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The effects of birth weight and postnatal linear growth retardation on body mass index, fatness and fat distribution in mid and late childhood

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Abstract

Objective: To determine the effects of birth weight and early childhood stunting on body mass index (BMI), body fat and fat distribution at ages 7 and 11 years, and the change from 7 to 11 years.
Design: Prospective cohort study
Setting: Kingston, Jamaica.
Subjects: One hundred and sixteen stunted children (height-for-age below two standard deviations (<−2SD) of the National Center for Health Statistics (NCHS) references) and 190 non-stunted children (height-for-age > −1SD), identified at age 9–24 months. The stunted group was divided into a previously stunted group (height-for-age at 11 years ≥ −1SD) and a chronically stunted group (height-for-age < −1SD).
Results: Birth weight was positively related to the children's BMI but not to measures of body fat. Birth weight was negatively associated with the subscapular/triceps skinfold (SSF/TSF) ratio at age 11 years, and to the change between 7 and 11 years. Controlling for birth weight, the chronically stunted group remained significantly smaller than the non-stunted children at both ages and increased less from 7 to 11 years in all measures except the SSF/TSF ratio, which was significantly greater at age 7 years. The previously stunted group had significantly lower BMI and percentage body fat at age 7 years than the non-stunted group. Change from 7 to 11 years was not significantly different from that of the non-stunted group except for a smaller increase in TSF. At age 11 years they had significantly lower TSF and percentage body fat.
Conclusions: Children stunted in early childhood had less fat and lower BMI than non-stunted children but had a more central fat distribution that was partially explained by their lower birth weights. The association between birth weight and central fat distribution developed between 7 and 11 years.

Keywords
Linear growth retardation
Birth weight
Body mass index
Fat distribution
Skinfolds

The prevalence of obesity among adults is increasing in many developing countries[1,2]. This trend is ascribed to the nutrition transition with increasing access to Westernised diets and less physical activity[2,3]. In many countries this increase in adult overweight coexists with significant levels of childhood undernutrition. It has been suggested that growth retardation *in utero*[4,5] and in early childhood[6,7] may increase the risk of obesity later in life. Thus present conditions in countries experiencing the nutrition transition may encourage a rapid increase in adult obesity and related chronic diseases.

In Jamaica, overweight (body mass index (BMI) $\geq 25\,kg\,m^{-2}$) is common among adults, particularly women, affecting 33% of men and 67% of women, and obesity is an important risk factor for cardiovascular disease in this population[8]. Although overweight in Jamaican adults is probably related to adult diet and lifestyle[9], childhood undernutrition may also contribute to the problem.

In 1986 we began a longitudinal study of children aged 9–24 months whose linear growth was retarded (stunted group; height-for-age below two standard deviations (<−2SD) of the National Center for Health Statistics (NCHS) references[10]) and a comparison group of children from the same poor communities in Kingston, Jamaica whose growth was normal (non-stunted group; height-for-age > −1SD). In the first two years of the study we evaluated the effects of supplementation and psychosocial stimulation on the stunted children's growth and mental development[11,12]. Since the end of the intervention the children have been re-evaluated at ages 7–8 years and 11–12 years. The objectives of this work were to

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determine whether birth weight and linear growth retardation in early childhood are related to BMI and body fatness at these ages and whether children who experienced catch-up growth differed in these measures from those who remained stunted.

Subjects and methods

Subjects

All children were identified at age 9–24 months by house-to-house survey of several poor Kingston neighbourhoods. At that time, 127 stunted children (height-for-age < -2SD of the NCHS references[10]) participated in a two-year randomised trial of nutritional supplementation and psychosocial stimulation[11,12]. A comparison group of 32 non-stunted children (height-for-age > -1SD), matched to every fourth stunted child for age and gender, was also studied. Four years later, when the children were aged 7–8 years, we located 122 of the stunted children and all of the non-stunted children for a follow-up study of their growth and cognitive development. To increase the number of non-stunted children we also studied an additional 175 children from the same neighbourhoods who had been identified during the original survey and were non-stunted (height-for-age > -1SD) in early childhood. At age 7–8 years, the height-for-age of the stunted children had increased from a mean score of -3.0SD on enrolment to a score of -1.1SD[13]. We located 116 of the stunted children (67 boys, 49 girls) and 190 non-stunted children (102 boys, 88 girls) at age 11–12 years. Loss to follow-up is due primarily to migration.

