogeneous evidence and no consistent association between sleep consolidation and physical growth indicators such as BMI or weight gain. These findings support the interpretation that total sleep duration alone may not adequately capture the aspects of sleep most relevant to physical growth (Bemanalizadeh *et al.*, 2024).

The findings should also be interpreted within the broader 24-hour behavioral context. In children aged 6–23 months, sleep interacts with feeding, breastfeeding, physical activity, and daily routines. Longer daytime sleep may potentially alter the timing of feeding opportunities or other daytime activities, although such mechanisms cannot be established from the present cross-sectional data. This consideration is particularly relevant in the present study because feeding and chrononutrition characteristics were also associated with HAZ. WHO emphasizes that healthy development in early childhood depends on the integrated pattern of sleep, physical activity, sedentary behavior, and other daily behaviors rather than on sleep duration considered in isolation (WHO, 2019).

The negative association between total sleep duration and HAZ should therefore be interpreted cautiously. The magnitude of the correlations was weak (r = 0,183–0,241), indicating that sleep duration explained only a small proportion of the variation in HAZ. Moreover, HAZ is influenced by multiple nutritional, infectious, socioeconomic, environmental, and caregiving factors. For children aged 6–23 months, the period of complementary feeding is particularly important because it coincides with a high risk of growth faltering and nutrient deficiencies, and adequate complementary feeding is required to meet increasing nutritional requirements alongside continued breastfeeding (WHO, 2023).

Overall, the present findings indicate that longer first-nap duration, total nap duration, and total sleep duration were weakly associated with lower HAZ, whereas second-nap, third-nap, and nighttime sleep duration were not significantly associated with HAZ. These findings do not support the interpretation that longer sleep causes poorer linear growth. Rather, they suggest that daytime sleep and total sleep duration may reflect complex differences in sleep regulation, health status, or daily behavioral patterns among children in this population. Given the cross-sectional design and the weak magnitude of the correlations, the findings should be considered hypothesis-generating. Longitudinal studies that distinguish age-specific sleep requirements and assess sleep quality, consolidation, nighttime awakenings, and other 24-hour behaviors would be valuable for clarifying the temporal and biological relationship between sleep characteristics and linear growth in children aged 6–23 months (Chaput *et al.*, 2017; Bemanalizadeh *et al.*, 2024; Zaffanello *et al.*, 2024).

CONCLUSIONS AND RECOMMENDATIONS

a. Conclusions

Significant differences between stunted and non-stunted children were observed in sex, age, birth length, and age gap with the nearest older sibling. Stunted children had a higher proportion of boys, were older, had shorter birth length, and showed a different distribution of age gap with the nearest older sibling (≤5 years) compared with non-stunted children. Most fathers and mothers had completed senior high school education, with fathers predominantly employed as laborers and mothers primarily being housewives. Per capita household income tended to be lower among families of stunted children, although the difference was not statistically significant, and household expenditure across all assessed components did not differ significantly between groups. Nevertheless, expenditure on dairy products tended to be higher among families of non-stunted children, providing an initial indication of differences in household circumstances that may be relevant to children's nutritional status.

Against this socioeconomic and demographic background, the prevalence of stunting in the present study was 30.0. The prevalence of underweight, wasting, chronic energy deficiency, overweight, and obesity was 15.0, 3.8, 2.5, 1.2, and 2.5, respectively. Stunted children had a significantly higher risk of being underweight than their non-stunted counterparts, indicating that stunting in this population frequently co-occurred with other manifestations of undernutrition. More than half of the respondents (53.1) experienced household food insecurity; however, the severity of food insecurity did not differ significantly between households with stunted and non-stunted children. Thus, differences in child nutritional status were not accompanied by statistically significant differences in household food security in this study.

Child health conditions provided another dimension of the differences observed between the two groups. Morbidity was relatively common, with acute respiratory infection (ARI) being the most frequently reported condition, while stunted children had significantly higher frequencies of dysentery and toothache. Moreover, higher frequencies of ARI, urinary tract infection (UTI), and toothache were significantly associated with lower HAZ, indicating that children experiencing these conditions more frequently tended to have lower linear-growth status. In contrast, more frequent dermatological disorders were associated with higher HAZ. Regarding illness duration, longer duration of dermatological disorders was associated with lower HAZ, whereas longer duration of varicella was associated with higher HAZ. Despite these specific associations, cumulative illness duration did not differ significantly between stunted and non-stunted children, suggesting that the relationship between morbidity and linear growth may vary according to the type and characteristics of illness rather than cumulative morbidity alone.

In addition to health conditions, infant and young child feeding practices showed several areas of suboptimal practice, particularly low early initiation of breastfeeding, exclusive breastfeeding, and minimum milk feeding frequency. Nevertheless, most breastfeeding and complementary feeding indicators did not differ significantly according to stunting status. Mixed milk feeding was the only breastfeeding-related practice that differed significantly between

groups, occurring less frequently among stunted children. Although some differences in dietary patterns were observed, including slightly higher sugar-sweetened beverage consumption among stunted children, none of the complementary feeding indicators or individual food-group consumption was significantly associated with stunting status. These findings indicate that conventional indicators of feeding practice alone may not fully capture the nutritional differences related to linear growth in this population.

