Energy expenditure of stunted and nonstunted boys and girls living in the shantytowns of São Paulo, Brazil[1–3]

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ABSTRACT

Background: Stunting increases the risk of obesity in developing countries, particularly in girls and women, but the underlying reason is not known.

Objective: Our objective was to test the hypothesis that stunted children have lower energy expenditure than do nonstunted children, a factor that has predicted an increased risk of obesity in other high-risk populations.

Design: A cross-sectional study was conducted in shantytown children from São Paulo, Brazil. Twenty-eight stunted children aged 8–11 y were compared with 30 nonstunted children with similar weight-for-height. Free-living total energy expenditure (TEE) was measured over 7 d by using the doubly labeled water method. In addition, resting energy expenditure (REE) was measured by indirect calorimetry and body composition was measured by dual-energy X-ray absorptiometry.

Results: There were no significant associations between stunting and any measured energy expenditure parameter, including REE adjusted for weight ($\bar{x}$ ± SEM: 4575 ± 95 compared with 4742 ± 91 kJ/d, in stunted and nonstunted children, respectively) and TEE adjusted for weight (8424 ± 239 compared with 8009 ± 221 kJ/d, in stunted and nonstunted children, respectively). In multiple regression models that included fat-free mass and fat mass, girls had significantly lower TEE than did boys ($P < 0.05$) but not significantly lower REE ($P = 0.17$).

Conclusions: There was no association between stunting and energy expenditure after differences between groups in body size and composition were accounted for. However, the girls had lower TEE than did boys, which may help to explain the particularly high risk of obesity in stunted adolescent girls and women in urban areas of developing countries. *Am J Clin Nutr* 2000;72:1025–31.

KEY WORDS Energy requirements, children, stunting, developing countries, girls, obesity, shantytowns, Brazil

INTRODUCTION

The prevalence of obesity is increasing in most countries worldwide, even those that have traditionally had high rates of undernutrition (1, 2). Childhood nutritional stunting, an indicator of chronic malnutrition (3, 4), has been suggested as one factor contributing to high rates of obesity in developing countries because of the observed association between stunting and adolescent and adult obesity (5, 6). In studies done by our own research group, stunted girls appeared to be at even greater risk of obesity than boys, with a 35% prevalence of obesity compared with 11% in stunted adolescent boys (and 13% in nonstunted girls and 8% in nonstunted boys) (5). However, the mechanisms underlying the apparent association between stunting and obesity have received little attention.

We recently observed that, compared with nonstunted children from the same community, stunted children have a lower fasting fat oxidation rate (7), a factor that strongly predicts excess weight gain in other at-risk populations (8–11). Reduced fat oxidation can contribute to obesity over time because fat that is not oxidized must be stored. In addition, the increased carbohydrate oxidation that may occur in parallel with decreased fat oxidation should increase hunger according to the energy regulation theory of Flatt (12), which is based on the assumption that carbohydrate stores are a signal for hunger. Increased hunger would further favor excess weight gain by promoting increased energy intake under conditions of a plentiful food supply.

In addition to impaired fat oxidation, other factors have been suggested as promoting obesity. In particular, low energy expenditure has been evaluated in several studies (13) and has predicted excess weight gain in some (14–16) but not in all (17, 18) prospective studies in children and adults; one study predicted weight gain in boys but not girls (19). However, relatively little is known about the effects of stunting on energy expenditure (20). Such information would not only help to determine the underlying causes of obesity associated with stunting, but it would also contribute to answering the question of whether the energy

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TABLE 1
Characteristics of the study children and their parents[1]