The stunted children were divided into those with height-for-age ≥ -1SD at age 11 years (previously stunted group, $n = 57$, 53% boys) and those with height-for-age < -1SD (chronically stunted group, $n = 59$, 63% boys). The mean ($\pm$SD) height-for-age score of the chronically stunted group was -1.67SD (± 0.48), that of the previously stunted group was -0.33SD (± 0.46), and for the non-stunted group the mean score was 0.52SD (± 0.81). Only 11.2% of the children who had been stunted in early childhood still had height-for-age < -2SD.

The study was approved by the Ethics Committee of the University of the West Indies, and the parents or guardians gave written informed consent for their children to participate.

Anthropometry

The children's height, weight, arm circumference, triceps skinfold (TSF) and subscapular skinfold (SSF) were measured using standard procedures[14]. Skinfolds were measured in triplicate and the average used. Inter-observer reliability was determined before beginning the study, and intra-class correlation coefficients exceeded 0.99 for all measures. Body mass index (weight/height^2) was calculated and percentage body fat was estimated from skinfolds with equations for black children, using those for prepubescent and pubescent children as appropriate[15]. The subscapular/triceps skinfold ratio (SSF/TSF) was calculated as an indicator of fat distribution. Birth weights were obtained from hospital records (73%) or maternal recall[16] and were available for all of the stunted children and 181 of the non-stunted children.

Pubertal status

The children's pubertal status was determined using the cut-offs recommended by World Health Organization (WHO)[17]: breast stage 2 for girls and genitalia stage 3 for boys. Children who have reached these stages have entered their pubertal growth spurt. The cut-offs were therefore used to classify children into prepubertal and pubertal groups.

Socio-economic status

Information on housing conditions (crowding, household possessions, sanitation and water supply) was collected at age 11 years by questionnaire administered to the children's caregiver in their home. Factor analysis produced a single factor that was used as an indicator of socio-economic status.

Statistical analyses

The distributions of all measurements were checked for normality and log transformations of BMI, triceps and subscapular skinfolds and percentage body fat were calculated for use in all analyses. There were no effects of the early childhood interventions on the children's size at age 7–8 years or 11–12 years[13,18]. The intervention group was therefore not included in the analyses. Separate multiple regression analyses were calculated with each anthropometric measurement as the dependent variable to determine the differences among the groups. The independent variables in all regression models were age, gender, dichotomous variables for the two stunted groups (chronically stunted and previously stunted) with the non-stunted group as the reference, and pubertal status at age 11 years (prepubertal $= 0$, pubertal $= 1$). For the analyses of change from age 7 to 11 years, the 11-year measurement was the dependent variable and the measurement at age 7 years was included in the regression model. The change models thus included gender, age at both 7 and 11 years, pubertal status, the measurement at age 7 years and the two dummy variables for the stunted groups. All analyses were repeated with the addition of birth weight to the models. Eight children for whom birth weight was not available were omitted from all analyses to allow comparison of the results with or without birth weight.

