The Consequences of Linear Growth Stunting: Influence on Body Composition Among Youth in the Bolivian Amazon

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ABSTRACT Stunting, or linear growth retardation, has been documented in up to half of all children in rural indigenous populations of South America. Stunting is well understood as a signal of adverse conditions during growth, and has been associated with developmentally induced modifications to body composition, including body fat and muscularity, that stem from early growth restriction. This article examines the relation between short stature and three anthropometric indicators of body composition during childhood and adolescence among a rural, indigenous population of forager-horticulturalists. Anthropometric data were collected annually from 483 Tsimane’ youth, ages 2–10 years, in 13 communities in the Beni region of Bolivia for 6 consecutive years (2002–2007). Baseline height-for-age was used to indicate stunting (HAZ < −2.0) and compared with z-scores of body mass index (BMI), sum of two skinfolds, and arm muscle area. Multilevel regression models indicate baseline stunting is associated with lower BMI z-scores (B = −0.386; P < 0.001), body fatness (ZSkinfold, B = −0.164; P < 0.001), and arm muscularity (AMAZ, B = −0.580; P < 0.001) in youth across a period of 6 years. When split by sex, there was a stronger relation between baseline stunting and lower skinfold body fat scores among girls (B = −0.244; P < 0.001) than boys (B = −0.080; P = 0.087). In contrast, baseline stunting was associated with lower arm muscularity in both girls (B = −0.498; P < 0.001) and boys (B = −0.646; P < 0.001). The relation between linear growth restriction and indicators of body composition persist into adolescence, providing additional insight into the influence of adverse conditions during growth. Am J Phys Anthropol 153:92–102, 2014. © 2013 Wiley Periodicals, Inc.

Stunting, or linear growth retardation (< −2.0 SD in height-for-age Z-score), is common in many rural indigenous populations of South America, and most recent estimates suggest that about 165 million children under the age of five worldwide were growth stunted (de Onis et al., 2012; Black et al., 2013). The bulk of research on growth patterns in biological anthropology (Kuzawa, 1998; Begun, 1999; Walker et al., 2006; Bogin et al., 2007) draws on evolutionary life history, which explicitly grapples with how trade-offs between overlapping domains of growth, reproduction, and maintenance serve to enhance survival and reproduction in heterogeneous environments. In terms of understanding human phenotypic variation, these developmental trade-offs connect early growth deficits to long-term consequences including shorter stature (Stein et al., 2010; Sterling et al., 2012) and increased risk of developing metabolic diseases including cardiovascular disease and type 2 diabetes (Barker et al., 2002). While there is considerable evidence linking growth restriction in utero to adult outcomes, research on the associations between postnatal stunting and later-life body composition has produced more variable results. Body fat reserves play important roles in survival and reproduction and developmental oscillations in body composition throughout childhood and adolescence may have evolutionary consequences (Zafon, 2007; Adair, 2008; Wells, 2010). Therefore, research examining how growth restriction or stunting in childhood is related to body composition may deserve additional attention.

Over the past several decades, research has shown links between early growth stunting and later body composition but the direction of the association likely varies with local context. Among some urbanizing populations, childhood stunting exists alongside rapidly rising rates of obesity. This dual nutritional burden is hypothesized to stem from developmental adaptations in which metabolic adjustments early in life result in increased risk of metabolic disease, including obesity, later in life (Frisancho, 2003; Leonard et al., 2009; Wilson et al., 2012). Several large studies have identified the co-occurrence of both stunting and overweight or obesity in children (Popkin et al., 1996; Fernald and Neufeld, 2007) and...
evidence for metabolic trade-offs associated with linear growth stunting is mounting (Hoffmann et al., 2000a,b). For example, in large surveys from Russia, Brazil, China, and the Republic of South Africa, Popkin et al. (1996) found that stunted children had a 1.7–7.8 times increased risk of being overweight than their non-stunted peers, depending on country and household income. Research among Mayans in urban Mexico has also demonstrated associations between child stature, lean body mass, and total energy expenditure (Wilson et al., 2012) and that the effects of shortness and stunting may persist across generations (Varel-Silva et al., 2009; Azcorra et al., 2013).