	Nonstunted children		Stunted children	
	Boys	Girls	Boys	Girls
Children				
Age (mo)	122 ± 19 [15]	120 ± 15 [15]	120 ± 16 [15]	124.1 ± 15 [13]
Height (cm)[2]	134 ± 8 [15]	136 ± 10 [15]	126 ± 10 [15]	127 ± 9 [13]
Weight (kg)[2]	32.0 ± 5.2 [15]	30.9 ± 6.2 [15]	25.6 ± 5.2 [15]	26.1 ± 4.3 [13]
HAZ[2]	−0.75 ± 0.74 [15]	−0.39 ± 0.84 [15]	−2.01 ± 0.63 [15]	−2.30 ± 0.76 [13]
WHZ	0.55 ± 0.63 [15]	0.28 ± 0.83 [15]	−0.35 ± 1.0 [15]	0.24 ± 1.2 [13]
Body fat (% of wt)[3]	17.1 ± 5.9 [15]	23.5 ± 6.0 [15]	15.0 ± 3.7 [15]	19.8 ± 5.3 [13]
Fat-free mass (% of wt)[3]	82.9 ± 5.9 [15]	76.5 ± 6.0 [15]	85.0 ± 3.7 [15]	80.2 ± 5.3 [13]
Fathers				
Height (cm)	165 ± 8 [16]		160 ± 12 [10]	
BMI (kg/m^2)	25.6 ± 3.5 [16]		24.7 ± 2.7 [10]	
Mothers				
Height (cm)	153 ± 7 [20]		151 ± 9 [18]	
BMI (kg/m^2)	25.3 ± 4.1 [20]		26.2 ± 4.9 [18]	

[1] $\bar{x}$ ± SD; *n* in brackets. HAZ, height-for-age *z* score; WHZ, weight-for-height *z* score.
[2] Significant effect of group, $P < 0.05$.
[3] Significant effect of sex, $P < 0.05$.

requirements of impoverished communities are lower than are those of well-nourished populations. We therefore tested the hypothesis that stunted prepubertal children have lower energy expenditures than do nonstunted children living in the same environment.

SUBJECTS AND METHODS

Subjects

Fifty-eight children were selected from population surveys of 3 shantytowns in the city of São Paulo, Brazil. More than 300 children aged 8–11 y were screened to participate in the study and were eligible if they had a weight-for-height *z* score (WHZ) of −2.0 to 1.50 according to the National Center for Health Statistics standards (21). Stunting is widely considered to be an index of long-term childhood undernutrition and was used as such in this study (4, 22). Children recruited for the stunted group ($n = 28$) had height-for-age *z* scores (HAZs) ≤ −1.50 and those recruited for the nonstunted control group ($n = 30$) had HAZs > −1.50. In addition, the 2 groups were selectively recruited to ensure group similarities in mean age and WHZ. Although it was not possible to match stunted and nonstunted children for parental height and weight, these values were measured in all families in which the biological parents lived with the children, and there were no significant differences between the groups in parental height or body mass index (BMI; in kg/m^2), as shown in **Table 1**.

Subjects apparently eligible for the study on the basis of weight, height, and the willingness to participate were given a screening examination that included a medical history, a physical examination, an evaluation of Tanner stage (23), blood collection for measurement of glucose and iron concentrations, and fecal and urine screening for acute infection. Children were excluded from the study if they were taking any medication, if Tanner staging for more than one criterion was >1, or if any past medical problems were identified that might influence current health status or metabolic condition (eg, hyperthyroidism or chronic anemia). Any child with acute health problems, such as intestinal or urinary tract infections, was treated according to the usual procedures of the São Paulo Hospital and began the study only after successful completion of treatment.

Ethical approval for the study was obtained from the Federal University of São Paulo Hospital and Human Investigations Review Board at the New England Medical Center, Tufts University. Written, informed consent was obtained from the children and their parents before the start of the study.

