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Successive 1-Month Weight Increments in Infancy Can Be Used to Screen for Faltering Linear Growth[1,2]

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Abstract

Background: Linear growth faltering in the first 2 y contributes greatly to a high stunting burden, and prevention is hampered by the limited capacity in primary health care for timely screening and intervention.

Objective: This study aimed to determine an approach to predicting long-term stunting from consecutive 1-mo weight increments in the first year of life.

Methods: By using the reference sample of the WHO velocity standards, the analysis explored patterns of consecutive monthly weight increments among healthy infants. Four candidate screening thresholds of successive increments that could predict stunting were considered, and one was selected for further testing. The selected threshold was applied in a cohort of Bangladeshi infants to assess its predictive value for stunting at ages 12 and 24 mo.

Results: Between birth and age 12 mo, 72.6% of infants in the WHO sample tracked within 1 SD of their weight and length. The selected screening criterion (''event'') was 2 consecutive monthly increments below the 15th percentile. Bangladeshi infants were born relatively small and, on average, tracked downward from approximately age 6 to <24 mo (51% stunted). The population-attributable risk of stunting associated with the event was 14% at 12 mo and 9% at 24 mo. Assuming the screening strategy is effective, the estimated preventable proportion in the group who experienced the event would be 34% at 12 mo and 24% at 24 mo.

Conclusions: This analysis offers an approach for frontline workers to identify children at risk of stunting, allowing for timely initiation of preventive measures. It opens avenues for further investigation into evidence-informed application of the WHO growth velocity standards.   *J Nutr* 2015;145:2725–31.

Keywords: growth velocity, weight increments, linear growth, stunting prevention, growth faltering, malnutrition screening

Introduction

In 2012, the World Health Assembly adopted a resolution on maternal, infant, and young child nutrition that includes 6 global targets to reduce the high burden of disease associated with malnutrition (1). The first target addresses stunting, with the aim to reduce by 40% the number of stunted children <5 y of age by 2025 (2).

A large part of the linear growth faltering that contributes to the high burden of stunting in developing countries accrues in the period from conception to age 2 y (3, 4). That period represents a window of opportunity for active case identification and timely interventions that could help prevent stunting before it becomes irreversible, with long-term negative consequences (5, 6). Growth assessment at these young ages focuses mainly on weight, however, with little active screening for stunting in primary health care services. The lack of appropriate equipment (accurate and robust infantometers for measuring length) and limited capacity of health workers to take reliable and accurate

length measurements are largely responsible for this situation. If routine weight measurements could be used to detect the risk of linear growth faltering, the role of primary health care services in stunting prevention could be enhanced.

Scientific evidence exists to support such an approach. A study in severely undernourished children in Jamaica found in most of the sample that linear growth remained stagnated until a substantial recovery of their weight-for-length had been achieved (7). Similarly, in Nepal, patterns of weight and height velocity in children recovering from undernutrition suggested that weight recovery preceded the resumption of linear growth (8). More recently, Richard et al. (9) reported a correlation between wasting or fluctuating weight-for-length at ages 6–17 mo, with an increased risk of linear growth retardation by 18–24 mo. This suggests that changes in weight could serve to predict subsequent adverse linear growth tendencies.

