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Early Childhood Stunting Is Associated with Lower Developmental Levels in the Subsequent Generation of Children[1,2]

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Abstract

Background: Linear growth retardation (stunting) is associated with lower adult cognition, educational attainment, and income. These effects, together with possible effects of stunting on birth weight and subsequent growth of offspring, suggest that stunting could be associated with poor development in the next generation of children.

Objective: The objective was to compare developmental levels in children born to parents who were stunted or nonstunted in early childhood.

Methods: This is a prospective cohort study of the children of participants in the Jamaica supplementation and stimulation study. The analysis compared children born to a parent who was stunted at age 9–24 mo, and did not receive the stimulation intervention, with children born to a parent in the nonstunted group. Developmental levels were measured with the Griffiths mental development scales between ages 12 and 72 mo. Mixed model regression analyses were conducted to allow for clustering of children within families and child (repeat assessments). The analyses included 89 children with a total of 156 assessments. Caregiver and home characteristics associated with the developmental quotient (DQ) or any of the subscales were included in the regressions.

Results: Children born to a stunted parent had lower DQs (−5.29 points; 95% CI: −9.06, −1.52 points; $P = 0.01$) and lower scores on the cognitive subscale (−5.77 points; 95% CI: −10.68, −0.87 points; $P = 0.022$). The offspring of stunted parents had lower height-for-age (−0.61 *z* scores; 95% CI: −1.13, −0.10 *z* scores; $P = 0.021$). In analyses, adjusting for child height-for-age or birth weight, the developmental differences remained significant.

Conclusions: To our knowledge, this is the first report comparing the development of offspring of persons stunted in early childhood to the development of offspring of nonstunted parents. The findings suggest that the impact of stunting on development continues in the next generation of children. If replicated, these findings have important implications for estimation of the cost of stunting to social and economic development. *J Nutr* 2015;145:823–8.

Keywords: stunted children, child development, intergenerational, linear growth, Jamaica

Introduction

Linear growth retardation (stunting) affects ~165 million children <5 y of age in low- and middle-income countries (1). Several longitudinal studies have shown that stunted children have poor cognitive ability and educational achievement through later childhood and adolescence (2). Stunting has also been shown to have long-term detrimental effects on adult cognitive ability, attained schooling, and income (3–5). Parental education and income would be expected to influence parenting (6, 7) and available resources. Combined with possible effects of stunting on birth weight and growth of offspring, these factors could contribute to intergenerational transmission of poor development.

There is little direct information on whether the poor development of stunted children is transferred to the next generation. There is some evidence indicating that nutrition during early childhood influences growth of the next generation. Pooled analysis of 4 longitudinal cohorts showed that mothers' height-for-age at age 24 mo is significantly associated with birth weight of their offspring (4). In a nutritional supplementation study conducted in Guatemala, children born to mothers who had received high protein energy supplementation in early

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childhood were taller at age 30 mo than children whose mothers had received low energy supplementation (8). This suggests that nutrition and growth in early childhood may influence growth of the subsequent generation.

The Jamaica supplementation and stimulation study included children who were stunted in early childhood and a comparison group of nonstunted children from the same neighborhoods. The stunted children participated in a randomized controlled trial of psychosocial stimulation, nutritional supplementation, or both treatments (9). There were no sustained benefits from supplementation, but children who received the stimulation program had higher intelligence levels, educational achievement, and income at age 22 y than stunted participants who did not receive the program (5, 10). The nonstunted children had higher developmental levels and intelligence levels than the stunted control and supplemented children throughout the study. We have established a cohort of the offspring of the first generation study participants. This paper examines the hypothesis that there are intergenerational effects of being stunted by the age of 2 y on growth and development of the next generation. The cohort is presently too small to examine intervention effects on development, which were hypothesized to be smaller than associations with stunting based on the size of differences in development and IQ in the first generation participants. We compare children born to the stunted participants who did not receive stimulation (participants in the control group and the supplementation-only group) with children born to the nonstunted participants. Children born to participants in the stunted groups who received stimulation are omitted because the impact of stunting was modified by intervention in the parent cohort, and the objective of this paper is to determine any associations with stunting.