Protocol

The study consisted of a 7-d period of field measurements and a 3-d inpatient study. The children were studied in groups of 4 during the 7-d field period, in which food intake and total energy expenditure (TEE) were measured within the shantytowns. Each child was weighed in light clothing and stocking feet by using a portable electronic scale (model SD-150; Country Technologies, Gays Mills, WI) on the day before the doubly labeled water measurement of TEE began (to estimate isotope dose requirements). Resting energy expenditure (REE) and body composition were measured during the 3-d inpatient period in the metabolic research unit of the Federal University of São Paulo. Each child was brought by car to the metabolic research unit on the morning of inpatient study day 1, and REE was measured after an ≈12-h fast on 3 occasions as described below.

Measurement of total energy expenditure

TEE was measured under free-living conditions over the 7-d field period by using the doubly labeled water technique in 53 of the 58 subjects. An oral mixed and diluted dose of 0.20 ^{18}O g/kg body wt and 0.08 g ^{2}H/kg body wt was given to each child after measurement of body weight on outpatient study day 1 and a baseline (predose) urine specimen was collected. Subsequent urine samples were collected 2, 3, 4, and 5 h postdose. In addition, urine from the second void of the day (when possible; a later sample when not possible) was also collected by the investigators on outpatient study days 2, 3, 6, 7, and 8. All samples were dispensed as aliquots into freezer tubes and kept refrigerated until frozen at −20°C at the end of each study day. Frozen aliquot tubes were sent by courier to MRC Human Nutrition Research,

TABLE 2
Dilution spaces (*N*) and disappearance rates (*K*) for ^{2}H (d) and ^{18}O (o), carbon dioxide production rate, and food quotient in nonstunted and stunted children[1]

	Nonstunted		Stunted	
	Boys (n = 14)	Girls (n = 14)	Boys (n = 13)	Girls (n = 12)
N_d (mol)[2]	1063 ± 46	982 ± 48	924 ± 38	885 ± 46
N_o (mol)[2]	1033 ± 45	955 ± 47	897 ± 37	860 ± 45
K_d (1/d)	0.1359 ± 0.0059	0.1233 ± 0.063	0.1314 ± 0.0079	0.1212 ± 0.0094
K_o (1/d)	0.1775 ± 0.0062	0.1630 ± 0.0128	0.1751 ± 0.0086	0.1610 ± 0.0109
Carbon dioxide production (mol/d)[3]	17.4 ± 0.7	15.5 ± 0.8	16.1 ± 0.5	14.1 ± 0.9
Food quotient	0.89 ± 0.00	0.87 ± 0.00	0.89 ± 0.01	0.88 ± 0.00

[1] $\bar{x}$ ± SEM.
[2] Significant effect of group, $P < 0.05$.
[3] Significant effect of sex, $P < 0.05$.

Cambridge, United Kingdom. For the measurements of ^{2}H and ^{18}O enrichments in the urine samples and samples of dilute doses, a Sira 10 dual-inlet mass spectrometer modified to use the OS/2 OPTIMA or PRISM software by means of the PRISMUP system (Micromass, Wythenshaw, United Kingdom) was used.

For ^{2}H, the aqueous samples were equilibrated in duplicate with hydrogen gas by means of an interface with a MultiPrep automated sampling system based on the Gilson 222 XL liquid handler (Middleton, WI). This system provides for *1*) automated evacuation of vials containing aqueous samples and their filling with hydrogen gas, *2*) gas sampling after equilibration and the removal of water from equilibrated gas samples by means of a cryogenic probe, and *3*) temperature control of equilibrating samples to ±0.1 °C. For the equilibration, 0.4-mL samples were placed in disposable glass vials with rubber septa (50 × 12.5 mm, catalog number 471284; Labco Ltd, High Wycombe, United Kingdom) containing a reusable catalyst (platinum coated on a rod of hydrophobic porous resin; Finnigan MAT, Bremen, Germany) that promotes rapid exchange of ^{2}H between water and $^2H_2$ gas, and complete equilibration after 3 h. The equilibrations were performed at 25 °C with 3 mL hydrogen gas at 200 kPa. All the measurements were made relative to a sample of hydrogen gas similarly equilibrated with water of natural abundance for $^2H_2$ and were corrected for interference of tritium in the ratio measurements. The internal precision (SD) of the mass spectrometer measurements was <0.5‰. Laboratory standards calibrated with values of −51.45 and 763.24‰ relative to Vienna Standard Mean Ocean Water/Standard Light Arctic Precipitation (V-SMOW/SLAP) were run as unknowns and the true enrichment of the analyzed samples was calculated. Precision (SD) of duplicate samples was 2.1‰. After use, the catalyst rods were washed with deionized water, dried in air at 40 °C for ≥1 d, and stored at the same temperature.