Results

The mean ($\pm$SD) birth weight of the chronically stunted children (2.93 ± 0.46 kg) was not significantly different

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Table 1 Age and anthropometry in chronically stunted, previously stunted and non-stunted children*

	Age 7 years						Age 11 years					
	Chronically stunted ($n = 58$)†		Previously stunted ($n = 57$)		Non-stunted ($n = 189$)		Chronically stunted ($n = 59$)		Previously stunted ($n = 57$)		Non-stunted ($n = 190$)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age (year)	7.8	0.3	7.7	0.3	8.0	0.4	11.8	0.4	11.7	0.3	11.9	0.3
Body mass index (kg m^{-2})	14.5	1.1	14.4	1.5	15.4	1.6	16.0	1.7	16.8	2.2	18.2	3.2
Triceps skinfold (mm)	6.0	1.2	6.4	2.3	7.4	2.8	7.7	3.0	8.6	4.1	11.2	5.8
Subscapular skinfold (mm)	5.1	0.8	5.4	1.6	5.9	2.1	6.5	2.0	7.6	3.5	9.0	5.0
SSF/TSF ratio	0.87	0.14	0.86	0.12	0.82	0.12	0.88	0.18	0.90	0.16	0.83	0.18
Percentage body fat‡	11.2	4.3	12.4	5.2	13.8	5.5	14.3	6.3	16.4	7.3	19.0	8.3

* *Stunted groups*: height-for-age < -2SD at age 9–24 months; chronically stunted group, height-for-age < -1SD at age 11 years; previously stunted group, height-for-age at age 11 years ≥ -1SD. *Non-stunted group*: height-for-age > -1SD at age 9–24 months.
† $n = 57$ for skinfolds, ratio and percentage fat.
‡ Percentage body fat not calculated at age 11 years for six children with missing pubertal status.

from that of the previously stunted children (2.84 ± 0.50 kg) and both groups had significantly lower birth weight than the non-stunted children (3.33 ± 0.52 kg; $P < 0.001$). The children's mean age and anthropometry at ages 7 and 11 years are shown in Table 1. The non-stunted children were slightly older at both ages. The mean BMI of the non-stunted children was slightly higher than the NCHS median[19], while that of the stunted children was slightly above (previously stunted group) or slightly below (chronically stunted group) the 25th percentile.

The BMI distribution of the non-stunted children approximated that of the US reference population[19] with 4.2% below the 5th percentile (thin), 14.2% above the 85th percentile (overweight) and 4.7% above the 95th percentile. The US references for BMI exclude more recent data and the prevalence of overweight was substantially less than in the US black population, in which 23–30% of children are overweight[20]. Only two of the stunted children were overweight (1.7%) while 17.2% of them were thin.

Significantly more of the non-stunted children (59.9%) had entered their pubertal growth spurt than the stunted children (39.8%, $P < 0.001$). As would be expected, more girls (87.5%) than boys (22.7%, $P < 0.001$) were in puberty.

There were no significant correlations between socio-economic status and any of the anthropometric measurements at age 11 years. Socio-economic status was therefore not included in subsequent analyses.

Differences between the groups in BMI, skinfolds, SSF/TSF ratio and percentage body fat were determined by multiple regression analyses. Separate regressions were calculated with each measurement in turn as the dependent variable. The independent variables were age, gender, group and pubertal status at age 11 years. Dummy codes were entered for the two stunted groups with the non-stunted group as the reference group. The regression coefficients for the groups in Tables 2–4 indicate the difference between each stunted group and the non-stunted children. The analyses were repeated with the inclusion of birth weight in the model. The regression coefficients for birth weight (Tables 3 and 4) indicate the effect of a 1 kg change in birth weight.

Girls had significantly larger triceps ($P < 0.001$) and subscapular ($P < 0.01$) skinfolds and thus a higher percentage body fat ($P < 0.001$) than boys at ages 7 and

Table 2 Regression coefficients (*B*) and standard errors (SE) comparing stunted groups with non-stunted group from multiple regressions of BMI, skinfolds, percentage body fat and SSF/TSF ratio†