This pattern was also reflected in the assessment of dietary adequacy. No statistically significant differences were observed in energy or nutrient adequacy or in mean nutrient intakes between stunted and non-stunted children. However, inadequate energy, calcium, iron, and vitamin C intake tended to be more prevalent among stunted children. Notably, iron adequacy was significantly and positively correlated with HAZ, indicating that greater iron adequacy was associated with better linear growth. Calcium adequacy also showed a positive but non-significant correlation with HAZ, whereas energy and macronutrient adequacy were not significantly related to HAZ. Thus, although overall dietary adequacy did not clearly distinguish the two groups, the findings highlight iron adequacy as a potentially relevant nutritional dimension of linear growth in this population.

Beyond the quantity and adequacy of food intake, the temporal organization of feeding provided additional differences between stunted and non-stunted children. In terms of chrononutrition, stunted children more frequently had earlier dinner and evening eating, a shorter eating window, shorter breastfeeding and main-meal durations, and shorter peak breastfeeding duration than non-stunted children. Several of these temporal characteristics were also significantly correlated with HAZ: longer peak breastfeeding duration, longer breakfast, lunch, and dinner durations, and a wider eating window were associated with higher HAZ, whereas longer evening latency was weakly associated with lower HAZ. In contrast, most meal and snack timing variables, breastfeeding frequency, and the interval between waking and first eating were not significantly associated with HAZ. Collectively, these findings suggest that the temporal pattern and duration of feeding may provide additional information about the relationship between feeding behavior and linear growth beyond conventional measures of feeding frequency and dietary adequacy.

Sleep characteristics further complemented this temporal perspective. Sleep timing was generally similar between stunted and non-stunted children; however, daytime nap duration and total sleep duration differed between groups. Longer first-nap duration, total nap duration, and total sleep duration were weakly and negatively correlated with HAZ, whereas nighttime sleep duration was not significantly associated with HAZ. Taken together with the feeding-related findings, these results suggest that the relationship between child growth and daily temporal organization is multidimensional, involving feeding duration and timing as well as sleep patterns, rather than being explained by a single behavioral characteristic.

b. Recommendation

Nutrition education and child-growth monitoring should continue to emphasize adequate and diverse diets, with particular attention to iron-rich foods and other micronutrient sources that may support optimal linear growth. Feeding counseling should also encourage appropriate breastfeeding and complementary feeding practices while considering the quality, duration, and timing of feeding rather than focusing solely on feeding frequency or dietary adequacy.

Child nutrition programs should consider incorporating temporal aspects of feeding and sleep into routine counseling and assessment. Parents and caregivers may benefit from guidance on establishing consistent daily routines for breastfeeding, meals, snacks, and sleep that are appropriate for the child's developmental stage. However, because the observed associations were cross-sectional and generally weak, specific recommendations regarding meal timing, eating window duration, or sleep duration should be applied with caution and should not be interpreted as established causal determinants of stunting.

Routine growth monitoring may be strengthened by integrating information on morbidity, dietary intake, feeding practices, and household socioeconomic conditions. Children with recurrent illnesses, particularly respiratory infections, urinary tract infections, or other conditions associated with lower HAZ in this study, may require closer monitoring of nutrition and growth. Early identification of growth faltering could facilitate timely nutrition counseling and appropriate referral for further assessment.

Future research should use longitudinal or prospective designs to clarify the temporal and potentially bidirectional relationships between feeding patterns, sleep, morbidity, dietary adequacy, and linear growth. Intervention studies are particularly needed to determine whether modifications to feeding and sleep routines, alongside improvements in dietary quality and micronutrient adequacy, can improve growth outcomes. Future studies should also account for potential confounding factors, including age, sex, socioeconomic status, morbidity, and overall dietary intake, when examining chrononutrition-related determinants of child growth.

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APPENDICES

a. Ethical Clearance Letter

ETHICS COMMITTEE APPROVAL

Ref. No. 001/KE/04/2026

Title of the Research Protocol

"HOUSEHOLD FOOD SECURITY, FEEDING PRACTICES, AND CHRONONUTRITION BETWEEN STUNTED AND NON-STUNTED CHILDREN AGED 6-23 MONTHS"

Principal Investigator:
Prof. Dr. Ir. Ali Khomsan, MS
Participating Investigator(s):
Prof. Dr. Ir. Hadi Riyadi, MS
Desiani Rizki Purwaningtyas, S.Gz., M.Si
As-Syaffa Amalia Adha, S.Gz., M.Sc
Date of Approval:
April 2, 2026

The Health Research Ethics Committee (HREC) states that the document above meets the ethical principal outlined in the International and National Guidelines on ethical standards and procedures for research with human being.