With regard to the timely detection of faltering or excessive growth, velocity may be better able than attained growth alone to detect patterns that signal risk (10–13). Whereas pathogenic factors affect growth velocity directly, their impact on attained size takes longer to become measurable (10). Unlike the assessment of attained growth in which serial measurements of individual children tend to "track" along fixed centiles, there is little correlation between successive increments (i.e., estimates of growth velocity) in healthy, normally growing children (14, 15). Oscillations between high and low centiles in successive intervals make it difficult to judge the risk of long-term growth outcomes from a single increment, even if extreme. Moreover, a velocity estimate requires 2 measurements, and if the interval between these is relatively short, the contribution of measurement errors to the increment can be substantive (16). However, whereas a negative measurement error in the second measurement will underestimate growth in a given interval, the authors argue that such an underestimation will tend to be balanced by a high increment over the next interval and that successive small increments likely indicate a real slowing down in growth. Therefore, some authors have proposed the use of 2 consecutive increments below specified thresholds as an approach to detecting risk. Brook et al. (14) and Healy et al. (17) proposed the use of the 25th percentile (P^{12}; P25) on the premise that the probability of obtaining 2 independent consecutive increments below this cutoff would be 6.25%, whereas Roche and Sun (18) proposed the use of P5. Similarly, Cole (19, 20) demonstrated by using the concept of the thrive line overlay on weight-for-age charts that velocity below P5 in 2 successive months would identify 0.5–2% of infants as failing to thrive depending on age and size.

The objective of this study was to determine a feasible approach to predicting long-term linear growth faltering from consecutive 1-mo weight increments in the first year of life on the basis of the WHO growth velocity standards (15, 21). (Monthly increments in the WHO standards were developed only for infancy because follow-up in the second year was in bimonthly intervals.)

To achieve the objective, a 2-part analysis was implemented. In the first part, data from the WHO reference sample were used to examine patterns and establish benchmarks of successive growth velocities and associated changes in attained size in a healthy population. Four combinations of successive increment percentiles were considered and one selected as the candidate screening

threshold for further testing. In the second part of the analysis, this candidate screening threshold was applied in a sample of predominantly undernourished Bangladeshi children (22) to examine its association with subsequent length-for-age.

Methods

Subjects. The WHO sample consisted of 882 children (428 boys and 454 girls) whose mothers complied fully with the Multicentre Growth Reference Study (MGRS) infant feeding and no-smoking criteria and completed the follow-up period of 24 mo (from a total of 1743 enrolled). The children were measured at birth; at weeks 1, 2, 4, and 6; monthly from 2–12 mo; and bimonthly in the second year (23).

The Maternal and Infant Nutrition Intervention in Matlab (MINI-Mat) trial in Bangladesh enrolled mothers early in pregnancy, assigned them to 6 intervention groups, and followed their infants after birth from age 1 to 24 mo (22, 24). The infants were not targeted with any intervention. The sample included 1788 boys and 1741 girls. Weight and length measurements were taken at scheduled monthly visits from 1 to 12 mo and then at 15, 18, 21, and 24 mo. To study overall length-for-age changes between early infancy and age 2 y in relation to 1-mo weight increments below P15 in the first year, analysis focused on 2900 children (1472 boys and 1428 girls) who had *1*) their first length measurement taken before age 6 mo and the last one between 18 and 24 mo and *2*) at least one block of 3 consecutive monthly weight measurements taken during the first year of life. Sample sizes varied by follow-up visit, because in addition to missed visits, infants entered the protocol at variable ages after birth.

Statistical methods. For both samples, we derived length-for-age z score (LAZ) and weight-for-age z score (WAZ) at each visit on the basis of the WHO Child Growth Standards (25). Similarly, monthly weight increments (birth to 12 mo) were calculated for each child. These were adjusted according to the same rules that were applied when constructing the WHO velocity standards. The target ages of the MGRS measurement schedule in the first year were 0, 28, 61, 91, 122, 152, 183, 213, 244, 274, 304, 335, and 365 d, with tolerable variations of ±3 d in the first 6 mo and ±5 d from 7 to 12 mo. To construct the standards, measurements taken outside the above-stated tolerable variations were estimated to the exact target age by linear interpolation (15). In the present analysis, increments were discarded if the interval between measurements was <15 or >45 d.

It was important to first establish centile-crossing patterns in the WHO standard sample as a benchmark for normal variations in WAZ and LAZ among healthy infants. To this end, changes in WAZ and LAZ from birth to 12 mo were calculated for each child and analyzed in 3 strata defined by WAZ at birth ($z \le -1$ SD, $-1 < z \le 1$ SD, and $z > 1$ SD).

