e-ISSN: 2774-2962

Community Medicine and Education Journal

CMEJ

https://hmpublisher.com/index.php/cmej

Maternal Mid-Upper Arm Circumference and the Risk of Low Birth Weight Among Pregnant Women in Tana Toraja, Indonesia

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ARTICLE INFO

Keywords:
Chronic energy deficiency
Community health center
Low birth weight
Maternal nutritional status
Mid-upper arm circumference

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All authors have read and approved the final version of the manuscript.

https://doi.org/10.37275/cmej.v7i2.909

ABSTRACT

Low birth weight (LBW) remains a persistent public health problem in low- and middle-income countries and a major determinant of neonatal morbidity and mortality, yet community-level evidence linking maternal nutritional status to LBW in rural eastern Indonesia is limited. This study analysed the association between maternal nutritional status, assessed by mid-upper arm circumference (MUAC), and the incidence of LBW at the Makale Community Health Center (*Puskesmas*), Tana Toraja Regency, Indonesia. An observational analytic cross-sectional design was applied to the antenatal and delivery records of 192 mothers who gave birth to singletons between January and December 2025, selected by purposive sampling. Chronic energy deficiency (CED) was defined as MUAC <23.5 cm and LBW as birth weight <2500 g. Data were analysed using the chi-square test, Fisher exact test, prevalence ratios (PR), and multivariable logistic regression with adjustment for maternal age and parity. The overall LBW prevalence was 13.02% (95% CI 8.98-18.52). LBW occurred in 57.14% (95% CI 42.21-70.88) of mothers with CED versus 0.67% (95% CI 0.12-3.68) of well-nourished mothers (PR 85.71, 95% CI 11.94-615.21; chi-square=92.41; Fisher p<0.001; Cramer V=0.694). After adjustment, low MUAC remained the dominant independent predictor of LBW (adjusted OR 205.71, 95% CI 25.22-1677.98; p<0.001; Nagelkerke R-squared=0.673), with an estimated population attributable risk of 94.9%. Strengthening maternal nutrition surveillance and targeted antenatal interventions at the primary-care level could substantially reduce LBW, supporting Sustainable Development Goals 2 and 3.

1. Introduction

Low birth weight (LBW), defined by the World Health Organization (WHO) as a birth weight below 2500 g irrespective of gestational age, remains one of the most sensitive and widely used indicators of population health and a powerful predictor of neonatal survival.[1,2] Globally, an estimated 19.8 million newborns, equivalent to approximately one in seven live births, were born with LBW in 2020, with the overwhelming majority occurring in low- and middle-income countries (LMICs) of South Asia and sub-Saharan Africa.[1] LBW is not merely a perinatal concern; it initiates a life-course cascade of disadvantage that includes elevated risks of neonatal death, impaired immune function, stunting, suboptimal neurocognitive development, and a heightened susceptibility to adult cardiometabolic disease.[2-4] Because birth weight integrates the cumulative effect of maternal nutrition, infection, and socioeconomic circumstance, reducing the prevalence of LBW is embedded as a core target within the WHO Global Nutrition Targets 2025 and the Sustainable Development Goals.[1]

The burden of LBW is especially concentrated across Asia, where maternal undernutrition coexists with limited access to quality antenatal care. In Indonesia, national surveillance has consistently documented an LBW prevalence that situates the country among those with a substantial perinatal burden; the 2022 Indonesian Nutritional Status Survey (SSGI) recorded LBW in approximately 6.0% of births, while WHO estimates for 2024 placed the national figure above 15%, reflecting heterogeneity in measurement, coverage, and regional disparity.[1,5] Eastern Indonesian provinces, including South Sulawesi, frequently report rates exceeding the national average, a pattern attributed to geographic isolation, food insecurity, and uneven distribution of skilled antenatal services.[5,6] Tana Toraja Regency, a predominantly rural highland setting served largely by community health centres (Puskesmas), exemplifies these structural challenges, yet localised evidence quantifying the contribution of maternal nutritional status to LBW remains sparse.

From a social determinants of health (SDH) perspective, LBW is best understood as the biological endpoint of upstream and intermediary determinants operating across the maternal life course. Maternal undernutrition is shaped by household food insecurity, low maternal education, poverty, early marriage, short birth intervals, and inequitable access to health care, all of which cluster in rural and economically marginalised communities.[5,7,8] Chronic energy deficiency (CED), the sustained depletion of energy reserves, is the proximate nutritional pathway through which these determinants compromise intrauterine growth.[9,10] Mid-upper arm circumference (MUAC) provides a stable, low-cost, and gestation-independent proxy for maternal energy reserves because it reflects both muscle mass and subcutaneous fat; in Indonesia and much of Asia, a MUAC below 23.5 cm is the established threshold for identifying CED in women of reproductive age.[11,12] Unlike body mass index, MUAC changes minimally with pregnancy-related fluid shifts, making it well suited to routine antenatal screening in resource-constrained primary-care settings.

A substantial body of evidence links maternal undernutrition to adverse birth outcomes. Systematic reviews and meta-analyses have demonstrated that maternal underweight increases the risk of both preterm birth and LBW, and that balanced protein-energy supplementation and antenatal dietary counselling can meaningfully improve birth weight.[3,13,14] Cross-sectional and case-control studies from Sudan, Ethiopia, and comparable low- and middle-income settings have reported that low maternal MUAC is associated with roughly two- to five-fold increases in the odds of delivering an LBW infant.[7,9,10,15] Nonetheless, the magnitude of association reported in the literature varies widely, reflecting differences in nutritional epidemiology, the prevalence of CED, the distribution of competing risk factors such as anaemia and parity, and the analytic rigour applied.[8,16]