The Health Research Ethics Committee (HREC) has the right to monitor the research activities at anytime.

Kartika Nugraheni, S.Gz, M.Gizi, Ph.D
Chairperson

b. Questionnaire

Kode responden: | | | | |

"KETAHANAN PANGAN RUMAH TANGGA, PRAKTIK PEMBERIAN MAKAN, DAN CHRONONUTRITION PADA BALITA STUNTING DAN NON-STUNTING USIA 6-23 BULAN"

PENJELASAN SEBELUM PERSETUJUAN

Pendahuluan

Selamat pagi/siang, perkenalkan kami tim peneliti Departemen Gizi Masyarakat Fakultas Kedokteran dan Gizi IPB yang diketuai oleh Prof. Dr. Ir. Ali Khomsan, MS. Dalam rangka pengembangan ilmu pengetahuan Bidang Gizi Masyarakat dan membantu mengatasi masalah stunting, kami melakukan penelitian mengkaji ketahanan pangan rumah tangga, praktik pemberian makan, dan chrononutrition pada baduta usia 6-23 bulan.

Sebelum Ibu ikut serta dalam kegiatan ini, kami akan menjelaskan mengenai tujuan, apa saja yang akan dilakukan, manfaat, dan hal penting lainnya. Semua informasi dan data pribadi peserta akan dirahasiakan. Data yang dikumpulkan hanya digunakan untuk keperluan penelitian dan tidak akan disebarluaskan dalam bentuk yang dapat mengungkap identitas peserta. Setelah mendapatkan penjelasan ini, dipersilahkan untuk memutuskan keikutsertaan dalam pengisian kuesioner ini. Keikutsertaan dalam penelitian ini bersifat sukarela. Penjelasan ini dibuat untuk memastikan bahwa semua peserta memahami sepenuhnya sebelum menyatakan kesediaannya. Jika ada hal-hal yang belum jelas, silakan bertanya kepada petugas yang memberikan penjelasan atau kepada tim peneliti: Desiani Rizki P. (081212276590)

Tujuan dan Manfaat Penelitian

Penelitian ini bertujuan untuk menganalisis faktor-faktor yang diduga berkaitan dengan stunting seperti ketahanan pangan, praktik pemberian makan, dan waktu atau kronologis pemberian makan (*chrononutrition*) pada baduta. Hasil penelitian ini diharapkan dapat memberikan informasi ilmiah untuk pengembangan intervensi gizi yang lebih efektif, khususnya dalam upaya pencegahan stunting melalui pendekatan pola makan dan waktu makan yang tepat.

Prosedur Penelitian

Apabila Anda bersedia berpartisipasi, Anda akan diminta untuk:

- Menjawab pertanyaan terkait karakteristik anak dan rumah tangga, ketahanan pangan rumah tangga, konsumsi pangan, praktik pemberian makan anak termasuk ASI, riwayat menyusui, serta riwayat imunisasi
- Dilakukan pengukuran antropometri anak yang meliputi panjang/tinggi badan, berat badan, dan lingkar lengan atas (LILA)
- Mengisi kuesioner **food record** yang akan dibawa pulang untuk mencatat seluruh makanan dan minuman yang dikonsumsi anak selama periode pengamatan (misalnya 1x24 jam atau sesuai desain penelitian). Pada pengisian food record, Anda diminta mencatat secara **rinci**, meliputi: waktu makan/minum (jam), jenis makanan/minuman yang dikonsumsi, jumlah/ukuran porsi, pemberian ASI (waktu dan durasi menyusui). Anda juga diminta untuk **memfotokan makanan dan minuman anak** setiap kali sebelum dikonsumsi (dan bila memungkinkan setelah makan jika terdapat sisa) kemudian mengirimkan foto tersebut kepada enumerator sebagai bahan verifikasi data konsumsi

Seluruh rangkaian kegiatan penelitian ini diperkirakan memerlukan waktu sekitar 30–45 menit untuk wawancara dan pengukuran, serta tambahan waktu untuk pengisian food record di rumah.

INFORMED CONSENT

Saya yang bertanda tangan di bawah ini dengan penuh kesadaran menyatakan bersedia untuk mengisi kuesioner dengan selengkap-lengkapnya dan sebaik mungkin serta menjunjung tinggi nilai kejujuran. Selanjutnya saya dengan sukarela mempercayakan data pribadi saya digunakan untuk kepentingan penelitian dan pengembangan ilmu pengetahuan.

Nama :

Hari/Tanggal Wawancara :

Tanda Tangan Responden

(.....)

