Longitudinal changes in energy expenditure in girls from late childhood through midadolescence\textsuperscript{1–3}

Jennifer L Spadano, Linda G Bandini, Aviva Must, Gerard E Dallal, and William H Dietz

ABSTRACT

Background: Longitudinal data on energy expenditure in children and adolescents are scarce.

Objective: The purpose of this study was to examine changes in energy expenditure and physical activity in girls from late childhood through midadolescence.

Design: We measured total energy expenditure (TEE) by doubly labeled water, resting metabolic rate (RMR) by indirect calorimetry, body composition by \textsuperscript{18}O dilution, and time spent in activity by an activity diary in 28 initially nonobese girls at \textasciitilde10, \textasciitilde12, and \textasciitilde15 y of age. Changes with age in TEE, RMR, and activity energy expenditure (AEE), both in absolute terms and in adjusted analyses, and in physical activity level (PAL) and time spent sleeping, being sedentary, and in moderate and vigorous activity were evaluated by mixed-model repeated-measures analyses.

Results: Absolute TEE and AEE increased significantly from age 10 to 15 y (\(P<0.0001\) for both). Absolute RMR at ages 12 and 15 y did not differ significantly, despite significant increases in fat-free mass and fat mass between the visits. PAL was significantly higher (\(P<0.0001\)) at age 15 y than at age 10 or 12 y, whereas time spent being sedentary increased significantly from age 10 to age 15 y (\(P<0.001\)), and AEE adjusted for fat-free mass appeared to decrease over the same interval.


KEY WORDS Energy expenditure, resting metabolic rate, physical activity, parental overweight, puberty, adolescents, female, obesity

INTRODUCTION

Most data on energy expenditure (EE) in children and adolescents are cross-sectional in nature. Among the few longitudinal EE studies that have been conducted in children (1–6), only one study (6) has, to our knowledge, published data on changes in total EE (TEE), resting metabolic rate (RMR), and activity EE (AEE) during adolescence. Because it is believed to be a critical period in the development of obesity (7), adolescence is an important time in which to study changes in the components of EE. Such study is particularly important for females because obesity in adolescence is more likely to persist into adulthood in girls than in boys (8).

Obesity results from a chronic state of positive energy balance, in which energy intake exceeds EE. A decline in the most variable component of EE, physical activity (9), may play a role in the increasing prevalence of childhood overweight (10). Longitudinal (11) and cross-sectional (12) questionnaire data have shown a decline in leisure time and vigorous physical activity, respectively, during adolescence in females. Cross-sectional accelerometry data from children in grades 1–12 showed an inverse relation between school grade and the number of minutes per day of moderate to vigorous physical activity (13). Among the 3 published studies with longitudinal measures of physical activity based on EE, 1 study presented data on changes in AEE adjusted for race and obesity status rather than changes in absolute AEE or AEE adjusted for weight or body composition (6). The other 2 studies followed children from age 5 y to age 10 y.

Although AEE directly reflects the energy spent in activity, the energy cost of many activities is influenced by body weight (14–16). Consequently, absolute AEE is not the most appropriate indicator of relative physical activity. Several different approaches have been advocated to correct AEE for differences in body size and composition (16–18). Physical activity level (PAL), AEE per kg of fat-free mass (FFM) or per kg of body weight, and AEE adjusted for FFM or weight in statistical models have all been used.

The purpose of the current study was to examine in 28 females the changes that occur from late childhood to midadolescence in TEE, RMR, and AEE, and in physical activity as assessed by AEE adjusted for FFM, PAL, and time spent in activity as recorded in an activity diary. Although the relatively small size of our study sample means that any findings should be evaluated

\textsuperscript{1} From the General Clinical Research Center, Massachusetts Institute of Technology, Cambridge, MA (JLS and LGB); the Gerald J and Dorothy R Friedman School of Nutrition Science and Policy (JLS, AM, and GED), and the Jean Mayer US Department of Agriculture Human Nutrition Research Center on Aging (AM and GED), Tufts University, Boston, MA; the Department of Health Sciences, Boston University, Boston, MA (LGB); the Eunice Kennedy Shriver Center, University of Massachusetts Medical School, Waltham, MA (LGB); the Department of Public Health and Family Medicine, Tufts University School of Medicine, Boston, MA (AM); and the Division of Nutrition and Physical Activity, Center for Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Atlanta, GA (WHD).

\textsuperscript{2} Supported by NIH grants DK-HD50537, MO1-RR-00088, MO1-RR-01066, and 5-PD30-DK46200.