For ^{18}O enrichments, single 3-mL samples were equilibrated with 13 mL CO_2 at 40.5 kPa and 25 ± 1 °C for 4 h in an Isoprep system (Micromass) as described by Wong et al (24) and measured relative to tank carbon dioxide and corrected for isotopic interferences according to Craig (25) and expressed relative to V-SMOW. The internal precision (SD) of the mass spectrometer measurement was <0.1‰. Sample analyses were done singly and the precision (SD) of the sample measurements was 0.47‰ calculated from replicate measurements of an enriched sample made with each batch of samples.

Carbon dioxide production rates were calculated from the doubly labeled water data by using a modification of the equation of Roberts et al (26), incorporating the recommended assumptions of a fixed ratio of dilution spaces (^2H_2O:$H_2{}^{18}O$) of 1.0342 (27) with a standard fractionation correction (28). TEE values were calculated from rates of carbon dioxide production by assuming estimates for the respiratory quotient to be equal to the food quotients determined from food records obtained during 6 d of the doubly labeled water study (29). For this component of the study, all foods and beverages consumed were weighed by field staff until 1700 every day; after that time, a literate member of the family made records that were checked for accuracy by the field staff the next morning, as described elsewhere (30). One subject was excluded from data analysis because of a physiologically impossible TEE value.

Energy expenditure for physical activity (EEPA) was calculated as EEPA = 0.9TEE − REE. This equation assumes that energy expenditure for postprandial thermogenesis is 10% of TEE. REE was also predicted from the standard equations used in current dietary recommendations (31), and TEE was predicted from World Health Organization (WHO) recommendations (32) and recommended dietary allowances (RDAs; 33) for energy intake. The energy requirement for growth was ignored in this comparison because mean weight gain at this age is only 7 g/d on average, which is equivalent to <2% of TEE, assuming an energy requirement for weight gain of 21 kJ/g (32).

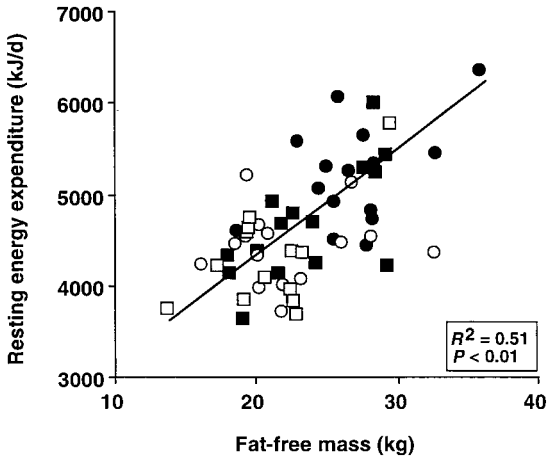

FIGURE 1. Relation between resting energy expenditure and fat-free mass in stunted (○, □) and nonstunted (●, ■) boys (○, ●) and girls (□, ■). n = 58.