	Age 7 years				Age 11 years			
	Chronically stunted		Previously stunted		Chronically stunted		Previously stunted	
	B	SE	*B*	SE	*B*	SE	*B*	SE
BMI (log kg m^{-2})	−0.023	0.006***	−0.025	0.006***	−0.041	0.010***	−0.028	0.010**
Triceps skinfold (log mm)	−0.068	0.017***	−0.058	0.018***	−0.108	0.027***	−0.094	0.027***
Subscapular skinfold (log mm)	−0.044	0.015**	−0.037	0.016*	−0.082	0.024***	−0.055	0.024*
SSF/TSF ratio	0.048	0.019*	0.040	0.020*	0.048	0.028	0.076	0.028**
Percentage body fat (log %)	−0.059	0.015***	−0.051	0.016***	−0.087	0.023***	−0.063	0.023**

† BMI, TSF, SSF, SSF/TSF ratio and percentage body fat were the dependent variables in separate multiple regressions. Independent variables in all regressions were age, gender, dummy codes for the two stunted groups with the non-stunted group as the reference group, and pubertal status at age 11 years. Eight non-stunted children for whom birth weights were missing were excluded from all analyses. *Sample sizes*: chronically stunted age 7, $n = 57$; age 11, $n = 59$; previously stunted, $n = 57$; non-stunted, $n = 182$.
*, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

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Table 3 Regression coefficients (*B*) and standard errors (SE) for birth weight and comparing stunted groups with non-stunted group from multiple regressions of BMI, skinfolds, percentage body fat and SSF/TSF ratio†

	Age 7 years						Age 11 years					
	Birth weight		Chronically stunted		Previously stunted		Birth weight		Chronically stunted		Previously stunted	
	B	SE	*B*	SE	*B*	SE	*B*	SE	*B*	SE	*B*	SE
BMI (log kg m^{-2})	0.018	0.005***	−0.016	0.006*	−0.016	0.007*	0.021	0.007**	−0.032	0.010***	−0.018	0.010
Triceps skinfold (log mm)	0.012	0.013	−0.063	0.018***	−0.052	0.019**	0.027	0.020	−0.096	0.028***	−0.082	0.029**
Subscapular skinfold (log mm)	0.007	0.012	−0.041	0.016*	−0.034	0.017*	0.007	0.018	−0.079	0.026**	−0.051	0.026*
SSF/TSF ratio	−0.009	0.015	0.045	0.020*	0.036	0.021	−0.043	0.021*	0.030	0.029	0.055	0.030
Percentage body fat (log %)	0.011	0.011	−0.055	0.016***	−0.046	0.017**	0.018	0.017	−0.079	0.024***	−0.054	0.025*

† BMI, TSF, SSF, SSF/TSF ratio and percentage body fat were the dependent variables in separate multiple regressions. Independent variables in all regressions were age, gender, birth weight, dummy codes for the two stunted groups with the non-stunted group as the reference group, and pubertal status at age 11 years. Eight non-stunted children for whom birth weights were missing were excluded from all analyses. *Sample sizes*: chronically stunted, age 7, $n = 57$; age 11, $n = 59$; previously stunted, $n = 57$; non-stunted, $n = 182$.
*, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

11 years. The SSF/TSF ratio was higher in boys at both ages ($P < 0.01$). Pubertal children had greater BMI and SSF at age 11 years than the prepubertal children (both $P < 0.01$). Their increase in these measures and in the SSF/TSF ratio from age 7 to 11 years were also greater (all $P < 0.05$).

At age 7 years both stunted groups were significantly smaller than the non-stunted children in all measures except the SSF/TSF ratio, which was significantly larger compared with the non-stunted children, indicating a more central pattern of fat distribution in the stunted groups (Table 2). At this age, birth weight was related only to BMI (Table 3). After adjustment for birth weight, the SSF/TSF ratio was no longer significantly larger in the previously stunted group compared with the non-stunted children.

At age 11 years both stunted groups had significantly smaller BMI, skinfolds and percentage body fat than the non-stunted children (Table 2). The SSF/TSF ratio was significantly larger in the previously stunted group compared with the non-stunted children. Birth weight was positively related to BMI at age 11 years and negatively related to the SSF/TSF ratio, but was not related to skinfolds or percentage body fat (Table 3). After adjustment for birth weight, the previously stunted group was no longer significantly different from the non-stunted children in BMI or SSF/TSF ratio ($P = 0.065$).