For growth velocity analyses, only the 1-mo weight increments from birth to age 12 mo were selected because the focus was on timely screening for growth faltering. Four potential screening thresholds were considered, namely 2 consecutive increments below P25, P15, and P10 and the combination of one increment below P5 followed by another below the median (P50). The threshold of P15 was used for subsequent analyses.

A binary variable was created to indicate the occurrence (yes or no) of 2 consecutive 1-mo weight increments below the P15 threshold (referred to hereafter as "the event"). To examine links between the event and continued downward trajectories in attained size in the WHO sample, mean changes in WAZ and LAZ associated with the event were studied at 4 alternative postevent observation points, that is at 0, 1, 4, and 6 mo after the end of the event. These changes were calculated by using, as a starting point, the child's age at the start of the first of the 2consecutive below-threshold weight increments.

For the Bangladeshi sample, patterns of stunting (LAZ < −2) prevalence and mean WAZ and LAZ from early infancy to age 2 y were studied, as were frequencies of consecutive increments below the test threshold and their association with LAZ. Means were calculated with their respective 95% CIs. To evaluate the influence of initial WAZ, a logistic model was fitted to assess the impact of the occurrence of the event between 6 and 9 mo (period in which we observe the steepest decrease in

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[12] Abbreviations used: LAZ, length-for-age z score; MGRS, Multicentre Growth Reference Study; MINIMat, Maternal and Infant Nutrition Intervention in Matlab; P, percentile; WAZ, weight-for-age z score; WHA, World Health Assembly.

TABLE 1 Frequency of 2 consecutive 1-mo weight increments below selected thresholds in the WHO growth velocity standards reference sample[1]

Consecutive age intervals, mo	Total *n*	Consecutive increments below specified thresholds, % (*n*)			
		25th Percentile	15th Percentile	10th Percentile	5th and 50th Percentiles
0–1,1–2	855	7.7 (66)	3.0 (26)	1.5 (13)	4.0 (34)
1–2, 2–3	846	9.1 (77)	3.0 (25)	1.5 (13)	3.2 (27)
2–3, 3–4	835	8.1 (68)	2.9 (24)	1.2 (10)	3.5 (29)
3–4, 4–5	835	5.6 (47)	2.6 (22)	1.1 (9)	2.0 (17)
4–5, 5–6	834	5.8 (48)	3.1 (26)	1.9 (16)	2.2 (18)
5–6, 6–7	837	5.6 (47)	2.3 (19)	1.4 (12)	1.4 (12)
6–7, 7–8	833	6.1 (51)	2.5 (21)	1.4 (12)	2.3 (19)
7–8, 8–9	817	4.0 (33)	2.1 (17)	0.7 (6)	1.1 (9)
8–9, 9–10	803	5.0 (40)	1.5 (12)	0.8 (6)	2.7 (22)
9–10, 10–11	806	4.2 (34)	1.1 (9)	0.5 (4)	1.6 (13)
10–11, 11–12	800	4.0 (32)	1.6 (13)	0.5 (4)	2.1 (17)

[1] Data are derived from reference 15.

LAZ) on the risk of growth faltering during late infancy. The response variable for this model was defined as a binary variable equal to 1 (and zero otherwise) if the decrease in LAZ exceeded −0.78 between age 5 and 12 mo (i.e., baseline measurement taken just before the event with allowance for up to 3 mo postevent follow-up to detect faltering). The cutoff of −0.78 LAZ corresponds to the first quartile of the sample's distribution of change in LAZ. The pattern of faltering across initial WAZs was examined. Furthermore, the risk of being stunted at 12 and 24 mo that was attributable to the event—in the total population and in the group who experienced the event—was calculated, differentiating those in whom the event had or had not occurred. The attributable risk at 12 mo for the total population = $100(P_T - P_N)/P_T$, where P_T is the proportion stunted at 12 mo in the total population and P_N is the proportion stunted at 12 mo of those who did not experience the event (26). The attributable risk at 12 mo for the group who experienced the event = $100(P_E - P_N)/P_E$, where P_E is the proportion stunted at 12 mo of those who experienced the event. Parallel calculations were conducted for the proportion stunted at 24 mo. The significance level used was 0.05.

