ARTICLE

Birth weight mediates the association of maternal undernutrition with child undernutrition prevalence in West Africa

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BACKGROUND: Maternal nutritional status before and during pregnancy is an important determinant of foetal health. In West Africa, maternal and child undernutrition remains a major public health problem and it is important to establish the mechanistic pathway linking the two disorders to help address the problem. We therefore assessed the mediating role of low birth weight (LBW) in the relationship of maternal undernutrition with child undernutrition in West Africa.

METHODS: We included recent (2010–2019) DHS data from thirteen West African countries. Poisson regression model with robust standard errors was used to assess the relationship between maternal undernutrition (body mass index and anaemia) and child undernutrition (stunting, wasting, underweight, and anaemia). Structural equation modelling was used to conduct the mediation analysis.

RESULTS: Prevalence of stunting, wasting, underweight, and anaemia among under-five children in West Africa was found to be 32.4%, 8.1%, 20.1%, and 71.5%, respectively. We found children of underweight mothers to be more likely to be undernourished (stunted, wasted, and underweight) and anaemic compared to children of normal-weight mothers. Also, children of anaemic mothers were more likely to be stunted and anaemic but not wasted compared with children of non-anaemic mothers. LBW mediated the observed relationships between maternal BMI and childhood stunting (22.6%), and maternal anaemia and childhood stunting (24.9%), wasting (11.7), and anaemia (6.6%).

CONCLUSION: We found maternal undernutrition to be associated with child undernutrition in West Africa with LBW noted to be a mediator of the observed relationship. We recommend that, to address the child undernutrition problem in West Africa, governments and policymakers must integrate measures to address the burden of LBW.

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INTRODUCTION

Maternal nutritional status before and during pregnancy is an important determinant of foetal health [1–3]. In many low- and middle-income countries, pregnant women frequently suffer from malnutrition as a result of inadequate dietary intake, poverty, weak healthcare system, recurring infections, and lack of awareness of healthy dietary patterns during pregnancy [4, 5]. In Africa, about 40% of women of reproductive age are deficient in iron, 15–20% of pregnant women are deficient in vitamin A, and 15–50% are deficient in zinc [6]. A systematic review by Desyibelew et al. [7] reported the prevalence of maternal malnutrition in Africa to be 23.5% (95% CI: 17.72, 29.32). The prevalence of maternal undernutrition has been found to show an increasing trend in West Africa [8]. The prevalence of maternal anaemia in West Africa has been reported to be 51.8% [9]. Underweight prevalence among women of reproductive age (20–49 years) in West Africa has been estimated to be 10% with South Asia, the only region with a higher estimate [10]. Maternal undernutrition has been documented to lead to poor child health outcomes including low birth weight (LBW) and child undernutrition [1, 2, 11–13]. Meta-analyses by some authors [13, 14] have reported maternal anaemia and underweight to be associated with higher risk of LBW. Similar reviews have also found these maternal undernutrition indicators to be associated with child undernutrition [8, 15, 16].

LBW defined by the World Health Organisation (WHO) as a birth weight less than 2500 g, is an important determinant of childhood undernutrition [15, 17, 18]. According to WHO, West Africa's LBW prevalence of 15.2% is the highest in Africa [19]. LBW has been associated with increased odds of stunting, wasting and

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Table 1. Characteristics of the study population.

Country	Year of survey	Estimated population of women aged 15–49 years[a]	Number of eligible women aged 15–49 years interviewed	Number of under-five children born in the five years preceding the survey *n* (%)	Number of children alive at the time of the survey *n* (%)	Eligible women response rate
Benin	2017	2,635,019	15,928	13,589 (9.35)	12,651 (9.55)	98.1%
Burkina Faso	2010	3,579,388	17,087	15,044 (10.35)	13716 (10.36)	98.4%
Cote d'Ivoire	2011	4,858,181	10,060	7776 (5.35)	7093 (5.36)	92.7%
Gambia	2013	471,456	10,233	8088 (5.56)	7788 (5.88)	90.7%
Ghana	2014	6,797,068	9396	5884 (4.05)	5595 (4.22)	97.3%
Guinea	2018	3,015,710	10,874	7951 (5.47)	7273 (5.49)	99.0%
Liberia	2019	1,189,170	9239	7606 (5.23)	5245 (3.96)	96.4%
Mali	2018	4,237,636	10,519	9940 (6.84)	9275 (7.00)	97.6%
Niger	2012	3,719,064	11,160	12,558 (8.64)	11,602 (8.76)	95.4%
Nigeria	2018	44,911,135	41,821	33,924 (23.34)	30,713 (23.19)	99.3%
Senegal	2019	3,890,229	34,850	6125 (4.21)	5899 (4.45)	96.1%
Sierra Leone	2019	1,914,452	15,574	9899 (6.81)	9063 (6.84)	96.7%
Togo	2013	1,677,648	9480	6979 (4.80)	6535 (4.93)	97.8%
Total		82,703,757	206,221	143,461	132,448	96.7%

% Unweighted percentages.
[a]https://www.who.int/data/maternal-newborn-child-adolescent-ageing/indicator-explorer-new/mca/women-of-reproductive-age-(15–49-years)-population-(thousands).

underweight among children under five in Sub-Saharan Africa [15, 17]. We have previously shown that even though there has been a consistent decline in the prevalence of these child undernutrition indicators in the West African region over the last three decades, the estimates remain consistently high in the region in comparison to global estimates [20]. WHO in 2019 estimated the number of stunted children in West Africa to be 18 million children, a prevalence rate of 27.7% and is the second highest in the African region and higher than the global prevalence of 21.3% [21]. More than 25% of the world's wasted children, 12.7 million children are found in Africa with West Africa recording the highest prevalence at 7.5% and higher than the global estimate of 6.9% [21]. In 2020, the prevalence of underweight among children under five was 16.3% in West Africa and was the highest in the African region [22]. We recently reported the prevalence of child anaemia in West Africa to be 71.7% and was the highest in Sub-Saharan Africa (SSA) [20].