Kode responden: | | | | |

A. IDENTITAS PENGUMPULAN DATA			
No	**Pertanyaan**	**Jawaban**	**Kode**
1	Nama responden		Nama_resp
2	Hubungan dengan baduta		Hub_resp
3	Kontak responden		Kontak_resp
4	Nama enumerator		Nama_enum
5	Tanggal pengambilan data		Tgl_survey
6	Jam mulai		Jam_mulai
7	Jam selesai		Jam_selesai
8	Tanggal entri data		Tgl_entri
9	Tanda tangan enumerator		TTD_enum

B. KARAKTERISTIK BADUTA DAN ORANG TUA				
No	**Pertanyaan**	**Pilihan Jawaban/ Unit Satuan**	**Jawaban**	**Kode**
Karakteristik Baduta (Lingkari/Isi)				
1	Nama lengkap			Nama_bdt
2	Jenis kelamin	0. Laki-laki; 1. Perempuan		JK_bdt
3	Tempat lahir			Tmpt_lhr
4	Tanggal lahir	…. (dd/mm/yyyy)		Tgl_lhr_bdt
5	Umur baduta **(diisi saat *entry* data)**	…. Bulan		Umur_bdt
6	Subjek baduta merupakan anak ke berapa?			Anak_ke
7	Jika bukan anak pertama, berapa bulan jarak usia subjek baduta dengan kakak terdekat?	…. Bulan		Jrk_usia
8	Bagaimana proses kelahiran baduta?	1. Kelahiran per vaginam spontan 2. Kelahiran per vaginam dibantu alat (*vacuum extraction,dll*) 3. Section caesarea		Prs_lahir
9	Pada usia kehamilan berapa baduta dilahirkan? **(Lihat KIA jika ada)**	1. Prematur (<37 minggu) 2. Lebih bulan (> 42 minggu) 3. Cukup bulan (37-42 minggu)		
10	Berat lahir	….. kg		BBL
11	Panjang lahir	….. cm		PBL
Karakteristik Orang tua				
12	Nama ayah			Nama_ayah
13	Tanggal lahir ayah	…. (dd/mm/yyyy)		Tgl_lhr_ayah

Kode responden: | | | | |

B. KARAKTERISTIK BADUTA DAN ORANG TUA				
No	**Pertanyaan**	**Pilihan Jawaban/ Unit Satuan**	**Jawaban**	**Kode**
14	Umur ayah **(diisi saat *entry* data)**	…. Tahun		Umur_ayah
15	Pekerjaan ayah	1. Petani (Pemilik/Buruh Tani) 2. Pedagang/Wiraswasta 3. Buruh lainnya (sopir, tukang cukur, PRT, dll) 4. Aparat Desa 5. Pengajar/Guru/Ustadz 6. PNS/TNI/Polri/BUMN/BUMD 7. Pegawai swasta 8. Tidak bekerja 99. Lainnya **(sebutkan)**		Kerja_ayah
16	Pendidikan terakhir ayah	1. Tidak/belum pernah sekolah 2. Tidak tamat SD/MI 3. Tamat SD/MI 4. Tamat SLTP/MTS 5. Tamat SLTA/MA 6. Tamat diploma/sarjana/pasca sarjana		Pend_ayah
17	Nama ibu			
18	Tanggal lahir ibu	…. (dd/mm/yyyy)		Tgl_lhr_ibu
19	Umur ibu **(diisi saat *entry* data)**	…. Tahun		Umur_ibu
20	Pekerjaan ibu	1. Petani (Pemilik/Buruh Tani) 2. Pedagang/Wiraswasta 3. Buruh lainnya (sopir, tukang cukur, PRT, dll) 4. Aparat Desa 5. Pengajar/Guru/Ustadz 6. PNS/TNI/Polri/BUMN/BUMD 7. Pegawai swasta 8. Ibu rumah tangga 99. Lainnya **(sebutkan)**		Kerja_ibu
21	Pendidikan terakhir ibu	1. Tidak/belum pernah sekolah 2. Tidak tamat SD/MI 3. Tamat SD/MI 4. Tamat SLTP/MTS 5. Tamat SLTA/MA 6. Tamat diploma/sarjana/pasca sarjana		Pend_ibu
22	Jumlah balita dalam keluarga	…… Orang		Jml_Balita
23	Jumlah anggota keluarga	……. Orang		Jml_klrg
24	Pendapatan Ayah (per bulan)	Rp …		Pndptn_Ayah
25	Pendapatan Ibu (per bulan)	Rp …		Pndptn_Ibu
26	Total pendapatan keluarga dalam Sebulan	Rp …		Pndptn_Klg

Kode responden: | | | | |

C. PENGELUARAN KELUARGA						
No.	Jenis Pengeluaran	Pengeluaran (Rp) per			
		Harian	Mingguan	Bulanan*	Tahunan
J1	J2			J3	
1.	PANGAN				
	1. Pangan pokok, sayuran, lauk pauk, buah-buahan, minyak goreng, bumbu, bumbu-bumbuan				
	2. Makanan jajanan (bakso, siomay, cilok)				
	3. Minuman (air kemasan, air galon, dll.)				
	4. Kopi, teh, gula				
	5. Susu				
2.	NON-PANGAN				
	1. Kesehatan (BPJS)/Pengobatan				
	2. Rokok				
	3. Kebersihan (sabun cuci, sabun mandi, shampoo, pasta gigi, dll)				
	4. Transportasi (bensin, transportasi umum)				
	5. Biaya sekolah (SPP, buku, seragam)				
	6. Uang saku sekolah				
	7. Pakaian				
	8. Telekomunikasi (pulsa/kuota internet/WiFi/warnet)				
	9. Cicilan/kredit/arisan				
	10. Listrik				
	11. Air (PAM/pikulan/beli)				
	12. Sewa/kontrak rumah				
	13. Gas LPG				