Results

Analysis of length-for-age tracking in the WHO sample revealed that, of 869 children with nonmissing data at birth and at 12 mo,

LAZ at 12 mo was within 1 SD of LAZ at birth for 631 children (72.6%). Of 127 (14.6%) whose LAZ declined by >1 SD, only 1 (0.8%) had WAZ < −1 at birth. Of the 111 infants (12.8%) whose LAZ shifted upward by >1 SD, only 2 (1.8%) had a WAZ >1 at birth. In other words, the majority of children in the sample tracked within 1 SD of their birth LAZ, and when larger shifts were experienced, they tended toward the mean. The patterns in WAZ were similar, in which 67.3% remained within 1 SD of their birth WAZ, whereas 16.3% shifted downward and 16.4% shifted upward beyond 1 SD of their birth size. As observed for LAZ, the latter shifts in WAZ also tended overwhelmingly toward the mean.

Table 1 presents occurrences (from birth to age 12 mo) of 2 consecutive 1-mo weight increments below 4 candidate thresholds in the WHO sample. The probability of a child experiencing 2 consecutive weight increments below P25 ranged between 4.0% and 9.1% in approximately inverse order with increasing age. Comparative probability ranges for the other thresholds were 1.1–3.1% for P15, 0.5–1.9% for P10, and 1.1–4.9% for P5 and P50. The 2 thresholds that resulted in average proportions close to the statistically acceptable false-positive error margin in a 1-tailed test were P15 (2.3%) and P5 and P50 (2.4%).

TABLE 2 Mean changes in weight-for-age and length-for-age *z* scores after 2 consecutive 1-mo weight increments below the 15th percentile (event) in the WHO growth velocity standards reference sample[1]

Age intervals, mo	*n*	Change in *z* score at time *t* postevent compared with pre-event weight-for-age				Change in *z* score at time *t* postevent compared with pre-event length-for-age			
		0 mo	1 mo	4 mo	6 mo	0 mo	1 mo	4 mo	6 mo
0–1,1–2	26	−1.60	−1.68	−1.38	−1.29	−0.55	−0.63	−0.69	−0.72
1–2, 2–3	25	−1.12	−1.21	−1.32	−1.30	−0.23	−0.38	−0.71	−0.77
2–3, 3–4	24	−0.81	−0.79	−0.77	−0.71	−0.13	−0.25	−0.38	−0.47
3–4, 4–5	22	−0.74	−0.77	−0.82	−0.68	−0.15	−0.29	−0.34	−0.18
4–5, 5–6	26	−0.67	−0.68	−0.58	−0.64	−0.25	−0.33	−0.39	−0.42
5–6, 6–7	19	−0.66	−0.61	−0.57		−0.28	−0.30	−0.50	
6–7, 7–8	21	−0.62	−0.51	−0.50		−0.20	−0.22	−0.26	
7–8, 8–9	17	−0.59	−0.53			−0.18	−0.15		
8–9, 9–10	12	−0.64	−0.51			−0.21	−0.27		
9–10, 10–11	9	−0.61	−0.50			−0.20	−0.32		
10–11, 11–12	13	−0.63				−0.07			

[1] Data are derived from reference 15. The baseline (pre-event) weight-for-age *z* score and length-for-age *z* score for calculating this change is the child's status at the earliest age of the interval in column 1. Time *t* postevent is counted from the latest age value in column 1.

Clinically, successive increments at P15 may suggest moderate but protracted faltering in weight gain, whereas P5 and P50 may suggest a more severe initial faltering with incomplete catch-up, and both could predict stunting. The P15 threshold was selected for further study on the basis that, for clinical application, reference to the same percentile would be simpler than the combination of 2 (P5 and P50).