Given the high burden of child undernutrition in West Africa, there is an urgent need to elucidate the mechanistic pathway underlying these disorders. Several factors including social, environmental and maternal health factors [23–26], proximate and distal factors as profiled in the adapted WHO framework of child undernutrition [27] have been associated with child undernutrition in the epidemiological literature. Yet, there has been little attempt to identify the mediators of these relationships to guide interventions and policy decisions for addressing the child undernutrition problem. It is against this background that, we sought to assess the mediating role of LBW in the relationship of maternal undernutrition with child undernutrition in West Africa. The findings of the study will provide child health policy decision-makers with the evidence base for evaluating the effectiveness of existing child nutrition policies and interventions, and make the necessary refinement for addressing the high burden of child undernutrition in West Africa and other developing regions.

METHODS

Data source

The study used the most recent Demographic and Health Surveys (DHS) datasets for the respective West African countries. DHS are nationally representative household surveys that collect data on women of reproductive age (15–49 years). The DHS sampling design is based on a two-stage stratified cluster sampling. Briefly, the sampling procedure sees the country stratified by region, province, or state followed by a random sampling of enumeration areas (EAs) within each stratum. The EAs are a cluster of households within a well-defined geographical boundary. The second stage of sampling involves systematic sampling of approximately 20–30 households within the selected EAs. The detailed sampling design for each country-specific DHS can be found in the appendix of the final DHS report available for download at the DHS website (www.dhsprogram.com).

Data used in this study has one record for every child of an interviewed woman, born within the five years preceding the survey. It contains information on the child's anthropometric measurements (stunting, wasting, underweight), anaemia status, and socio-demographic characteristics of the child, mother and the household. The data also has information on postnatal care, immunisation and other health indicators. The unit of analysis is children of women born in the last 5 years (0–59 months).

Our study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline. Table 1 presents the characteristics of the study population.

Study population and sampling size

Thirteen West African countries (Benin, Burkina Faso, Cote d'Ivoire, Ghana, Gambia, Guinea, Liberia, Mali, Niger, Nigeria, Sierra Leone, Senegal, Togo) were included in the study. The DHS survey years ranged from 2010–2019. The eligibility criteria for the study were as follows: children aged 0 to 59 months who were born within the five years preceding the survey, alive at the time of the survey, and had valid data on their nutritional status (stunting, underweight, wasting and anaemia). We included a total of 132,448 children from the 13 West African countries in the final sample. Figure 1 is a flowchart of the sample included in the study.

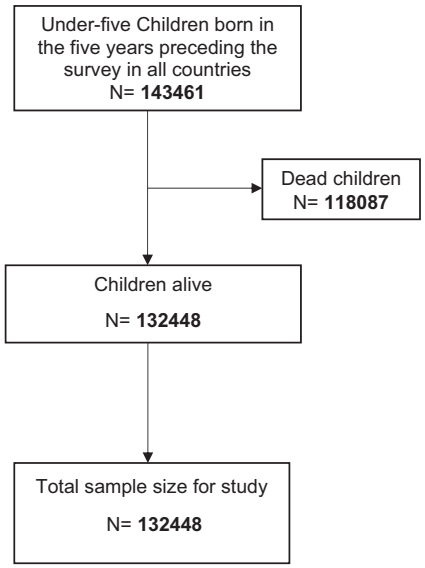

Fig. 1 Study flowchart of participants included in the analysis.

Outcome measures

The outcomes of interest were childhood stunting, wasting, underweight, and anaemia. Based on the 2006 WHO child growth standards, children were classified as stunted if their height-for-age z-score were below −2 standard deviations (−2SD) from the median of the reference population [28]. Children were classified as wasted if their weight-for-height z-scores were below −2SD from the median of the reference population. Children were classified as underweight if their weight-for-age z-score were below −2SD from the median of the reference population. In the DHS survey, these childhood nutritional indicators were among children aged 0–59 months. Children aged 6–59 months with haemoglobin level less than 11.0 g/dl were classified as anaemic [28].

The DHS reports have information on nutritional status of children under 5 years of age which the survey computes from measurement of height and weight of the children using a Shorr Productions measuring board and SECA 878 digital scale, respectively, together with information on ages of the children

Exposures of interest

The exposure of interest was maternal body mass index (normal weight, underweight, overweight/obesity) and anaemia status. Anaemia was classified as haemoglobin level less than 11.0 g/dl among pregnant women and less than 12.0 g/dl among non-pregnant women. The body mass index (BMI) classification were as follows: underweight (BMI < 18.5 kg/m^3), normal weight (BMI 18.5–24.9 kg/m^3), Overweight (BMI 25–29.9 kg/m^3), and obese (BMI ≥ 30.0 kg/m^3). All analyses pertaining to maternal BMI focused only on normal and underweight categories.

