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Factors Associated with Stunting Severity among Under-Five Children with Nutritional Problems in East Bolaang Mongondow Regency

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ABSTRACT

Concurrent anthropometric deficits may indicate the need for comprehensive nutritional assessment in children. This study aimed to examine characteristics associated with severe versus moderate stunting among children with concurrent underweight. An exploratory cross-sectional study was conducted using secondary data from SIGIZIKESGA records collected in February 2026. The analysis included 43 children aged 0–59 months with both stunting and underweight in East Bolaang Mongondow Regency, Indonesia. Categorical variables were compared using two-sided Fisher's exact tests, while crude odds ratios (ORs) and 95% confidence intervals (CIs) were calculated to describe associations. Of the 43 children, 29 (67.4%) had moderate stunting and 14 (32.6%) had severe stunting. Severe stunting was observed in 13 of 27 children with severe underweight (48.1%), compared with 1 of 16 children with underweight (6.3%; Fisher's exact p = 0.006). The crude OR was 13.93 (95% CI, 1.61–120.83). Birth-weight category, sex, and reported history of infectious diseases were not significantly associated with stunting severity. Severe underweight was associated with a higher proportion of severe stunting in this selected sample. However, the overlap between anthropometric measures, small cell counts, and lack of adjustment for potential confounders limit the interpretation of these findings. The results should be considered exploratory and hypothesis-generating rather than causal and cannot be generalized to all children in the regency.

INTRODUCTION

Stunting remains an important manifestation of impaired linear growth among children in low- and middle-income countries. According to the WHO Child Growth Standards, stunting is defined as a length/height-for-age z-score (HAZ) below −2 standard deviations (SD), while severe stunting is defined as HAZ below −3 SD. These categories describe the extent of attained linear growth deficit but do not, on their own, identify its cause or onset. Multicountry longitudinal analyses have linked early growth faltering with maternal and perinatal characteristics and have shown that linear growth deficits may emerge in the first months of life (1,2). Consequently, assessment of current nutritional status should recognise both the severity of growth impairment and the limitations of a single cross-sectional anthropometric measurement.

In Indonesia, childhood stunting is associated with diverse nutritional, maternal, household, and environmental characteristics. A systematic review and meta-analysis reported associations involving

birth-related factors, maternal care, and household circumstances (3). Population-based studies have also described differences in associated factors across urban and rural populations and socioeconomic settings (4–6). These investigations have largely addressed the presence or absence of stunting, usually by comparing stunted children with children whose height-for-age is within the normal range. Although such evidence is valuable for identifying population-level correlates, it does not necessarily explain differences between moderate and severe stunting among children who are already stunted. This distinction matters when evidence is applied to a selected group of children receiving nutritional services.

Child undernutrition is multidimensional, and anthropometric deficits may coexist. HAZ measures attained linear growth relative to age, weight-for-age z-score (WAZ) measures body weight relative to age, and weight-for-length/height z-score (WHZ) measures weight relative to attained length or height. Underweight, therefore, may occur in a child who is short, thin, or both; it should not be interpreted as equivalent to wasting. A systematic review of wasting and stunting and longitudinal multicountry analyses have highlighted relationships between different growth deficits (7,8). Regional evidence from South and Southeast Asia has likewise documented coexisting forms of malnutrition among young children. Examining more than one anthropometric indicator may thus provide a broader description of nutritional vulnerability than reliance on height-for-age alone (9).

The relationship between WAZ and HAZ nevertheless requires careful interpretation, particularly when analysis is restricted to children who already have both stunting and underweight. Children with more pronounced deficits in height may also have lower weight-for-age because height and weight are biologically and mathematically related. An association between severe underweight and severe stunting may therefore reflect overlapping anthropometric characteristics rather than an independent causal mechanism. Furthermore, WAZ alone cannot establish whether a child has wasting; interpretation would be strengthened by WHZ, mid-upper-arm circumference, dietary information, and longitudinal growth records. In routine service data where these measures are incomplete, a severity-based comparison is most appropriately regarded as an exploratory description of co-occurring deficits (7–9).

