

Linear Growth spurts are Preceded by Higher Weight Gain Velocity and Followed by Weight Slowdowns Among Rural Children in Burkina Faso: A Longitudinal Study

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ABSTRACT

Background: The temporal relationship between length (linear) and weight (ponderal) growth in early life is important to support optimal nutrition program design. Studies based on measures of attained size have established that wasting often precedes stunting, but such studies do not capture responsiveness of growth to previous compared with current conditions. As a result, the temporality of linear and ponderal growth relationships remain unclear.

Objectives: We used growth velocity indicators to assess the temporal bidirectional relationships between linear and ponderal growth in children.

Methods: Using monthly anthropometric measurements from 5039 Burkinabè children enrolled at 6 months of age and followed until 28 months from August 2014 to December 2016, we employed multilevel mixed-effects models to investigate concurrent and lagged associations between linear and ponderal growth velocity, controlling for time trends, seasonality, and morbidity.

Results: Faster ponderal growth is associated with faster concurrent and subsequent linear growth (0.21–0.72 increase in length velocity z-score per unit increase in weight velocity z-score), while faster linear growth is associated with slower future weight gain (0.009–0.02 decrease in weight velocity z-score per unit increase in length velocity z-score), especially among children 9–14 months. Ponderal growth slows around the same time as peaks in morbidity, followed roughly a month later by slower linear growth.

Conclusions: Use of velocity measures to assess temporal dependencies between linear and ponderal growth demonstrate that the same growth-limiting conditions likely affect both length and weight velocity, that slow ponderal growth likely limits subsequent linear growth, and that linear growth spurts may not be accompanied by sufficient increases in dietary intake to avoid slowdowns in weight gain. *J Nutr* 2022;152:1963–1973.

Keywords: linear growth, ponderal growth, growth faltering, growth velocity, children, Burkina Faso, stunting, wasting

Introduction

Malnutrition in all forms remains a prevalent global health issue, with 21.9% of children estimated to be stunted [length-for-age z scores (LAZ) < −2 SDs from the WHO growth reference standards], and 6.9% estimated to be wasted [weight-for-length z scores (WLZ) < −2 SDs] worldwide in 2019 (1, 2). Nutrition interventions have historically focused on each form of malnutrition as a separate problem (3–6), with wasting often thought to be a consequence of proximal factors, such as food shortages and illness, and stunting usually attributed to general underlying conditions of poverty

and food insecurity (7). This approach limits the efficiency and effectiveness of malnutrition prevention and treatment, since conditions may affect length (linear) and weight (ponderal) growth concurrently (8).

Recently, increased attention has been paid to the relationships among the different forms of malnutrition, including how knowledge of interdependencies can be leveraged to increase program effectiveness (9, 10). Several studies have shown that stunting and wasting likely have overlapping causal pathways (10–14). The consensus from studies using longitudinal data to investigate temporality between stunting and wasting is that sufficient ponderal growth, and thus ample energy reserves,

may be necessary for linear growth (11, 15, 16). As such, early episodes of wasting or deficits in weight gain may substantially affect attained height and contribute to linear growth faltering (6, 14, 17–25). Additionally, there is evidence that the relationship may be bidirectional, with poor linear growth associated with subsequent higher levels of wasting, though the mechanism through which this happens remains unclear (26).

These studies have advanced our understanding of temporal associations between linear and ponderal growth but are largely limited by the rigidity of the definitions of stunting and wasting, which were established for assessing undernutrition at the population level, but do not accurately reflect undernutrition at the individual level (27). While a limited number of studies have used the entire range of attained size z-scores (LAZ, WLZ) or investigated the relationship between attained WLZ and linear growth velocities (6, 15, 24, 28), indices of attained size still limit understanding of temporal dependencies, as they reflect the cumulative effects of all environmental growth-limiting conditions up to the point of measurement (29). We aimed to evaluate the bidirectional relationships between linear and ponderal growth trajectories among young children in Burkina Faso using length and weight velocities (absolute and velocity z-scores). We assessed whether increases (or decreases) in 1 growth velocity parameter affect the rates of the other parameter and identified sensitive age ranges when growth rates most influence each other at the individual level.

Methods

Study design and data source

Anthropometric data were collected between August 2014 and December 2016 as part of a geographically clustered trial comparing the cost effectiveness of 4 supplementary foods in preventing stunting and wasting in children aged 6–23 months in Sanmatenga Province, Burkina Faso. All children in the catchment area were eligible for enrollment as they reached 6 months, except those with a midupper arm circumference < 11.5 cm, who were referred for severe acute malnutrition treatment, and children > 12 months at enrollment. Participating children were measured monthly (recumbent length, weight, and midupper arm circumference) for 18 months during the intervention and for an additional 3 months postintervention, with most measurements (99%) occurring when children were between 6–28 months. Recumbent length was used for all children up to 28 months; thus, adjustments were applied to analyze z-scores of children > 24 months. Details of the original trial are found elsewhere (30). Children from all study arms are pooled for this analysis, and the study arm is controlled for as a covariate.

Implausible anthropometric values were identified using jackknifed residuals, as described by Shi et al. (31). Imputation using predicted values from linear interpolations of measurement on age was then done for any missing or implausible values between the first and last measurement taken, such that each child had no missing windows for measurements. After imputation and restriction to children with at least 20 repeated measurements to maximize the analysis of full cases, the final data set included 108,802 measurements on 5,039 children

between 5–28 months and comprised 82% of the original sample of 129,944 measurements on 6,112 children (see **Supplemental Figure 1** for the participant flowchart).

Variable specification

Indices for linear and ponderal growth served as both the dependent and independent variables; when linear growth was the dependent variable, ponderal growth indices were the independent variables, and vice versa. Primary analyses were conducted using velocity z-scores that compare growth velocity among children in this study to the 2009 WHO Child Growth Velocity Standards. Velocity z-scores were calculated using the Lambda-Mu-Sigma parameters supplied by the WHO (32). Growth velocity standards are limited to 2-month intervals among children under 24 months; as such, growth velocity z-scores are constant for each 2-month age period. As sensitivity analyses, indices of absolute length and weight velocity were calculated as the difference between 2 consecutive measurements, divided by the time difference between measurements in days and multiplied by 30.44, to obtain standardized estimates in both units as cm/month and kg/month. In order to compare our results to those of previous studies, we also looked at temporality between age- and sex-standardized indices of attained length or height using LAZ and weight using WLZ, derived using the 2006 WHO Child Growth Standards (2, 33).