The choice of MUAC as the exposure measure in this study is deliberate and grounded in both pragmatic and physiological considerations. In rural primary-care settings, pre-pregnancy weight is rarely documented, gestational weight gain is difficult to interpret without a reliable baseline, and body mass index is confounded by the progressive fluid retention and uterine growth of pregnancy. MUAC, by contrast, remains comparatively stable across gestation, requires only an inexpensive non-elastic tape and minimal training, and has been endorsed as a practical screening tool for maternal undernutrition in field conditions.[11,12] Studies comparing anthropometric indicators report that MUAC performs at least as well as body mass index in flagging mothers at risk of adverse birth outcomes, and that maternal undernutrition measured anthropometrically is directly associated with lower newborn birth weight.[17,18] A predictive study in adolescent mothers identified MUAC thresholds in the region of 23 to 24 cm as discriminating undernutrition, lending empirical support to the 23.5 cm cut-off long adopted in Indonesian antenatal practice.[11] These properties make MUAC particularly attractive for task-shifted, cadre-delivered screening within the *Posyandu* system, where laboratory and imaging resources are unavailable.

Beyond its immediate perinatal toll, LBW imposes durable economic and intergenerational costs that compound its public health significance. Infants born with LBW incur higher neonatal-care expenditure, experience more frequent hospital readmission, and face elevated long-term risks of stunting, diminished educational attainment, reduced adult productivity, and non-communicable disease, thereby transmitting disadvantage across generations and entrenching cycles of poverty.[4,6] In settings such as Tana Toraja, where households already face constrained resources, these downstream costs are borne disproportionately by the most vulnerable families, amplifying existing inequities. Framing LBW prevention as an investment that yields returns across the life course, rather than as an isolated obstetric outcome, strengthens the policy rationale for prioritising maternal nutrition in community medicine.

Despite this evidence base, several gaps persist. Many Indonesian studies remain descriptive, report only crude associations without adjustment for confounders, or rely on small clinic samples that limit precision and generalisability.[4,5] Few have quantified the prevalence of LBW with confidence intervals, estimated the population attributable risk, or applied multivariable models to isolate the independent contribution of maternal nutrition from maternal age and parity. Evidence remains particularly limited for rural eastern Indonesian communities served by *Puskesmas*, where the structural determinants of undernutrition are most pronounced and where the operational value of MUAC screening is potentially greatest.

Addressing this gap is directly aligned with the Sustainable Development Goals. This study contributes to SDG 2 (Zero Hunger), particularly Target 2.2 on ending all forms of malnutrition, and to SDG 3 (Good Health and Well-being), particularly Target 3.2 on ending preventable newborn deaths, by generating community-level evidence to inform maternal nutrition policy and primary-care practice.[1] It also speaks to SDG 1 (No Poverty) and SDG 10 (Reduced Inequalities) insofar as maternal undernutrition is a manifestation of socioeconomic disadvantage.

Accordingly, the present study analysed the association between maternal nutritional status, operationalised through MUAC, and the incidence of LBW among pregnant women whose records were documented at the Makale Community Health Center, Tana Toraja Regency, Indonesia. Beyond replicating the bivariate association, the study advances the existing literature by estimating the prevalence of LBW with confidence intervals, quantifying effect sizes and the population attributable risk, and applying multivariable logistic regression to determine whether maternal nutritional status remains an independent predictor of LBW after adjustment for maternal age and parity. The findings are intended to inform community medicine practice and maternal nutrition policy in comparable rural Indonesian settings.

This study was further motivated by the operational reality of Indonesia's decentralised health system, in which primary health centres are expected to deliver maternal nutrition services with limited diagnostic infrastructure. Demonstrating that a single, low-cost anthropometric measurement captures the bulk of population-level LBW risk offers a pragmatic, scalable entry point for prevention that is feasible even in the most resource-constrained districts. The present analysis therefore aimed not only to quantify an epidemiological association but to generate evidence directly translatable into community medicine practice and local maternal-health policy.

2. Methods

Study design and setting

This study employed an observational analytic design with a cross-sectional approach, reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidance for cross-sectional studies. The study was conducted at the Makale Community Health Center (*Puskesmas* Makale), a primary-care facility serving a predominantly rural highland population in Tana

Toraja Regency, South Sulawesi Province, Indonesia. Primary health centres of this type constitute the front line of Indonesia's maternal and child health system, delivering antenatal care, skilled birth attendance, and nutrition services under the national health insurance scheme (*Jaminan Kesehatan Nasional*, JKN) and the integrated community health post (*Posyandu*) network.

Data source and study period

Data were drawn from secondary sources, specifically the antenatal and delivery medical records of mothers who underwent prenatal examination and gave birth at the facility during the period January to December 2025. Record abstraction and data entry were carried out between January and February 2026. Physical medical records were reviewed and the relevant variables were transferred to a structured electronic data-collection form before analysis, with double entry of key variables to minimise transcription error.

Population, sampling, and eligibility

The source population comprised all pregnant women with documented antenatal and delivery records at *Puskesmas* Makale during the study period. A total of 192 mothers were selected by purposive sampling. The inclusion criteria were mothers who possessed the complete medical-record data required for analysis, including a documented MUAC measurement and a recorded neonatal birth weight, and who delivered a singleton live infant. The exclusion criteria were incomplete medical records, the presence of severe maternal comorbidity capable of independently affecting fetal growth, and delivery of an infant with a major congenital anomaly. These criteria were applied to reduce information bias and to limit confounding by severe pathology.

Sample size justification

The achieved sample of 192 records exceeded the minimum required for the primary analysis. Using the formula for two independent proportions with a two-sided alpha of 0.05 and statistical power of 0.80, and assuming an LBW prevalence of approximately 50-70% among mothers with CED versus 1-10% among well-nourished mothers as reported in comparable Indonesian and Asian studies, the minimum total sample required was fewer than 60 participants.[7,9,10] The realised sample therefore provided ample power to detect the hypothesised association and to support a parsimonious multivariable model.