Mediators

The mediator of interest was birth weight categorised into normal and low birth weight based on the WHO definition [28, 29].

Covariates

The following exposure – outcome confounders were controlled in the multivariable models; age and sex of the child, place of residence (urban vs. rural), wealth index, household size, employment status of mother, maternal education, paternal education, current breast feeding status, exclusive breast feeding, minimum diet diversity (MDD), minimum meal frequency (MMF), minimum acceptable diet (MAD), receipt of vitamin A supplement, and child immunisation status. Household environmental quality (HEQ) served as the mediator – outcome cofounder in the models. We previously reported how these covariates were measured and categorised [30]. The DHS programme uses principal component analysis to derive household wealth for each country and categorised it into five levels (poorest, poor, moderate, rich and richest). In our study, we classified poorest and poor as poor, moderate as moderate, and rich and richest as rich. This re-classification was undertaken to reduce dimension of the covariates to help render the model more parsimonious and facilitate interpretation of the determinant of interest. HEQ is a latent trait variable generated from household sanitation (improved vs. unimproved), water sources (improved vs. unimproved) and household cooking fuel (clean vs. solid fuel) using item response theory with the one-parameter logistic regression model that adjusts for complex survey design characteristics. We used empirical Bayes means to predict the latent HEQ index. The HEQ index was categorised into low, medium and high levels based on quantile distribution.

Statistical analysis

This study pooled data from the 13 West African countries for the analysis. We first determined the prevalence of the mediator and the exposure of interest across the 13 West African countries. We assessed the relationship between maternal body mass index and perceived birth weight of the child controlling for possible confounders using Poisson regression model with robust standard errors and log link function. Prevalence ratios (PR) and their corresponding 95% confidence intervals (CI) were estimated from the models. All the analyses were adjusted for the complex survey design characteristics (sampling weight, clustering, and stratification) as provided by DHS to ensure that the effect estimates were unbiased and representative at the national level and the sub-region. In the pooled analyses, we re-weighted observations by a country's population size (i.e., de-normalise the sampling weight) and further adjusted for country and year of survey fixed-effects to account for the unobservable country-level factors and time trend.

We conducted a causal mediation analysis with X representing the vector of covariates/confounders, W the vector of exposures/treatment variables, M the vector of mediators, and Y the vector of outcome variables. We previously reported a DAG of the association of socio-economic status with child undernutrition which takes care of the present relationship [30]. Figure 2 is a comprehensive DAG of the relationship between W (maternal undernutrition), M (low birth weight), Y(child undernutrition) and X1 (exposure – outcome confounders) and X2 (mediator – outcome confounders). Under these circumstances, the direct effect is the effect (W→Y), not through M, and the indirect effect is the effect (W > M > Y). Moreover, each causal mediation effect represents a contrast of potential outcomes [31] that are a function of both M and W. The mediation analysis is based on the following three equations:

$$logit(Y = 1|X, M, W) = a_0 + a_1 W + \varepsilon_1 \quad (1)$$

$$Y = b_0 + b_1 W + b_2 M + \varepsilon_2 \quad (2)$$

$$M = c_0 + c_1 W + \varepsilon_3 \quad (3)$$

Where $a_0, b_0, and\, c_0$ denote intercepts, a_1 is coefficient linking the exposure/treatment variables (W) and outcome variables (Y) and is the total causal effect, b_1 is the coefficient for the effect of exposures on the outcome variable adjusting for the mediator variable M (direct effect), b_2 is the effect of M on Y adjusting for exposure variables, and c_1 is the coefficient relating to the effect of exposures on M. The $\varepsilon_1, \varepsilon_2 and \varepsilon_3$ are residuals [32] that are independent to each other and uncorrelated with variables in the right hand side of the equation.

Substitute Eq. [3] in Eq. [2], the causal mediation effect (CME) is represented by the product coefficient of $b_2 c_1$.

$$Y = b_0 + b_2 c_0 + (b_1 + b_2 c_1) W + b_2 \varepsilon_3 + \varepsilon_2 \quad (4)$$

RESULTS

Children born in the five years preceding the survey in the 13 included countries was 143,461 (Table 1). Of these children, 132,448 (92.3%) were alive at the time of the survey and constituted the study sample. The mean age of the children was 1.96 years (Standard deviation: 1.43). About 51% of the children were boys.