Keterangan : Semua pengeluaran dikonversi ke bulan

D. KEPEMILIKAN FASILITAS (beri tanda ceklis (V) pada kolom jawaban yang sesuai)				
No	Nama barang	Jawaban		Kode
		Tidak Punya	Punya	
1	Laptop/Komputer			As1
2	Motor			As2
3	Mobil			As3
4	Mesin cuci			As4
5	Televisi			As5
6	Kulkas			As6
7	AC/pendingin ruangan			As7
8	Rumah			As8
9	Tanah			As9
10	Ladang/sawah/kebun			As10

Kode responden: | | | | |

E. KETAHANAN PANGAN RUMAH TANGGA				
No	**Pertanyaan**	**Pilihan Jawaban**	**Jawaban**	**Kode**
1	**Selama setahun terakhir,** apakah Anda/anggota rumah tangga lainnya **khawatir tidak akan memiliki cukup makanan** untuk disantap karena kekurangan uang atau sumber daya lainnya?	0. Tidak 1. Ya		FIES1
2	**Selama setahun terakhir,** apakah ada saat dimana Anda/anggota rumah tangga lainnya **tidak dapat menyantap makanan sehat dan bergizi** karena kekurangan uang atau sumber daya lainnya?	0. Tidak 1. Ya		FIES2
3	**Selama setahun terakhir,** apakah Anda/anggota rumah tangga lainnya hanya **menyantap sedikit jenis makanan** karena kekurangan uang atau sumber daya lainnya?	0. Tidak 1. Ya		FIES3
4	**Selama setahun terakhir,** apakah Anda/anggota rumah tangga lainnya **pernah melewatkan makan** karena tidak memiliki uang atau sumber daya lain yang cukup untuk mendapatkan makanan?	0. Tidak 1. Ya		FIES4
5	**Selama setahun terakhir,** apakah Anda/anggota rumah tangga lainnya **makan lebih sedikit daripada seharusnya** karena kekurangan uang atau sumber daya lainnya?	0. Tidak 1. Ya		FIES5
6	**Selama setahun terakhir,** apakah **rumah tangga Anda kehabisan makanan** karena kekurangan uang atau sumber daya lainnya?	0. Tidak 1. Ya		FIES6
7	**Selama setahun terakhir,** apakah Anda/anggota rumah tangga lainnya **pernah merasa lapar tetapi tidak makan** karena kekurangan uang atau sumber daya lainnya untuk mendapatkan makanan?	0. Tidak 1. Ya		FIES7
8	**Selama setahun terakhir,** apakah Anda/anggota rumah tangga lainnya **pernah tidak makan seharian** karena kekurangan uang atau sumber daya lainnya?	0. Tidak 1. Ya		FIES8

F. PENGUKURAN STATUS GIZI				
No	**Pertanyaan**	**Pilihan Jawaban**	**Jawaban**	**Kode**
1	Bagaimana kondisi kesehatan baduta saat pengukuran?	1. Oedema 2. Hidrosefalus 3. Diare 4. Sakit berat/kronis 5. Sakit ringan 6. Sehat		Kshtn_resp
2	Tanggal pengukuran			
STATUS GIZI				
3	Pengukuran tinggi badan 1	……. cm		TB1
4	Pengukuran tinggi badan 2	……. cm		TB2
5	Pengukuran berat badan 1	…… kg		BB_1
6	Pengukuran berat badan 2	…… kg		BB_2
7	Lingkar lengan atas 1	….. cm		LLA1
8	Lingkar lengan atas 2	….. cm		LLA2

Kode responden: | | | | |

G. IMUNISASI (CEK BUKU KIA)				
No.	**Pertanyaan**	**Pilihan Jawaban**	**Jawaban**	**Kode**
1	Apakah anak pernah menerima imunisasi?	1. Tidak 2. Ya		IMUN1
2	Apa alasan anak tidak menerima imunisasi?	1. Tidak penting 2. Isu larangan agama/budaya 3. Isu efek samping 4. Tidak tahu/lupa jadwal 5. Tidak tahu tempatnya 6. Sulit akses 99. Lainnya:		IMUN2

No	**Imunisasi**	**Jumlah imunisasi**	**Tempat Imunisasi**
3a	HB0		
3b	BCG		
3c	DPT-HB / DPT-HB-HiB		
3d	Polio oral		
3e	Polio suntik		
3f	Campak / Mumps-Rubella		
3g	Lainnya:		
3h	Lainnya:		

