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Risk Factors for Stunting Among Children Aged 6–24 Months in Uluere Subdistrict, Bantaeng Regency, Indonesia

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ABSTRACT

Stunting remains an important child health problem in Indonesia, particularly during the first 1,000 days of life. This study aimed to identify factors associated with stunting among children aged 6–24 months in Uluere Subdistrict, Bantaeng Regency. A frequency-matched case-control study was conducted by child age group and sex. Child age and sex were subsequently retained as adjustment variables in the multivariable logistic regression to account for the matching design. Cases were children with a length-for-age z-score (LAZ) <-2 SD, whereas controls had LAZ ≥-2 SD based on WHO standards. The study included 69 cases and 136 controls. Child age was retained as an adjustment variable, while sex was included as a prespecified covariate in multivariable logistic regression. Recent illness history showed the strongest statistical association with stunting. This can be seen in the statistical analysis odds ratio for stunting (aOR 4.23; 95% CI 1.91–9.36; $p<0.001$), followed by low birth weight (aOR 3.59; 95% CI 1.43–8.99; $p=0.006$) and uncovered household waste bins (aOR 2.01; 95% CI 1.06–3.80; $p=0.033$). Family food consumption was associated with lower odds of stunting (aOR 0.45; 95% CI 0.20–0.99; $p=0.049$), although this estimate was close to the significance boundary. The final model demonstrated acceptable discrimination (AUC = 0.722), although its explanatory power remained modest (Nagelkerke R^2 = 18.6%). These findings support closer monitoring of children with low birth weight, morbidity prevention, improved household waste management, and age-appropriate feeding support, without implying causal relationships.

Keywords: Case-control study; Child morbidity; Household sanitation; Low birth weight; Stunting

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INTRODUCTION

Stunting is a chronic manifestation of impaired linear growth and remains an important child health problem in low- and middle-income countries. Global estimates show that the burden remains concentrated in Africa and Asia, while nutritional deficits, recurrent infections, and suboptimal caregiving during the first 1,000 days may have persistent effects on physical growth and development.[1–4]

Linear growth failure results from cumulative interactions between prenatal growth restriction, inadequate nutrient intake, recurrent infections, intestinal inflammation, and adverse environmental exposures. These biological mechanisms interact with socioeconomic and behavioral determinants, making the causes of stunting highly context-specific despite global consistency in its overall burden.

Indonesia has made progress in reducing stunting, but the burden remains uneven across regions.[5] South Sulawesi continues to show geographic heterogeneity, indicating that provincial or district-level improvement may conceal persistent problems in specific subdistricts. In Bantaeng Regency, Uluere Subdistrict remained a priority setting and recorded the highest stunting burden in the regency in 2023[5]. Local risk-factor analysis is therefore required, as district-level progress may not be distributed uniformly across subdistricts.

Although many determinants of stunting have been consistently identified across countries, their relative contributions vary substantially depending on local epidemiological context, health service coverage, environmental sanitation, cultural feeding practices, and socioeconomic conditions. Therefore, identifying locally dominant determinants remains essential for designing context-specific interventions.

Stunting is shaped by interacting prenatal, child-level, household, and environmental factors. The UNICEF conceptual framework places diet and health as immediate determinants, while feeding practices, food security, health services, and the household environment operate at underlying levels.[4] Indonesian evidence also shows that the statistical importance of these factors varies across populations and levels of analysis.[6,7] Consequently, determinants established in national or international studies cannot be assumed to have the same relative importance in every local context.

Previous studies have reported inconsistent findings regarding the dominant determinants of stunting. Several studies identified maternal education, household income, and food insecurity as major predictors, whereas others demonstrated that biological factors such as low birth weight, recurrent infections, and prenatal growth restriction remained stronger predictors after multivariable adjustment. These inconsistencies indicate that the relative importance of risk factors may vary across local populations and measurement approaches.

Children aged 6–24 months are a critical group because they are transitioning from breastfeeding to complementary foods and family foods, while exposure to infection and the household environment increases.[3,4] Despite the continuing burden in Uluere, no analytic study has specifically estimated the relative magnitude of maternal, birth-related morbidity, feeding, food security, and household environmental factors among children aged 6–24 months in this setting. We hypothesized that biological vulnerability, feeding practices, household sanitation, and socioeconomic factors would each be independently associated with stunting among children aged 6–24 months. This study aimed to estimate the associations between these factors and stunting in Uluere Subdistrict.