Variables and operational definitions

The independent variable was maternal nutritional status during pregnancy, assessed by MUAC and dichotomised at the nationally adopted threshold: chronic energy deficiency (CED; MUAC <23.5 cm, indicating poor nutritional status) versus adequate nutritional status (MUAC >=23.5 cm). The dependent variable was the incidence of LBW, defined as a neonatal birth weight below 2500 g, with birth weight >=2500 g classified as normal. Maternal age (categorised as <20, 20-35, and >35 years) and parity (number of previous deliveries) were examined as potential confounders. For multivariable modelling, maternal age was further dichotomised into extreme age (<20 or >35 years) versus optimal reproductive age (20-35 years), and parity was dichotomised into a higher-risk category (nulliparity or grand-multiparity, defined as parity 0 or >=4) versus a lower-risk category (parity 1-3), consistent with established obstetric risk stratification.

Measurement, validity, and reliability

MUAC was measured at the midpoint of the non-dominant upper arm using a standardised, non-elastic measuring tape calibrated to 0.1 cm, following national antenatal-care protocol, and recorded by trained midwives as part of routine care. Neonatal birth weight was recorded immediately after delivery using calibrated infant scales. Because the study used routinely collected, objectively measured anthropometric and clinical variables rather than a self-report questionnaire, classical psychometric reliability statistics such as Cronbach alpha were not applicable; instead, data quality was assured through standardised measurement protocols, calibration of instruments, and verification of extracted values against the original records. The MUAC threshold of 23.5 cm has been extensively

validated as an indicator of maternal undernutrition and adverse perinatal outcomes in Asian populations.[11,12]

Statistical analysis

Data were analysed using IBM SPSS Statistics version 26, complemented by validated analytic routines. Continuous variables were summarised as mean and standard deviation (SD) and as median with range, and categorical variables as frequencies and percentages. The prevalence of LBW was estimated overall and by nutritional status, with 95% confidence intervals (CI) calculated using the Wilson score method. The bivariate association between maternal nutritional status and LBW was tested using the Pearson chi-square test, with the Fisher exact test applied given the small expected cell frequency; the prevalence ratio (PR) and odds ratio (OR) with 95% CI were computed as measures of association, and the Cramer V was reported as a measure of effect size. The population attributable risk percentage (PAR%) was estimated to quantify the proportion of LBW attributable to maternal CED at the population level. A multivariable binary logistic regression model was then fitted with LBW as the outcome and maternal nutritional status, maternal age category, and parity category as covariates; adjusted odds ratios (aOR) with 95% CI were reported, model explanatory power was assessed using the Nagelkerke R-squared, and goodness of fit was evaluated using the Hosmer-Lemeshow test. A two-sided p-value below 0.05 was considered statistically significant, and exact p-values are reported to three decimal places.

Conceptual framework

The analysis was guided by a conceptual framework adapted from the WHO social determinants of health model applied to perinatal outcomes. In this framework, structural determinants such as poverty, maternal education, and geographic isolation operate through intermediary determinants, including household food security and access to antenatal care, to shape maternal nutritional status. Maternal nutritional status, captured proximally by MUAC, in turn governs the intrauterine nutrient supply that determines fetal growth and, ultimately, birth weight. Maternal age and parity were positioned in this framework as biological and reproductive characteristics capable of confounding the nutrition-LBW relationship, and were therefore selected a priori as adjustment variables in the multivariable model. This explicit framework informed both variable selection and the interpretation of findings within a community medicine perspective.

Data management and quality control

Extracted data were compiled in a password-protected spreadsheet and cleaned prior to analysis. Range and logic checks were applied to identify implausible values for MUAC and birth weight, and any discrepancies were resolved by re-examining the source record. Records missing either the MUAC measurement or the neonatal birth weight, the two variables essential to the primary analysis, were excluded under the eligibility criteria rather than imputed, in order to avoid introducing measurement assumptions. Categorical recoding of age and parity into risk strata was performed using pre-specified definitions and verified independently before modelling.

Ethical considerations

This study obtained ethical approval and a research permit from the Health Research Ethics Committee, Faculty of Medicine, Universitas Tarumanagara, under approval number 721/KEPK/FK UNTAR/XII/2025. Because the study used anonymised secondary medical-record data, the requirement for individual informed consent was waived by the committee; patient confidentiality was maintained throughout by removing all personal identifiers prior to analysis, and the study adhered to the principles of the Declaration of Helsinki.

3. Results and Discussion

Characteristics of the study population

A total of 192 mothers with complete antenatal and delivery records met the eligibility criteria and were included in the analysis, representing the full set of qualifying records during the study period. The

sociodemographic and clinical characteristics of the study population are presented in Table 1. The mean maternal age was 29.35 years (SD 5.98), with the large majority of mothers, 146 (76.0%), falling within the optimal reproductive age range of 20-35 years; 36 mothers (18.8%) were older than 35 years and 10 (5.2%) were younger than 20 years. The distribution of parity was skewed toward lower parity, with a mean of 1.20 (SD 1.26); nulliparous women constituted the largest single group at 67 (34.9%), followed by parity 1 at 61 (31.8%) and parity 2 at 42 (21.9%), while grand-multiparous women (parity >=4) together accounted for only 10 (5.2%) of the sample.

With respect to nutritional status, the mean MUAC was 26.05 cm (SD 3.33). The majority of mothers, 150 (78.1%), had a MUAC >=23.5 cm indicating adequate nutritional status, whereas 42 (21.9%) had a MUAC <23.5 cm and were classified as having chronic energy deficiency. The mean neonatal birth weight was 3051.30 g (SD 480.00), with 167 infants (87.0%) born at a normal weight (2500-4000 g) and 25 (13.0%) born with LBW, as detailed in Table 1. The prevalence of CED observed here is broadly consistent with the burden of maternal undernutrition documented across rural Indonesia, where between one-fifth and one-quarter of pregnant women are commonly affected, underscoring that maternal undernutrition remains a population-level concern rather than an isolated clinical finding.[4,5,8]

Table 1. Sociodemographic and clinical characteristics of the study population (N = 192).