The percentage of variance explained by age, gender, pubertal status and group was low, particularly for the SSF/TSF ratio (6% at age 7 years and 4% at age 11 years). For BMI the variance explained was 10% at age 7 years and 16% at age 11 years, and for the skinfolds the variance explained ranged from 14 to 21%. Birth weight explained an additional 3–4% of the variance in BMI at ages 7 and 11 years and 2% of the variance in the SSF/TSF ratio at age 11 years.

The chronically stunted children increased significantly less from age 7 to 11 years than the non-stunted children in all measures except the SSF/TSF ratio (Table 4). The increase in TSF was significantly less in the previously stunted children, while their SSF/TSF ratio increased more than that of the non-stunted children. There were no other differences between the previously stunted children and the non-stunted group. Birth weight was related only to

Table 4 Regression coefficients (*B*) and standard errors (SE) for birth weight and comparing stunted groups with non-stunted group from multiple regressions of change in BMI, skinfolds, percentage body fat and SSF/TSF ratio, from age 7 to 11 years†

	Without birth weight				With birth weight					
	Chronically stunted		Previously stunted		Birth weight		Chronically stunted		Previously stunted	
	B	SE	*B*	SE	*B*	SE	*B*	SE	*B*	SE
BMI (log kg m^{-2})	−0.016	0.006**	0.000	0.006	−0.001	0.004	−0.016	0.016**	0.001	0.006
Triceps skinfold (log mm)	−0.057	0.019**	−0.048	0.019*	0.015	0.014	−0.052	0.020**	−0.041	0.020*
Subscapular skinfold (log mm)	−0.050	0.017**	−0.027	0.017	−0.001	0.012	−0.050	0.017**	−0.027	0.018
SSF/TSF ratio	0.023	0.024	0.051	0.025*	−0.036	0.018*	0.008	0.025	0.035	0.026
Percentage body fat (log %)	−0.044	0.016**	−0.026	0.016	0.007	0.012	−0.041	0.017*	−0.023	0.017

† BMI, TSF, SSF, SSF/TSF ratio and percentage body fat at age 11 years were the dependent variables in separate multiple regressions. Independent variables in all regressions were age, gender, measurement at age 7 years, dummy codes for the two stunted groups with the non-stunted group as the reference group, and pubertal status at age 11 years. Analyses were conducted with and without birth weight. Eight non-stunted children for whom birth weights were missing were excluded from all analyses. *Sample sizes*: chronically stunted, age 7, $n = 57$; age 11, $n = 59$; previously stunted, $n = 57$; non-stunted, $n = 182$.
*, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

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change in the SSF/TSF ratio, which showed greater increases in children with lower birth weights. Adjustment for birth weight did not alter the findings for the chronically stunted group but the change in SSF/TSF ratio in the previously stunted children was no longer significantly different from that of the non-stunted children.

Although the measurements for the previously stunted group tended to be greater than those of the chronically stunted group, the only significant difference was in the change in BMI from age 7 to 11 years, which was greater in the previously stunted group ($P < 0.02$).

Discussion

In this prospective study, children who were stunted in early childhood had lower mean BMI and less body fat at ages 7 and 11 years than children who were never stunted, and only two of the stunted children had BMI greater than the 85th percentile of the US references[19]. BMIs were lower than those of the non-stunted children, both in children who remained stunted and in those who had achieved catch-up growth in height. The findings therefore do not support an increased risk for overweight at these ages in children stunted in early childhood, which has been found in cross-sectional studies in other populations[6]. In contrast, in a longitudinal Guatemalan study childhood stunting was associated with lower BMI in adult males but there was no relationship in women[7]. The association between stunting and later overweight thus appears to be inconsistent and may depend on environmental factors such as the change in conditions sufficient to produce a shift from dietary deficit to excess[21]. All children in the present study came from low-income areas of Kingston, Jamaica. Nonetheless, the socio-economic status (housing quality) of the stunted children was significantly lower than that of the non-stunted children in early childhood and at follow-up. It is thus possible that stunted children are more likely to come from food insecure homes and this may contribute to few of them becoming overweight.