Focusing, therefore, on consecutive increments below P15 for further analysis, **Table 2** summarizes the frequency of these events experienced during infancy (by 1.1–3.1% of the WHO sample) and associated changes in WAZ and LAZ assessed at 0, 1, 4, and 6 mo thereafter. Events occurring in the first 3 mo were associated with larger overall downward shifts in WAZ and LAZ than were events that happened in later infancy. Compared

with WAZ changes, which ranged between −1.6 and −0.6 but then shifted upward thereafter, immediate changes in LAZ were smaller (−0.6 to −0.1), but they continued in slight downward trends thereafter.

For the Bangladeshi sample, stunting prevalence and mean LAZ and WAZ patterns are plotted in **Figure 1**A with the use of all available measurements at each observation age. Stunting rates in the sample increased from 24% at age 1 mo to 51% at 24 mo. Both mean LAZ and the prevalence of stunting remained relatively stable until age 7 mo. Thereafter, the mean LAZ declined steadily to −2.0 SDs by age 24 mo. Mean WAZ originated and tracked at approximately −1.1 and −1.2 SDs in the first months, slightly peaked from age 4 to 5 mo and then decreased, less precipitously than LAZ, to reach −1.7 SDs at 24 mo.
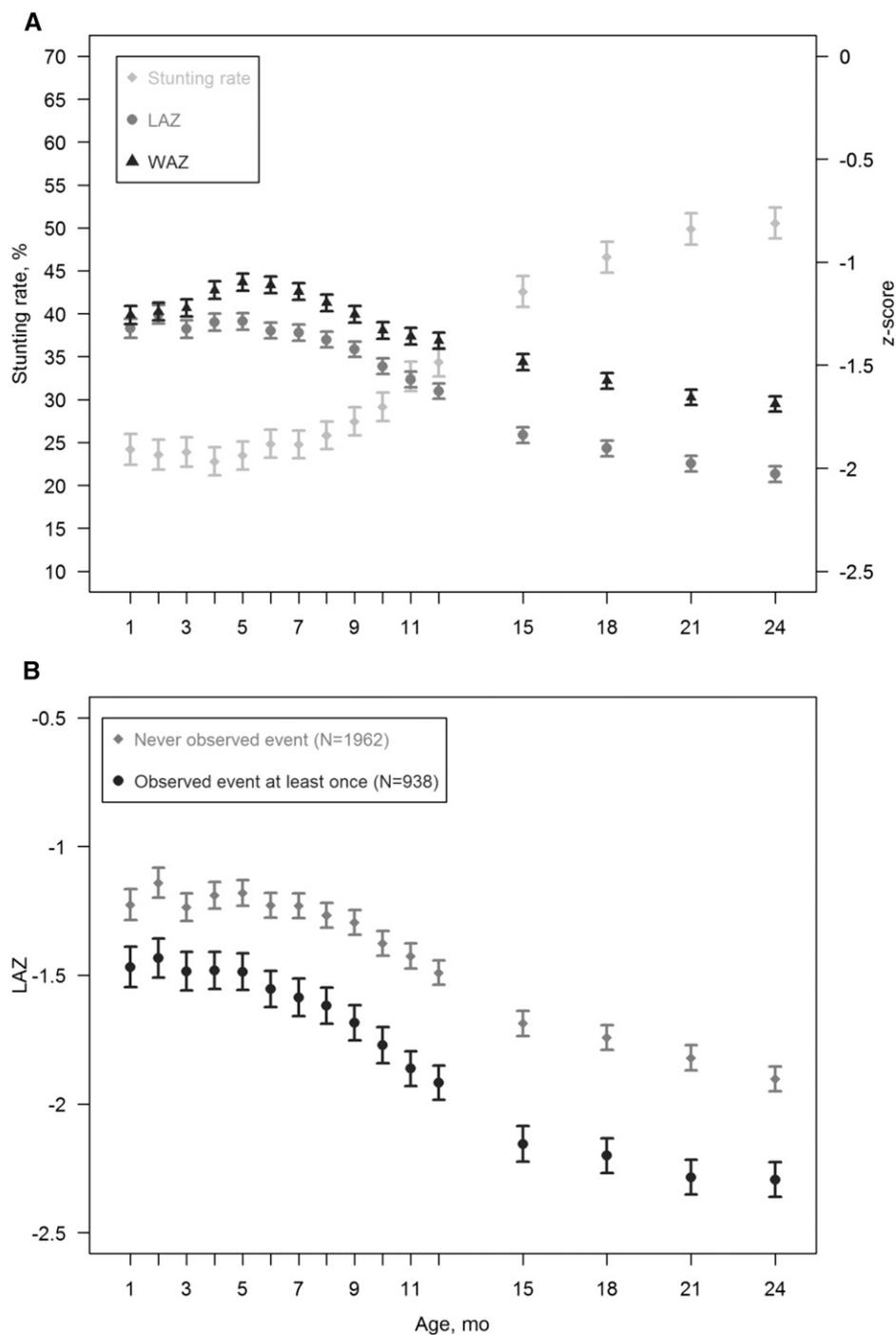