Several longitudinal or cohort studies have failed to find clear associations between stunting and weight or body fat during childhood or adolescence. For example, in their longitudinal research among South African urban children, Cameron et al. (2005) found children who were stunted at 2 years of age were shorter and lighter than their non-stunted peers at 9 years of age, but they did not identify associations between stunting and later BMI, body composition, or fat patterning. Research from Guatemala (Li et al., 2003) and Jamaica (Walker et al., 2002, 2007) has also suggested early growth restriction is associated with less body fat and musculoskeletal development. The results in this literature indicate that the consequences of linear growth restriction are complex and likely depend on local environment, diet, and developmental timing. Therefore, as several researchers have noted (Baker et al., 2009; Varel-Silva et al., 2012), longitudinal research in rural areas of low-income countries is needed to better understand the full range of consequences of growth trade-offs.

The goal of this article is to examine the short-run biological consequences of linear growth stunting throughout childhood and adolescence in a foraging-horticultural group with high rates of stunting and infection but limited evidence for overnutrition. We explore the consequences of growth stunting on both muscularity and body fatness using anthropometric data collected from Tsimane’ youth from 2002 to 2007. Prior work among Bolivia’s Tsimane’ has documented both frequent stunting (Foster et al., 2005) and evidence for catch-up growth during childhood associated with household composition and income (Godoy et al., 2010a). Additionally, previous research suggests that growth stunting is associated with linear growth retardation and reduced body fat reserves (McDade et al., 2008). Given these associations, we first predict that tracking childhood growth will identify associations between growth stunting and body composition across a 6-year period. Second, we predict that sex will modify the relations between growth stunting, muscularity, and body fat based on literature suggesting that boys may be more sensitive to the nutritional environment than girls (Stinson, 1985; Kuzawa, 2007). Third, we predict that the association between stunting and body composition will vary with age and, more specifically, become stronger at later ages as growth becomes canalized (Bogin, 1999). Finally, we predict that the association between stunting and body fatness will remain after controlling for maternal, household, and community effects.

METHODS

Background

Research was conducted among Tsimane’, an indigenous group of ~15,000 people living in the Beni Depart-
they estimate that the average survivorship to age 5 was 79.3%. In more recent years, Gurven (2012) found that infant mortality rates in the first year of life were high (~13%) but had declined from 1990 to 2002.

In order to examine catch-up growth, a recent paper explored the growth trajectories of 2- to 7-year-old children in the 2002–2006 TAPS panel dataset (Godoy et al., 2010a). Using year-to-year change in age and sex-standardized height, this research found children who were stunted at baseline exhibited faster growth than their non-stunted peers. Growth rates were not statistically different between boys and girls: 25% of girls and only 13% of boys who were stunted at baseline were no longer stunted in 2006. Variables that were associated with slower linear growth were 1) living in a community close to a Bolivian town, 2) having additional younger siblings, and 3) having higher household income levels. Here, we build on these findings by adding an additional year of data and by considering the association between linear growth stunting and anthropometric indicators of body composition.

Recent research has suggested people throughout the Amazon are experiencing increasing rates of overweight and obesity (Benefice et al., 2007; Lourenco et al., 2008), although others have documented modest changes in nutritional status (Piperata et al., 2011). In one of a small number of studies to consider child body composition in rural lowland Bolivia, Benefice et al. (2007) documented the rates of overweight among Amerindian children living along the Beni River to be about 12%, with less than 1% of children obese according to BMI. Research among Tsimane’ has found evidence for gain in weight over the past decade among adults; however, the significance of this trend is elusive. Weight increases have not clearly been linked to any of the commonly invoked explanations including wealth, degree of cultural consonance, acculturation, or household measures of food availability (Reyes-García et al. 2010; Zeng et al., 2012).

**Methods**

In 2002, a baseline survey was conducted during May–August to capture demographic, socio-economic, cultural, and health data along with anthropometric measurements of all participating individuals. The initial survey attempted to recruit all individuals over 2 years of age living in the 13 selected communities and follow-up surveys were conducted annually. In this article, we limit our analyses to 483 children (225 girls and 258 boys) who were between the ages of 2 and 10, inclusive, in 2002. Children were tracked for 6 consecutive years and, at the end of data presented here, the children were 7–15 years of age. The age bracket in this study follows children through a period in which they are sensitive to growth faltering due to infection and undernutrition (Bogin, 1999).