TABLE 3
Energy expenditure in nonstunted and stunted children[1]

Energy expenditure measures	Nonstunted		Stunted	
	Boys (n = 15)	Girls (n = 15)	Boys (n = 15)	Girls (n = 13)
	kJ/d			
REE[2]	5206 ± 143	4659 ± 161	4432 ± 105	4298 ± 157
REE adjusted for weight[3]	4990 ± 121	4502 ± 118	4653 ± 121	4473 ±127
REE adjusted for fat-free mass	4906 ± 127	4630 ± 115	4584 ± 118	4513 ± 134
TEE[3]	9029 ± 368 [14][4]	8079 ± 401 [14]	8395 ± 270 [13]	7325 ± 479 [12]
TEE adjusted for weight[3]	8499 ± 283 [14]	7641 ± 280 [14]	8957 ± 294 [13]	7845 ± 304 [12]
EEPA[3]	2938 ± 297	2658 ± 236	3056 ± 241	2067 ± 323
EEPA adjusted for weight[3]	2645 ± 226	2416 ± 223	3376 ± 235	2400 ± 266

[1] $\bar{x}$ ± SEM. REE, resting energy expenditure; TEE, total energy expenditure; EEPA, energy expenditure of physical activity.
[2] Significant effects of group and sex, $P < 0.05$.
[3] Significant effect of sex, $P < 0.05$.
[4] n in brackets.

Measurement of resting energy expenditure

REE was measured as described previously (7). Briefly, after an ≈12 h overnight fast and a specific 20-min period during which subjects rested supine in bed, REE was measured on 3 occasions under thermoneutral conditions by indirect calorimetry using a DeltaTrac metabolic monitor (SensorMedics, Yorba Linda, CA). One of the measurements was made for 30 min and the other 2 were made for 15 min each. The mean CV for the repeat measurements of REE ranged from 2.8% for stunted girls to 4.0% for stunted boys (control boys and girls had intermediate values). Subjects were instructed to lie prone, relax, and avoid hyperventilating, fidgeting, and sleeping during the measurements. The calorimeter was calibrated by using a standard gas mixture (96% O_2 and 4% CO_2) before each measurement and alcohol burn tests were conducted every 2 wk to ensure the accuracy of the calorimeter (defined as values within the range 97–103% of predicted on each occasion). REE values were determined from values for oxygen consumption and carbon dioxide production on each measurement day (34) and the mean of the 3 d was used in data analysis.

Body-composition analysis

Height and weight were measured as described elsewhere (5). Fat mass and fat-free mass were measured by dual-energy X-ray absorptiometry with a Hologic X-ray densitometer (model QDR-4500A; Hologic Inc, Bedford, MA) with an adult quick-scan program shown to be accurate for this age and weight group (35).

Statistical analysis

Values are expressed as means ± SDs and means ± SEMs. Statistical analyses were performed by using SPSS 7.0 for WINDOWS and SYSTAT 7.0 for WINDOWS (SPSS Inc, Chicago). Differences between nonstunted and stunted boys and girls were tested by analysis of variance by using general linear models univariate analysis. Analysis of covariance was used to determine differences between nonstunted and stunted boys and girls when controlling for weight or fat-free mass. Stepwise forward multiple linear regression analysis was performed to determine the best predictors of TEE. Statistical significance was accepted at $P < 0.05$.

RESULTS

The physical characteristics of the subjects and their parents are summarized in Table 1. The nonstunted and stunted groups were not significantly different in age or WHZ. The control group was taller and had a greater HAZ and weight than did the stunted group, but body fat expressed as a percentage of weight was not significantly different between the groups. The parents' heights and BMIs did not differ significantly between groups.

The doubly labeled isotope data are summarized in **Table 2**. The stunted group had isotope-dilution spaces and mean rates of carbon dioxide production that were significantly smaller than those of the control group, but there were no significant differences in the rate constants for isotope disappearance between the 2 groups.