In addition to metrics of child growth, we coded a dichotomous variable identifying whether the child's caregiver reported any illness in the 2 weeks prior to the day the child was measured. Illnesses measured included upper-respiratory symptoms (cough, difficulty breathing, rapid breathing), fever, diarrhea, confirmed malaria, accident or injury, or burn. We also descriptively examined the temporal relationship between reported fever or diarrhea on the day of measurement and growth metrics. Last, the original trial was geographically clustered, so by controlling for study arm, we are also controlling for geographic region.

Data analysis

Prior to modeling, we examined correlations between anthropometric indices using Pearson correlation coefficients, looking at concurrent relationships and up to 9 lag periods. We present up to 9 lag periods for the ease of presentation and interpretation, as relationships remain the same after this point. To estimate the relationships between linear and ponderal growth indices, we used multilevel mixed-effects linear regression models with child-level random intercepts. **Table 1** summarizes the primary outcomes and predictors by model and indicates the corresponding figures and tables. Analyses were conducted in Stata version 16.1 (StataCorp) (34).

To control for the effects of seasonality and overall time trends on child growth, we included harmonic terms representing trigonometric sine and cosine functions and a continuous daily time indicator, centered at 0 to represent the first day the study was initiated (35–37). We investigated multiple functional forms to control for age in our models and decided for ease of interpretation to stratify the models by narrow age categories, as well as by sex of the child, since our sample size is large enough to handle fine stratifications. Age categories were set in 3-month intervals, with separate models for children 6–8, 9–11, 12–14, 15–17, 18–20, 21–23, and 24–28 months. Models were specified for the entire sample, as well as each age and sex category as follows:

$$Y_{it} = \beta_0 + \beta_1 G + \beta_2 PG + \beta_3 I + \beta_4 S + \beta_5 d + \beta_6 \sin(2\pi\omega d) + \beta_7 \cos(2\pi\omega d) + \beta_8 \sin(4\pi\omega d) + \beta_9 \cos(4\pi\omega d) + \alpha_i + \varepsilon_{it} \quad (1)$$

Here, Y_{it} is the growth outcome for child i at time point t , which is either a contemporaneous measurement or a first difference (velocity). Indices for linear and ponderal growth are paired together by type (velocity z-scores, absolute velocity, attained size) and serve as each other's outcomes and primary explanatory variables in separate models. Pairs include length velocity z-scores (LVZ) and weight velocity z-scores (WVZ), length and weight velocity, and LAZ and WLZ. The coefficient β_1 represents the explanatory growth variable, G , which is the opposing linear or ponderal growth metric to the outcome (e.g.,

Financial support was provided by the United States Agency for International Development, through the Food Aid Quality Review project under contract number AID-OAA-C-16-00020.

Author disclosures: The authors report no conflicts of interest.

Supplemental Figures 1–3 and Supplemental Tables 1–12 are available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at <https://academic.oup.com/jn/>.

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Abbreviations used: LAZ, length-for-age z-score; LVZ, length velocity z-score; WLZ, weight-for-length z-score; WVZ, weight velocity z-score.

when the outcome is LVZ, the explanatory growth variable is WVZ). The relationship between the previous month's measurement and the current measurement for the outcome is represented by the coefficient β_2 , where PG is the previous month's measurement for the outcome variable. The effect of the explanatory growth variable on the outcome growth variable is therefore conditional on previous growth of the outcome variable. Morbidity in the previous 2 weeks is controlled for by coefficient β_3 , where I is a dichotomous indicator for any self-reported (by the caregiver) illness in the previous 2 weeks and study arm (S) is controlled for by β_4 . The coefficient β_5 represents a control for the overall daily time trend in the outcome of interest. Seasonality is controlled for by the sine and cosine terms, where ω is a constant equal to $1/365.25$, representing the frequency of the annual cycle in days, accounting for the 2016 leap year. Individual child-level random effects are accounted for by α_i , and ε_{it} is the time-varying error term.

Each model was fit with concurrent metrics from the same month for both the outcome and explanatory growth indices, as well as lagged (by 1 month) explanatory variables. For LVZ and WVZ, which are constant for 2-month periods of time, lagged growth indices were from the previous 2 months in chronological time.

In addition to these primary models, we examined the seasonality and overall time trends of linear and ponderal growth indices and morbidity indicators using predictions from harmonic regression models, with each of the growth indices and morbidity indicators as outcomes and only the trigonometric functions and daily trend terms as the predictors:

$$Y_{it} = \beta_0 + \beta_1 t + \beta_2 \sin(2\pi\omega d) + \beta_3 \cos(2\pi\omega d) + \beta_4 \sin(4\pi\omega d) + \beta_5 \cos(4\pi\omega d) + \alpha_i + \varepsilon_{it} \quad (2)$$

Here Y_{it} is the growth or morbidity outcome for child i at time point t , as in Equation 1 above. The coefficient β_1 represents the overall daily time trend in the outcome of interest, and seasonality is modeled using the sine and cosine terms, as described for Equation 1 above.

We focused on the entire distribution of LAZ and WLZ as opposed to drawing inferences about the relationships between linear and ponderal growth based on categorical indicators of undernutrition. However, as a secondary analysis, we fitted ordinary least squares regression models to examine the relationship between LAZ or WLZ at the end of the study and total months of wasting or stunting measurements throughout the study period, respectively. We fitted these models to increase generalizability of our analyses and to be able to compare our results with those from previous studies that examined the relationships between early episodes of wasting (between 0–17 months) and later stunting or LAZ (after 18 months) and to expand on those findings by also looking at the reverse (how early stunting effects later WLZ). These models took the form:

$$Y_i = \beta_0 + \beta_1 N_i + \beta_2 SA_i + \beta_3 S_i + \beta_4 ITotal_i + \beta_5 M_i + \beta_6 Sex_i + \varepsilon_i \quad (3)$$

Here, Y_{it} is the end (~ 27 months of age) LAZ or WLZ for child i ; the primary independent variable (N) is the total number of wasted or stunted months throughout the study period; SA represents the starting anthropometric z-scores; S indicates 3 binary dummy variables for the original study arm; $ITotal$ is an indicator of the total number of months in which an episode of illness was reported; M is an indicator from 1–12 for calendar month at the end of the child's participation in the study (~ 27 months of age); Sex is the child's sex; and ε is a random error term.