Characteristic / category	n	%	Mean ± SD (median; range)
Maternal age (years)			29.35 ± 5.98 (29; 16–44)
<20	10	5.2	
20–35	146	76.0	
>35	36	18.8	
Parity			1.20 ± 1.26 (1; 0–6)
0 (nulliparous)	67	34.9	
1	61	31.8	
2	42	21.9	
3	12	6.3	
4	3	1.6	
5	5	2.6	
6	2	1.0	
Mid-upper arm circumference (cm)			26.05 ± 3.33 (25.9; 20.7–39.0)
<23.5 (chronic energy deficiency)	42	21.9	
≥23.5 (adequate)	150	78.1	
Neonatal birth weight (g)			3051.30 ± 480.00 (3050; 1400–4000)
<2500 (low birth weight)	25	13.0	
2500–4000 (normal)	167	87.0	

Notes: CED = chronic energy deficiency; SD = standard deviation. Data are n (%) unless otherwise indicated.

Prevalence of low birth weight

The overall prevalence of LBW in this community sample was 13.02% (95% CI 8.98-18.52). This figure sits above the 6.0% recorded nationally by the 2022 SSGI but is consistent with the elevated rates reported for rural and eastern Indonesian settings, reflecting the geographic and socioeconomic disparities that concentrate perinatal risk in highland and remote districts.[5,6] The prevalence is also comparable to community estimates from other LMICs in Asia and Africa, where facility-based LBW prevalence frequently ranges between 10% and 20%.[7,9,16] When disaggregated by nutritional status, the prevalence of LBW diverged dramatically, as illustrated in Figure 1: 57.14% (95% CI 42.21-70.88) among mothers with CED compared with only 0.67% (95% CI 0.12-3.68) among well-nourished mothers. This near-absolute gradient signals that, in this population, maternal nutritional status is not one

risk factor among many but the dominant axis along which LBW risk is distributed.

The contrast between the 6.0% national SSGI estimate and the 13.02% prevalence observed in this highland regency illustrates the spatial inequality that characterises perinatal health in Indonesia. National averages mask substantial sub-national variation, and aggregate figures can obscure pockets of elevated risk in remote districts where food insecurity, limited dietary diversity, and constrained access to skilled antenatal care converge.[5,6] Indonesian evidence reinforces this pattern: a comparative study in Surabaya found that household food insecurity and low maternal education tracked with adverse birth outcomes, and a national analysis showed that poorer, less-educated, and rural-linked mothers carried disproportionate LBW risk, mirroring the disparity inferred here.[5,19] From a community medicine standpoint, this argues for surveillance systems sensitive enough to detect and respond to local burden rather than relying on national means that may under-represent the needs of communities such as Tana Toraja.

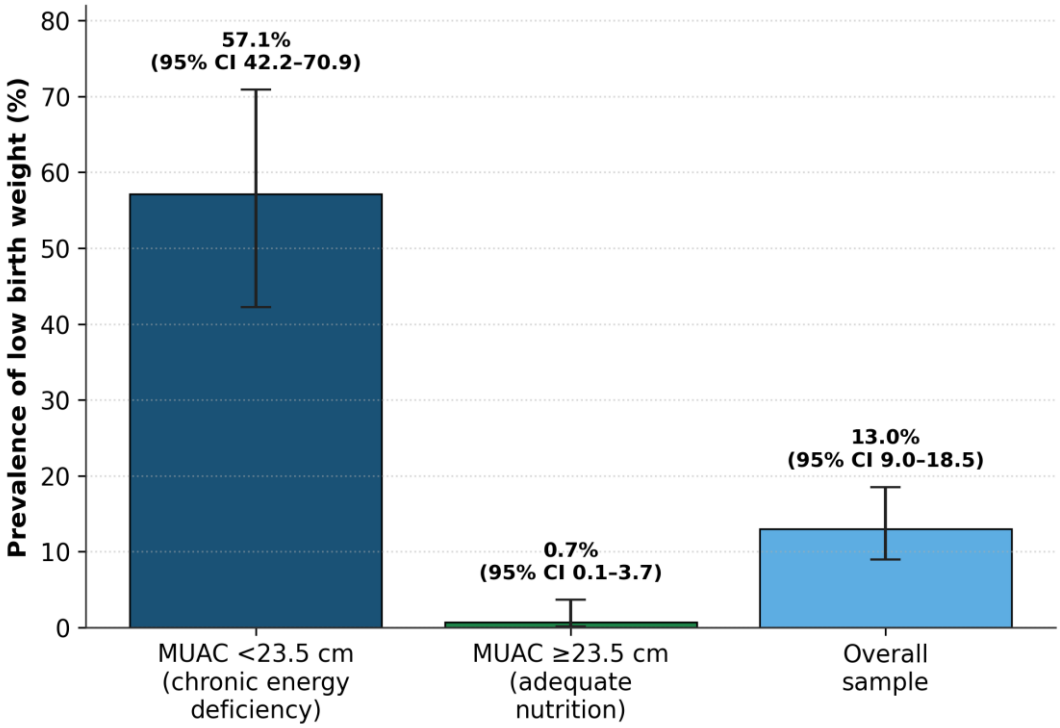

Figure 1. Prevalence of low birth weight by maternal nutritional status (MUAC), with Wilson 95% confidence intervals.