Overweight may develop in our cohort during puberty[22] or in adulthood. Forty-eight per cent of the children had entered their pubertal growth spurt; however, significantly fewer of the stunted children had done so. Although we controlled for puberty in the analyses the children were classified only by whether they had begun their pubertal growth spurt or not, and this was thus a relatively crude index. Nonetheless, the significant associations of pubertal status with BMI and central fat deposition suggest that the index was valid. Further follow-up of the children after puberty is complete is necessary to determine whether overweight will develop among this group.

There may also be some concern about the equations used to estimate percentage body fat from skinfolds. The equations used are appropriate for prepubertal and pubertal black children but were developed with a sample of well-nourished children[15]. There is some evidence that body composition is altered in stunted children[23]. The estimates of percentage body fat for the stunted children should therefore be interpreted with caution.

Early childhood linear growth retardation was associated with a more central pattern of fat distribution. This was evident despite the relative leanness of the children. The significance of this in relation to long-term risk remains to be established. Fat distribution is not strongly associated with risk factors for cardiovascular diseases in early adolescence[24] but change in the SSF/TSF from age 13 to 27 years is associated with change in blood pressure and high-density lipoprotein-cholesterol[25]. The main importance of the more central fat distribution among the stunted children is whether it is predictive of later fat distribution. The SSF/TSF ratio at age 7 years was significantly correlated with the ratio at age 11 years ($r = 0.54$, $P < 0.001$), which is suggestive of tracking. The correlation was lower than for BMI and skinfolds ($r = 0.77$ to 0.80) but was similar to the correlations of the SSF/TSF ratio from ages 13 to 29 years in Dutch adolescents and adults[26].

Birth weight was positively associated with BMI at age 7 and 11 years. This is in agreement with other studies in which higher birth weight has been associated with higher BMI or weight later in childhood[27,28]. The relationship between birth weight and skinfolds is less consistent. Positive associations have been reported[28] as well as no association[29]. In this study, birth weight was not related to these more direct measures of body fat or to percentage body fat. The lack of relationship may be due in part to the relatively few overweight children.

Birth weight was not related to the SSF/TSF ratio at age 7 years but was related to change from age 7 to 11 years and to the ratio at age 11. Children with lower birth weights moved towards a more central distribution of fat during this interval. Thus, unlike BMI in which the positive association with birth weight begins in early childhood and tends to diminish with age, the association between birth weight and fat distribution only became evident late in childhood. This may be related to the changes in fat distribution that occur with maturation and it is possible that the association may become stronger after puberty is completed.

Lower birth weights have been reported to be predictive of higher SSF/TSF ratios in children aged 7–12 years[30] and adolescent girls[31], and of higher waist/hip ratios in adults[7]. In all three of these studies the relationship with birth weight was seen only after controlling for BMI. In contrast, in our relatively lean subjects there was no association between BMI and SSF/TSF ratio and its addition to the regression models did not alter the findings.

After adjusting for birth weight, the association between stunting and SSF/TSF ratio was significant only for the

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chronically stunted group at age 7 years. For the previously stunted group the trends remained the same but were no longer statistically significant. Thus lower birth weights among the stunted children are in large part responsible for the differences in fat distribution between the stunted and non-stunted children, although the results are suggestive of an additional effect of growth retardation in childhood.

In summary, in our cohort, stunted children did not show any tendency towards overweight at age 11–12 years. Children with lower birth weight had lower BMI but more central fat distribution and this accounted for most of the differences in fat distribution seen between stunted and non-stunted children.

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