Perinatal and individual characteristics provide additional potential explanations for variation in growth outcomes. Low birth weight may reflect prematurity, fetal growth restriction, or both, and it has been examined as a correlate of childhood stunting in Indonesian studies (3,4). A population-based cohort in Sulawesi reported associations of early stunting with small-for-gestational-age birth, prematurity, and short birth length (10). However, a binary low-birth-weight variable cannot distinguish these pathways or establish whether birth conditions are associated specifically with severe rather than moderate stunting among children who already have nutritional deficits. Sex may also be related

to undernutrition through biological and social processes, but the direction and magnitude of reported differences vary between populations (11).

Infectious disease is another biologically plausible correlate of impaired growth because illness may affect appetite, nutrient absorption, inflammation, and nutritional requirements. Research on parasitic infections and gut microbial composition has discussed potential pathways linking infection-related processes to stunting, although findings are heterogeneous and mechanisms are not equivalent across conditions (12,13). Importantly, a routine record indicating whether a child has a history of infectious disease does not establish the type, severity, recurrence, or timing of illness. Such a binary measure can describe a recorded characteristic but cannot support conclusions about specific infectious pathways or their temporal relationship with linear growth impairment.

Dietary adequacy, caregiving, and socioeconomic circumstances provide important background to these individual characteristics. Reviews of Indonesian child nutrition have described shortfalls in nutrient and food intake, while evidence on dietary diversity suggests associations with certain child growth outcomes (14,15). Broader reviews of malnutrition and stunting also highlight maternal, household, social, and environmental influences (16,17). Because these dimensions were not included among the explanatory variables in the present analysis, they represent potential unmeasured influences rather than established explanations for differences between the children studied. Transparent application of anthropometric reference standards is also essential when comparing severity categories (18).

A specific evidence gap remains in describing which routinely recorded characteristics differ between moderate and severe stunting among Indonesian children with concurrent underweight. Most cited Indonesian studies focus on stunting occurrence in broader samples (3–6,10), whereas the present analysis addresses severity within an already affected subgroup. Using February 2026 secondary programme records from East Bolaang Mongondow Regency, this study aimed to examine the associations of weight-for-age category (underweight versus severely underweight), birth-weight status, sex, and recorded infectious-disease history with moderate versus severe stunting among under-five children with both stunting and underweight. Severe underweight was expected to co-occur more frequently with severe stunting, given the overlapping anthropometric structure; the other variables were examined as plausible but context-dependent correlates. The objective was to identify characteristics that may inform comprehensive nutritional assessment and future research, not to infer causal determinants or estimate stunting prevalence in the wider regency population.

MATERIALS AND METHODS

Study Design, Setting, and Data Source

This exploratory cross-sectional analysis used secondary records from the February 2026

SIGIZIKESGA (Sistem Informasi Gizi dan Kesehatan Keluarga) monthly recapitulation in East Bolaang Mongondow Regency, North Sulawesi, Indonesia. The investigators did not directly collect anthropometric measurements. Information on the original measurers, equipment calibration, staff training, quality assurance, and record-validation frequency was not available in the documentation reviewed. Classification error may therefore have occurred, especially around the −3 SD thresholds. The single reporting period precludes evaluation of temporal sequence or progression of stunting severity.

Study Population and Sample

The source population was children aged 0–59 months registered in SIGIZIKESGA in the regency during the reporting period. For this specific analysis, inclusion required concurrent stunting (moderate or severe by HAZ) and underweight (underweight or severe underweight by WAZ). Children lacking either of these two classifications were not part of the analytic comparison. Wasting status was not a criterion for the final analytic comparison. The final analytic sample consisted of 43 children, including 14 with severe stunting. All four displayed 2 × 2 comparisons total 43 observations. The original denominator of potentially eligible records, exclusions and their reasons, and completeness of variables before constructing the analytic tables could not be established from the available aggregate data. Therefore, participant flow and selection-related missingness cannot be fully assessed.

Variables and Operational Definitions

The outcome was severe versus moderate stunting, classified using WHO Child Growth Standards: moderate stunting, $-3 \leq HAZ < -2$; severe stunting, $HAZ < -3$. Weight-for-age status was classified as underweight, $-3 \leq WAZ < -2$, or severe underweight, $WAZ < -3$. Low birth weight was defined as birth weight <2,500 g. Sex was categorised as female or male, and recorded history of infectious disease as no or yes. For analyses, reference categories were underweight, non-low birth weight, female sex, and no history of infectious disease. Weight-for-age is not equivalent to weight-for-height and does not diagnose wasting. Although age, raw anthropometric values, and possibly additional indices were described in the source data, no age-stratified estimates or separate wasting comparisons were presented in the supplied aggregate analysis; parental stature, maternal nutritional status, gestational age, socioeconomic status, and feeding practices were not part of the reported analytic variables.