\textsuperscript{3} Reprints not available. Address correspondence to J. Spadano, Jean Mayer USDA HNRCA at Tufts University, Dietary Assessment and Epidemiology Research Program, 711 Washington Street, Boston, MA 02111. E-mail: jennifer.spadano@tufts.edu.

Received July 20, 2004.

Accepted for publication January 10, 2005.
cautiously and explored further in larger longitudinal studies, these data provide a rare opportunity to examine possible changes in EE during adolescence.

SUBJECTS AND METHODS

Between September 1990 and June 1993, 196 girls aged 8–12 y were enrolled in the Massachusetts Institute of Technology (MIT) Growth and Development Study, a prospective cohort study with annual follow-up visits from study entry until after menarche. The criteria for enrollment were menarchean status and a triceps skinfold thickness <85th percentile for age and sex (19). Girls were recruited from the Cambridge and Somerville (Massachusetts) public school systems and the MIT summer day camp; other recruits were friends and siblings of enrollees. All subjects were initially healthy and were not taking any medications known to affect body composition or metabolic rate.

Subjects in the current study were a subgroup (EE subcohort) of the MIT Growth and Development Study. Girls who enrolled in the MIT Growth and Development Study during year 2 or 3 of recruitment and were ≈10 y of age at study entry were asked to participate in a substudy designed to examine longitudinal changes in EE; 28 girls from different families agreed to participate.

Measurements of TEE by doubly labeled water, RMR by indirect calorimetry, body composition by total body water (TBW), and time spent in activity as recorded in an activity diary were taken at the baseline (year 0), year 2, and year 5 visits when the girls were ≈10, ≈12, and ≈15 y of age, respectively. The year 2 and year 5 visits were scheduled with ±1 mo of the 2nd and 5th anniversary of the girl’s baseline visit, respectively. All 3 study visits were conducted during the school year. All 28 girls had a year 2 visit, and 24 of the 28 girls had a year 5 visit. Of the 4 girls missing data at year 5, 1 had moved out of the country, 1 dropped out of the study, and the remaining 2 could not schedule a visit before the end of the school year because of weekend extracurricular activities.

As part of the larger MIT Growth and Development Study cohort, the girls also had a 4th measure of RMR and TBW performed at their study completion visit, scheduled for 4 y (±1 mo) after menarche. Twenty-three of the 28 girls came in for this final visit. However, the study completion visit coincided with the year 5 visit for 1 girl and preceded the year 5 visit for another girl; only data from the year 5 visit of those 2 girls were included in these analyses. In addition, 1 girl was missing TBW data at study completion. Consequently, a 4th measure of RMR and TBW was available for only 20 of the 28 girls. Of the 5 girls without this study completion visit, 2 dropped out of the study before their year 5 visit, 2 dropped out of the study between their year 5 visit and their scheduled study completion visit, and 1 had moved and could not be located for her study completion visit (referred to below as the visit 4 y after menarche).

Written informed consent was obtained from both the subject and a parent or legal guardian (when subject was <18 y old) at each study visit. The study was approved by both the Committee on the Use of Humans as Experimental Subjects at MIT (Cambridge, MA) and the Institutional Review Board at the Tufts-New England Medical Center (Boston, MA).

Total energy expenditure and body composition

For all 4 study visits, subjects were admitted to the General Clinical Research Center (GCRC) at MIT in the late afternoon for an overnight stay. On the girl’s arrival, the study physician obtained a medical history and performed a brief medical examination to assess the girl’s health. At the baseline, year 2, and year 5 visits, a baseline urine sample was collected, and an overnight fast was initiated approximately 1 h before the administration of $^2$H$_2$, $^{18}$O. In the evening, between 1900 and 2000, a dose of 0.25 g H$_2$$^{18}$O and 0.1–0.12 g $^2$H$_2$O per kg of estimated TBW was administered to the study subject. Urine was collected until 0600 the next morning to determine urinary losses of isotope. The second urine void of the morning was used to measure $^{18}$O and $^2$H enrichment above the baseline values. This sample was used to determine TBW and served as the initial time point of the EE period (initial sample). Subjects returned to the GCRC as outpatients 2 wk after admission. At this time, the 2nd urine void of the day (endpoint sample) was collected to complete the EE period. Isotopic enrichments of the urine samples were measured on a Hydra Gas Isotope Ratio Mass Spectrometer (PDZ Europa Ltd, Northwich, United Kingdom) at the mass spectrometry laboratory at the Jean Mayer US Department of Agriculture Human Nutrition Research Center on Aging at Tufts University (Boston). Criteria for acceptance values were replicate-measures SEs of 0.35 for $^{18}$O and 1.5 for $^2$H.