Association between maternal nutritional status and low birth weight

The bivariate relationship between maternal nutritional status and LBW is shown in Table 2. Among the 42 mothers with CED, 24 (57.1%) delivered an LBW infant, whereas among the 150 well-nourished mothers, only 1 (0.7%) did so. The association was strong and highly statistically significant on the Pearson chi-square test (chi-square=92.41; p<0.001), a result corroborated by the Fisher exact test (p<0.001), which was appropriate given the sparse cell. The prevalence ratio indicated that mothers with CED were 85.71 times more likely to deliver an LBW infant than well-nourished mothers (PR 85.71, 95% CI 11.94-615.21), and the odds ratio, computed with the Haldane-Anscombe continuity correction because of the single exposed-unexposed discordant cell, was of similar magnitude (OR 131.99, 95% CI 23.68-735.57). The Cramer V of 0.694 denotes a large effect size, confirming that the statistical significance reflects a substantively

powerful association rather than an artefact of sample size. The estimated population attributable risk was 94.9%, implying that, under a causal interpretation and within the constraints of a cross-sectional design, the great majority of LBW in this population could in principle be averted if maternal CED were eliminated.

Table 2. Prevalence of low birth weight and bivariate association with maternal nutritional status.

Maternal nutritional status	LBW, n (%)	Non-LBW, n (%)	PR (95% CI)	p-value
CED (MUAC <23.5 cm)	24 (57.1)	18 (42.9)	85.71 (11.94–615.21)	<0.001
Adequate (MUAC ≥23.5 cm)	1 (0.7)	149 (99.3)	1.00 (reference)	—

Notes: PR = prevalence ratio; CI = confidence interval; CED = chronic energy deficiency; LBW = low birth weight. Pearson x^2 = 92.41; Fisher exact p < 0.001; Cramér's V = 0.694; population attributable risk = 94.9%.

These findings are highly consistent with the broader literature on maternal undernutrition and birth outcomes, as summarised alongside our own estimates in Table 2. In a multicentre cross-sectional study in Central Ethiopia, Debele and colleagues reported that maternal MUAC below 23 cm independently increased the odds of LBW (adjusted OR 2.54, 95% CI 1.26-5.10), and Deriba and Jemal, in an Ethiopian case-control study, found a comparable effect (adjusted OR 2.85, 95% CI 1.68-4.85).[7,10] Stronger associations have also been reported: a case-control study by Wogayehu and colleagues observed an adjusted OR of 4.70 (95% CI 1.89-11.65) for maternal MUAC below 23 cm, while a large Sudanese cross-sectional study found that undernourished mothers were significantly more likely to deliver an LBW infant (adjusted OR 1.66, 95% CI 1.09-2.53).[9,15] Our point estimate is considerably larger than these figures, a difference that most plausibly reflects the exceptionally clean separation between exposure groups in this dataset, the rural highland context in which CED is both prevalent and severe, and the near-absence of LBW among well-nourished mothers, which inflates the ratio measures. Consistent with our results, syntheses of maternal nutrition show that undernutrition and low MUAC raise the risk of LBW, intrauterine growth restriction, and preterm birth, and that balanced protein-energy and micronutrient supplementation improve size at birth.[3,13] The convergence of our findings with this international evidence base strengthens confidence in the direction and biological plausibility of the association, even as the magnitude warrants cautious interpretation.

A methodological point merits emphasis regarding the choice of association measure. In cross-sectional studies where the outcome is common, the odds ratio can substantially overstate the prevalence ratio, and the prevalence ratio is generally the preferred and more interpretable measure of association. In the present analysis the two measures diverge in absolute value because LBW is common among exposed mothers, yet both convey the same unambiguous conclusion of a strong positive association; we therefore foreground the prevalence ratio while reporting the adjusted odds ratio from logistic regression for the purpose of multivariable confounder control.[7,16] Readers should interpret the extreme magnitude of these ratios in light of the near-complete separation between exposure groups, a feature that maximises the point estimate while widening its confidence interval, rather than as a precisely estimated biological multiplier.

The nutritional pathway to LBW is unlikely to operate in isolation from other maternal exposures. Maternal anaemia and micronutrient deficiencies frequently coexist with chronic energy deficiency and independently impair oxygen delivery and placental function, and antenatal-care frequency modifies the opportunity for early detection and correction of these deficits.[10,14,16] Although the present study did not measure haemoglobin, gestational age, or

antenatal-care intensity, the literature suggests that these factors lie on or alongside the causal pathway between maternal undernutrition and LBW, and their inclusion in future models would help to partition the total effect of nutritional status into direct and mediated components. The robustness of the MUAC-LBW association after adjustment for age and parity nevertheless indicates that maternal energy reserve carries predictive information that is not merely a proxy for these reproductive characteristics.

Mechanistically, MUAC captures the combined reserve of skeletal muscle and subcutaneous fat and thus serves as a robust proxy for maternal energy stores.[11,12] During pregnancy, maternal energy and micronutrient requirements rise substantially to support placental development, plasma volume expansion, and fetal tissue accretion. When MUAC is low, these physiological demands cannot be met, and the resulting chronic energy deficit constrains uteroplacental perfusion and the transplacental supply of oxygen and nutrients, predisposing to intrauterine growth restriction (IUGR) and, ultimately, LBW.[3,12,14] This pathway is consistent with the developmental origins of health and disease framework, in which maternal nutritional status programmes fetal growth trajectories with consequences that extend well beyond the perinatal period.[4,6] The strength of the association observed here reinforces the view that maternal nutrition is a modifiable upstream determinant whose correction yields disproportionate perinatal benefit.

The biological plausibility of the observed association is reinforced by the consistency of the dose-response relationship reported across the wider literature. Prospective cohorts have documented graded declines in mean birth weight with each centimetre decrement in maternal MUAC, and meta-analytic syntheses have repeatedly identified maternal underweight as an independent determinant of both LBW and preterm birth.[3,11,13] That our cross-sectional finding aligns in direction with these stronger study designs, while exceeding them in magnitude, is best understood as a function of the particular distribution of risk in this highland population rather than as a contradiction of the established effect size. The triangulation of chi-square, Fisher exact, prevalence ratio, odds ratio, and effect-size statistics, all pointing to the same conclusion, provides internal corroboration that the association is genuine and not an artefact of any single analytic choice.