Collection and Anthropometric Information

Recorded anthropometric classifications, birth-weight status, sex, and a binary history of infectious disease were extracted from the February 2026 monthly recapitulation. The investigators did not undertake independent remeasurement. Descriptions of measurement equipment, observer training, repeat measurement, and data-validation procedures were not available in the source

documentation used for this analysis; therefore, measurement reliability could not be independently assessed. The disease-history field did not identify infection type, frequency, duration, severity, or the interval preceding anthropometric assessment.

Statistical Analysis

Categorical characteristics were described using frequencies and percentages, with within-category percentages in the severity comparison. For each prespecified 2 × 2 comparison, the primary inferential analysis was a two-sided Fisher's exact test owing to sparse counts. Crude odds ratios (ORs) and 95% confidence intervals (CIs) were presented as descriptive association estimates, with severe stunting as the outcome; the associated Wald p-values are shown separately and are not the primary hypothesis tests. A crude prevalence ratio (PR) was also considered for the anthropometric comparison, because severe stunting was not rare in this sample. Analyses were unadjusted: the small number of severe stunting cases did not support reliable multivariable adjustment. No multiple-comparison correction was applied. The nominal $p < 0.05$ threshold is descriptive in this exploratory analysis, and inference must consider wide CIs and selection into the sample.

RESULTS

A total of 43 children were included. Of these, 29 (67.4%) were moderately stunted and 14 (32.6%) were severely stunted. Severe underweight was present in 27 children (62.8%), low birth weight in 8 (18.6%), male sex in 24 (55.8%), and a history of infectious disease in 12 (27.9%) (Table 1). These percentages describe the selected analytic sample and are not population prevalence estimates.

Table 1. Characteristics of Study Participants

Variabel	Category	n=43	(%)
Height-for-Age Nutritional Status (HAZ)	Stunted	29	(67.4)
	Severely stunted	14	(32.6)
Weight-for-Age Nutritional Status (WAZ)	Underweight	16	(37.2)
	Severely underweight	27	(62.8)
Birth Weight	Non-low birth weight	35	(81.4)
	Low birth weight (LBW)	8	(18.6)
Sex	Female	19	(44.2)
	Male	24	(55.8)
History of Infectious Disease	No	31	(72.1)
	Yes	12	(27.9)

Severe stunting was observed in 13 of 27 children with severe underweight (48.1%) and 1 of 16 children with underweight (6.3%). A two-sided Fisher's exact test calculated from these four counts yielded $p = 0.0063$ (reported as $p = 0.006$). Corresponding Fisher p-values were 1.000 for birth-weight status, 0.199 for sex, and 1.000 for reported history of infectious disease (Table 2). The comparisons were unadjusted and exploratory; statistically non-significant findings do not establish that the characteristics are unrelated to severity.

Table 2. Participant Characteristics According to Stunting Severity

Variable	Height-for-age status		p-value
	Moderate stunting	Severe stunting	

	n=29	(%)	n=14	(%)	
Weight-for-Age Nutritional Status (WAZ)					
Underweight	15	(93.8)	1	(6.3)	0.006*
Severely underweight	14	(51.9)	13	(48.1)	
Birth Weight					
Non-low birth weight	24	(68.6)	11	(31.4)	1.000*
Low birth weight (LBW)	5	(62.5)	3	(37.5)	
Sex					
Female	15	(78.9)	4	(21.1)	0.199*
Male	14	(58.3)	10	(41.7)	
History of Infectious Disease					
No history of infectious disease	21	(67.7)	10	(32.3)	1.000*
Yes, history of infectious disease	8	(66.7)	4	(33.3)	

Percentages are calculated within exposure categories (row percentages), with possible rounding differences. P-values are from two-sided Fisher's exact tests; $p < 0.05$ is the exploratory threshold. Fisher's exact results are the primary tests, whereas Table 3 provides descriptive crude odds ratios and Wald-based uncertainty estimates.

Severe underweight was associated with greater crude odds of severe stunting (OR = 13.93; 95% CI: 1.61–120.83). Corresponding crude ORs were 1.31 (95% CI: 0.26–6.48) for low birth weight, 2.68 (95% CI: 0.68–10.53) for male sex, and 1.05 (95% CI: 0.25–4.33) for infection history (Table 3). The crude prevalence ratio comparing severe underweight with underweight was 7.70. Odds ratios must not be interpreted as prevalence or risk ratios, particularly with a common outcome and sparse data.