Age was reported by the child’s principal caretaker in each year of the survey. When possible, parents reported the child’s exact birth date or showed a birth certificate. When the parent was not able to provide an exact age or birthdate, surveyors estimated age in years based on surveys with parents that focus on season of birth, birth order within a household, and age in relation to locally important events. The study protocol was reviewed by the Institutional Review Boards of Northwestern and Brandeis Universities.

In addition to annual surveys with primary caretakers on a range of individual-level and household-level characteristics including demographics, income, and wealth (Leonard and Godoy, 2008), anthropometric measurements were taken annually on all participating individuals following Lohman (1988). Stature was measured with a portable stadiometer. Body weight was measured with a Tanita scale and recorded to the nearest 0.2 kg. Mid-arm circumference was measured to the nearest millimeter, using plastic tape measures. Triceps and subscapular skinfold thicknesses of were measured to the nearest 0.5 mm using Lange calipers. The technical error of measurement for anthropometry is not available. Across the study period, measurements were taken by multiple surveyors who received training in anthropometric methods before data collection.

Anthropometric measures of body composition were examined through three derived anthropometric indices. Body Mass Index was calculated as weight (kg)/height (m$^2$). A skinfold measure of body fatness was calculated using the sum of tricep and subscapular skinfolds (Sum 2 skinfolds). Upper arm muscle area was calculated following Frisancho (2008). For analysis, height-for-age and weight-for-height measures were standardized relative to the National Center for Health Statistics (NCHS) percentiles as $z$-scores (i.e., HAZ, WHZ; Hamill et al., 1979). BMI $z$-scores were calculated for all children older than 5 years of age also, using the 2007 WHO growth reference based on NCHS data (Hamill et al., 1979; de Onis et al., 2007). Finally, age- and sex-standardized $z$-scores for the sum of two skinfolds (ZSkinfold) and arm muscle area $z$-score (ZAMA) were calculated according to Frisancho (2008), which provide growth references values based on the NHANES III databases.

**Data analysis**

Analysis was conducted in Stata 10.0 in two stages. First, descriptive statistics were used to examine the association between stunting and body composition using a sub-sample of 321 youth who participated in both 2002 and 2007. Here we consider if the youth was stunted in 2002 (baseline stunting) and patterning in the outcomes of interest: a) BMI, b) skinfold measurements, and c) arm muscle area. Because boys and girls differ in body composition and rates of growth, descriptive data are presented for each sex separately. Second, panel linear multiple regressions with individual random effects, clustering by child, and robust standard errors are used to estimate the association between baseline stunting and measures of body composition. Random effects modeling allows the inclusion of time invariant characteristics (sex, baseline stunting) and examines if differences across individuals are related to the outcome variable of interest (Rabe-Hesketh and Skrondal, 2008). Of the 483 children measured at baseline, ~49% were present during all 6 surveys and ~21% were present in 5 survey years. About 6% were present only in the first year and 5% only in the first 2 years. The remaining ~19% were present in 3 or 4 survey years. Children who left the sample during the study were not more likely to be stunted than children who remained in sample (Chi-square test of base stunting vs. eventual attrition $P = 0.792$). There was also no difference in stunting rates among children who participated in <4 years of anthropometric measurements.
than those who participated in 4 or more years (Chi-square = 0.617; $P = 0.432$).

Other publications have discussed rounding error or digit heaping (Godoy et al., 2010a) and there is evidence for digit heaping in height and skinfold measurements in this study. For example, digit heaping in skinfold measurements occurred with values ending in 5, 6, or 7 mm (they account for nearly 50% of values instead of the 30% that would be expected with a normal distribution. The consequences of this measurement error are discussed in the conclusions.

Byron (2003) found that Tsimane’ girls reach puberty by 12–13 years of age and, although puberty is a confounding factor in the associations examined in this article, we do not have accurate information on puberty. Therefore, in order to facilitate data presentation and minimize the confounding effects of puberty, sex and age categories are used for descriptive data presentation: a) 2–4 years at baseline (7–9 at follow-up), b) 5–7 at baseline (10–12 at follow-up), and c) 8–10 at baseline (13–15 at follow-up). We eliminated six measurements from young women who were either pregnant or lactating.