The prevalence of child undernutrition in West Africa stratified according to the exposures of interest, mediator and covariates are presented in Table 2. Overall, 32.4% (95% CI: 31.5%, 33.3%) of children were stunted, 8.1% (95% CI: 7.8%, 8.5%) were wasted,

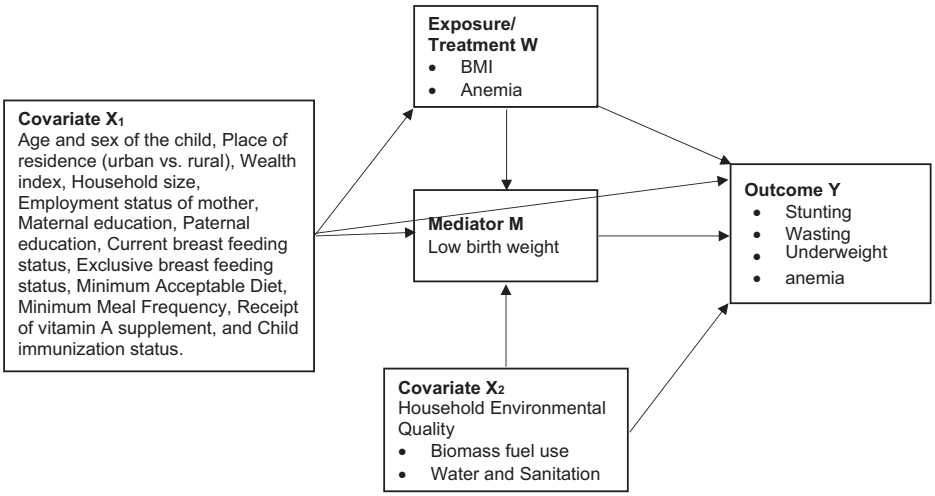

Fig. 2 Directed Acyclic Graph (DAG) of the relationship between maternal undernutrition (W), low birth weight (M) and child undernutrition (Y) with identification of exposure – outcome (X_1) and mediator – outcome (X_2) confounders.

20.1% (95% CI: 19.4%, 20.8%) were underweight, and 71.5% (95% CI: 70.6%, 72.4%) were anaemic. Prevalence of undernutrition was higher among anaemic and underweight mothers. Prevalence of undernutrition was higher among children born low birth weight and those two years and below. Prevalence of undernutrition was higher among children who were not currently breastfeeding, were not exclusively breastfed, and were classified as having inadequate MAD, MDD and MMF as well as children who resided in a rural area and whose mothers were unemployed. Prevalence of undernutrition was higher in households using solid fuels for cooking and unimproved water sources, and with unimproved sanitation and low household air quality. Prevalence of undernutrition increased with decreasing maternal and paternal education and wealth status, and increasing household size. Prevalence of undernutrition with the exception of wasting was high among non-immunised children. Prevalence of undernutrition with the exception of stunting was higher among children who received vitamin A supplementation. The relationship between maternal undernutrition indicators (maternal BMI and anaemia) and child undernutrition indicators (stunting, wasting, underweight and anaemia) is presented in Table 3. Children born to underweight mothers had 30% (adjusted PR (aPR) = 1.30, 95% CI: 1.19, 1.42), 101% (aPR = 2.01, 95% CI: 1.74, 2.33), 89% (aPR = 1.89, 95% CI: 1.71, 2.09) and 7% (aPR = 1.07, 95% CI: 1.01, 1.14) increased risk of becoming stunted, wasted, underweight and anaemic, respectively compared to children born to normal weight mothers. Children born to anaemic mothers had 7% (aPR= 1.07, 95% CI: 1.02, 1.12) and 13% (aPR = 1.13, 95% CI: 1.10, 1.17) increased risk of becoming stunted and anaemic, respectively and 14% (aPR = 0.86, 95% CI: 0.78, 0.94) decreased risk of becoming wasted compared to children born to non-anaemic mothers.

Results of the mediation analysis are presented in Table 4. Low birth weight mediated 22.6% of the observed relationship between maternal underweight and stunting. Low birth weight also mediated 24.9%, 11.7%, and 6.6% of the relationship between maternal anaemia and stunting, wasting, and anaemia, respectively. LBW did not mediate the relationships between maternal BMI and wasting, underweight and anaemia, and maternal anaemia and underweight.

DISCUSSION

The pooled prevalence of stunting, wasting, underweight, and anaemia among children under-five years in West Africa was 32.4%, 8.1%, 20.1%, and 71.5% respectively. We found children of underweight mothers to be more likely to be undernourished (stunted, wasted, and underweight) and anaemic compared to children of normal weight mothers. Also, children of anaemic mothers were more likely to be stunted and anaemic but not wasted compared with children of non-anaemic mothers. LBW mediated the observed relationships between maternal BMI and childhood stunting (22.6%), and maternal anaemia and childhood stunting (24.9%), wasting (11.75), and anaemia (6.6%).

Our study findings also underscore the key role of maternal nutritional and health status in early child growth and development. Previous reviews [8, 15, 16] and multi-country studies [33–36] have documented maternal BMI as a key risk factor for childhood undernutrition. Li et al. [33] found low maternal BMI (<18.5 kg/m^2) to be associated with increased odds of stunting (OR = 1.6; 95% CI: 1.6, 1.7), underweight (OR = 4.8; 95% CI: 4.6, 5.0) and wasting (OR = 2.3; 95% CI: 2.1, 2.4) in low and middle-income countries (LMICs). Studies by Razak et al. [35] and Ahmed et al. [36] also found children born to underweight mothers in SSA to have an increased odds of being underweight and stunted when compared to children born to mothers with normal weight. Very few studies have investigated the relationship between maternal BMI and childhood anaemia. In a large prospective cohort study conducted in China that included 17,193 mother-infant pairs, maternal underweight was found to be associated with increased odds of childhood anaemia at both 6 months (OR = 1.08; 95% CI: 0.85, 1.37)) and 12 months (OR = 1.02; 95% CI: 0.78, 1.34), albeit the confidence interval included the null value.