H. MORBIDITAS **Apakah dalam satu bulan terakhir anak Anda mengalami penyakit berikut?**				
No	**Jenis Penyakit**	**Frekuensi**	**Lama Sakit**	
			Hari	**Minggu**
1	ISPA (batuk, pilek, atau sakit tenggorokan)			
2	TB paru/flek paru			
3	Hepatitis			
4	Tifus			
5	Demam berdarah			
6	Kaki gajah			
7	Infeksi saluran kemih			
8	Diare			
9	Disentri (diare disertai darah, lendir)			
10	Cacar air			
11	Campak			
12	Sakit kulit (bisul, borok, gatal, panu)			
13	Sakit gigi			
14	Sakit mata			
15	Lainnya: ……			
16	Lainnya: ……			

Kode responden: | | | | |

G. IMUNISASI (CEK BUKU KIA)				
No.	**Pertanyaan**	**Pilihan Jawaban**	**Jawaban**	**Kode**
1	Apakah anak pernah menerima imunisasi?	1. Tidak 2. Ya		IMUN1
2	Apa alasan anak tidak menerima imunisasi?	1. Tidak penting 2. Isu larangan agama/budaya 3. Isu efek samping 4. Tidak tahu/lupa jadwal 5. Tidak tahu tempatnya 6. Sulit akses 99. Lainnya:		IMUN2

No	Imunisasi	Jumlah imunisasi	Tempat Imunisasi
3a	HB0		
3b	BCG		
3c	DPT-HB / DPT-HB-HiB		
3d	Polio oral		
3e	Polio suntik		
3f	Campak / Mumps-Rubella		
3g	Lainnya:		
3h	Lainnya:		

H. MORBIDITAS Apakah dalam satu bulan terakhir anak Anda mengalami penyakit berikut?				
No	**Jenis Penyakit**	**Frekuensi**	**Lama Sakit**	
			Hari	**Minggu**
1	ISPA (batuk, pilek, atau sakit tenggorokan)			
2	TB paru/flek paru			
3	Hepatitis			
4	Tifus			
5	Demam berdarah			
6	Kaki gajah			
7	Infeksi saluran kemih			
8	Diare			
9	Disentri (diare disertai darah, lendir)			
10	Cacar air			
11	Campak			
12	Sakit kulit (bisul, borok, gatal, panu)			
13	Sakit gigi			
14	Sakit mata			
15	Lainnya: ……			
16	Lainnya: ……			

Kode responden: | | | | |

I. PRAKTIK PEMBERIAN MAKAN ANAK				
No.	**Pertanyaan**	**Pilihan Jawaban**	**Jawaban**	**Kode**
13	Apakah anak diberikan makanan atau minuman sebelum ASI keluar?	1.Ya 2.Tidak **>Lompat ke no. 15**		PREL1
14	Sebutkan minuman/makanan yang pertama kali diberikan kepada anak sebelum mulai disusui atau sebelum ASI keluar/lancar?	……..		PREL3
15	Pada saat anak umur berapa ibu mulai MENGENALKAN makanan atau minuman (cairan) selain ASI?	…… hari atau ……. bulan **(Jika jawaban ≥6 bulan, lompat ke pertanyaan no. 17)**		MPASI1
16	Apa alasan UTAMA dikenalkan makanan atau minuman selain ASI sebelum 6 bulan?	1.ASI sudah tidak keluar/sedikit 2.Anak tidak mau menyusu 3.Anak terpisah dari ibunya 4.Anak selalu rewel 5.Anak lapar 6. Alasan budaya/agama/norma 99. Lainnya:		MPASI2
17	Apa makanan/minuman (cairan) selain ASI yang diberikan kepada anak di usia tersebut?	……		MPASI3
18	Apakah anak mengonsumsi susu atau cairan pengganti atau pendamping ASI?	1. Ya 2. Tidak		MPASI4
19	Sejak umur berapa anak mengonsumsi susu atau cairan pengganti atau pendamping ASI?	…… hari atau ….. bulan		MPASI5
20	Susu atau cairan pengganti atau pendamping ASI apa yang diberikan?	1.Air tajin 2.Air gula 3.Susu kental manis 4.Susu sapi segar/pasterurisasi 5.Susu kotak UHT 6.Susu formula 99. Lainnya……		MPASI6
21	Bagaimana frekuensi pemberian susu atau cairan pengganti atau pendamping ASI tersebut?	….. per hari		MPASI7
22	Media apa yang digunakan untuk pemberian susu atau cairan pengganti atau pendamping ASI	1.Dot 2.Gelas 3.Sendok 3. Sedotan 99. Lainnya		MPASI8