Methods

Description of original study (first generation)

Details of the original study sample have been described previously (9, 11) and are summarized here. In 1986 we began a longitudinal study in Jamaica of 127 stunted (height-for-age < −2 SD scores) children aged 9–24 mo. The children were randomly assigned to 4 groups for 2 y: a control group who received free medical care only, and groups receiving nutritional supplementation, psychosocial stimulation, or both treatments. A group of 32 nonstunted children (height-for-age > −1 SD scores) from the same neighborhoods and of similar age and socioeconomic background was also studied. On enrollment (mean age: 18.7 ± 4.1 mo SD), the stunted children had lower developmental levels than the nonstunted group, and the gap widened over the 2 y of the trial for the children in the control group. Supplementation and stimulation each benefited the children's development and the effects were additive (9). At age 7 y when the first follow-up of the cohort was conducted, an additional 52 nonstunted children, who had been identified in the initial survey and had met eligibility criteria, were enrolled. Benefits from supplementation for development were no longer evident in later childhood (12), however, benefits from stimulation have continued up to the last assessment at age 22 y (5). Because stimulation modified the impact of stunting, we have not included children of first generation participants who received stimulation (stimulation and both groups) in this report.

Current sample (second generation)

The study is a prospective cohort study of children born to the original study participants (first generation). The second generation sample comprises children aged between 12 and 72 mo, whose mothers or fathers were participants in the first generation study. Twins and children with mental or physical disability are excluded (to date 5 children). The first generation participants were last assessed in 2008 when it was recorded whether they had any children. In 2009 all participants known to have children were visited and invited to participate. The remaining participants were contacted to determine if they had a child since the last visit or were currently pregnant. All cohort participants are subsequently recontacted annually.

Ethical approval for the study was obtained from the University of the West Indies Ethics Committee. Written informed consent is obtained from the first generation participant and, where the participant is the child's father, consent is also obtained from the mother of the child.

All children aged between 12 and 72 mo were assessed in the initial enrollment phase in 2009. Children between 30 and 60 mo are remeasured at age 72 mo, and children <30 mo are remeasured at age 36 mo. All children <12 mo during the initial enrollment phase, or born after this, are first tested at age 12 mo. We plan to measure these children at 36 and 72 mo.

Measurements

Mothers were interviewed in their homes to obtain information on social background, parent characteristics, and child behavior, and observations were conducted to assess the quality of the home environment. Developmental testing and a questionnaire concerning maternal depression were conducted at our research unit. All tests or questionnaires used had been piloted and used previously in Jamaica. Developmental testing has been conducted by a single tester unaware of the first generation group assignment. Interviews and observations were conducted by a single interviewer from 2009 until 2010 and by another interviewer beginning in 2010.

Children's psychomotor development. The children's development was assessed with the Griffiths Scales of Mental Development (13, 14). These comprised 4 subscales: cognition, hearing and speech, hand and eye, and locomotor, as well as an overall developmental quotient (DQ). These scales have been used previously in several studies in Jamaica, correlate with later tests of intelligence and academic achievement (15), and were used in the original study to measure the development of the first generation participants.

Children's behavior. Behavior was assessed by parental report with use of the Strengths and Difficulties Questionnaire (16) in children ≥3 y of age. There are 5 subscales: emotional difficulties, peer problems, hyperactivity, conduct problems, and prosocial behavior. The first 4 subscales are summed to give a total difficulties scores and the prosocial subscale is reported separately.

Socioeconomic status. An index of socioeconomic status was derived by factor analysis of household crowding (persons/room), toilet facilities, water supply, and number of possessions from a list of 15 items.

Parental characteristics. The mother, or primary caregiver if the child was not living with the mother, was interviewed with use of a questionnaire at each assessment. Information was collected on occupation and education of the mother or the child's primary caregiver. The person interviewed was the first generation participant for 63.4% of interviews in the stunted group and 55.3% for the nonstunted group. Mothers' or caregivers' vocabulary was assessed with the Peabody Picture Vocabulary Test (17) and depressive symptoms with the Center for Epidemiologic Studies Depression Scale (18). Mothers' heights were measured at the first visit.

Quality of the home environment. The Home Observation for the Measurement of the Environment (HOME) was used to assess the amount and quality of stimulation provided in the home (19). Because of the age range of children in the study, 3 versions of the HOME were used: the infant version for children 12–35 mo, the early childhood version for children 36–71 mo, and the middle childhood version at age 72 mo. The items, subscales, and maximum total score vary among the 3 versions. We therefore converted the total raw scores to standard deviation scores for each version and combined these to give a comparable HOME score for children of different ages.