Patient and public involvement

Prior to implementation of this study, community leaders and members were consulted about the presence of the study teams in the communities, and leaders gave their approval and understanding of the importance of the study for the community priority of reducing the burden of undernutrition. Informed consent was obtained from all participants prior to enrollment in the study. In January 2018, study results from the original study were disseminated to members of the participant communities; at a meeting in Kaya, Burkina Faso; and to government officials of the Ministry of Health at a meeting in Ouagadougou.

Ethics

The Tufts University Health Sciences Institutional Review Board (IRB #: 10899) and the ethics board of the Ministry of Health in Burkina Faso (#: 2013–10-090) approved the original study in which anthropometric data were collected. Secondary analysis of the same anthropometric data for this paper was exempt by the Tufts University Health Sciences Institutional Review Board (IRB ID: STUDY00000255).

Results

Description of growth and morbidity

The descriptive analysis of growth and morbidity among children in this study revealed poor growth overall, and stark differences by both sex and age categories (Table 2). Males fared worse than females in all growth and morbidity parameters, regardless of the age category. Age-related patterns varied by growth indices. Both WVZ and LVZ increased as children aged, from -0.27 ± 0.49 to -0.05 ± 0.37 and -0.50 ± 1.05 to -0.16 ± 0.79 , respectively. The average LAZ value started negative (-0.66 ± 1.06) and declined as children aged, reaching a mean of -1.32 ± 0.98 by the time children were 24–28 months old. Absolute length velocity also declined as children aged, as expected in young children, starting at 1.29 ± 0.70 cm/month between 6–8 months of age and ending at 0.72 ± 0.51 cm/month between 24–28 months of age. Absolute weight velocity remained relatively constant, as children aged at a mean of 0.18 ± 0.34 kg/month, and WLZ values fluctuated between -1.03 ± 0.98 and -0.71 ± 1.05 , demonstrating no apparent overall age trend. Instances of morbidity decreased as children aged, with the highest prevalences for all morbidities when children were 6–8 months and decreasing prevalences thereafter.

We visualized these patterns, along with their seasonality components, using graphs of predicted growth from harmonic regression models of each index over time. We overlaid each pair of corresponding linear and ponderal growth indices to look at the average timing of peaks and nadirs in the indices in relation to each other (Figure 1). The seasonality of linear and ponderal growth indices do not differ by child sex; therefore, a single curve for each growth index is displayed, showing the averages over time for the entire sample. Each anthropometric measure shows 2 peaks and nadirs per year. Peak timing of the fastest length and weight velocities (both z-scores and absolute values) occur at almost the same time, at the end of December or beginning of January (cooler, dry season) and again near the end of July (near the peak of the rainy season; Figure 1A and B). The slowest points for weight velocity, in mid-April (hot, dry season) and again in mid-October (hot, dry season), are followed slightly over a month later (39–42 days) by the slowest points in length velocity, at the end of May (hot, pre-rainy season) and the end of November (hot, dry season). LAZ values did not demonstrate seasonal trends, so the LAZ average temporal relationship with WLZ is harder to distinguish (Figure 1C). Peaks in fever and diarrhea prevalences happen within 19 days of the nadirs in ponderal growth indicators, though they sometimes occur before and sometimes right after the periods of slowest ponderal growth (Figure 1D).

Relationship between linear and ponderal growth

Figure 2 shows the Pearson correlation coefficients between each combination of corresponding linear and ponderal growth indices at different lag periods. Each lag period is a 1-month time interval between the current month's measurement and the lagged measurement. Current length is highly positively corre-

TABLE 2 Characteristics of study children, Sanmatenga Province, Burkina Faso, 2014–2016