We also found maternal anaemia to be associated with childhood stunting and anaemia as reported by previous studies [37–42]. Studies by Nadhiroh et al. [39] and Vaivada et al. [40] identified maternal anaemia as an important risk factor of childhood stunting. A multi-country study conducted in Southern Africa [42] found children of anaemic mothers in four study countries to have an increased odds of becoming anaemic (Malawi: OR = 1.69 [95% CI:1.37, 2.13], Mozambique: OR = 1.71 [95% CI: 1.37, 2.13] Namibia: OR = 1.55 [95% CI: 1.08, 2.22] and Zimbabwe: OR = 1.52 [95% CI: 1.25, 1.84]). We found children of anaemic mothers to have a lower risk of being wasted which is surprising given the strong link between maternal nutritional factors, such as anaemia and child growth indicators as documented by Victoria et al. [8]. Epidemiological evidence from previous studies assessing these relationships have however been largely inconclusive [43, 44]. For instance, in a prospective study conducted in India, average maternal haemoglobin level during pregnancy was not associated with child stunting, wasting, or underweight. A multi-country analysis of cross-sectional data from India and Bangladesh also found no association between the

Table 2. Prevalence of child undernutrition according to exposures of interest, mediator, and covariates among children under-five in West Africa.

	Stunting %	95% CI	Wasting %	95% CI	Underweight %	95% CI	Anaemia %	95% CI
Overall prevalence	32.41	(31.5, 33.3)	8.1	(7.8, 8.5)	20.1	(19.4, 20.8)	71.5	(70.6, 72.4)
Exposures								
Maternal Anaemia								
No anaemia	30.7	(29.8, 31.9)	7.9	(7.4, 8.4)	18.5	(17.7, 19.4)	65.0	(63.8, 66.2)
Anaemic	35.0	(33.9, 36.2)	8.4	(7.9, 9.0)	22.0	(21.1, 23.0)	76.9	(75.8, 77.9)
Maternal BMI								
Normal	36.4	(35.3, 37.4)	8.4	(8.0, 8.9)	22.1	(21.3, 23.0)	74.2	(73.3, 75.2)
Underweight	45.1	(42.6, 47.5)	16.3	(14.7, 17.9)	38.0	(35.7, 40.4)	78.1	(76.1, 80.0)
Overweight/Obese	22.0	(20.7, 23.2)	4.9	(4.3, 5.6)	11.1	(10.2, 12.1)	63.0	(61.2, 64.7)
Mediator								
Birth weight								
Normal	32.2	(31.3, 33.1)	8.4	(8.0, 8.9)	22.1	(21.3, 23.0)	74.2	(73.3, 75.2)
LBW	36.6	(34.0, 39.3)	16.3	(14.7, 18.0)	38.0	(35.7, 40.4)	78.1	(76.1, 80.0)
Covariates								
Current breastfeeding								
No	29.48	(28.7, 30.2)	11.0	(10.0, 12.2)	20.1	(19.2, 21.1)	85.9	(69.6, 94.2)
Yes	25.6	(24.6, 26.6)	8.1	(6.5. 10.1)	14.3	(13.7, 14.9)	81.4	(75.7, 86.0)
Exclusive breastfeeding								
No	15.8	(14.4, 17.4)	11.0	(9.9, 12.2)	15.9	(14.4, 17.4)	8.4	(8.0, 8.8)
Yes	14.8	(12.8, 17.2)	8.1	(6.5, 10.1)	10.6	(8.9, 12.5)	8.3	(6.3, 9.4)
Minimum Acceptable Diet								
Inadequate	35.0	(34.0, 36.0)	8.9	(7.3, 10.8)	21.2	(20.5, 22.0)	75.5	(72.7, 78.2)
Adequate	23.9	(21.6, 26.3)	7.9	(7.5, 8.3)	14.5	(12.7, 16.4)	71.3	(70.4, 72.2)
Minimum Meal Frequency								
Inadequate	35.6	(34.6, 36.6)	11.5	(10.7, 12.5)	20.9	(20.0, 21.6)	79.8	(78.4, 81.1)
Adequate	29.1	(27.7, 30.6)	7.1	(6.7, 7.5)	20.8	(19.8, 22.1)	69.5	(68.5, 70.5)
Minimum Dietary Diversity								
Inadequate	33.4	(32.9, 33.9)	8.8	(8.5, 9.1)	18.7	(18.4, 19.1)	78	(77.7, 78.4)
Adequate	29.3	(28.8, 29.7)	6.3	(6.05, 6.6)	18.4	(18.0, 18.8)	78.1	(77.8, 78.5)
Employment status of mother								
No	35.3	(34.0, 36.7)	9.8	(9.2, 10.4)	23.9	(22.9, 25.0)	73.2	(72.0, 74.5)
Yes	31.0	(30.1, 32.0)	7.3	(6.9, 7.7)	18.2	(17.4, 18.9)	70.7	(69.7, 71.7)
Maternal education								
None	40.2	(39.1, 41.3)	10.1	(9.6, 10.6)	26.1	(25.2, 27.0)	77.4	(76.4, 78.4)
Primary	31.3	(30.0, 32.8)	6.7	(6.0, 7.5)	17.0	(16.0, 18.2)	72.3	(70.4, 74.1)
Secondary	19.4	(18.3, 30.6)	5.4	(4.9, 6.0)	11.3	(10.5, 12.2)	67.7	(60.1, 63.3)
Paternal education								
None	39.5	(38.4, 40.6)	10.4	(9.9, 10.9)	26.0	(25.1, 27.0)	78.5	(77.4, 79.5)
Primary	34.8	(33.0, 36.7)	7.1	(6.3, 7.9)	20.0	(18.6, 21.4)	72.5	(70.5, 74.4)
Secondary	23.7	(22.5, 24.9)	6.0	(5.5, 6.5)	13.6	(12.6, 14.5)	63.7	(62.2, 65.2)
Wealth Index								
Poor	42.2	(40.9, 43.5)	9.6	(9.0, 10.1)	26.7	(25.6, 27.8)	78.8	(77.7, 79.8)