**RESULTS**

**Measures of body composition in 2002 and 2007**

Anthropometric assessments of body composition show that Tsimane’ children and adolescents are relatively short, but not experiencing acute nutritional stress (Tables 1 and 2). BMI scores among the sub-set of children and adolescents ($n = 321$) that participated in both 2002 (baseline) and the last year of data used here, 2007, indicate that Tsimane’ youth are not experiencing acute malnutrition (mean BMI range from 16.4–19.7 for boys, 15.8–21.4 for girls). Similarly, the age- and sex-standardized BMI $z$-scores also indicate Tsimane’ youth are comparable to international reference populations (mean BMIs for all boys $= 0.26$; girls $= 0.25$ in 2007). In 2002, 11% of girls (11 of 86) and 25% of boys (26 of 104) over the age of 5 years had a BMI $z$-score above 1.0, indicating at risk of overweight (de Onis et al., 2010). By 2007, the frequency of overweight remained constant in girls (11%, 116 of 145) and declined for boys (11%, 19 of 176). Thus, Tsimane’ youth were lean, but skinfold measurements of body fatness were not so low as to indicate severe energy stress (mean ZSkinfold $= -0.40$ for all girls and $-1.0$ for all boys in 2007) and masculinity levels do not indicate acute protein malnutrition (ZAMA $= -0.52$ for girls and $-0.93$ for boys in 2007).

Overall, by 2007, youth who were stunted (S) at baseline had lower measures of adiposity (Fig. 1) and masculinity (Fig. 2) than their peers who were not stunted (NS) at baseline. Children who were stunted in 2002 had lower average skinfold body fat measurements than their non-stunted peers in 2007, although the difference is more apparent during the ages of 10 and 14 for both boys and girls.

Similar to body fatness, Tsimane’ boys (Fig. 2a) and girls (Fig. 2b) who were stunted at baseline had lower average values of upper arm muscularity in 2007 than their non-stunted peers. In contrast to skinfold measures of body fatness, the difference in average arm muscularity between stunted and non-stunted children appears similar for all youth and consistent from ages 2 through 10 years.

Youth who were stunted in both 2002 and 2007 had significantly poorer indicators of body composition than their peers who were not stunted in either year or those who changed stunting status from 2002 to 2007 (Table 3). Those who were never stunted had a higher average BMI $z$-score than youth who were stunted in both years (ANOVA $F = 9.46$, df = 3; $P < 0.001$). When considering arm muscularity, youth who were not stunted in either 2002 or 2007 also had significantly higher AMA $z$-score than their peers who were stunted in both years (mean values of $-0.43$ vs. $-1.23$; $F = 22.06$; df = 3; $P < 0.001$). A similar pattern emerged with respect to skinfold thickness (ANOVA $F = 5.31$, df = 3; $P = 0.001$).
Although the comparisons above suggest that stunting is associated with lower BMI, skinfold measurements, and arm muscularity over time, panel data allow an examination of year-to-year patterns. In the entire sample of youth, descriptive analyses of the panel data from 2002 to 2007 suggest two points: a) stunting appears to be associated with smaller skinfold measurements in girls; and b) while all Tsimane' children diverge from
Their US peers with respect to arm muscularity, stunted children have lower scores of arm muscularity than their non-stunted peers.

Figure 3 shows the average yearly values of age- and sex-adjusted z-scores of skinfold measurements and arm muscularity for youth who were stunted and non-stunted at baseline in 2002. For boys, skinfold fatness does not appear to differ between boys who were stunted and non-stunted in 2002 but, by 2007, boys who were stunted at baseline had significantly lower age- and sex-adjusted skinfold measurements (NS = −0.94; S = −1.13; t = 2.184 (181); P = 0.030 (Fig. 3a). Similarly, among girls, there is little difference in z-scores of body fatness between those who were stunted and non-stunted at baseline (NS = −0.39; S = −0.51; t = 1.468 (200); P = 0.144). However, by 2007, girls who were not stunted at baseline have significantly higher average values of skinfold body fat than girls who were stunted at baseline (ZSkinfold: −0.28 vs. −0.58; t = 3.542 (153); P = 0.001). Finally, descriptive analysis of each year shows that both boys (Fig. 3a) and girls (Fig. 3b) who were stunted in 2002 had lower average z-scores for arm muscularity than their peers who were not stunted in 2002 across all years in this sample.