	Overall	Age Block						
		6–8 months	9–11 months	12–14 months	15–17 months	18–20 months	21–23 months	24–28 months
<i>n</i>	5039	5037	5020	5017	4984	4993	5002	5033
Total number of child-months, observations	108,802	16,539	14,491	15,010	15,230	15,177	15,198	16,452
Outcomes								
Ever concurrently stunted ¹ and wasted ² during time period, %	16.25	3.77	6.15	8.35	8.24	7.92	6.39	6.19
Females	11.15	1.81	3.54	5.11	5.27	4.95	3.90	4.43
Males	21.21	5.68	8.69	11.51	11.12	10.80	8.81	7.91
Length velocity z-score, mean ± SD	−0.37 ± 0.92	−0.50 ± 1.05	−0.55 ± 0.96	−0.50 ± 0.91	−0.27 ± 0.86	−0.20 ± 0.82	−0.16 ± 0.79	—
Females	−0.38 ± 0.94	−0.51 ± 1.07	−0.57 ± 0.98	−0.50 ± 0.93	−0.27 ± 0.86	−0.21 ± 0.84	−0.17 ± 0.82	—
Males	−0.36 ± 0.90	−0.50 ± 1.04	−0.53 ± 0.95	−0.50 ± 0.89	−0.26 ± 0.85	−0.19 ± 0.80	−0.15 ± 0.77	—
Weight velocity z-score, mean ± SD	−0.10 ± .42	−0.27 ± 0.49	−0.13 ± 0.43	−0.05 ± 0.39	−0.04 ± 0.38	−0.03 ± 0.38	−0.05 ± 0.37	—
Females	−0.09 ± .41	−0.23 ± 0.46	−0.12 ± 0.42	−0.04 ± 0.40	−0.05 ± 0.38	−0.04 ± 0.37	−0.05 ± 0.36	—
Males	−0.11 ± .43	−0.30 ± 0.51	−0.15 ± 0.44	−0.05 ± 0.39	−0.03 ± 0.38	−0.02 ± 0.38	−0.05 ± 0.38	—
Length velocity, cm/month, mean ± SD	0.94 ± 0.59	1.29 ± 0.70	1.07 ± 0.58	0.95 ± 0.57	0.93 ± 0.56	0.89 ± 0.54	0.83 ± 0.50	0.72 ± 0.51
Females	0.95 ± 0.59	1.30 ± 0.70	1.08 ± 0.59	0.97 ± 0.57	0.96 ± 0.57	0.90 ± 0.55	0.84 ± 0.50	0.73 ± 0.51
Males	0.92 ± 0.58	1.29 ± 0.69	1.05 ± 0.58	0.94 ± 0.58	0.91 ± 0.55	0.87 ± 0.53	0.82 ± 0.49	0.70 ± 0.50
Weight velocity, kg/month, mean ± SD	0.18 ± 0.34	0.19 ± 0.30	0.18 ± 0.31	0.18 ± 0.33	0.20 ± 0.33	0.18 ± 0.35	0.17 ± 0.35	0.15 ± 0.39
Females	0.18 ± 0.33	0.20 ± 0.29	0.19 ± 0.30	0.18 ± 0.32	0.19 ± 0.32	0.18 ± 0.34	0.17 ± 0.34	0.15 ± 0.38
Males	0.18 ± 0.35	0.18 ± 0.31	0.18 ± 0.33	0.17 ± 0.33	0.20 ± 0.33	0.17 ± 0.35	0.18 ± 0.35	0.16 ± 0.39
Length-for-age z-score, mean ± SD	−1.11 ± 1.04	−0.66 ± 1.06	−0.90 ± 1.04	−1.11 ± 1.02	−1.23 ± 1.02	−1.27 ± 1.01	−1.28 ± 0.99	−1.32 ± 0.98
Females	−1.00 ± 0.98	−0.55 ± 0.98	−0.79 ± 0.96	−0.99 ± 0.95	−1.11 ± 0.95	−1.15 ± 0.94	−1.17 ± 0.94	−1.22 ± 0.93
Males	−1.22 ± 1.09	−0.77 ± 1.11	−1.02 ± 1.09	−1.23 ± 1.08	−1.35 ± 1.07	−1.39 ± 1.06	−1.39 ± 1.03	−1.42 ± 1.01
Weight-for-length z-score, mean ± SD	−0.86 ± 0.97	−0.71 ± 1.05	−0.95 ± 1.01	−1.03 ± 0.98	−0.91 ± 0.94	−0.85 ± 0.94	−0.78 ± 0.91	−0.80 ± 0.91
Females	−0.77 ± 0.92	−0.66 ± 0.98	−0.86 ± 0.96	−0.91 ± 0.93	−0.80 ± 0.90	−0.74 ± 0.90	−0.70 ± 0.87	−0.76 ± 0.89
Males	−0.94 ± 1.01	−0.76 ± 1.11	−1.05 ± 1.06	−1.14 ± 1.01	−1.02 ± 0.97	−0.96 ± 0.96	−0.86 ± 0.93	−0.85 ± 0.94
Illness in last 2 weeks, %	32.74	41.42	38.23	33.42	32.32	30.88	26.55	25.98
Females	31.74	39.89	36.77	32.59	31.77	30.09	26.04	24.86
Males	33.65	42.89	39.64	34.23	32.85	31.64	27.03	26.94
Ever fever during time period, %	60.85	22.71	15.34	13.89	12.90	11.90	9.54	12.76
Females	59.22	21.30	14.22	12.90	12.74	11.07	9.31	12.06
Males	62.43	24.08	16.42	14.85	13.06	12.70	9.76	13.44
Ever diarrhea during time period, %	49.39	16.40	15.32	13.43	10.53	8.35	6.40	7.15
Females	45.81	13.77	13.82	12.22	9.32	7.26	6.38	6.57
Males	52.88	18.95	16.78	14.62	11.71	9.42	6.41	7.72
Total number stunted months, mean ± SD	3.78 ± 6.59	—	—	—	—	—	—	—
Females	2.92 ± 6.59	—	—	—	—	—	—	—
Males	4.61 ± 7.18	—	—	—	—	—	—	—
Total number wasted months, mean ± SD	2.39 ± 4.44	—	—	—	—	—	—	—
Females	1.80 ± 3.79	—	—	—	—	—	—	—
Males	2.96 ± 4.92	—	—	—	—	—	—	—

¹ Stunting was defined as a length-for-age z-score < −2.

² Wasting was defined as a weight-for-length z-score < −2.