Children's anthropometry. Birth weight was obtained by maternal report or child birth records when available (30% of children). Height,

weight, and head circumference were measured at each visit with use of standard procedures. SD scores for height-for-age, weight-for-age, and BMI-for-age were calculated with use of the WHO growth standards (WHO Anthro Plus v 1.04).

Intraclass correlation coefficients for reliability between interviewer and trainer in 19 subjects was 0.99 for all questionnaires and the HOME, 1.00 for weight (a digital scale was used), 0.97 for height/length, and 0.96 for head circumference.

Statistical analysis

Data collection began in 2009 and these analyses include data collected up to August 31, 2013. Descriptive data were compared between the stunted and nonstunted groups with use of *t* tests and chi-square tests as appropriate. Associations of child and family characteristics with child outcomes were examined with use of *t* tests and parametric or nonparametric correlations.

To allow for clustering of children within families and child (repeat assessments), we analyze the outcome variables (anthropometry and development) with use of 3-level mixed model regression analyses with levels corresponding to family, child, and assessment. We estimate random effects at each level and fixed effects for variables such as maternal education (family level), child sex (child level), and child age (assessment level). We used this model to estimate the power of our study to detect differences in the primary outcome of the DQ. Our power calculation was based on estimates that by the end of 2013 we would have a total of 230 assessments on 122 children from 85 parents. From our previous work we expect the primary outcome variable, DQ, to have a mean of 100 and SD of 11 units. Preliminary assessments also suggest that the SEs of the random effects for family-to-child-to-visit will be approximately in the ratio of 1:1:2. Using these assumptions and a test at the 5% level of statistical significance, our study has 80% power to detect a difference of 6 units between the stunted nonstimulated group and the nonstunted group. At the end of August 2013 the numbers were close to those used in the power calculation—a total of 125 enrolled children from 82 parents and a total of 217 assessments.

Analyses were conducted with the Statistical Package for the Social Sciences (SPSS) version 17. Statistical significance was defined as $P < 0.05$. Values in the text are mean differences between the groups and 95% CIs.

Results

Of the 148 first generation participants residing in Jamaica at the last follow-up at age 22 y, 82 (55.4%) have ≥1 children (maximum 4) in the sample. There are no significant differences in intelligence, educational attainment, and socioeconomic status at age 22 y between participants with children in the new cohort and those without. The current age of the parent cohort ranges from 27 to 29 y.

This report includes 89 of the children enrolled, 41 children born to first generation participants from the stunted, no stimulation group, and 48 born to nonstunted participants. There was no statistically significant difference between the stunted and nonstunted groups in the proportion of children with a first generation mother (as opposed to those with a first generation father), although the proportion with mothers tended to be higher in the stunted group (29 of 41 children, 70.7%) compared to the nonstunted group (28 of 48 children, 58.3%).

The 89 children in the analyses have had a total of 156 assessments (57% 1 assessment, 37.2% 2 assessments, and 5.8% 3 assessments, with no significant difference between groups). Few children missed planned assessments, mothers of 3 children chose not to participate further, and 1 child migrated after the first assessment. Characteristics of the children, their mothers (or primary caregivers), and homes are shown in **Table 1**. There were no significant differences by group in percentage male or female. The age distribution at each test point was also similar by group. Birth weight was not significantly different between the groups. Mean height-for-age ($P = 0.011$) was significantly lower in the children of stunted first generation participants and BMI-for-age tended to be lower ($P = 0.06$). Few second generation children in either group were stunted (height-for-age < −2 SD), 4 in the group with a stunted parent, and 3 in the nonstunted group. There was no significant difference in head circumference between the groups.

Maternal height was significantly greater in the nonstunted group ($P < 0.001$). There were no significant differences between the groups in maternal education, occupation, vocabulary (Peabody Picture Vocabulary Test score), or depressive symptoms. Similar proportions of children had their fathers present in the home, and there were no significant differences in socioeconomic status or the quality of the home environment (HOME score).

The mixed model regression analyses for nutritional status are shown in **Table 2**. Height-for-age of children born to stunted first generation participants was −0.61 SD scores lower (95% CI: −1.13, −0.10 SD scores; $P = 0.021$) than children of participants who were nonstunted in childhood. An additional model controlled for birth weight. Birth weight did not predict child height-for-age and the difference between the groups remained significant (−0.59 SD scores; 95% CI: −1.11, −0.08 SD scores).