Regression results

Tables 4 and 5 contain the main regression results. First, consistent with the graphical and descriptive analysis presented above, stunting early in life is related to later body composition for Tsimane’ youth. Across the study period, youth who were stunted in 2002 had lower BMI z-scores (B = −0.386; P < 0.001; Table 4, model 1) than their non-stunted peers. The association remained consistent when boys and girls were analyzed independently and there was no evidence for interactions between sex and baseline stunting. Because BMI z-scores are most commonly used for children over 5 years of age, additional models using weight-for-height as the outcome variable for children who were 2–4 years old at baseline showed that, among the younger cohort of the panel, there was little relation between baseline stunting and weight-for-height z-score during 2002–2007 (B = −0.084; P = 0.271, Table 4, Model 1).

The association between baseline stunting and body fatness and arm muscularity from 2002 to 2007 (Table 5) was similar to the pattern observed with BMI. Youth who were stunted at baseline had significantly lower skinfold z-score (B = −0.164; P < 0.001; Model 1) and arm muscle area z-score (B = −0.580; P < 0.001; Model 1) than their non-stunted peers after controlling for covariates in the model. Additionally, results show evidence for a stronger relation between baseline stunting and lower skinfold z-score among girls (B = −0.244; P < 0.001; Table 5, model 2) than among boys (B = −0.080; P = 0.087; model 3) when the models were run separately for boys and girls. An additional regression model for the full sample including an interaction term (male × baseline stunting) indicated that the difference in favor of boys was statistically significant at conventional levels (full results not shown, P = 0.026). For arm muscularity, the results observed with the full sample remained consistent when boys and girls were analyzed separately. Baseline stunting was associated with lower average scores of arm muscularity in girls (B = −0.498; P < 0.001; Table 5, model 2) and boys (B = −0.646; P <
The association between stunting and anthropometric measurements of body composition differed across early life. Regression models that examined skinfold measurements and muscularity independently for both the youngest ages (2–4 at baseline) and oldest ages (8–10 at baseline) show the effects discussed above remained only for the older children (Table 6, rows 1 and 2). Among the youngest children, baseline stunting was associated with lower values of arm muscularity from 2002 to 2007, but the associations with skinfold z-scores were not statistically significant at conventional levels. In contrast, among the older age category, which is most likely to include youth in multiple stages of puberty, baseline stunting remained inversely associated with lower scores of both arm muscularity and body fatness among girls.

Finally, the results presented above remain robust to additional analysis that included maternal characteristics (mother’s age, height, weight, and years of completed education) and household conditions (number of children in the household, household income, and wealth). Including these covariates slightly strengthened the relation between stunting and lower skinfold z-scores (Table 6, row 3). Adding additional covariates to account for community level effects also had only a small effect on the associations (Table 6, row 4). For example, children stunted at baseline had significantly lower AMA z-scores than their non-stunted peers (B = 0.580; P < 0.001) in the original model, but controlling for maternal, household, and community effects slightly reduces the effect size (B = 0.518; P < 0.001). Household wealth, number of children in the household, and mother’s height and weight were statistically associated with measures of body composition among youth (results not shown), but the associations between stunting and body composition remained robust after controlling for these confounding effects.

**DISCUSSION AND CONCLUSION**

Indigenous groups throughout South America suffer disproportionately high rates of infection and undernutrition (Hurtado et al., 2005), but several studies also

**TABLE 4. Random-effect panel linear regression models examining the relation between baseline stunting (2002) and the child’s BMI or weight-for-height z-score from 2002 to 2007**