The best single predictor of REE was fat-free mass; the relation between REE and fat-free mass is shown in **Figure 1**. As shown in **Table 3**, there were significant associations of both stunting and sex with absolute values for REE. REE adjusted for weight was not significantly different between stunted and nonstunted children, although it was lower in girls than in boys. REE adjusted for fat-free mass was not significantly different

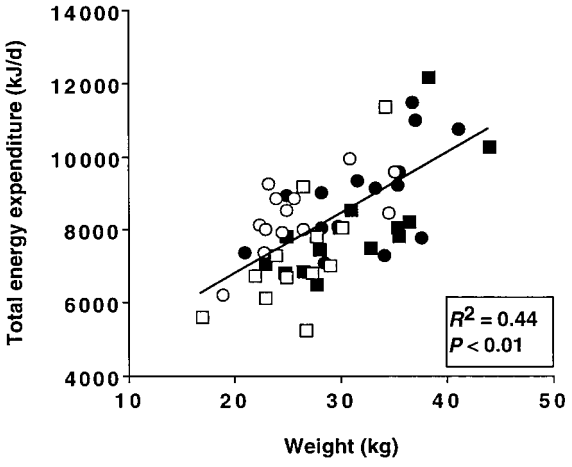

FIGURE 2. Relation between total energy expenditure and weight in stunted (○, □) and nonstunted (●, ■) boys (○, ●) and girls (□, ■). n = 53.

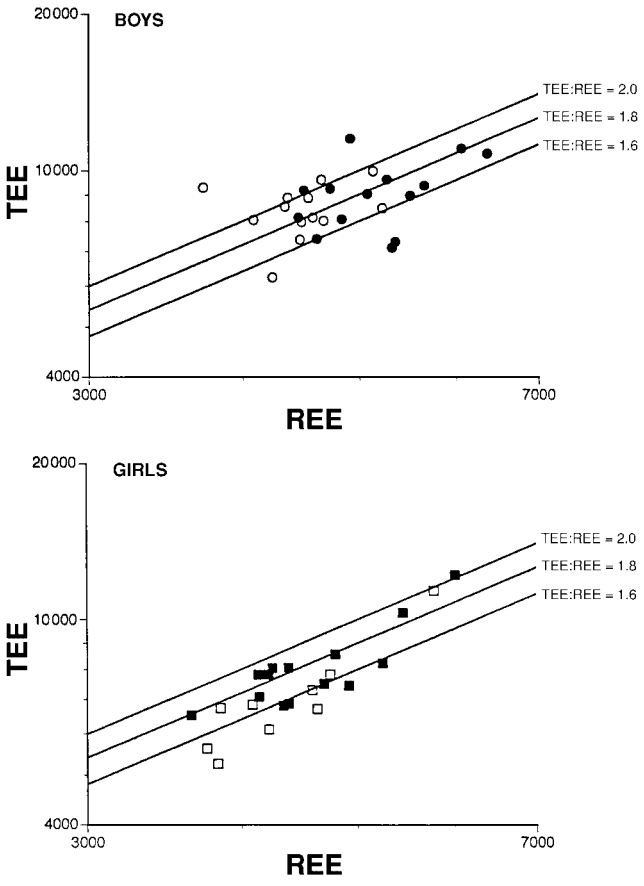

FIGURE 3. Relation between total energy expenditure (TEE) and resting energy expenditure (REE) in stunted (○, □) and nonstunted (●, ■) boys and girls. The ratio of TEE to REE at values of 1.6, 1.8, and 2.0 are indicated. Values for TEE and REE are plotted on natural log scales. n = 53.

between groups or sexes. EEPA and EEPA adjusted for weight were not significantly associated with either stunting or sex, as summarized in Table 3.

The best single predictor of TEE was weight (**Figure 2)**. Both unadjusted TEE and TEE adjusted for weight were significantly lower in girls than in boys, but there was no influence of stunting on either variable (Table 3). The relation between TEE and REE is shown in **Figure 3** and again illustrates the tendency of TEE to be lower in girls than in boys, in this case when expressed relative to REE.