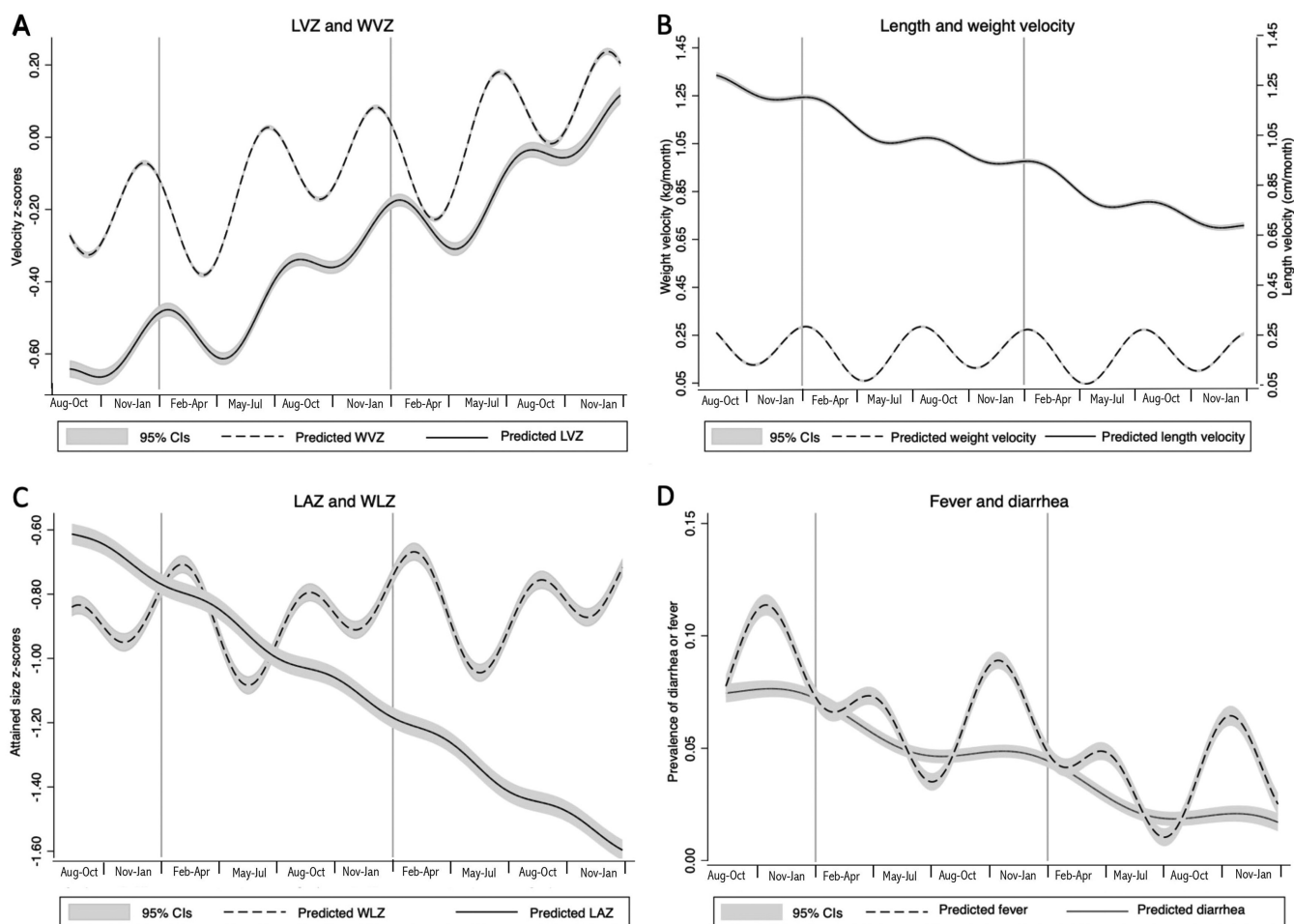


FIGURE 1 Seasonality of linear and ponderal growth indicators and morbidity indicators among children 6–28 months in Burkina Faso, based on model predictions from harmonic regression models. Vertical reference lines signify calendar years; (A) LVZ compared to WVZ; (B) length velocity (cm/month) compared to weight velocity (kg/month); (C) LAZ compared to WLZ; and (D) fever prevalence compared to diarrhea prevalence. LAZ, length-for-age z-score; LVZ, length velocity z-score; WLZ, weight-for-length z-score; WVZ, weight velocity z-score.

lated with previous weight, even after 9 lag periods. Similarly, current weight is positively correlated with previous length, but to a lesser degree, with a decreasing correlation as lag periods increase (Figure 2A). The current LAZ remains positively correlated with the previous WLZ at roughly the same level, regardless of the lag period, with slight increases in the magnitude of correlation with lags 3–4 of WLZ. The current WLZ is also positively correlated with the previous LAZ, with small declines as the lag periods increase (Figure 2C). Velocity indices have much lower correlations with each other than attained size indices, and have less consistent relationships (Figure 2B and D).

Figure 3 reveals how individual children's linear and ponderal growth velocity z-scores relate to each other in each age category, stratified by sex. Overall, the WVZ increases modestly if the average LVZ increases in the same month (0.07–0.13 increase in WVZ per unit increase in concurrent LVZ). However, the WVZ decreases slightly if the LVZ increased in the previous month (0.009–0.02 decrease in WVZ per unit increase in lagged LVZ; Figure 3A and C). This relationship is strongest among children 9–14 months old. The LVZ increases greatly if the WVZ increases either concurrently or in the previous time period (0.21–0.72 increase in LVZ per unit increase in concurrent or lagged WVZ). The highest increases in LVZ associated with an increased WVZ happen in children 6–8 months, and relationships are slightly stronger among females compared to males in

infancy (Figure 3B and D). The full model results, including CIs and *P* values from all models that make up Figure 3, are available in Supplemental Tables 1–12. Sensitivity analyses using absolute length and weight velocities (cm/month and kg/month, respectively) reveal similar results to the velocity z-scores (Supplemental Figure 2). Results from attained LAZ or WLZ models show small increases in LAZ after concurrent and previous increases in WLZ, as well as small increases in WLZ after concurrent or previous increases in LAZ (Supplemental Figure 3).

Ordinary least squares regression of LAZ and WLZ on the total number of wasted or stunted months revealed that conditional on the starting z-scores, month of measurement, study arm, total episodes of illness, and sex, each additional wasted month throughout the study period is associated with a 0.023 decrease (95% CI, –0.027 to –0.019) in LAZ at study end and each additional stunted month is associated with a 0.015 decrease (95% CI, –0.018 to –0.011) in WLZ at study end (Table 3).