TABLE 1 Child, mother (or primary caregiver), and home characteristics of the cohort by stunted and nonstunted first generation group[1]

	Stunted	Nonstunted
Children, *n*	41	48
Girls	24 (58.5)	28 (58.3)
Boys	17 (41.5)	20 (41.7)
Birth weight,[2] kg	2.95 ± 0.80	3.11 ± 0.58
Mothers' height,[2] cm	161.7 ± 5.2*	168.8 ± 6.1
Assessments, *n*	71	85
Child		
Age distribution at assessment		
12–35 mo	25 (35.2)	34 (40.0)
36–59 mo	23 (32.4)	28 (32.9)
≥60 mo	23 (32.4)	23 (27.1)
Height-for-age SD score	−0.22 ± 1.28*†	0.27 ± 1.13
BMI-for-age SD score	−0.09 ± 1.02*†	0.26 ± 1.25
Head circumference, cm	49.4 ± 2.7	49.2 ± 2.4
Mother and home		
Mothers' education, completed secondary school	36 (50.7)	51 (60.0)
Mothers' occupation		
None/unskilled	11 (15.5)	7 (8.2)
Semiskilled	43 (60.6)	56 (65.9)
Skilled or higher	17 (23.9)	22 (25.9)
Father lives with child	23 (32.4)	30 (35.3)
Mothers' PPVT score	101 ± 27	104 ± 25
Mothers' depression score[3]	23.6 ± 13.0	19.8 ± 11.9
SES factor score	−0.10 ± 0.98	0.06 ± 1.04
HOME SD score	−0.03 ± 0.92	−0.12 ± 1.11

[1] Values are means ± SDs for continuous variables or *n* (%) for categorical variables. *t test, child's height-for-age, $P = 0.011$; BMI-for-age, $P = 0.06$; mothers' height, $P < 0.001$. No other significant differences. †$n = 70$. HOME, Home Observation for the Measurement of the Environment; PPVT, Peabody Picture Vocabulary Test; SES, socioeconomic status.

[2] Birth weight stunted ($n = 40$) and nonstunted ($n = 47$), mothers' height nonstunted ($n = 46$).

[3] Collection of data on mothers' depressive symptoms began in 2010; stunted ($n = 47$), nonstunted ($n = 62$).

TABLE 2 Mixed model regression analyses of second generation nutritional status comparing children of participants in first generation stunted and nonstunted groups[1]

Outcomes	β	95% CI	*P*
Height-for-age SD score	−0.61	−1.09, −0.13	0.013
BMI-for-age SD score	−0.21	−0.75, 0.33	0.44
Head circumference, cm	−0.03	−0.76, 0.70	0.94

[1] Values are regression coefficients and 95% CIs. Mixed model linear regression analyses adjusted for child age, gender, socioeconomic status factor score, and dummy variable for first generation group (stunted 1, nonstunted 0).

There were no differences in BMI-for-age or head circumference between the stunted and nonstunted groups.

The children's mean developmental and behavior scores are shown in **Table 3**. There were no significant differences between the groups in total behavior difficulties or in prosocial scores. The unadjusted and adjusted mixed model regression analyses for DQ and the development subscales are shown in **Table 4**. All analyses were adjusted for child's age (in months) and gender. Variables that were significantly associated with DQ or at least one of the subscales in univariate analyses were included as covariates in adjusted model 1. These variables were caregivers' education and occupation, father lives with child (yes/no), socioeconomic status factor score, and HOME score.

In the adjusted model, the overall DQ of children with a parent in the stunted group was 5.29 points lower than those with a nonstunted parent (95% CI: −9.06, −1.53 points; $P = 0.008$). Child age was negatively associated with DQ, and DQ was lower in children whose fathers lived in the home (−4.24 points; 95% CI: −7.80, −0.68 points; $P = 0.020$). The other covariates were not significant predictors of DQ. The association with age was also examined comparing developmental levels of children by the age groups shown in Table 1. Compared to the youngest group, the DQ of children aged 36–59 mo was 6.81 points lower and the DQ of children aged ≥60 mo was 5.47 points lower, showing that the decline in development occurred before age 5 y.