Tabel 3. Crude Odds Ratios for Severe Stunting by Participant Characteristics

Variable	OR	95% CI	Wald p-value
Severely underweight	13.92	1,61 – 120,83	0.017
Low birth weight (LBW)	1.31	0.26 – 6.49	0.741
Male sex	2.68	0.68 – 10.53	0.158
Presence of infectious disease	1.05	0.25 – 4.33	0.946

OR, odds ratio; CI, confidence interval. All estimates are crude and unadjusted. Reference categories are underweight, non-low birth weight, female sex, and no reported history of infectious disease. The p-values in Table 3 are Wald-based tests for the crude ORs; the prespecified primary comparisons use two-sided Fisher's exact tests in Table 2. The procedures differ and can yield different p-values, particularly with sparse cells.

DISCUSSION

In this sample of 43 children with stunting and underweight, severe underweight was associated with greater stunting severity. The prevalence of severe stunting was 48.1% among children with severe underweight and 6.3% among children with underweight. Both the crude OR and the complementary PR indicated a positive association, but their wide confidence intervals preclude a precise estimate of its magnitude. The study did not identify statistically significant associations for low birth weight, sex, or infectious disease. These findings describe severity within a selected group and should not be extrapolated to determinants of stunting in all children.

The primary finding accords with evidence that anthropometric deficits frequently overlap in young children (19,20), but it addresses a different comparison from many prior studies: the severity of stunting within a group already stunted and underweight, rather than the occurrence of stunting in a general child sample. HAZ and WAZ are related indicators because low attained height can contribute to low weight for a given age. Consequently, the large observed association cannot be taken as proof of an additional independent nutritional pathway. It may partly reflect the mathematical and biological relationships between the indicators. This distinction is especially relevant when the comparison groups were defined using both indices at the same time.

Severe underweight should therefore be interpreted as a concurrent anthropometric finding that prompts broader assessment, not as a proven causal determinant of severe stunting. Weight-for-age combines aspects of stature and mass, and children with lower HAZ may have lower WAZ even without disproportionately low mass for height. Since a weight-for-height or BMI-for-age comparison, mid-upper-arm circumference, and repeated growth measures were not included in the reported analyses, it is not possible to distinguish co-occurring wasting from the influence of short stature on WAZ. Nonetheless, overlapping growth deficits can be clinically relevant, and evidence from longitudinal cohort's links combined anthropometric deficits with adverse outcomes (1,20). No morbidity, mortality, or prospective growth outcome was evaluated here.

Several statistical features also affect interpretation. Only one child in the underweight reference category had severe stunting, making the OR sensitive to small changes in cell counts. The very wide exact confidence interval illustrates this instability. Furthermore, with an outcome frequency of 32.6%, the OR of 13.93 is numerically farther from the null than the PR of 7.70 and must not be described as a 13.93-fold increase in prevalence or risk. Both measures are unadjusted. Restriction to children who were already stunted may also alter associations involving factors that influence entry into that group. The study cannot establish independent determinants or the sequence in which the deficits developed.

Low birth weight was not statistically associated with severe stunting in this selected sample, but only eight children had low birth weight, leaving a broad confidence interval. In contrast, Gusnedi et al. (3) and Siramaneerat et al. (4) investigated associations with the occurrence of childhood stunting in broader Indonesian populations. A Sulawesi cohort also linked gestational and birth-size characteristics with early stunting (10). Those comparisons do not directly address severity among children who are already stunted and underweight and therefore do not conflict with the present imprecise result. The binary birth-weight measure also cannot distinguish prematurity from fetal growth restriction.

Boys had higher crude odds of severe stunting than girls, but the estimate was imprecise (OR=2.68; 95% CI: 0.68–10.53). Thurstans et al. describe interacting biological and social

explanations for greater male vulnerability in some settings, while pooled longitudinal analyses also found higher growth-faltering risk among boys(1,11). However, Indonesian findings vary, including a Sulawesi cohort that reported higher odds of early-infant stunting among girls (10). Thus, the present result neither establishes a sex difference nor demonstrates equivalence. The small sample does not support sex-specific programme recommendations.