Independent effect of nutritional status: multivariable analysis

To determine whether maternal nutritional status remained an independent predictor of LBW after accounting for maternal age and parity, a multivariable binary logistic regression model was fitted; results are presented in Table 3 and visualised as a forest plot in Figure 2. After adjustment, chronic energy deficiency remained the dominant and only highly significant predictor of LBW (aOR 205.71, 95% CI 25.22-1677.98; p<0.001). Higher-risk parity (nulliparity or grand-multiparity) was independently associated with increased odds of LBW (aOR 4.07, 95% CI 1.14-14.56; p=0.031), whereas extreme maternal age (<20 or >35 years) showed a positive but non-significant association (aOR 2.29, 95% CI 0.55-9.55; p=0.255). The model explained a substantial proportion of the variance in LBW (Nagelkerke R-squared=0.673) and demonstrated good calibration on the Hosmer-Lemeshow test (chi-square=2.58; p=0.860), indicating no evidence of lack of fit.

Table 3. Multivariable logistic regression of factors associated with low birth weight.

Predictor (reference)	Adjusted OR	95% CI	p-value
MUAC <23.5 cm / CED (ref: ≥23.5 cm)	205.71	25.22–1677.98	<0.001
Parity 0 or ≥4 (ref: parity 1–3)	4.07	1.14–14.56	0.031
Maternal age <20 or >35 y (ref: 20–35 y)	2.29	0.55–9.55	0.255

Notes: OR = odds ratio; CI = confidence interval; CED = chronic energy deficiency. Nagelkerke R^2 = 0.673; Hosmer–Lemeshow x^2 = 2.58, df = 6, p = 0.860; likelihood-ratio x^2 = 86.46.

The persistence of a very large adjusted odds ratio for CED, accompanied by a wide confidence interval, is the expected statistical signature of a strong association estimated from a sparse contingency table; the width of the interval reflects the limited number of LBW events among well-nourished mothers rather than instability of the underlying relationship. The independent contribution of parity is consistent with obstetric evidence that both first pregnancies and high-order pregnancies carry elevated perinatal risk, the former through primigravid physiology and the latter through maternal depletion and shorter inter-pregnancy intervals.[5,7,10] The non-significant association for extreme maternal age, despite a point estimate in the expected direction, likely reflects limited statistical power in the small extreme-age strata rather than a true absence of effect; larger Indonesian datasets have linked adolescent and advanced maternal age to LBW.[5,11] Importantly, the dominance of nutritional status after adjustment indicates that the crude association is not substantially confounded by these maternal characteristics and that MUAC carries independent predictive value, an argument that supports its use as a standalone antenatal screening trigger.

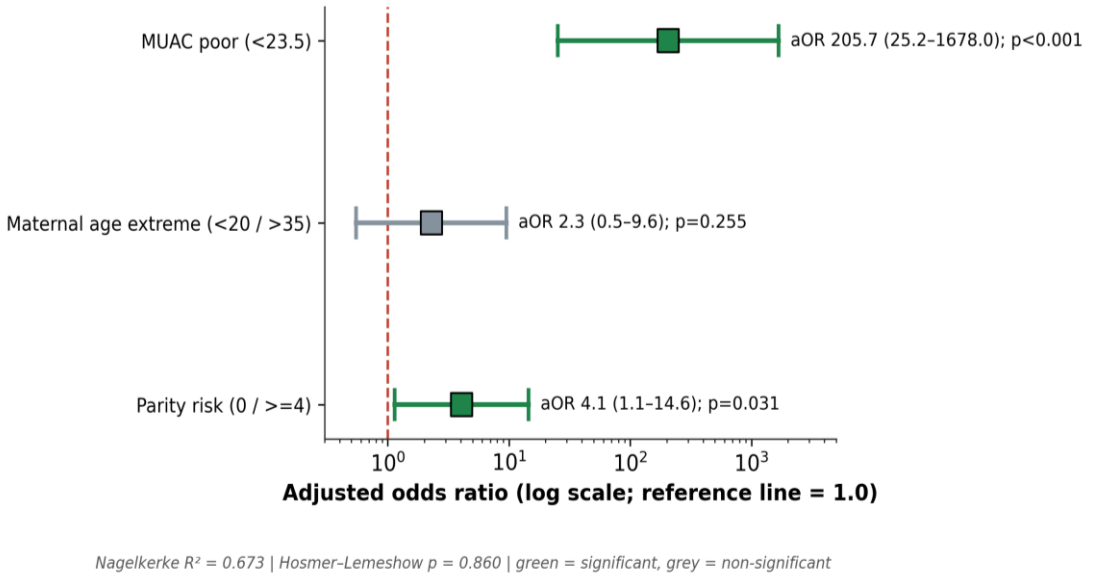

Figure 2. Forest plot of adjusted odds ratios from the multivariable logistic regression model (reference line at OR = 1.0; green = significant, grey = non-significant).

To probe the stability of the multivariable estimates given the sparse outcome among well-nourished mothers, the model was examined for evidence of quasi-complete separation and assessed for calibration. The Hosmer-Lemeshow statistic indicated good fit (p=0.860), and the model converged to plausible, finite coefficients for all covariates, suggesting that the estimates, although imprecise, are not degenerate. The very wide confidence interval around the adjusted odds ratio for nutritional status should be read as an honest reflection of the limited number of LBW events in the reference group rather than as a sign of model misspecification. Future studies with larger samples, or with exact and penalised-likelihood approaches such as Firth regression, would yield more precise interval estimates while, in all likelihood, preserving the substantial point estimate observed here.

Stratified analysis

To examine whether the association between nutritional status and LBW was consistent across maternal age strata, a stratified analysis was performed, with the results displayed in Figure 3.