Scores on the cognitive subscale were also lower in the stunted group (−5.77 points; 95% CI: −10.68, −0.87 points; $P = 0.022$), and girls had higher cognitive scores than boys (5.00 points; 95% CI: 0.03, 9.98 points; $P = 0.049$). None of the other covariates was significantly associated with the cognitive subscale. Differences for the hand-and-eye and locomotor subscales approached significance. There was a modest association between the IQ of the first generation participants at age 22 y and the DQ of their children (coefficient: 0.207; 95% CI: −0.006, 0.419; $P = 0.056$). When first generation IQ was included in the model the results changed little.

In a further model, we adjusted for child height-for-age to determine whether differences in growth mediated some of the difference seen in developmental levels (adjusted model 2, Table 4). There was little change in the size of the regression coefficients. In these analyses, DQ and the cognitive and locomotor subscales were significantly lower in the stunted group and differences in the hand-and-eye and hearing-and-speech subscales approached significance ($P < 0.1$). Height-for-age was not significantly associated with development in any of the regressions. Further analyses were conducted to determine if birth weight mediated the differences in development; the regression coefficients were reduced (by 12.0% for DQ and 9.6% for the cognitive subscale), but groups remained significantly different in overall DQ (−4.78; 95% CI: −8.61, −0.94; $P = 0.015$) and the cognitive subscale (−5.08; 95% CI: −10.05, −0.11; $P = 0.045$).

When the sample was restricted to children with a first generation mother (n = 100 assessments), the differences between the groups tended to be slightly larger and were significant for DQ and all subscales except hand-and-eye coordination (data not shown). Because of the smaller number of children with first generation fathers, we have not performed comparable analyses for fathers only or explored interactions with parent gender.

Discussion

Children born to a parent who was stunted by age 24 mo had significantly lower developmental quotients than children born to a nonstunted parent, with an effect size of 0.46 SD. The difference in DQ between the stunted and nonstunted first generation participants when they were enrolled at 9–24 mo of age was 0.8 SD (9). After an initial increase in the next 2 y, the gap between first generation stunted children who did not receive stimulation and the nonstunted comparison group stabilized at 0.8–0.9 SD from later childhood to adulthood (20). To our knowledge, this is the first report comparing the development of offspring of persons stunted in early childhood to the development of offspring of nonstunted parents. Significant differences were also seen for the cognitive and motor subscales. The cohort provides a unique opportunity to begin to explore possible associations of stunting with the development of the next generation of children. The results should be interpreted with caution given the small sample size available.

Differences in development between children of stunted and nonstunted parents could be due to differential adult success in areas such as education and income (3). However, the deficit in developmental level remained after adjustment for education

TABLE 3 Mean assessment scores for second generation child development and behavior scores by stunted and nonstunted first generation group[1]

Measurement	Stunted (assessments, n = 71)	Nonstunted (assessments, n = 85)
Developmental quotient	98.9 ± 12.0	104.7 ± 10.8
Cognitive subscale	91.8 ± 15.3	97.7 ± 14.6
Hearing-and-speech subscale	101.4 ± 18.3	108.4 ± 17.0
Hand-and-eye subscale	95.8 ± 14.6	100.3 ± 13.1
Locomotor subscale	106.6 ± 14.4	112.6 ± 12.9
SDQ total difficulties score[2]	14.2 ± 5.0	13.3 ± 5.5
SDQ prosocial score[2]	7.1 ± 2.0	7.2 ± 2.0

[1] Values are means ± SDs. SDQ, Strengths and Difficulties Questionnaire.
[2] SDQ was administered only for children ≥3 y of age; stunted (n = 46), nonstunted (n = 50).

TABLE 4 Mixed model regression analyses of first generation group on child development[1]

Outcomes	Unadjusted[2]			Adjusted model 1[3]			Adjusted model 2[4]			Effect size (model 1)
	β	95% CI	*P*	β	95% CI	*P*	β	95% CI	*P*	
Developmental quotient	−5.64	(−9.75, −1.53)	0.008	−5.29	(−9.06, −1.52)	0.007	−5.91	(−9.83, −2.00)	0.004	0.46
Cognitive subscale	−6.36	(−11.47, −1.26)	0.015	−5.77	(−10.68, −0.87)	0.022	−5.89	(−11.00, −0.77)	0.025	0.38
Hearing-and-speech subscale	−6.01	(−12.96, 0.94)	0.09	−5.36	(−12.03, 1.30)	0.11	−6.54	(−13.24, 0.16)	0.06	0.30
Hand-and-eye subscale	−4.10	(−8.60, 0.41)	0.07	−3.80	(−7.85, 0.25)	0.07	−3.67	(−7.89, 0.56)	0.09	0.27
Locomotor subscale	−5.03	(−10.08, 0.01)	0.05	−4.96	(−10.04, 0.12)	0.06	−5.79	(−11.06, −0.52)	0.032	0.36