<table>
<thead>
<tr>
<th>Explanatory variable</th>
<th>All children (1)</th>
<th>Girls (2)</th>
<th>Boys (3)</th>
<th>All children (1)</th>
<th>Girls (2)</th>
<th>Boys (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline stunting (yes)</td>
<td>-0.386*** (0.066)</td>
<td>-0.467*** (0.104)</td>
<td>-0.310*** (0.086)</td>
<td>-0.084 (0.076)</td>
<td>-0.192* (0.110)</td>
<td>0.023 (0.104)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>-0.313*** (0.048)</td>
<td>-0.396*** (0.062)</td>
<td>-0.261*** (0.064)</td>
<td>0.149*** (0.054)</td>
<td>0.189*** (0.069)</td>
<td>0.105 (0.084)</td>
</tr>
<tr>
<td>Age²</td>
<td>0.014*** (0.002)</td>
<td>0.020*** (0.003)</td>
<td>0.010*** (0.003)</td>
<td>-0.005 (0.005)</td>
<td>-0.008 (0.006)</td>
<td>-0.001 (0.007)</td>
</tr>
<tr>
<td>Male</td>
<td>0.106 (0.067)</td>
<td></td>
<td></td>
<td>0.013 (0.076)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>2.015*** (0.238)</td>
<td>2.203*** (0.305)</td>
<td>2.037*** (0.330)</td>
<td>-0.128 (0.165)</td>
<td>-0.184 (0.205)</td>
<td>-0.046 (0.253)</td>
</tr>
</tbody>
</table>

Number of observations: 1,418 (646) 646 (772) 772 (952) 465 (487) 465 (487) 487 (102)

Robust standard errors in parentheses.

* P < 0.1, ** P < 0.05, *** P < 0.01.

**TABLE 5. Random-effect panel linear regression models examining the relation between baseline stunting (2002) and the measures of body fatness (ZSkinfold) and arm muscularity (AMAZ) from 2002 to 2007 among Tsimane’ youth**

<table>
<thead>
<tr>
<th>Explanatory variable</th>
<th>All children (1)</th>
<th>Girls (2)</th>
<th>Boys (3)</th>
<th>All children (1)</th>
<th>Girls (2)</th>
<th>Boys (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline stunting (yes)</td>
<td>-0.164*** (0.038)</td>
<td>-0.244*** (0.058)</td>
<td>-0.080 (0.047)</td>
<td>-0.580*** (0.057)</td>
<td>-0.498*** (0.080)</td>
<td>-0.646*** (0.077)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>-0.011 (0.028)</td>
<td>-0.148*** (0.040)</td>
<td>-0.097*** (0.033)</td>
<td>-0.206*** (0.035)</td>
<td>-0.202*** (0.052)</td>
<td>-0.220*** (0.046)</td>
</tr>
<tr>
<td>Age²</td>
<td>0.002* (0.002)</td>
<td>0.007*** (0.002)</td>
<td>0.011*** (0.002)</td>
<td>0.011*** (0.002)</td>
<td>0.013*** (0.003)</td>
<td>0.010*** (0.002)</td>
</tr>
<tr>
<td>Male</td>
<td>0.426*** (0.039)</td>
<td></td>
<td></td>
<td>-0.293*** (0.056)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>-0.063 (0.127)</td>
<td>0.452*** (0.181)</td>
<td>-0.677*** (0.149)</td>
<td>0.549*** (0.164)</td>
<td>0.327 (0.235)</td>
<td>0.497** (0.210)</td>
</tr>
</tbody>
</table>

Number of observations: 2,339 (1,092) 1,247 (2,341) 1,244 (1,097) 1,244 (1,244)

Robust standard errors in parentheses.

* P < 0.1, ** P < 0.05, *** P < 0.01.
have found evidence of overweight and obesity among adults (Benefice et al., 2007; Zeng et al., 2012). This study draws on panel data to examine the relation between childhood stunting and body composition among a sample of Tsimane’ youth. Overall, we found that linear growth retardation early in life has implications for body composition throughout adolescence. Among Tsimane’ youth, stunting was associated with lower age and sex adjusted scores of BMI, skinfold body fat reserves, and arm muscularity after a period of 6 years. The effects of linear growth retardation are particularly evident among girls and older youth.