Multiple regression models predicting TEE are summarized in **Table 4**. The best fitting models incorporated either fat-free mass, fat mass, and sex or weight and sex. A comparison of measured REE and TEE with values predicted from standard equations is shown in **Table 5**. There was not a significant effect of stunting or sex on the difference between measured and predicted REE (32). Measured TEE was compared with WHO (32) and US RDA (33) estimates of energy requirements. WHO (32) estimates of energy requirements significantly overpredicted TEE in nonstunted children, and RDA estimates of energy requirements significantly underpredicted TEE in stunted children.

DISCUSSION

The results of this study indicate that nutritional stunting, a factor previously reported to increase the risk of later obesity (5, 6, 36), is not associated with low TEE determined by using the doubly labeled water technique (37, 38). After adjustment for body weight or fat-free mass and fat mass, there was no significant difference in TEE between the stunted and nonstunted subjects. This finding of normal TEE in relation to body composition in stunted children relative to nonstunted control subjects from the same population is consistent with similar findings by Wren et al (20) and suggests that the reported high risk of later obesity in stunted adolescents and adults (5, 6, 36, 39) is not due to a long-term effect of stunting on energy requirements per se.

We did find, however, that the girls had significantly lower TEEs than did the boys, independent of body-composition differences between sexes, a finding that may help explain the greater apparent susceptibility to obesity of Brazilian shantytown girls than shantytown boys (5). The fact that TEE was lower in girls but REE was not suggested that the cause of low TEE was primarily reduced physical activity. Low energy expenditure (TEE in some studies and REE in others) predicted excess weight gain in some (14–16) but not in all (17, 18) prospective studies of factors influencing excess weight gain, and in one case predicted excess weight gain in boys but not in girls (19).

The reason for the discrepancy between the results of different previous studies is not known, but one possible explanation is that low energy expenditure may be a strongly predisposing factor for obesity only in persons with an underlying genetic susceptibility. For example, in populations such as Pima Indians living on reservations, who are almost all extremely obese and therefore by definition highly susceptible to obesity, low energy expenditure predicts obesity (14). Similarly, low energy expenditure predicted obesity in 2 groups of English infants of overweight parents who were recruited during a period when only a minority of families within the general community were overweight (15, 16, 40), suggesting that only particularly susceptible families were included in the study. However, in recent studies of infants of overweight parents in the United States, low energy expenditure was not found to be a measurable risk factor for obesity (17, 18), perhaps because the increasing prevalence of obesity since the early 1980s (41)

TABLE 4
Multiple regression models of the association of total energy expenditure with body composition and sex[1]

Model and parameters	Regression coefficient	SE	P	Overall R^2
Model 1			0.000	0.56
Constant	4505	1062	0.000	
Sex	−914	342	0.010	
Fat-free mass (kg)	183	40	0.000	
Fat mass (kg)	149	69	0.035	
Model 2			0.000	0.56
Constant	4742	828	0.000	
Sex	−985	277	0.001	
Weight (kg)	172	24	0.000	
Model 3			0.000	0.49
Constant	2611	822	0.003	
Fat-free mass (kg)	244	35	0.000	

[1] Boy = 1; girl = 2.

TABLE 5
Measured and predicted values for energy expenditure in nonstunted and stunted children[1]

	Nonstunted		Stunted	
	Boys (n = 15)	Girls (n = 15)	Boys (n = 15)	Girls (n = 13)
	kJ/d			
Measured REE[2]	5206 ± 143 [15]	4659 ± 161 [15]	4432 ± 105 [15]	4298 ± 157 [13]
Predicted REE[3,4]	5082 ± 119 [15]	4778 ± 81 [15]	4503 ± 101 [15]	4462 ± 72 [13]
Measured REE − predicted REE	124 ± 143 [15]	−118 ± 126 [15]	−72 ± 85 [15]	−127 ± 126 [13]
Measured TEE[5]	9029 ± 368 [14]	8079 ± 401 [14]	8395 ± 270 [13]	7325 ± 479 [12]
Predicted TEE				
by US RDA[3]	8509 ± 356 [14]	8706 ± 535 [14]	6853 ± 235 [13]	6741 ± 410 [12]
by WHO[2]	9571 ± 360 [14]	8575 ± 444 [14]	7984 ± 235 [13]	7177 ± 282 [12]
Measured TEE − US RDA TEE[2]	521 ± 336 [14]	−627 ± 358 [14]	1542 ± 240 [13]	584 ± 495 [12]
Measured TEE − WHO TEE[3]	−542 ± 351 [14]	−496 ± 298 [14]	410 ± 190 [13]	148 ± 419 [12]