METHOD

This study used an observational analytic unmatched case-control design with frequency matching by child age group (6–11, 12–17, and 18–24 months). Sex was not used as a matching variable. The study was conducted in six villages within Uluere Subdistrict, Bantaeng Regency, South Sulawesi, from June to December 2025. The cases were children aged 6–24 months with LAZ <−2 SD, whereas the controls were children in the same age range with LAZ ≥−2 SD according to the WHO Child Growth Standards.[8] Routine Posyandu and primary health center records were used to identify potentially eligible children. Final case and control classifications were based on anthropometric assessment conducted during study data collection, and controls were selected from the same source population and geographic area as the cases. The minimum sample size was calculated using the formula for unmatched case-control studies with a case-control ratio of 1:2, a 95% confidence level, 80% statistical power, an expected odds ratio derived from previous Indonesian studies, and an anticipated exposure prevalence among controls. The minimum required sample was 68 cases and 136 controls; the final analysis included 69 cases and 136 controls.

All anthropometric measurements were repeated by trained research personnel using standardized equipment, following the WHO Child Growth Standards, to minimize misclassification of outcomes. Data were collected through anthropometric measurements and structured interviews with mothers or caregivers. The child's age was verified using available birth or health records. Recumbent length was measured using a standardized length board, and LAZ was calculated according to the WHO 2006 Child Growth Standards.[8] Measurements were performed by trained data collectors following standardized anthropometric procedures. The variables covered child biological characteristics, including birth weight and illness history within the previous 30 days; maternal and pregnancy history; socioeconomic characteristics; feeding practices; minimum dietary diversity; household food security; access to health services; and sanitation or household environmental indicators. Frequency matching was performed during sampling to improve comparability between cases and controls with respect to child age group and sex. These variables were subsequently retained in all multivariable models as forced adjustment variables. Univariable logistic regression was used to estimate crude odds ratios (ORs), 95% confidence intervals (CIs), and p-values. Variables with $p<0.25$ were considered for multivariable modeling. Exclusive breastfeeding was excluded from the primary multivariable model because its observed direction was inconsistent with established biological evidence. The variable was therefore interpreted cautiously to avoid potentially misleading conclusions. Nevertheless, the bivariable results are presented transparently. Multivariable logistic regression with backward elimination was performed. Child age was retained in all models because it was used for frequency matching, whereas sex was retained as a prespecified adjustment covariate based on substantive considerations. Adjusted odds ratios (aORs) with 95% CIs were reported, and $p<0.05$ was considered statistically significant. Model performance was assessed using the omnibus test, Nagelkerke R2, area under the receiver operating characteristic (ROC) curve (AUC), classification accuracy, sensitivity, specificity, and events per variable. Written informed consent was obtained from each mother or primary caregiver before participation.

RESULTS

Participant Characteristics

The study included 205 children comprising 69 cases (33.7%) and 136 controls (66.3%). The proportion of cases reflected the predefined sampling ratio rather than the prevalence of stunting in the study population. This proportion reflects the sampling ratio rather than population prevalence. The largest number of respondents came from Bonto Lojong (44.9%). Frequency matching produced comparable age distributions between cases and controls: mean age was 14.7 ± 5.8 months among cases and 14.9 ± 5.5 months among controls ($p=0.903$). Sex distributions were also similar, although sex was not used as a matching variable: males accounted for 46.4% of cases and 47.8% of controls ($p=0.848$).