Discussion

In this analysis of the temporal relationships between individual children's length and weight growth, we find that episodes of faster ponderal growth are associated with concurrent

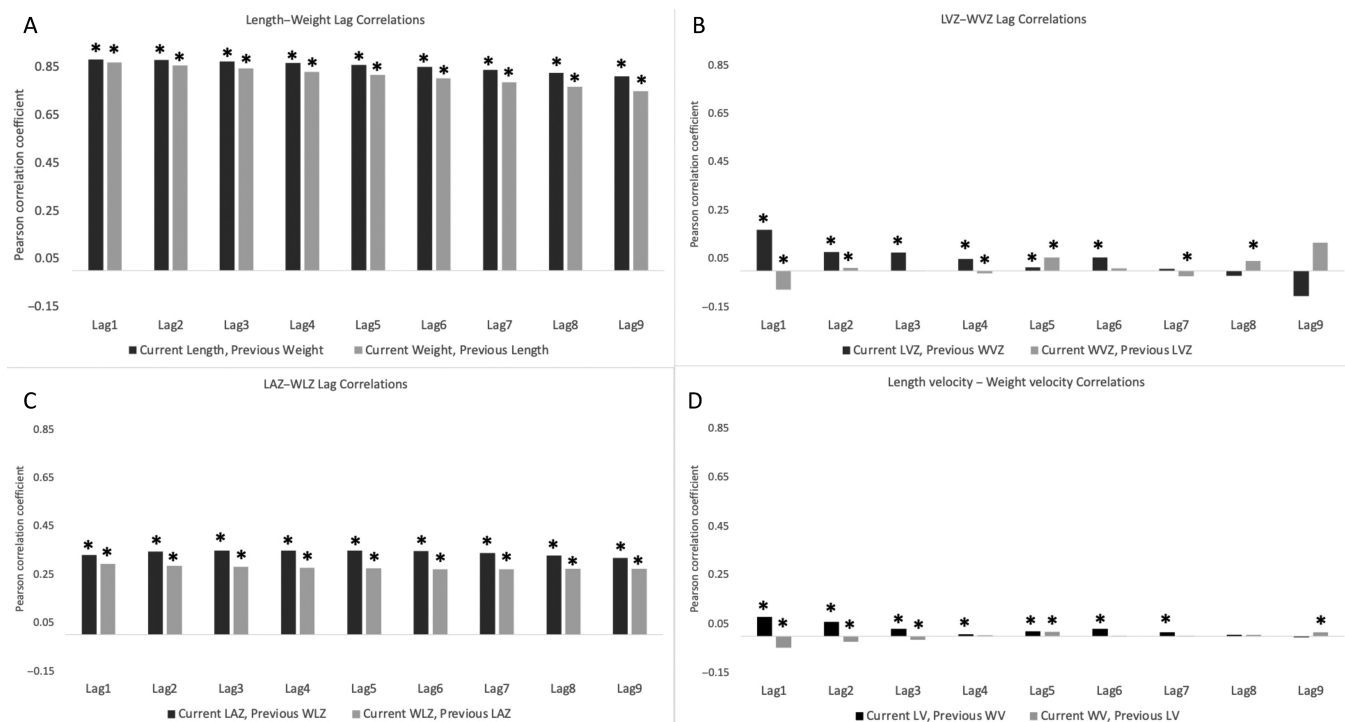


FIGURE 2 Correlations between length and weight indicators of children 6–28 months in Burkina Faso, at different lag periods. Values are Pearson correlation coefficients. (A) Length (cm) and weight (kg) correlations; (B) LVZ and WVZ correlations; (C) LAZ and WLZ correlations; and (D) LV (cm/month) and WV (kg/month) correlations. * $P < 0.05$. LAZ, length-for-age z-score; LV, length velocity; LVZ, length velocity z-score; WLZ, weight-for-length z-score; WV, weight velocity; WVZ, weight velocity z-score.

and subsequent faster linear growth, but that episodes of faster linear growth are associated with slower future ponderal growth. Recently, the Wasting-Stunting Technical Interest Group has produced several articles and policy briefs outlining the important connections between stunting and wasting (38), and a new systematic review has confirmed the growing evidence supporting such relationships, making the case for aggregating interventions that address both forms of undernutrition (10). These papers and reports have begun to change the paradigms for addressing undernutrition (39). While they present convincing evidence that there is a significant relationship between earlier episodes of wasting and later linear growth faltering (6, 14, 17–25), the focus on indicators of undernutrition rather than the distribution of anthropometric indices limits interpretation of temporality between linear and ponderal growth. Our analysis confirms previous findings that episodes of wasting are associated with lower LAZ values and adds important insights into this temporal relationship, using velocity measures to show that linear and ponderal growth faltering may result from common environmental influences.

The strong association between linear and ponderal growth velocities in the same month at the individual level suggests that conditions that affect 1 anthropometric parameter affect the other at roughly the same time. Many have suggested, based on findings using attained size, that linear growth faltering often happens following ponderal growth faltering, and that linear growth faltering is partially a biological response to being wasted (23, 39). However, given the strong concurrent associations between linear and ponderal growth velocities, the 2- to 3-month temporal delay observed between low WLZ and low LAZ measurements in previous studies may be an indication of the relative speeds of these 2 processes. Linear

growth faltering is a slower process than wasting, in that the body loses mass (fat- and fat-free masses) relatively quickly in growth-limiting conditions, in contrast to the lag period between growth-limiting conditions and a lower LAZ (11). Put differently, the 2 parameters are affected at the same time but, given the slower process of linear growth, the effects are apparent in weight before they become apparent in length. Our investigation into the overall time trends and seasonality patterns of growth in different anthropometric parameters also supports the idea that the same growth-limiting conditions related to the proximal determinants of undernutrition (e.g., dietary intake and disease) affect both length and weight velocities. A comparison of the slowest average ponderal growth period to the slowest average linear growth period among all children in our sample separates the 2 by about 1 month, whereas previous studies that have looked at this question in terms of attained size indicators have found that LAZ declines roughly 2–3 months after declines in WLZ (25, 28, 40). As such, the association between linear and ponderal growth may be a consequence of shared environmental drivers. Whether the deficient environment affects ponderal growth velocity first, which then influences linear growth, or whether the processes happen independently of each other at different speeds requires further study. What is clear is that the biological mechanisms underlying the growth process are at least somewhat dependent on the temporal relationships between linear and ponderal growth.