FIGURE 1 Prevalence of stunting and mean LAZ and WAZ (95% CIs) from birth to age 2 y (A) and mean LAZ (95% CIs) trajectory in groups classified according to the occurrence of 2 consecutive 1-mo weight increments below the 15th percentile of the WHO velocity standards (15) (event) (B) in the Bangladeshi sample. LAZ, length-for-age z score; WAZ, weight-for-age z score.

Most children in the Bangladeshi sample (94.6%) experienced 1-mo weight increments below P15 at least once. That is, of the 2900 children included in these analyses, 556 (19.2%) had 1 increment below P15 and 2186 (75.4%) experienced 2–7 increments below that threshold.

With regard to the event of interest (1-mo weight increments below P15 in 2 consecutive age intervals), 938 children (32.3%) were affected. The analysis of postevent changes in WAZ and LAZ equivalent to those reported for the WHO sample in Table 2 was hampered by sample size fluctuations due to missing observations in the MINIMat sample, but it was possible to track comparative growth trajectories as plotted for LAZ until the end of follow-up (Figure 1B).

Mean LAZ in the no-event group was stable at approximately −1.2 in the first 7 mo and then declined thereafter to −1.9 by age 24 mo (average loss of 0.7 SDs). The percentage of children who were stunted increased from 21% at 1 mo to 46% at 24 mo. For the group who experienced the event, LAZ originated at approximately −1.4 and then declined to −2.3 by age 24 mo (average loss of 0.9 SDs). The percentage of stunted children increased from 30% at 1 mo to 60% at 24 mo.

The probability of "growth faltering" in the MINIMat sample, defined as a decrease in LAZ larger than the first quartile of change in LAZ between ages 5 and 12 mo (−0.78), increases with higher initial WAZ (just before the event occurs), in line with the phenomenon of regression to the mean (**Figure 2**). However, the risk of growth faltering in children who experienced the event between 6 and 9 mo (as an example) was persistently higher (approximately double) than those who did not observe the event, regardless of their initial WAZ. Logistic regression shows an overall OR of 2.3 (95% CI: 1.9, 2.9).

The proportion of stunting in the total population that was attributable to having experienced 2 consecutive below-threshold weight increments during infancy was 14% at 12 mo and 9% at 24 mo. That is, at 12 and 24 mo, 14% and 9% of the stunted children, respectively, were classifiable as such on the basis of the event of 2 consecutive below-threshold weight increments. The percentage of stunting seen in the subpopulation affected by the event of 2 consecutive below-threshold weight increments that was attributable to the event was 34% at 12 mo

and 24% at 24 mo. That is, at 12 and 24 mo, 34% and 24% of the stunted children in the group with the event, respectively, were stunted associated with the event.