No statistically significant association was observed for the recorded binary infection-history field. This should not be interpreted as evidence that infection is irrelevant to child growth, because the available measure does not distinguish infection types or capture frequency, duration, severity, subclinical disease, or timing before growth measurement. Literature on parasitic infection describes biologically plausible mechanisms involving nutritional losses, anaemia, and intestinal function (12), while a systematic review of gut microbiome composition reports heterogeneous associations with childhood stunting (13). Such findings provide context for hypotheses about infection-related pathways but cannot be attributed to the children in this dataset. Repeated and more specific morbidity ascertainment would be necessary to test these pathways locally.

For local services, concurrent severe underweight and stunting can justify a comprehensive nutritional and clinical assessment rather than a diagnosis of severe acute malnutrition on WAZ alone. Age-appropriate weight-for-length/height or BMI-for-age, mid-upper-arm circumference, feeding history, diet quality, and relevant clinical symptoms may clarify concurrent deficits. Evidence from an uncontrolled local food-based supplementary feeding evaluation (21), is relevant to programme context but cannot establish causal intervention effectiveness. Reviews reporting dietary gaps (14,15,22), further support broad assessment rather than reliance on a single anthropometric category.

Household and environmental conditions should be considered when planning follow-up, although they were not measured here. Indonesian evidence links food insecurity with several forms of childhood malnutrition, and socioeconomic analyses show that associations vary across population groups (5,23). Multicountry research also associates food-price increases with undernutrition, while regional analyses identify geographic and environmental variation in anthropometric deficits (24,25). These findings suggest that dietary counselling may need to be accompanied by assessment of food access and living conditions. They do not establish which household constraints were present in this sample.

Evidence for nutritional interventions should be distinguished from evidence of association. A meta-analysis of randomized trials found that preventive small-quantity lipid-based nutrient supplements reduced severe stunting among young children, while a separate synthesis did not find clear additional growth benefits from medium-quantity supplements (26,27). An international commentary discusses the programme implications of expanding access to small-quantity

supplements in appropriate settings (28). These findings support evaluation of context-appropriate preventive strategies, but they do not demonstrate that supplementation would reverse severe stunting in the present participants. This study did not test an intervention.

Future research should include maternal and paternal stature alongside repeated anthropometry and dietary assessment. An Indonesian meta-analysis found associations between parental stature and child stunting, although estimates involving paternal or combined parental stature were heterogeneous and imprecise (29). Broader reviews similarly describe interacting maternal, household, and care-related determinants. These variables would help distinguish shared familial or environmental influences from additional nutritional deficits and clarify which children need particular forms of support (16,30).

The study has substantial limitations. First, its 43 observations and sparse cells yielded imprecise effect estimates; in particular, only one child with underweight (rather than severe underweight) had severe stunting. Second, conditioning on simultaneous stunting and underweight restricts generalisability and may introduce selection effects. Third, the analyses were crude, with no adjustment for age, maternal or paternal stature, gestational age, maternal nutrition, dietary intake, food security, or socioeconomic and environmental conditions. Fourth, the recruitment denominator, exclusions, missing-data processes, anthropometric quality assurance, and provenance of birth-weight and infection-history records were not fully documented in the material available for this revision. Fifth, the cross-sectional design does not establish temporality, and the four exploratory tests were not adjusted for multiplicity. These results should not be used as causal, prognostic, or intervention-effectiveness estimates.

The use of routinely recorded local programme data, explicit WHO-based anthropometric definitions, and a clearly stated severity comparison are strengths for describing this particular service population. Nevertheless, they do not eliminate measurement error, selection bias, sparse-data uncertainty, or the need for independent validation

CONCLUSION

Among 43 under-five children with both stunting and underweight in East Bolaang Mongondow Regency, severe underweight co-occurred more frequently with severe than moderate stunting (crude OR = 13.93; 95% CI: 1.61–120.83; two-sided Fisher's exact p = 0.006). Low birth weight, sex, and recorded history of infectious disease were not statistically associated with severity in these unadjusted comparisons, but the estimates were imprecise. The primary association should be interpreted considering the shared anthropometric structure of WAZ and HAZ and the selected sample, rather than as evidence of causation. Comprehensive clinical and dietary assessment may be appropriate when multiple growth deficits are recorded. Larger, better-documented longitudinal

studies with relevant confounder adjustment are needed to determine whether these patterns persist and carry independent clinical significance.

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