[1] $\bar{x}$ ± SEM. Predicted resting energy expenditure (REE) is based on the prediction equations used by both the World Health Organization (WHO) and US recommended dietary allowances (RDA; 31), and total energy expenditure (TEE) is based on the RDA (33) and WHO (32) recommendations. n in brackets.
[2] Significant effect of sex and group, $P < 0.05$.
[3] Significant effect of group but not sex, $P < 0.05$. The US RDAs significantly underpredicted energy requirements in stunted children and WHO values overpredicted them in nonstunted children, $P < 0.05$.
[4] Significantly different from measured values in both groups and sexes, $P < 0.05$.
[5] Significant effect of sex but not group, $P < 0.05$.

resulted in recruitment of many families who were presumably obese for environmental rather than genetic reasons and therefore not especially susceptible to adverse effects of low energy expenditure. Further support for this suggestion is gained from the fact that very few of the infants born to overweight parents in the most recent study (18) actually became overweight during the initial 12-mo measurement period, suggesting that the infants lacked a strong underlying predisposition to obesity despite having overweight parents.

Note that measured REE values in our study population were similar to and not significantly different from values predicted from the standard equations developed for use in international recommendations on energy needs (32) and now used in national recommendations (33). Thus, equations developed in affluent populations to predict REE appear to accurately predict REE in stunted and nonstunted shantytown children. Considering all subjects together, values for TEE in our study also tended to be similar to international recommendations on energy requirements (32). However, US RDAs underpredicted energy expenditure in stunted children, and WHO estimates of energy needs overpredicted energy expenditure in nonstunted children. Thus, our results suggest that further research is needed to refine recommendations on energy requirements for urban children in developing countries, a population for which there has previously been little information.

An additional observation relating to recommendations on the energy needs of children in developing countries is that our finding of low TEE in these girls supports the general WHO approach of predicting lower energy values in girls than in boys. A recent study that directly compared the energy requirements of boys and girls in the United States found no sex effect (42), but the children in that study were younger than our subjects. In an analysis of 574 doubly labeled water measurements from multiple investigators, Black et al (29) observed that normalized values for TEE were 11% lower in women and girls than in men and boys across the life span, but the specific effect of the subject's sex during the prepubertal period was uncertain. These observations do not resolve the issue of whether the low energy expenditure of the prepubertal girls in our study was a natural consequence of sex-related differences in energy physiology or whether undesirable cultural factors were involved. We noticed that the girls in our study devoted less time to intensely vigorous games, such as soccer, than did the boys, but the underlying cause of this difference was not known. Nevertheless, in view of the possibility that low energy expenditure may increase the risk of obesity in susceptible individuals and that girls are at greater risk of becoming obese, particularly if they are stunted (5), further studies are needed to investigate the influence of sex on energy requirements and components of energy expenditure in different populations.

In conclusion, the results of this study suggest that there is no effect of nutritional stunting on energy expenditure after adjustment for body size, but that shantytown girls have lower energy expenditures than do shantytown boys. These observations, combined with previous work suggesting that low energy expenditure may be a risk factor for weight gain in susceptible populations, indicate that low energy expenditure may help to explain the increased risk of excess weight gain leading to obesity among shantytown girls compared with shantytown boys.

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