Discussion

Increasing commitments to the reduction in stunting and the narrow window available for its prevention in early human development heighten the need for timely identification of children at risk. The analyses in this article evaluate how weight velocity could serve such a purpose in the face of persistent challenges to obtaining frequent and sufficiently precise length measurements. They also provide a foundation for further investigation with the use of context-specific clinical and programmatic data to help establish evidence-informed applications of velocity standards, a need recognized at the time the WHO growth velocity standards were published (15). Once an effective screening strategy is established, the challenge still remains to identify effective interventions to prevent or minimize stunting, an issue not addressed by the present analysis.

The overall attained weight-for-age and length-for-age patterns in the healthy WHO sample showed that the majority of children remained within 1 SD of their size at birth through infancy, whereas the minority who experienced larger changes grew toward the mean. Compared with that standard distribution, children in the Bangladeshi sample were born with a mean WAZ and LAZ already below −1 SD, which then trended downward to −1.7 and −2.0 SDs, respectively, by age 2 y.

The patterns of successive increments observed in the WHO sample led to the selection of 2 consecutive 1-mo weight increments below P15 as a reasonable probabilistic criterion for screening for subsequent linear growth faltering. We observed in the Bangladeshi sample that consecutive weight increments below this threshold identified children with lower LAZ and a higher risk of stunting. In practical terms, this implies that monthly weight monitoring can help select infants who gain 2 consecutive increments below P15 at specified age intervals and that intervention with the goal of restoring them to their original weight-for-age could partially mitigate subsequent trends toward stunting. This serves the interest of timely initiation of interventions: that is, on first observing an increment below the threshold, the infant should be checked for reassessment in a month and if the velocity in the second month is also below the threshold intervention should be initiated. There is an important caveat to this goal. The feeding options available to the infant must be of sufficient quality to support linear growth, and other environmental shortfalls must be addressed (e.g., increased risk of infection). This will avoid focusing solely on adding weight to a progressively stunted frame.

In the WHO sample, the largest decreases in WAZ and LAZ after consecutive increments below P15 were observed in the first 2 mo. This is the period when regression to the mean was manifest (15). The larger decreases followed by recovery in WAZ (compared with LAZ) are consistent with fluctuations in weight associated with, for example, bouts of infection and recovery therefrom. Short-term insults have a lesser impact on length, hence the observed smaller yet more lasting decreases in LAZ, attesting to the lack of elasticity in length.

A comparable analysis in the Bangladeshi sample (data not shown) did not replicate a pattern suggestive of regression to the mean. Rather, mean WAZ and LAZ in the overall sample tracked downward. With regard to the event of interest in this analysis, it should be remembered that 75% of the Bangladeshi children had 2–7 intervals of monthly weight increments
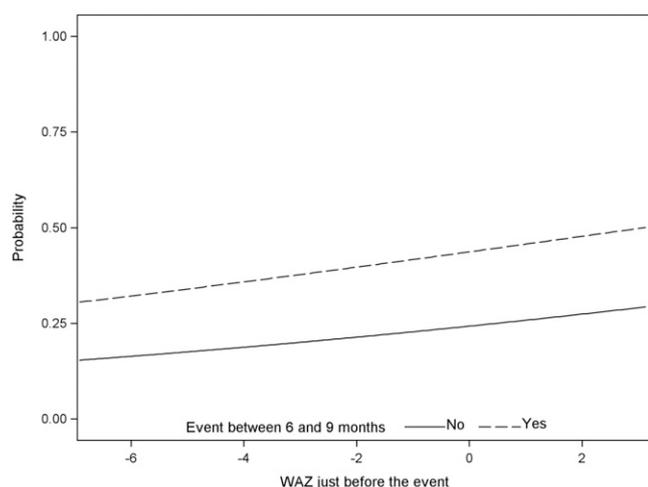