Kode responden: | | | | |

I. PRAKTIK PEMBERIAN MAKAN ANAK				
No.	**Pertanyaan**	**Pilihan Jawaban**	**Jawaban**	**Kode**
23	Apakah dalam sehari kemarin, (dari bangun tidur kemarin pagi hingga pagi tadi), anak Ibu memakan makanan padat, semi padat atau lumat?	1.Tidak 2.Ya		MPad
24	Berapa kali anak Ibu memakan makanan padat, setengah padat atau makanan lumat selama seharian kemarin (mulai bangun tidur kemarin pagi hingga tadi pagi)? JIKA 7 KALI ATAU LEBIH ISIKAN KODE "7", JIKA TIDAK TAHU ISIKAN KODE "88"	…… kali		Frekmak

25. Apakah dalam sehari kemarin (dari bangun tidur kemarin hingga tadi pagi) anak Ibu mengonsumsi bahan pangan berikut:

No	**Kelompok Bahan Pangan**	**Isikan Kode: 1.Ya 2.Tidak 8.Tidak tahu**	**Jumlah**	**Kode**
a	Air Susu Ibu (ASI)			ASI
b	Air putih			AP
c	Jus atau sari buah produk pabrikan			Jus
d	Air kaldu (seperti kaldu ayam, daging, atau kaldu ikan)			AK
e	Susu formula bayi/balita			Sufor
f	Susu lainnya, seperti: susu bubuk, susu segar			Susu
g	Minuman/cairan lainnya (seperti air gula, kental manis, the air tajin, susu kedelai, dll)			Mila
h	Yogurt (tidak termasuk Yakult, Vitacarm)			Yog
i	Makanan bayi bermerek seperti Sun, Milna, cerelac			MB
j	Nasi, roti, mie, bubur, jagung, sagu atau makanan lain yang dibuat dari padi-padian seperti beras, gandum, sorgum, dll			Cer

Kode responden: | | | | |

No	Kelompok Bahan Pangan	Isikan Kode: 1.Ya 2.Tidak 8.Tidak tahu	Jumlah	Kode
k	Labu kuning, wortel, atau ubi jalar yang berwarna kuning atau oranye didalamnya			VegA
l	Kentang, ubi kayu/ketela pohon/singkong, talas, dan makanan lain dari akar- akaran atau akar umbi			Ubi
m	Sayuran hijau (bayam, kangkung, katuk, daun singkong, daun labu dll.)			Veghi
n	Buah-buahan yang kaya vitamin A yang masak, seperti mangga, pepaya, nangka, cempedak, kesemek, melon kuning			BuA
o	Buah atau sayuran lainnya (seperti apel, alpukat, kapri, terong, oyong dll)			BuL
p	Ati, ampela, ginjal, jantung, atau jeroan lainnya			Jer
q	Daging: ayam, sapi, kambing, babi atau itik			Dag
r	Telur			Egg
s	Ikan/kerang segar atau asin			Fish
t	Makanan dari kacang-kacangan (kacang kedelai, kacang merah, kacang tolo, kacang jogo, kacang hijau, kacang babi, kacang tanah, tahu, tempe, dll.)			Nuts
u	Keju atau makanan lain yang terbuat dari susu			DP
v	Makanan padat, setengah padat, makanan lumat lainnya termasuk kue-kue seperti kue pisang, cucur, pancong, permen			Kue

E. CHRONONUTRITION				
No	**Pertanyaan**	**Opsi Jawaban**	**Jawaban**	**Kode**
1	Dalam satu hari berapa kali anak Anda menyusu?	…. kali		CN1
2	Dalam satu kali menyusu biasanya durasinya berapa lama?	…… menit		CN2
3	Berapa jarak antara waktu menyusu dengan waktu makan?	……. Jam …….. menit		CN3