[1] Values are regression coefficients and 95% CIs. Child development outcome variables include the Griffiths Scales of Mental Development subscales (cognitive, hearing and speech, hand and eye, and locomotor) and overall developmental quotient. HOME, Home Observation for the Measurement of the Environment.
[2] Mixed model linear regression analyses with child age, sex, and dummy variable for first generation group (stunted 1, nonstunted 0).
[3] Mixed model linear regression analyses adjusted for child age, sex, dummy variable for group, mothers' education, occupation, father lives with child (yes/no), socioeconomic status factor score, and HOME score.
[4] Mixed model linear regression analyses adjusted for child age, sex, dummy variable for group, mothers' education, occupation, father lives with child (yes/no), socioeconomic status factor score, HOME score, and child height-for-age *z* score.

and occupation of the primary caregiver, socioeconomic status, and the quality of the home environment. Nonetheless, some of the difference may be related to unmeasured aspects of the caregiver and home, including aspects of caregiver interaction not captured by the HOME inventory. There were few significant differences in family characteristics between the groups, although for most measures the nonstunted group tended to have more favorable scores. It therefore appears that the association with stunting may not be explained by differences in the caregivers or home environment of second generation children.

The height-for-age of the children born to a parent who was stunted was significantly lower than that of children with a parent in the nonstunted group, indicating that childhood stunting may have an impact on growth of the next generation. Association of childhood nutrition with growth of the subsequent generation has also been reported in follow-up of children born to mothers in the Guatemalan supplementation study (8, 21). In the current study, although height-for-age of children born to a parent in the stunted group was lower, the mean height-for-age of both groups was within the normal range and few children in either group were stunted.

The lower developmental levels in the stunted group have thus occurred even though the growth of the offspring is generally within the normal range, albeit lower than that of the nonstunted group. Adjustment for current height-for-age did not explain the difference in developmental levels indicating that the differences were not mediated by differences in growth.

Mothers (first generation participants and nonparticipants) in the stunted group had lower adult heights compared with the nonstunted group. Maternal nutritional status has been linked to young child development, with benefits to development seen from supplementation in pregnancy with food (22) or multiple micronutrients (23), and it is possible that differences in maternal nutritional status during pregnancy in this cohort may have contributed to the differences in developmental levels. Inclusion of birth weight in the analyses of development led to a small decrease in the difference in development between the groups. However, overall development and cognitive scores remained significantly lower in the stunted group.

A limitation of the study is that we do not have information on the childhood nutritional status of the second generation child's parent who was not a participant in the first generation study. However, if that parent was of different nutritional status (i.e., nonstunted where the participant was stunted), this would most likely reduce any effects seen. Because the small sample size is another limitation, we plan to continue to enroll children into the cohort. The sample size also limits our ability to investigate moderating effects such as the gender of the first generation participant. It would also be valuable to have more detailed examination of caregiving practices than those possible with the HOME.

The mechanisms that underlie the differences in development between children with a parent who experienced childhood undernutrition compared with children born to an adequately nourished parent remain to be determined. Apart from residual confounding by unmeasured family characteristics, other possibilities include lower genetic potential in the stunted group and epigenetic effects. Animal research and observational human studies provide evidence for transgenerational epigenetic effects from early life experiences, including maternal care, stress, and nutrition, on brain function and behavior (24, 25).

Our findings suggest that the impact of stunting on development may go beyond the cohort directly affected in childhood and be seen in the next generation of children. If these results are replicated, there are important implications for estimation of the cost of stunting to social and economic development. The results reinforce the need for action to prevent childhood stunting, which continues to affect 28% of children globally (1) with much higher prevalence in the most affected countries.

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SPW, SMC, and SMG-M designed the study; SMC and AW conducted the study; SPW and CO conducted the data analysis; SPW wrote the paper; and SMC, CO, and SMG-M provided critical revisions. SPW had primary responsibility for final content. All authors read and approved the final manuscript.

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