FIGURE 2 Predicted probabilities for growth faltering from age 5 to 12 mo by the occurrence of 2 consecutive 1-mo weight increments below the 15th percentile of the WHO velocity standards (15) (event), adjusted by initial WAZ, in the Bangladeshi sample. Growth faltering is defined as a decrease exceeding −0.78 length-for-age z scores between ages 5 and 12 mo. WAZ, weight-for-age z score.

below P15, but only one-third of the sample had 2 consecutive increments below the threshold. This may partly explain why two-thirds of the sample who did not experience the event also had a downward trend in LAZ. On the observation that, by age 1 mo, the event group already had an LAZ 0.2 SDs below that of the nonevent group of children, and in the absence of additional descriptive data, one can only speculate about the factors underlying this apparent preselection. Because stunting has an intergenerational component, it may be that mothers in the event group were shorter on average, had poorer nutritional status in pregnancy, and that growth constraints were more prevalent in their households. In the final analysis, however, logistic regression results showed a significant association between low monthly weight increments (or losses) and subsequent decreases in length-for-age until age 2 y, pointing to a window during which timely detection and intervention could minimize long-term deficits in linear growth. With little existing evidence for the treatability of stunting, the achievement of the WHO global targets depends on prevention, and this pattern also sets time-bound recovery targets to guide clinical monitoring once at-risk children are enrolled in intervention programs.

A possible tool for field use from these analyses could be a simple 3-column table (age interval and boys' and girls' increments at P15) for each monthly interval between birth and age 12 mo. An infant who experienced a weight increment below the threshold would be booked for review in a month and then enrolled into a stunting prevention program if a second below-threshold increment was observed. Tables of a similar kind were used in community programs in Central America [AIN (Atención Integral al Niño)] in which targets for minimum weight gain conditional on starting weight were set for periods of 30 or 60 d (27, 28). The proposed table would address the limitations of the AIN approach by providing sex-specific increments conditional on age, respecting both the sex differences in growth and the dramatic changes that characterize velocity in the 12 mo of infancy.

Attributable risk calculations quantified the potential yield (i.e., predictive value) from screening for 2 consecutive below-threshold weight increments in the first 12 mo. Under the assumption that the prevention of poor weight gain would prevent stunting, the proportion of stunting that could be averted in the group who experienced the event is estimated at up to 34% at 12 mo and 24% at 24 mo.

This analysis used data from a population with a high prevalence of stunting (~50% at 24 mo). It is possible that the percentage identified would be different in other contexts. Furthermore, the MINIMat data were collected in a research study conducted by an experienced and well-trained team; the yield from screening could be lower in programmatic settings if weight measurements are less reliable and accurate.

The results reported here identify new avenues for further investigation of growth velocity patterns in different populations and their relation to attained growth indicators and profiles. Such investigation should seek to understand what influences recovery after low increments and associated growth in the long term for different indicators of attained growth. Given the need to establish benchmarks for using the WHO velocity standards, our analyses focused on weight increments conditional on sex and age. Future analyses might condition recovery on weight-for-length, building on earlier research on the association between wasting, recovery, and subsequent stunting risk (7–9), as well as addressing other questions on the relation between weight and linear growth (29).

As the world moves on to the post–Millennium Development Goals era, global consensus has emerged on the importance of nutrition for the attainment of sustainable development. At the time of this writing, the UN High Level Panel on the post-2015 development agenda has proposed a goal on food security and adequate nutrition that includes a target on stunting. Similarly, the UN Secretary-General's Zero Hunger Challenge in 2012 included the goal of zero stunting for children below age 2. In line with that high-level political commitment to improve nutrition, the challenge for frontline workers is to identify individual children at risk of stunting and initiate timely measures to prevent it. This article provides a practical proposal for how they could begin to address the challenge with the use of resources and means already within their reach.

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