Findings in this article are consistent with literature suggesting early growth restriction may permanently constrain lean mass development and modify body fat throughout adolescence and into adulthood (Cameron, 2007; Wells, 2007). For example, in Guatemala, Li et al. (2003) found that growth retardation in early childhood (0–2 years of age) was associated with leanness and less body fat at 21–27 years of age. In a prospective cohort study in Kingston, Jamaica, Walker et al. (2002) found that, after controlling for the effects of low birth weight, children who were “chronically stunted” (stunted both at 9–24 months and 7 years) had less fat and lower BMI than non-stunted children at 11 years of age. Although stunted children had less body fat than non-stunted children, stunting was also associated with a more central fat distribution (as assessed by the ratio of subscapular:triceps skinfolds), indicating that stunting may modify how fat is distributed across the body.

Our findings and other research among Latin American children (Varella-Silva et al., 2012, Wilson et al., 2012) are relevant to understanding the co-occurrence of childhood stunting and overweight or obese status (Popkin et al., 1996). Although the relationship can vary with the location, study design, and methodology, there is a strong suggestion that important role of environment in human growth and downstream health (Cameron, 2007). A potential explanation for differences in the literature is that possible links between stunting and overweight or abdominal fat patterning may be visible only among youth with access to high-fat or calorically dense foods (Popkin et al., 1996; Frisancho, 2003; Kain et al., 2005) and moderate levels of daily activity. A high-fat diet may be particularly important in these associations, as Sawaya et al. (1998) found that a high-fat diet resulted in greater body fat gains in stunted girls than non-stunted girls after controlling for the effects of energy intake and activity levels in Brazil. Among the Tsimane’ communities participating in this study, households were relatively independent of market foods throughout the panel study. Although the pattern is beginning to change, food is primarily produced at the household level and consists of crops such as rice, corn, plantains, and manioc supplemented with wild game, fish, or household domestic animals such as chickens and pigs. Additionally, children and adolescents are active throughout the day, which would influence both muscularity and fatness. Some attend school in the mornings and youths spend the remainder of the day fishing or hunting, playing in groups throughout the community, or assisting parents or older siblings in household duties which may include child-care or working in fields (Aiello, 2013). If the consequences of linear growth retardation differ with context, we might expect that the increases in body fat or BMI for age may occur only when at least one of the double burdens of infection and moderate undernutrition is removed.

A second possible explanation may be population variation in stature. Tsimane’ adults are also relatively short and about 40% of Tsimane’ adults are classified as stunted when compared with sex and age peers in the US (Godoy et al., 2010b). Recently several scholars have suggested adaptive explanations for small body size (Perry and Dominy, 2008). Although previous research among Tsimane’ suggests catch-up growth is influenced

**TABLE 6. Prediction testing and results of random-effects panel linear regression models examining the relation between baseline stunting and measures of body composition in youth.**

<table>
<thead>
<tr>
<th>Explanatory variables</th>
<th>Skinfolds z-score</th>
<th>Arm muscle area z-score</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>All children</td>
<td>Girls</td>
</tr>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
</tr>
<tr>
<td>Youngest children</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(2–4 years in 2002; n = 195)</td>
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<td></td>
</tr>
<tr>
<td>All children</td>
<td>0.029 (0.059)</td>
<td>0.011 (0.095)</td>
</tr>
<tr>
<td>Older children</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(8–10 years in 2002; n = 142)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All children</td>
<td>0.266*** (0.087)</td>
<td>0.474*** (0.118)</td>
</tr>
<tr>
<td>Includes controls for mother and household condition*</td>
<td>0.189*** (0.038)</td>
<td>0.197*** (0.050)</td>
</tr>
<tr>
<td>(n = 477)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3+ community effectsb</td>
<td>0.174*** (0.036)</td>
<td>0.261*** (0.053)</td>
</tr>
</tbody>
</table>

Values are Beta (Robust standard error).
*a Control variables include mom’s age, education, height and weight, number of children in household, and household income and wealth.
*b Adds dummy variables for community of residence.
**P < 0.1, ***P < 0.05, ****P < 0.01.
by local ecology (Godoy et al. 2010a), research also sug-
gest that adult stunting does not carry negative conse-
quences for socioeconomic indicators of well-being such
as education or income (Godoy et al., 2010b; Undurraga
et al., 2012). Additional research is needed to under-
stand the longer-term consequences of shortness or
stunting.