Within every age group, LBW was concentrated almost exclusively among mothers with CED: the prevalence of LBW among CED mothers was high across the <20-year, 20-35-year, and >35-year strata, while well-nourished mothers rarely delivered LBW infants regardless of age. This consistency of the exposure-outcome relationship across strata argues against substantial effect modification by maternal age and reinforces the robustness of maternal nutritional status as a determinant of LBW. The pattern is congruent with the SDH framework, in which a proximate biological determinant, energy reserve, mediates risk across demographic subgroups that themselves reflect differing exposures to upstream social conditions.[5,7]

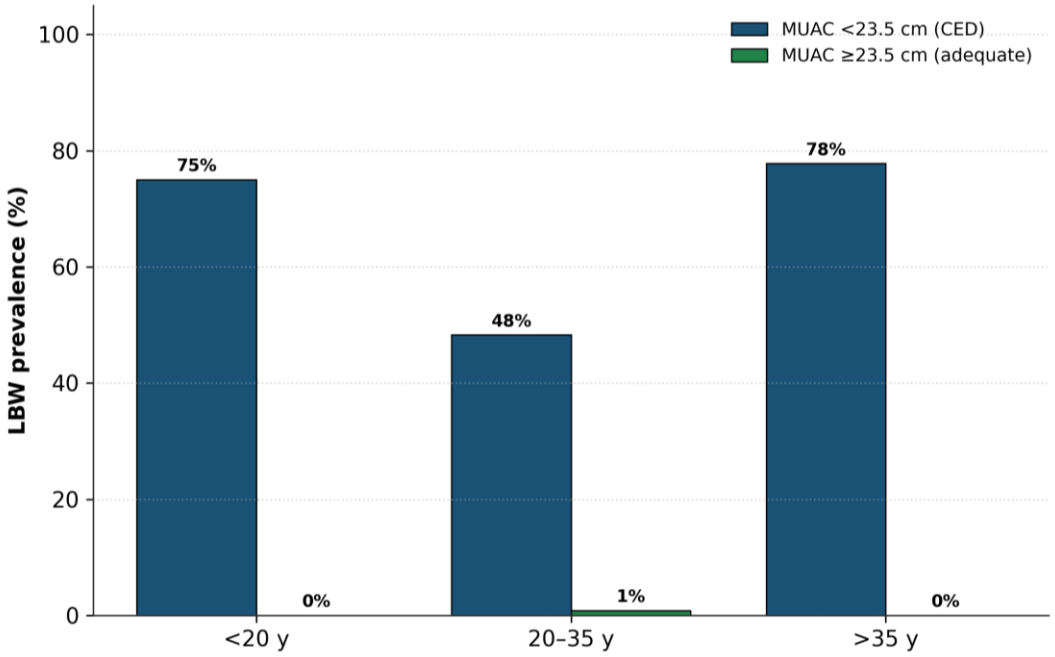

Figure 3. Low birth weight prevalence stratified by maternal age group and nutritional status.

Public health and policy implications

The public health implications of these findings are direct and actionable. Because MUAC measurement is inexpensive, rapid, non-invasive, and already embedded in Indonesian antenatal-care protocols, the dramatic risk gradient observed here makes a compelling case for systematic, repeated MUAC screening at every antenatal contact and at the Posyandu level, with MUAC <23.5 cm serving as an explicit trigger for nutritional intervention. Community health workers and village midwives should be empowered to identify CED early and to initiate balanced protein-energy supplementation, dietary counselling, and intensified antenatal monitoring, interventions whose efficacy in improving birth weight is supported by high-quality systematic-review and meta-analysis evidence.[3,13] Linking nutritional screening to the JKN financing mechanism and to existing supplementary feeding programmes for pregnant women would help ensure that detection translates into treatment, particularly for the food-insecure rural households in which CED clusters.[5,7] For the infants who are nevertheless born with low birth weight, the care pathway should extend to evidence-based newborn interventions such as kangaroo mother care, which substantially reduces neonatal mortality and improves maternal mental health and bonding and is feasible at the primary-care level.[20,21]

These actions map directly onto the Sustainable Development Goals. By targeting maternal undernutrition, the proposed interventions advance SDG 2 (Zero Hunger), specifically Target 2.2 on ending all forms of malnutrition including the

nutritional needs of pregnant women, and SDG 3 (Good Health and Well-being), specifically Target 3.2 on ending preventable neonatal deaths, given the role of LBW as a leading antecedent of neonatal mortality.[1,2] Because maternal undernutrition is itself a product of poverty and gendered inequality, sustained progress also contributes to SDG 1 (No Poverty) and SDG 10 (Reduced Inequalities). Situating maternal nutrition within this framework reframes MUAC screening not as a narrow clinical task but as a high-yield, equity-promoting investment in the health of the next generation.

Within the Indonesian community health system, the findings reinforce the strategic importance of the *Puskesmas-Posyandu*-cadre triad. The integrated community health post network offers an established platform for population-level MUAC surveillance, growth monitoring, and nutrition education delivered by trained village cadres, while the *Puskesmas* provides the clinical backbone for management of identified cases. Strengthening referral pathways between these tiers, ensuring uninterrupted supplies of measuring tapes and supplementary food, and incorporating maternal nutritional status into routine health-information reporting would convert the evidence presented here into durable practice in Tana Toraja and comparable rural regencies.[5,12]

Operationalising these findings within the Indonesian maternal-health architecture would involve embedding a structured MUAC-based decision rule into the antenatal-care card and the maternal-and-child health handbook (*Buku KIA*), such that any measurement below 23.5 cm automatically triggers a defined package of nutritional counselling, supplementary feeding, micronutrient supplementation, and intensified follow-up. Coupling this decision rule with the routine reporting that already flows from *Posyandu* to *Puskesmas* would create a near-real-time surveillance signal for maternal undernutrition at the district level, enabling health authorities to direct supplementary-feeding resources to the communities where the burden is greatest. Such a system would convert an individual screening act into a population-health instrument consistent with the principles of community medicine.