Table 1. Selected characteristics of study participants (n=205)

Variable	n	%
Child sex, male	97	47.3
Child age 6-12 months	82	40.0
Low birth weight (<2,500 g)	27	13.2
Stunted (LAZ -3 to < -2 SD)	64	31.2
Severely stunted (LAZ < -3 SD)	5	2.4
Illness history within last 30 days	142	69.3
Exclusive breastfeeding	159	77.6
Minimum dietary diversity not adequate	110	53.7
Food insecure household	31	15.1
Uncovered household waste bin	114	55.6
Child played barefoot on soil	142	69.3
Maternal age >35 years	22	10.7
Maternal education, secondary school or lower	113	55.1
Household income <IDR 1 million/month	39	19.0

Bivariable Analysis

In the bivariable analysis, recent illness history demonstrated the strongest crude association with stunting (OR 3.26; 95% CI 1.57–6.78), followed by maternal age above 35 years and low birth weight. Several variables, including food insecurity, family food consumption, and meal frequency, did not reach conventional statistical significance but met the predefined criterion ($p<0.25$) for inclusion in multivariable modeling. Several other variables met the candidate criterion for multivariable modeling despite not reaching conventional statistical significance, including low meal frequency, food insecurity, packaged snack consumption, family food consumption, maternal weakness or pallor during pregnancy, and iron tablet consumption. Exclusive breastfeeding showed an unexpected direction and was evaluated in a sensitivity analysis.

Table 2. Summary of bivariable logistic regression results

Variable	OR	95% CI	p-value	Status
Illness history	3.26	1.57–6.78	0.001	Significant
Maternal age >35 years	2.66	1.08–6.50	0.033	Significant
Low birth weight	2.41	1.06–5.47	0.035	Significant
Uncovered waste bin	1.82	1.00–3.31	0.050	Borderline significance
Barefoot soil play	1.96	1.00–3.84	0.049	Significant
Exclusive breastfeeding	3.56	1.50–8.46	0.004	Sensitivity analysis
Meal frequency <3/day	0.49	0.24–1.00	0.051	Candidate
Food-insecure household	2.08	0.96–4.52	0.063	Candidate
Packaged snack consumption	1.78	0.97–3.25	0.062	Candidate
Family food consumption	0.52	0.25–1.05	0.069	Candidate
Maternal weakness/pallor	0.57	0.30–1.05	0.073	Candidate
Iron tablet consumption	2.08	0.74–5.82	0.165	Candidate

Multivariable Analysis

Backward elimination was applied to the candidate variables. Child age was retained because it was used for frequency matching, whereas sex was retained as a prespecified adjustment covariate. The final model retained four substantive variables: low birth weight, recent illness history, uncovered household waste bins, and family food consumption. After adjustment for matching variables and candidate predictors, four variables remained independently associated with stunting. Recent illness history showed the largest adjusted odds ratio (aOR 4.23; 95% CI 1.91–9.36), followed by low birth weight (aOR 3.59; 95% CI 1.43–8.99) and uncovered household waste bins (aOR 2.01; 95% CI 1.06–3.80). Family food consumption remained inversely associated with stunting (aOR 0.45; 95% CI 0.20–0.99).

Table 3. Final multivariable logistic regression model (n=205)

Variable	B	SE	p-value	aOR	95% CI
Child age (months)*	0.053	0.029	0.068	1.05	1.00–1.12
Sex*	-0.155	0.332	0.639	0.86	0.45–1.64
Low birth weight (<2,500 g)	1.278	0.469	0.006	3.59	1.43–8.99
Illness history	1.443	0.405	<0.001	4.23	1.91–9.36
Uncovered waste bin	0.697	0.326	0.033	2.01	1.06–3.80
Family food consumption	-0.795	0.403	0.049	0.45	0.20–0.99

*Child age was retained because of frequency matching; sex was retained as a prespecified adjustment covariate. aOR was adjusted for all variables in the final model.

In the sensitivity model that included exclusive breastfeeding, exclusive breastfeeding was not statistically associated with stunting (aOR 2.22; 95% CI 0.88–5.59; p=0.092). The estimates for low birth weight, recent illness, and uncovered waste bins remained broadly stable, whereas the association with family food consumption weakened and was no longer statistically significant.

Table 4. Final model performance statistics

Parameter	Value
Omnibus test	G2 = 29.562; df = 6; p < 0.001
Nagelkerke R2	0.186 (18.6%)
AUC / c-statistic	0.722
Classification accuracy	69.3%
Sensitivity	31.9%
Specificity	88.2%
Null model accuracy	66.3%
Final model EPV	69 / 6 = 11.5

The omnibus test indicated that the final multivariable model significantly improved model fit compared with the null model ($p<0.001$). The model demonstrated acceptable discrimination (AUC = 0.722) but explained only a modest proportion of the variability in stunting (Nagelkerke R^2 = 18.6%). Although overall classification accuracy reached 69.3%, sensitivity remained relatively low (31.9%), whereas specificity was high (88.2%), indicating that the model identified non-stunted children more accurately than stunted children.