A second important finding of this study is that sex
appeared to modify the association between stunting
and body fatness as assessed through skinfold measure-
ments. When boys and girls were analyzed independ-
ently, baseline stunting was associated with both lower
age- and sex-adjusted measures of body fat and arm
muscularity among girls across a period of 6 years.
Among boys, baseline stunting was associated with arm
muscularity but the association with skinfold measures
of body fatness was not statistically significant at con-
ventional levels of P < 0.05. Although this result should
be interpreted with caution in this study, it is consistent
with research in Guatemala where women who
were stunted early in life had lower amounts of both lean and fat
mass than women who had not been stunted, while
men who experienced early childhood stunting had adult
reductions only in lean mass compared to their non-
stunted peers (Li et al., 2003).

Although research among Tsimane’ children has consis-
tently found little evidence for disparities between girls
and boys with respect to nutritional status, infection,
immune system activation, or rates of catch-up growth
(Foster et al., 2005; McDade et al., 2005; Godoy et al.,
2006; McDade et al., 2008), a few studies have also indi-
cated that there may be sex differences in responses to
environmental stressors. Prior research has indicated
that environmental conditions during growth, and specifi-
cally greater variation in yearly rainfall levels, are associ-
ated with reduced adult height of Tsimane’ women but
not of Tsimane’ men (Godoy et al., 2008b). In contrast, the
association between climatic conditions and child and ado-
lescent height was less clear as the height of boys 2–12
years of age was more susceptible to climate events than
the height of girls of the same age (Godoy et al., 2008a).
Life history theory and the developmental origins of
health research suggest that boys may be more sensitive
to environmental insults than girls, especially in utero
(Stinson, 1985; reviewed in Kuzawa and Pike, 2005). Evi-
dence for differential susceptibility to environmental
influences after birth is somewhat more conflicting, possi-
bly because of the buffering effects of parental behavior
and/or cultural traditions that give preference to boys or
girls (reviewed by Stinson, 1985).

Overall, our findings also suggest that additional
research examining diet, activity, and the metabolic con-
sequences of stunting in diverse contexts is needed.
Work by Hoffman and his colleagues in the shantytowns
of Brazil (Schroeder et al., 1999; Hoffman et al., 2000;
Martins et al., 2004; Hoffman et al., 2007) and others
(Schroeder et al., 1999; Walker et al., 2001; Fernald
and Neufeld, 2007; Adair, 2008) have found that child stun-
ting correlates with changes to substrate metabolism,
metabolic rate, and blood pressure in children and ado-
lescents. This research suggests that stunted children
have a reduced capacity to burn (oxidize) fat and this
enhanced fat storage capacity might predispose them to
increased body fatness later in life (Grillol et al., 2005;
Walker et al., 2007).

A strength of this study is the fact that this is one of
only a handful of studies to track body composition in an
indigenous group through puberty. Our finding that lin-
ear growth stunting at 2–10 years of age is associated
with body composition across a 6-year period indicates
the importance of considering multiple stages of the
growth process. An important shortcoming of this study
is that we do not have accurate birth weight data or fre-
quent measures of linear growth or weight gain during
the first 2 years of life; therefore, we are unable to eval-
uate or control for the role of prenatal growth, birth
weight, and early postnatal growth. We also do not have
data on puberty, which could be an important confound-
ing factor among the oldest age category. Additionally,
information on metabolic rates and activity level among
children and adolescents is needed to shed light on the
underlying mechanism linking growth perturbations to
life-long body composition. Finally, rounding error and
random measurement error in anthropometric measure-
ments would inflate standard errors and weaken the
results. These measurement errors suggest that the
inverse association between stunting and later anthropo-
metric measurements of muscle and skinfold body fat
likely underestimates the magnitude of the true rela-
tion in this sample.

In sum, these results present a short window of
insight into the connection between linear growth and
body composition in a rural, indigenous population. We
found evidence that linear growth stunting was associ-
ated with short-run anthropometric measures of body
composition among Tsimane’ youth, including lower BMI
z-scores and measures of arm muscularity and body fat-
ness. Given the importance of developmental plasticity
and the potential for intergenerational effects of linear
growth retardation (Kuzawa, 2007), additional research
that examines the full consequences of growth stunting
is needed to examine the links between nutrition,
health, and disease.

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