The cost-effectiveness implications are also favourable. Because MUAC screening is inexpensive and the intervention package draws on commodities already available within national programmes, the marginal cost of expanded screening is low relative to the substantial averted costs of neonatal intensive care, recurrent childhood illness, and the long-term productivity losses associated with LBW.[6] In settings facing the dual pressures of constrained health budgets and high perinatal burden, prioritising maternal nutrition represents an efficient allocation of scarce resources, an argument that strengthens the case for its inclusion in district health planning and JKN benefit design.

Equity, social determinants, and the intergenerational cycle

Interpreted through the social determinants of health lens, the concentration of LBW among undernourished mothers is the biological expression of inequity. Chronic energy deficiency does not arise randomly; it tracks household food insecurity, low maternal education, early childbearing, and the limited purchasing power that characterise rural highland economies, all of which are themselves shaped by structural forces of poverty and geographic marginalisation.[5,7,8] The very high population attributable risk estimated in this study therefore signals not only a clinical opportunity but a social one: investments that improve the material circumstances and dietary adequacy of pregnant women would be expected to yield large reductions in LBW precisely because nutritional status mediates so much of the risk in this setting.

This framing also clarifies the stakes of inaction. Because LBW predisposes to childhood stunting and impaired cognitive development, and because undernourished girls are more likely to become undernourished mothers, maternal CED perpetuates an intergenerational cycle of disadvantage.[4,6,8] Interrupting this cycle requires action that extends beyond the clinic to encompass food security programmes, maternal education, and social

protection, delivered in coordination with the health sector. Community medicine is well positioned to broker this intersectoral response, using the Puskesmas and Posyandu as platforms that connect biomedical screening with the broader determinants of maternal and child nutrition.

Strengths and limitations

This study has several strengths. It analysed a complete set of qualifying records over a full calendar year, applied objectively measured anthropometric and clinical variables rather than self-report, and advanced beyond the descriptive orientation of much of the existing Indonesian literature by reporting prevalence with confidence intervals, multiple effect-size measures, the population attributable risk, and a multivariable model with formal assessment of fit. The use of the nationally validated MUAC threshold enhances comparability and the operational relevance of the findings for primary care.

Several limitations must nevertheless be acknowledged. First, the cross-sectional design precludes causal inference; although the temporal logic of maternal nutrition preceding birth weight is biologically coherent, reverse causation and residual confounding cannot be excluded. Second, reliance on secondary medical-record data introduces the potential for information bias, as records with incomplete data were excluded, which may have selected a healthier or better-documented subgroup and limited generalisability. Third, the analysis was confined to maternal nutritional status, age, and parity, and did not capture other established determinants of LBW such as maternal haemoglobin and anaemia, gestational age, the frequency and quality of antenatal care, maternal smoking, infection, and socioeconomic indicators; their omission may have left residual confounding.[10,14,16] In particular, the absence of gestational age meant that growth restriction could not be distinguished from prematurity, an important distinction given that preterm birth remains a leading antecedent of LBW and neonatal mortality worldwide.[22] Fourth, the very wide confidence intervals around the ratio measures, a consequence of the sparse cell of LBW among well-nourished mothers, mean that the precise magnitude of the association should be interpreted with caution even though its direction and significance are robust. Finally, as a single-centre study in one rural regency, the findings may not generalise to urban populations or to regions with different nutritional epidemiology.

Several steps were taken to mitigate the limitations inherent in a secondary-data, single-centre design. Standardised measurement protocols and instrument calibration reduced measurement error in the principal exposure and outcome; pre-specified eligibility criteria and exclusion of records with severe maternal pathology limited confounding by serious illness; and the use of multiple, complementary statistical approaches guarded against over-reliance on any single estimator. The transparent reporting of confidence intervals and effect sizes, in line with contemporary epidemiological standards, allows readers to weigh the precision as well as the magnitude of the findings. Despite these measures, the residual confounding, selection, and information biases described above cannot be fully eliminated, and the results should be interpreted as establishing a strong association rather than a definitive causal effect.

Taken together, the strengths and limitations define a clear agenda for subsequent research. Prospective, multi-centre cohort studies that enrol women in early pregnancy, capture serial MUAC alongside haemoglobin, gestational age, antenatal-care utilisation, and household socioeconomic indicators, and follow infants to delivery would permit causal inference, mediation analysis, and the derivation of locally calibrated risk thresholds. Embedding such studies within the existing *Puskesmas-Posyandu* network would not only strengthen the evidence base but also build local research capacity, advancing the broader goal of evidence-informed community medicine in rural Indonesia.

4. Conclusion

In this community-based cross-sectional study of 192 mothers at a rural primary health centre in Tana Toraja, Indonesia, maternal nutritional status assessed by mid-upper arm circumference was

strongly and independently associated with low birth weight. The overall LBW prevalence was 13.02% (95% CI 8.98-18.52), but it reached 57.14% among mothers with chronic energy deficiency versus 0.67% among well-nourished mothers (PR 85.71; p<0.001), and low MUAC remained the dominant predictor after adjustment for maternal age and parity (adjusted OR 205.71; p<0.001), with an estimated population attributable risk of 94.9%. These results identify maternal undernutrition as a high-yield, modifiable target for LBW prevention and support the systematic use of MUAC screening at every antenatal and *Posyandu* contact, coupled with timely nutritional supplementation and intensified monitoring for mothers with CED, in direct support of SDG 2 and SDG 3. Future prospective, multi-centre studies that incorporate maternal haemoglobin, gestational age, antenatal-care quality, and socioeconomic determinants are recommended to refine effect estimates and to guide the design and evaluation of community nutrition interventions.

5. References

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