Table 2. continued

	Stunting %	95% CI	Wasting %	95% CI	Underweight %	95% CI	Anaemia %	95% CI
Moderate	33.2	(31.8, 34.6)	7.8	(7.1, 8.5)	19.1	(18.1, 20.3)	71.5	(69.8, 73.1)
Rich	21.6	(20.6, 22.7)	6.8	(6.3, 7.3)	13.6	(12.8, 14.4)	63.9	(62.4, 65.3)
Child's age								
0	17.8	(16.8, 18.9)	12.2	(11.5, 13.0)	16.7	(15.8, 17.6)	81.7	(80.0, 83.2)
1	34.4	(33.1, 35.9)	11.7	(10.9, 12.6)	23.7	(22.6, 25.0)	81.3	(80.1, 82.5)
2	42.5	(40.9, 44.0)	6.3	(5.7, 7.0)	22.8	(21.6, 24.1)	71.7	(70.2, 73.1)
3	37.8	(36.2, 39.4)	4.4	(3.9, 4.9)	19.6	(18.4, 20.9)	66.1	(64.3, 67.8)
4	31.5	(30.0, 32.9)	4.8	(4.3, 5.4)	17.7	(16.6, 18.8)	60.0	(58.3, 61.5)
Place of residence								
Urban	23.4	(22.1, 24.7)	6.1	(5.6, 6.6)	13.8	(12.9, 14.8)	63.9	(62.2, 65.5)
Rural	37.5	(36.5, 38.6)	9.2	(8.8, 9.7)	23.6	(22.8, 24.5)	76.0	(75.0, 77.0)
Household size								
1–5	28.1	(27.1, 29.2)	7.7	(7.2, 8.3)	17.6	(16.7, 18.4)	69.0	(67.7, 70.2)
6–9	32.9	(31.8, 34.0)	8.1	(7.6, 8.7)	20.2	(19.3, 21.1)	72.2	(70.9, 73.5)
10+	39.3	(37.7, 40.9)	8.8	(8.2, 9.5)	24.5	(23.2, 25.9)	75.1	(73.5, 76.7)
Type of cooking fuel								
Clean fuel	13.7	(11.7, 16.1)	4.5	(3.6, 5.7)	8.8	(7.2, 10.7)	51.3	(4.8, 5.5)
Solid fuel	34.2	(33.4, 35.1)	8.5	(8.1, 8.9)	21.2	(20.5, 21.9)	73.4	(72.6, 74.3)
Water source								
Improved	29.1	(28.2, 30.1)	7.9	(7.4, 8.3)	18.3	(17.6, 19.1)	69.4	(68.4, 70.4)
Unimproved	41.5	(40.0, 43.0)	8.9	(8.3, 9.6)	25.0	(23.8, 26.3)	77.1	(75.7, 78.5)
Sanitation								
Improved	26.2	(25.1, 27.3)	6.7	(6.2, 7.1)	15.5	(14.8, 16.4)	65.3	(64.0, 66.6)
Unimproved	38.4	(37.4, 39.5)	9.6	(9.1, 10.1)	24.5	(23.6, 25.4)	77.3	(76.3, 78.3)
Household environmental quality								
Low	41.8	(40.2, 43.4)	9.3	(8.6, 10.1)	25.8	(24.5, 27.2)	77.8	(76.2, 79.3)
Medium	36.8	(36.7, 38.0)	9.2	(8.7, 9.9)	23.3	(22.3, 24.4)	76.9	(75.8, 78.0)
High	24.0	(23.0, 25.2)	6.5	(6.1, 7.0)	14.5	(13.7, 15.3)	64.1	(62.7, 65.4)
Receipt of vitamin A supplement								
No	32.5	(31.6, 33.4)	7.6	(7.2, 7.9)	20.1	(19.4, 20.8)	70.5	(69.5, 71.5)
Yes	31.9	(30.4, 33.5)	8.5	(7.7, 9.4)	20.2	(19.0, 21.6)	75.4	(73.9, 76.8)
Child immunisation status								
Yes	30.6	(29.0, 32.2)	8.9	(8.1, 9.8)	16.6	(15.4, 17.9)	73.3	(71.7, 74.7)
No	37.2	(31.1, 43.8)	7.9	(7.5, 8.3)	25.1	(19.9, 31.2)	77.6	(71.6, 82.6)

BMI Body mass index, *CI* Confidence interval, *LBW* Low birth weight.

Table 3. Prevalence ratios (PR) estimated from modified Poisson regression for child undernutrition according to maternal nutrition indicators.