Kode responden: | | | | |

E. CHRONONUTRITION				
No	**Pertanyaan**	**Opsi Jawaban**	**Jawaban**	**Kode**
4	Dalam satu bulan terakhir manakah waktu yang merupakan waktu puncak menyusu anak Anda dalam sehari?	1. Pukul 01.00-05.00 2. Pukul 05.00-09.00 3. Pukul 09.00-13.00 4. Pukul 13.00-17.00 5. Pukul 17.00-21.00 6. Pukul 21.00-01.00		CN4
5	Dalam satu bulan terakhir berapa lama durasi waktu anak Anda menyusu pada waktu puncak menyusu tersebut?	…… jam ……. menit		CN5
6	Dalam satu bulan terakhir, berapa kali anak Anda biasanya tidur selain tidur malam (tidur pagi/siang/sore) dalam satu hari?	Jumlah dalam satuan kali per hari. Jika tidak melakukan tidur pagi/siang/sore maka isi 0	…… kali per hari	CN6
7	Dalam satu bulan terakhir, jam berapa biasanya anak Anda mulai tidur pagi/siang/sore untuk yang pertama kali (tidur pertama)?	Waktu (format 24 jam) misalkan 05:00 waktu setempat. Isikan – jika tidak melakukan		CN7
8	Dalam satu bulan terakhir biasanya berapa lama durasi tidur pagi/siang/sore yang pertama?	Durasi dalam menit, isikan 0 jika tidak melakukan	…. menit	CN8
9	Dalam satu bulan terakhir, jam berapa biasanya anak Anda mulai tidur pagi/siang/sore untuk yang kedua kali (tidur kedua)?	Waktu (format 24 jam) misalkan 05:00 waktu setempat. Isikan – jika tidak melakukan		CN9
10	Dalam satu bulan terakhir biasanya berapa lama durasi tidur pagi/siang/sore yang kedua?	Durasi dalam menit, isikan 0 jika tidak melakukan	…. menit	CN10
11	Dalam satu bulan terakhir, jam berapa biasanya anak Anda mulai tidur pagi/siang/sore untuk yang ketiga kali (tidur ketiga)?	Waktu (format 24 jam) misalkan 05:00 waktu setempat. Isikan – jika tidak melakukan		CN11
12	Dalam satu bulan terakhir biasanya berapa lama durasi tidur pagi/siang/sore yang ketiga?	Durasi dalam menit, isikan 0 jika tidak melakukan	…. menit	CN12
13	Biasanya anak Anda makan terakhir berapa jam sebelum tidur malam?	Durasi dalam menit, isikan 0 jika tidak melakukan	…… menit	CN13
14	Dalam satu bulan terakhir jam berapa anak Anda biasanya tertidur pada malam hari?	Waktu (format 24 jam) misalkan 20:00 waktu setempat		CN14
15	Dalam satu bulan terakhir biasanya berapa lama durasi waktu anak Anda tidur malam?	Durasi dalam menit, isikan 0 jika tidak	…… menit	CN15
16	Dalam satu bulan terakhir, jam berapa anak Anda biasanya bangun pagi?	Waktu (format 24 jam) misalkan 05:00 waktu setempat		CN16
17	Dalam satu bulan terakhir setelah bangun tidur pada pagi hari, seberapa cepat anak Anda melakukan makan pertama?	Durasi dalam menit, isikan 0 jika tidak melakukan	…… menit	CN17
18	Dalam satu bulan terakhir jam berapa biasanya anak Anda sarapan?	Format hh:mm / 0 jika tidak makan		CN18
19	Dalam satu bulan terakhir jam berapa biasanya anak Anda makan snack pagi?	Format hh:mm / 0		CN19
20	Dalam satu bulan terakhir jam berapa biasanya anak Anda makan siang?	Format hh:mm / 0		CN20
21	Dalam satu bulan terakhir jam berapa biasanya anak Anda makan snack sore?	Format hh:mm / 0		CN21

Kode responden: | | | | |

E. CHRONONUTRITION				
No	**Pertanyaan**	**Opsi Jawaban**	**Jawaban**	**Kode**
22	Dalam satu bulan terakhir jam berapa biasanya anak Anda makan malam?	Format hh:mm / 0		CN22
23	Dalam satu bulan terakhir jam berapa biasanya anak Anda makan snack malam?	Format hh:mm / 0		CN23
24	Dalam satu bulan terakhir berapa lama waktu sarapan?	Durasi dalam menit, isikan 0 jika tidak	…… menit	CN24
25	Dalam satu bulan terakhir berapa lama waktu snack pagi?	Durasi dalam menit	…… menit	CN25
26	Dalam satu bulan terakhir berapa lama waktu makan siang?	Durasi dalam menit	…… menit	CN26
27	Dalam satu bulan terakhir berapa lama waktu snack sore?	Durasi dalam menit	…… menit	CN27
28	Dalam satu bulan terakhir berapa lama waktu makan malam?	Durasi dalam menit	…… menit	CN28
29	Dalam satu bulan terakhir berapa lama waktu snack malam?	Durasi dalam menit	…… menit	CN29
30	Dalam satu bulan terakhir waktu makan apa yang merupakan makan terbesar anak Anda dalam sehari?	1. Sarapan 2. Makan siang 3. Makan malam 4. Lainnya, ……		CN30

Kode responden: | | | | |

FORM FOOD RECALL 1X24 JAM					
Waktu Makan	**Nama Masakan / Masakan**	**Metode Pemasakan:** 1. Kukus; 2. Rebus 3. Tumis 4. Goreng sedikit miyak 5. Goreng banyak minyak 6. Bakar/panggang	**Bahan Makanan**	**Berat Makanan**	
				URT	**Gram**

Kode responden: | | | | |

FORM FOOD RECALL 1X24 JAM					
Waktu Makan	**Nama Masakan / Masakan**	**Metode Pemasakan:** 1. Kukus; 2. Rebus 3. Tumis 4. Goreng sedikit miyak 5. Goreng banyak minyak 6. Bakar/panggang	**Bahan Makanan**	**Berat Makanan**	
				URT	**Gram**

Apakah konsumsi pangan tersebut sudah mencerminkan kebiasaan makan Baduta?
1. Tidak 2. Ya

Apakah baduta mengonsumsi suplemen? 1. Tidak 2. Ya

Suplemen yang dikonsumsi: ……..

c. List of Pictures


POSYANDU
TANJUNG. 3