We found that on average, fever and diarrhea prevalences peak around the same time as the slowest ponderal growth velocity, which is followed shortly by the period of slowest linear growth velocity. While we cannot establish the directionality of the effect between illness and slow ponderal growth in this analysis, the temporal proximity of the slowest ponderal

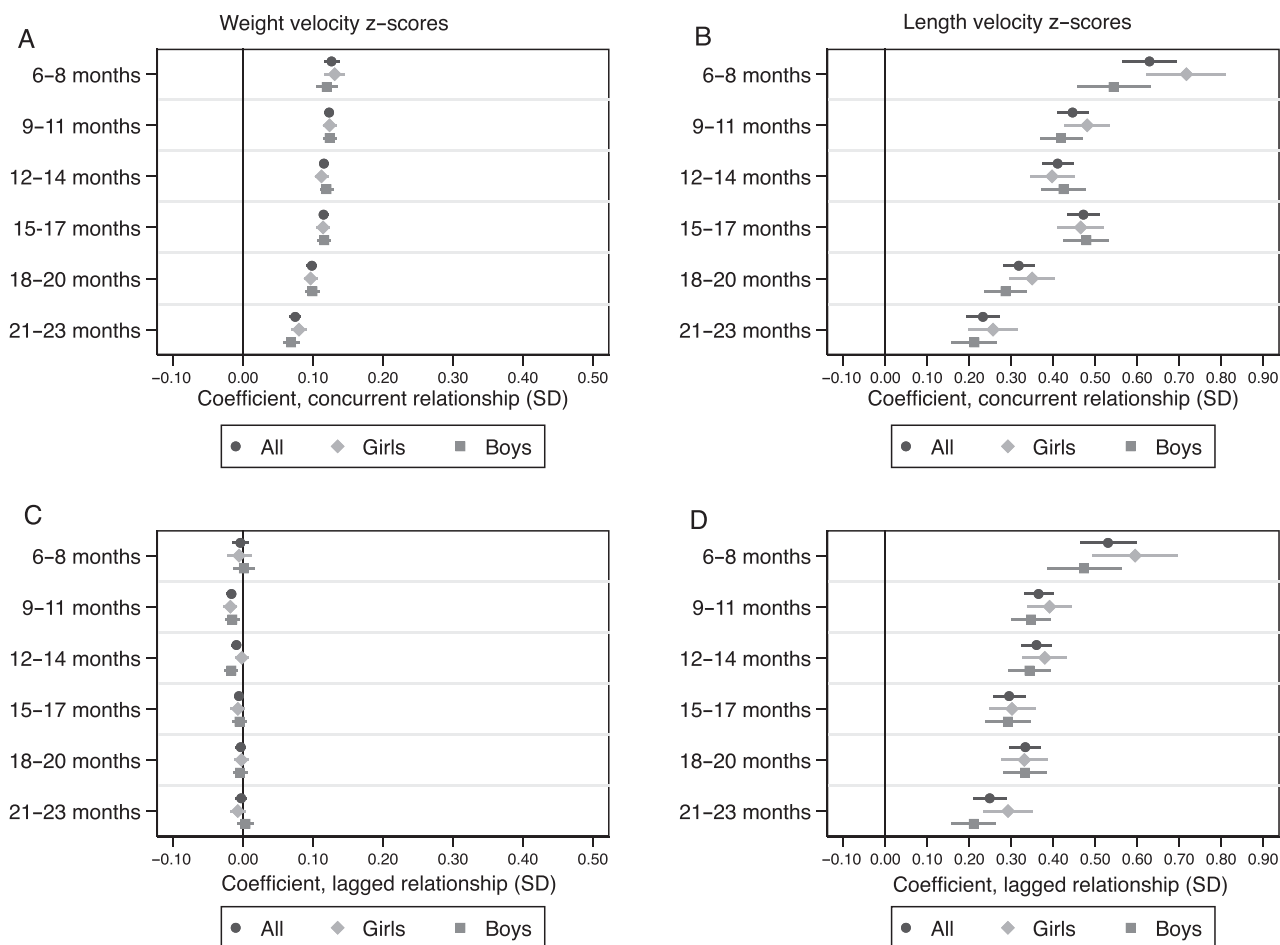


FIGURE 3 Relationships between linear and ponderal growth velocity z-scores among children 6-28 months in Burkina Faso, by age and sex. Coefficients derived from linear mixed-effects regressions, shown with 95% CIs. (A) Change in WVZ per unit increase in concurrent LVZ; (B) change in LVZ per unit increase in concurrent WVZ; (C) change in WVZ per unit increase in lagged LVZ from the previous period; and (D) change in LVZ per unit increase in lagged WVZ from the previous period. LVZ, length velocity z-score; WVZ, weight velocity z-score.

growth velocity to peaks in fever and diarrhea demonstrates the effects of the undernutrition-infection cycle, whereby illness leads to nutrient loss, malabsorption, and loss of appetite, which leads to growth faltering and compromised immunity (41–44). Environmental conditions may lead first to ponderal velocity slowdowns and then to linear velocity slowdowns through several potential mechanisms. Fat mass is important for the production of leptin, and leptin is needed for both the immune response and bone growth (11, 45). In addition, inflammation due to repeated infections from poor environmental conditions can lead to sustained higher levels of proinflammatory cytokines, which also inhibit growth (46). Multiple insults to ponderal growth velocity, perpetuated by the vicious cycle of infection and lowered immunity, will continuously lead to slower linear growth velocity, eventually manifesting as a low LAZ. This relationship is particularly relevant to the context of many low- and middle-income countries, such as Burkina Faso, in which environmental and socioeconomic conditions impact growth in the entire population (47, 48).

We also find, contrary to the study in which linear growth faltering was associated with a higher risk of future wasting (26), that faster linear growth is associated with slower future ponderal growth, conditional on the previous ponderal growth. While the magnitude of the relationship is small, it is helpful to interpret the finding in relation to the observed upper

limits of changes in z-scores. Few studies have investigated changes in growth velocity z-scores, but the largest observed changes in height-for-age z-scores or weight-for-age z-scores after complementary feeding interventions are limited to 0.25 and 0.41 increases in z-score, respectively (49). It would be informative if future studies could determine the upper limits of velocity changes in response to interventions. Regardless of the magnitude of the association or its clinical relevance, this study reveals an important temporal dependency between length velocity in 1 period and weight velocity in the next. This relationship is clear using multiple anthropometric metrics, and merits further exploration into its clinical significance. When dietary intake is inadequate to maintain metabolic processes, the body is forced to draw on nutritional reserves in fat and muscle tissues to ensure that organs have enough energy (50). In addition, linear growth increases energy requirements through the association with accretion of lean tissue (51). Growth spurts among children in the context of Burkina Faso may not be accompanied by sufficient increases in dietary intake to avoid slowdowns in weight gain due to these physiological adjustments. Future research is needed to determine the cumulative effects of such a temporal dependency between linear and ponderal growth, and any related implications. In the meantime, nutrition programs should consider the effects that linear growth spurts may have on weight gain in contexts where di-