Categories	Stunting (aPR)	95% CI	Wasting (aPR)	95% CI	Underweight (aPR)	95% CI	Anaemia (aPR)	95% CI
BMI								
Underweight	1.30	(1.19, 1.42)***	2.01	(1.74, 2.33)***	1.89	(1.71, 2.09)***	1.07	(1.01, 1.14)***
Normal	1.00		1.00		1.00		1.00	
Anaemia								
Yes	1.07	(1.02, 1.12)**	0.86	(0.78, 0.94)*	1.01	(0.94, 1.07)	1.13	(1.10, 1.17)***
No	1.00		1.00		1.00		1.00	
Random Effect Estimate (%)								
Identity variance: Estimate (95% CI)	10.0 (5.0, 23.0)		13.0 (6.0, 29.0)		9.0 (4.3, 22.0)		11.0 (4.6, 25.0)	
ICC: Estimate (95% CI)	3.0 (1.0, 7.0)		4.0 (2.0, 8.0)		2.0 (1.0, 5.0)		2.0 (1.0, 4.0)	

All models were adjusted for age and sex of the child, place of residence (urban vs. rural), wealth index, household size, household environmental quality, employment status of mother, maternal education, paternal education, current breastfeeding status, exclusive breastfeeding, minimum adequate diet, minimum dietary diversity, minimum meal frequency, receipt of vitamin A supplement, and child immunisation status.
aPR Adjusted Prevalence ratio, *CI* Confidence interval, *ICC* Inter-correlation Coefficient.
p-value notation: ***$p < 0.001$; **$p < 0.01$; *$p < 0.05$.

probability of maternal anaemia during pregnancy (first, second, and third trimester) and the probability of childhood wasting by birth month. However, probability of maternal anaemia during the postpartum period was significantly associated with probability of childhood wasting by birth month. This situation calls for more research to further validate the relationship.

We found LBW to mediate the observed relationships between maternal BMI and childhood stunting, and maternal anaemia and childhood stunting, wasting, and anaemia. Very few studies [45–47] have investigated the mechanistic pathway linking maternal health factors with child undernutrition relationship. Our study to the best of our knowledge is the first to evaluate the mediating role of LBW in the relationship between maternal undernutrition and child undernutrition. Majority of studies have investigated the independent relationships between maternal BMI and LBW [2, 14, 48], maternal anaemia and LBW [12, 13], LBW and child undernutrition indicators [17, 49, 50], and LBW and childhood anaemia [51]. The associations, however, substantiate our findings as they provide suggestive evidence of the mediating role of LBW in the relationship between maternal undernutrition and child undernutrition. There is, however, evidence from Botswana on the meditating role of LBW in the relationship between HIV infection among children and child undernutrition [52] which strengthens our findings. The authors found LBW to mediate 67% of the excess risk of stunting among HIV-exposed but non-infected children (children born to mothers with HIV but diagnosed as HIV negative). A recent decomposition analysis also found LBW to explain 14.8%, 10.4% and 9.6% of the differences in stunting, wasting and underweight, respectively, in India after adjusting for various individual-level characteristics [53]. A pooled analysis of 19 longitudinal birth cohort studies in LMICs also reported LBW to be attributed to a larger number of stunting cases than wasting [50]. The findings of these studies could explain our observation of the LBW mediation effect being greater for stunting compared to the other child undernutrition indicators. The low mediation effect of LBW observed in the relationship between maternal anaemia and childhood anaemia could be due to the fact that, in SSA, other factors besides maternal and perinatal health factors such as poor household environmental conditions and infectious disease transmission which are pervasive in the region could be more important in explaining the burden of child anaemia in SSA as highlighted by Moschovis et al. [54].

Biological plausibility

LBW resulting from maternal undernutrition (maternal underweight and anaemia) leads to reduced immune function, respiratory problems, and metabolic dysfunction which increases the risk of childhood infections and illnesses, and impairment in growth and development [55, 56]. It has been reported that differential occurrence of in-utero foetal growth restriction (a key determinant of LBW) due to maternal anaemia during early and late pregnancy results in symmetrical growth and non-asymmetrical growth patterns which contributes to stunting and wasting, respectively [57, 58]. This observation could also possibly explain the large mediation effect observed for stunting and wasting in comparison to childhood anaemia.

After birth, the detrimental effects of maternal undernutrition on neonatal and child health still persist throughout childhood, particularly the adverse birth outcomes (LBW, preterm birth) effect and explains the association of maternal anaemia with childhood anaemia.

Maternal undernutrition has also been reported to affect children's gut microbiota, consequently leading to increased susceptibility to infection and impaired nutrient uptake thus ultimately contributing to undernutrition [59]. Maternal undernutrition can also lead to epigenetic changes in the genes governing metabolism and growth in the developing foetus with implications for the nutrition of newborn. Early nutritional exposure has been proposed to affect epigenetic changes in the developing foetus [60].

Validity issues

Combining nationally representative data from multiple countries guaranteed a large sample size which renders our study sufficiently powered to test the study hypotheses and is a major strength of our study. The high response rates of DHS surveys also minimise selection bias. The potential for exposure, outcomes and covariates misclassification is also minimised owing to the standardised data collection protocols and well-calibrated

Table 4. Mediation effect of low birth weight in the association of maternal undernutrition with child undernutrition outcomes in West Africa.