DISCUSSION

This study found statistical associations between stunting and recent illness history, low birth weight, uncovered household waste bins, and family food consumption among children aged 6–24 months in Uluere Subdistrict. The sampled stunting pattern was more clearly differentiated by proximal child vulnerability, morbidity, and household microsanitation than by broader socioeconomic indicators in the final model. This does not mean that socioeconomic conditions are irrelevant; rather, the limited variability of some socioeconomic variables in a relatively homogeneous rural agricultural population may have reduced their statistical contrast.

The recent illness showed the largest adjusted odds ratio in the final model. This finding is biologically plausible because morbidity can reduce appetite, increase metabolic requirements, and interfere with nutrient absorption and utilization.[9,10] Evidence from Ethiopia also shows that diarrhea and fever are associated with concurrent wasting and stunting, supporting the role of morbidity in worsening nutritional vulnerability.[11] Nevertheless, the temporal interpretation must be conservative. Stunting is a chronic cumulative condition, while illness history in this study captured only the previous 30 days. The association may partly reflect reverse causation, in which children who are already stunted become more vulnerable to illness, or it may serve as a proxy for recurrent morbidity that was not fully measured longitudinally. Therefore, this variable should not be read as proof that a short recent illness episode directly caused chronic stunting. The relatively wide confidence interval also indicates that the magnitude of this association should be interpreted with caution.

Recurrent exposure to enteric pathogens may also contribute to environmental enteric dysfunction (EED), a chronic subclinical inflammatory condition characterized by impaired intestinal barrier integrity, reduced nutrient absorption, and persistent immune activation. Although EED was not directly measured in this study, it provides a biologically plausible mechanism linking repeated infections, poor sanitation, and impaired linear growth.

Low birth weight was the finding with the clearest temporal ordering because birth weight was established before assessment of the child's current growth status. The adjusted association remained significant after controlling for age, sex, recent illness, household waste management, and family food consumption. This is consistent with studies included in the thesis, which show that low birth weight is associated with later linear growth failure.[12–14] Low birth weight may also reflect underlying fetal growth restriction resulting from inadequate maternal nutrition, placental insufficiency, maternal anemia, or pregnancy-related illness. These prenatal conditions may influence skeletal growth before birth and increase susceptibility to subsequent postnatal growth faltering. These pathways may leave the infant with lower nutrient stores, reduced organ maturity, and a lower initial growth trajectory. However, low birth weight should not be treated as synonymous with fetal growth restriction because preterm birth may also result in a birth weight below 2,500 g. The finding supports the prevention and monitoring of these conditions beginning before and immediately after birth. Gestational age and birth length were not measured in sufficient detail, limiting differentiation between prematurity and restricted fetal growth.

The association between uncovered household waste bins and stunting should be interpreted as a marker of household hygiene and microsanitation. Uncovered waste may increase opportunities for exposure to flies, animals, faecal contamination, and enteric pathogens. Evidence reviewed in the thesis links contaminated household environments and poor WASH conditions with impaired child growth.[15–18] Repeated enteric exposure may also contribute to environmental enteric dysfunction, characterized by subclinical intestinal inflammation, impaired barrier function, and reduced nutrient absorption. [15–18] However, enteric pathogens, inflammatory biomarkers, and environmental enteric dysfunction were not measured directly in this study; this mechanism therefore remains biologically plausible rather than empirically demonstrated in the study population. Larger sanitation infrastructure indicators, such as piped water use and latrine defecation, were relatively homogeneous, whereas waste management varied more strongly across households. This may explain why waste-bin status remained visible in the model.

Family food consumption was associated with lower odds of stunting in the primary model, although the estimate was close to the significance boundary and weakened in the sensitivity analysis. In this study, the variable indicated whether the child consumed foods prepared for the household; it was not a standardized measure of dietary quality, nutrient density, portion size, or total energy intake. Indonesian studies included in the thesis show that dietary diversity and complementary-feeding adequacy are relevant to nutritional status, but these indicators are not equivalent to the simple consumption of family foods.[19,20] The variable may therefore reflect feeding transition, age, appetite, developmental readiness, or caregiver responsiveness. The finding should be interpreted as a possible marker of feeding transition rather than evidence that family foods themselves prevent stunting. Feeding counseling should address texture, frequency, dietary diversity, animal-source foods, meal safety, and responsive feeding.