TABLE 3 Relationship between attained size at study end (~27 months) and total numbers of wasted or stunted months among children 6–28 months in Burkina Faso¹

	End LAZ	Adjusted End LAZ	End WLZ	Adjusted End WLZ
Total # wasted months	– 0.060 ² (–0.066 to –0.054)	– 0.023 ² (–0.027 to –0.019)	—	—
Start LAZ	—	0.672 ² (0.655–0.688)	—	—
Total # stunted months	—	—	– 0.025 ² (–0.029 to –0.021)	– 0.015 ² (–0.018 to –0.011)
Start WLZ	—	—	—	0.475 ² (0.455–0.496)
Study arm (ref = CSB+)				
CSWB	—	– 0.155 ² (–0.206 to –0.104)	—	– 0.058 (–0.121, 0.004)
SC+	—	0.062 ³ (0.012–0.112)	—	– 0.086 ⁴ (–0.147 to –0.025)
RUSF	—	– 0.024 (–0.075, 0.028)	—	– 0.011 (–0.074, 0.052)
Total # illness episodes	—	– 0.008 ⁴ (–0.013 to –0.002)	—	– 0.018 ² (–0.024 to –0.011)
End month (ref = March)				
April	—	– 0.161 (–0.811, 0.490)	—	– 0.109 (–0.903, 0.685)
May	—	0.030 (–0.583, 0.643)	—	– 0.298 (–1.047, 0.450)
June	—	0.116 (–0.495, 0.728)	—	– 0.265 (–1.012, 0.482)
July	—	0.040 (–0.572, 0.652)	—	– 0.023 (–0.770, 0.724)
August	—	0.106 (–0.506, 0.718)	—	0.063 (–0.684, 0.811)
September	—	0.175 (–0.437, 0.787)	—	0.012 (–0.735, 0.760)
October	—	0.185 (–0.427, 0.798)	—	– 0.148 (–0.896, 0.599)
November	—	0.233 (–0.379, 0.844)	—	– 0.161 (–0.907, 0.586)
December	—	0.281 (–0.330, 0.892)	—	– 0.008 (–0.754, 0.738)
Female	—	0.020 (–0.015, 0.055)	—	0.022 (–0.020, 0.065)
Constant	– 1.208 ² (–1.237 to –1.178)	– 0.993 ⁴ (–1.606 to –0.381)	– 0.686 ² (–0.715 to –0.657)	– 0.229 (–0.977, 0.519)
<i>n</i>	5003	5003	5003	5003
R ²	0.076	0.590	0.032	0.330

¹Values are coefficients (95% CIs) from ordinary least squares regression models. CSB+, Corn Soy Blend Plus; CSWB, Corn Soy Whey Blend; LAZ, length-for-age z-scores; RUSF, Ready-to-use supplementary food; SC+, SuperCereal Plus; WLZ, weight-for-length z-scores.

²*P* < 0.001.

³*P* < 0.05.

⁴*P* < 0.01.

etary intake is insufficient, ensuring that children have adequate macro- and micronutrient intakes during times of intense linear growth.

Last, consistent with other studies, we find that male children are more vulnerable to growth insults than female children and are more likely to experience linear and ponderal growth faltering concurrently (38). In addition, the temporal relationships between linear and ponderal growth are strongest among the youngest children. Males and younger children also experience higher prevalences of illness, further bolstering the importance of the morbidity pathway's influence on linear and ponderal growth trajectories and their temporal dependencies.

A key limitation of this study is the lack of longitudinal data on food security and diet diversity, which may be important exposures that influence growth velocity in addition to morbidity. We were unable to explore the temporal relationships between these factors and linear and ponderal growth velocities, but doing so in future research could add to the understanding of the mechanisms through which linear and ponderal growth are related. In addition, given the strong association between LAZ at the beginning and end of the study, which indicates the continued effects of early growth faltering, it would be beneficial to have data on maternal characteristics, as well as the gestational age and birth size of the infants, to be able to examine intergenerational growth faltering. Last, all children in this study were receiving a supplemental food, which may have influenced linear and ponderal growth trajectories. Any influence of the supplemental foods on growth would likely have been protective of slowdowns, which would make our

results underestimations of the true relationship between linear and ponderal growth.

Our findings linking linear and ponderal growth to shared environmental drivers reinforce the importance of improving living conditions to address growth faltering through a systems approach. Programs and policies should target community-level deficiencies that constrain growth in both parameters through inflammatory and dietary challenges. This includes, for example, improving sanitation and hygiene infrastructures, providing clean water, eliminating mosquito breeding grounds, and improving market connectivity to enable year-round access to nutritious foods. Finally, assessing growth velocity and associated changes in growth drivers is the best way to understand the temporal dependencies between linear and ponderal growth. We have only scratched the surface of complex growth relationships, raising questions about the time series properties of growth velocity and the related physiological mechanisms along the way. Answering these questions requires observations on both growth and its drivers at multiple time points, and while it may require additional resources, researchers should prioritize systematic serial growth monitoring, as feasible.

Acknowledgments

Data used in this study were collected by researchers and enumerators at the Institut de Recherche en Sciences de la Santé in Burkina Faso. We thank the study participants and communities.

The authors' responsibilities were as follows – IRC: supervised data collection, conceived of the research questions

and analyses, performed the analyses, wrote the manuscript, and had primary responsibility for the final product; FG, LNO: supervised data collection and critically reviewed the manuscript; ENN, WAM, BLR, NP: advised on analyses and critically reviewed the manuscript; and all authors: read and approved the final manuscript and agree to be accountable for all aspects of the work.

Data availability

Anthropometric data described in the manuscript as well as codebooks are available at: <https://data.usaid.gov>. Analytic code will be made available upon request.

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