Exposures	Effects (%)	Stunting		Wasting		Underweight		Anaemia status	
		Log Odds	95% CI	Log Odds	95% CI	Log Odds	95% CI	Log Odds	95% CI
BMI	Total Direct effect	1.07	(−0.65, 2.78)						
	Total Indirect effect	1.02	(0.69, 1.35)**						
	Total effect	1.09	(−0.69, 2.9)						
Percentage of total effect of BMI that is mediated by low birth weight		22.6%							
Anaemia	Total Direct effect	1.08	(0.40, 1.76)***	1.13	(0.35, 1.91)***			1.90	(0.64, 3.15)**
	Total Indirect effect	1.03	(0.97, 1.08)***	1.02	(0.97, 1.06)***			1.03	(0.97, 1.10)**
	Total effect	1.12	(0.41, 1.80)***	1.15	(0.36, 1.94)***			1.96	(0.66, 3.27)**
Percentage of total effect of anaemia that is mediated by low birth weight		24.92%		11.72%				6.61%	

LBW did not mediate maternal BMI – wasting, underweight and anaemia relationships, and maternal anaemia – underweight relationship.
BMI Body mass Index, *CI* Confidence interval.
p-value notation: ***$p < 0.001$; **$p < 0.01$; *$p < 0.05$.

instruments used across all countries. The study outcomes were common and hence we used Poisson regression modelling for the analysis instead of logistic regression to prevent overestimation of the effect estimates. Ignoring missing data in mediation analysis may produce biased estimates of mediation effects. As a result, we excluded missing values of <10% of the original data as recommended by Newman [61] and used mean imputation methods for values above this threshold. We have incorporated random effects (country as a random effect) in the model with the amount of variation explained by the random component found to be very small for all study outcomes, between 2% to 4% (Table 3).

Given that the DHS survey adopts a cross-sectional design, this does not allow for the establishment of temporality, which is a major limitation of our study. We could not control for the potential confounding effect of individual-level factors such as maternal health-seeking behaviour, HIV status and other infectious diseases, and country-level unobserved factors such as health system access and utilisation. This is because DHS surveys does not measure these covariates. The sequential ignorability assumption of mediation analysis which requires that there are no unobserved confounders of the treatment – mediator or treatment – outcome relationships as well as no unobserved (baseline or post-treatment) mediator – outcome confounders have possibly been violated as a result and is another limitation of the study.

We were hampered from assessing exposure – mediator interaction in our study. This because previous studies on mediation analysis that have assessed exposure – mediation interaction have investigated either continuous exposure and continuous mediator or binary exposure and continuous mediator or continuous exposure and binary mediator. As a result, methods for examining binary exposure and binary mediator as in our study have not been well developed.

Mediator misclassification could be a potential problem in our study. According to Hermanussen [62], there is the need to constantly review the standards used to diagnose conditions such as LBW to help minimise the risk of misclassification and to also develop context-specific standards to help better address the burden of these adverse birth outcomes globally. In light of this, developing standards tailored to the West Africa region where the burden of LBW and child undernutrition is high is necessary to help better measure and quantify LBW in the region. However, in the absence of West Africa standards, use of the current WHO global standards applicable to all geographical regions is acceptable to guide child health programming and policies.

CONCLUSION

Our study provides evidence that child undernutrition is high among children born to undernourished mothers. We also demonstrated that LBW is a mediator of the relationship between maternal undernutrition and child undernutrition, especially stunting. The study findings suggest that, in order to address child undernutrition in West Africa, there is the need for policy action and interventions targeted towards the care of LBW babies to ensure optimal growth and development. This is very important as traditionally, policies seeking to address child undernutrition have primarily focused on improving complementary feeding and micronutrient supplementation and in spite of the sustained investment in these programmes, child undernutrition still bedevils many developing regions including West Africa and requires a policy shift. Our findings therefore highlight the need to give serious consideration to addressing the low birth weight burden in West Africa through improved maternal health and nutrition if we are to mitigate the burden of child growth faltering in West Africa. We recommend the conduct of longitudinal studies to further test our hypotheses and strengthen the epidemiological

evidence on the mediating role of LBW in the maternal undernutrition – child undernutrition relationship.

DATA AVAILABILITY

The Demographic and Health Survey datasets were available in the public domain (https://dhsprogram.com/data/available-datasets.cfm). All Stata codes used in the analysis are available upon request.

CODE AVAILABILITY

The Demographic and Health Survey datasets were available in the public domain (https://dhsprogram.com/data/available-datasets.cfm). All Stata codes used in the analysis are available upon request.

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AUTHOR CONTRIBUTIONS

AKA conceived the idea and designed the study. DD accessed and merged all the dataset for the study. HMF conducted the data analysis. AKA wrote the manuscript together with RA, KY, CS and SAM. SKA, SS and NBK reviewed draft manuscripts for important intellectual content.

COMPETING INTERESTS

The authors declare no competing interest.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

As this study leverages secondary data from the Demographic and Health Surveys (DHS), no ethical approval and consent were required. Notwithstanding, DHS, the program directors ensured that IRB-approved procedures were used with all women interviewed giving verbal consent before data were collected. In this study, before the use of the dataset, the DHS Program gave the authors permission to use the dataset.

ADDITIONAL INFORMATION

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