Several variables commonly discussed in the stunting literature did not remain independently associated in the final model, including maternal education, household income, household food security, access to health services, and latrine use. These null findings require careful interpretation because Indonesian evidence identifies socioeconomic, maternal-service, and household-level determinants as relevant in other populations.[6,7] Most respondents in this study used piped water and reported defecation in latrines, while household income and occupation were clustered within a narrow rural profile. Measurement limitations were also present for some variables, particularly maternal mid-upper arm circumference during pregnancy, which had a high proportion of unknown responses. Limited sample size, exposure homogeneity, measurement error, and residual confounding may also have reduced the ability to detect associations. A non-significant result in this context should not be translated into a claim that the factor is unimportant in general.

Exclusive breastfeeding showed an unexpected positive association with stunting in the univariable analysis. This direction differs from studies cited in the thesis that report a protective or potentially beneficial relationship between exclusive breastfeeding and child growth.[21,22] Because the variable was measured retrospectively and was susceptible to recall error, outcome-related reporting, social desirability bias, and residual confounding, it was not included in the primary parsimonious model. This decision was made to reduce the risk of misleading interpretation rather than to dismiss the observed result. Exclusive breastfeeding was subsequently included in a sensitivity analysis, in which the association weakened and was no longer statistically significant (aOR 2.22; 95% CI 0.88–5.59; $p=0.092$). The estimates for low birth weight, recent illness, and uncovered waste bins remained broadly stable, whereas the association with family food consumption weakened and was no longer statistically significant. The result should therefore not be interpreted as evidence against exclusive breastfeeding recommendations.

The main strength of this study is its local relevance and its assessment of multiple determinant domains in a case-control design with frequency matching by age group. Several limitations should nevertheless be considered. First, the case-control design limits causal and temporal inference, particularly for recent illness, current feeding practices, and household environmental indicators measured near the outcome assessment. Second, the 30-day illness history may represent reverse causation or may function as a proxy for recurrent morbidity that was not measured longitudinally. Third, breastfeeding, pregnancy history, illness, and feeding practices relied partly on caregiver recall and were susceptible to reporting and classification error. Fourth, recruitment through routine Posyandu and primary-health-center sources may have underrepresented children who did not regularly access community health services, creating potential selection bias. Fifth, residual confounding remains possible because parental height, gestational age, birth length, detailed nutrient intake, pathogen exposure, and environmental enteric dysfunction were not directly measured. Sixth, confidence intervals for several estimates were relatively wide, indicating limited precision. Finally, although the model had acceptable events per variable and moderate discrimination, its low sensitivity means that it should not be used as a stand-alone screening or prediction tool. Future studies should use prospective designs beginning in pregnancy or at birth, include children not routinely reached by Posyandu-based surveillance, and measure recurrent morbidity, gestational age, birth length, parental anthropometry, quantitative dietary intake, and more specific WASH or enteric-inflammation indicators.

CONCLUSIONS AND RECOMMENDATIONS

Among children aged 6–24 months in Uluere Subdistrict, stunting was statistically associated with low birth weight, recent illness history, uncovered household waste bins, and family food consumption after adjustment for age, sex, and other variables retained in the final model. Family food consumption was associated with lower odds in the primary model, but this finding was borderline and weakened in the sensitivity analysis. Program priorities should consider not only the magnitude of adjusted odds ratios but also temporality, biological plausibility, precision, and modifiability. Children with low birth weight warrant early growth monitoring because the exposure clearly precedes the current growth status. Prevention and follow-up of recurrent childhood illness should also be strengthened, although the 30-day illness measure cannot establish causality. Household waste management and play-area hygiene represent potentially modifiable environmental targets. Feeding counseling should emphasize age-appropriate texture, frequency, dietary diversity, nutrient density, food safety, and responsive feeding rather than simply encouraging family-food consumption. Further research should use prospective cohort designs and include children who are not routinely reached by Posyandu surveillance.

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