We used a modification (20) of the equation of Lifson and McClintock (21) to calculate the mean daily rate of carbon dioxide production (mol CO$_2$/d), as follows:

$$rCO_2 = \frac{(N/2.078)(1.01 k_o - 1.04 k_i) - 0.0246 r_{Gf}}{1}$$

where $N$ is TBW in mol, $k_o$ is the $^{18}$O elimination rate, $k_i$ is the $^2$H elimination rate, and $r_{Gf}$ is the estimated rate for isotopically fractionated water loss, equal to 1.05$N/(1.01 k_o - 1.04 k_i)$.

The elimination rates of the $^{18}$O and $^2$H isotopes were calculated according to the 2-point method using the difference in the atom percent excess (APE) of the initial ($i$) and endpoint ($f$) samples and the time between their urine collections, as follows:

$$k = (\ln APE_f - \ln APE_i)/\text{time}$$

where APE is the isotopic enrichment of a sample relative to that of the predose (baseline) sample. TEE was calculated by using Weir’s equation (22). Oxygen consumption was obtained by dividing rCO$_2$ by the food quotient (23) derived from a 7-d food diary that subjects collected during the 2nd wk of the EE period, as described elsewhere (24). For the 2 girls at baseline and the 3 girls at year 5 who lacked valid food diaries, we used the average food quotient for the entire prospective cohort at baseline (as reported previously in 25) and the average food quotient for the subcohort at year 5, respectively.

At the baseline visit, at years 2 and 5, and 4 y after menarche, body composition was estimated from TBW by using $^{18}$O dilution space. The dose of $^{18}$O administered at the visit 4 y after menarche was 0.07 g H$_2$$^{18}$O/kg estimated TBW, which followed the doubly labeled water protocol described earlier from the collection of the baseline urine sample to the collection of the 2nd urine void of the morning after the overnight stay. The oxygen dilution space was calculated according to the method of Halliday and Miller (26) and was assumed to be 1% higher than TBW. FFM was estimated from TBW by assuming a hydration constant of 0.73. Fat mass (FM) was calculated as the difference between body weight and FFM, and percentage body fat was calculated by dividing FM by body weight and multiplying by 100.
Resting metabolic rate

RMR was measured in the morning by using an indirect calorimeter with customized software and fitted with a ventilated hood, as described previously (25, 27). Each subject fasted overnight for a minimum of 12 h and engaged in minimal activity before the determination of metabolic rate. A 30-min rest and a 5-min equilibration period preceded the 30-min measurement period. On the morning of each measurement, the linearity of the gas analyzers was confirmed by calibrating the analyzers against 2 standard gases and checking the concentration of a third standard gas. In addition, the study technician checked the calibration of the entire system before each scheduled visit by pushing known amounts of a standard gas through the hood at a constant rate with a 3-L calibrated syringe (Warren E Collins Co, Braintree, MA). RMR was calculated from measures of oxygen consumption and carbon dioxide production according to modified Weir’s equation (22). At the baseline, year 2, and year 5 visits, RMR was calculated from measures of oxygen consumption and carbon dioxide production according to modified Weir’s equation (22). At the baseline, year 2, and year 5 visits, RMR was measured on the morning after subjects were admitted to the GCRC and ~2 wk later when they returned to end their EE period. The average of the 2 RMR values at each time point was used. The intraclass correlation of the 2 RMR measures at baseline for the entire prospective cohort was 0.96, which indicated that the measurements were highly reproducible (25). Consequently, RMR at the visit 4 y after menarche was based on a single measurement made the morning after the overnight stay.

Measures of physical activity

AEE was calculated as \((0.9 \times \text{TEE}) - \text{RMR}\), assuming that 10% of TEE is food-induced thermogenesis (9). PAL was calculated as TEE/RMR.

Subjects kept an activity diary during the 1st week of the EE period for the baseline, year 2, and year 5 visits. The diary was set up as a grid with the rows representing each hour of the day and the columns indicating sleep, sit, stand, walk, and play. The girls documented when they awoke and when they went to sleep. For each 1-h time block in between, the girls were instructed to place an X in the column(s) that best described their activity during that hour, recording up to 2 activities/h. In accordance with the baseline coding protocol for the larger prospective cohort, the time attributed to each activity within a given hour was based on the number of X markings within a row (eg, 1.0 h for 1 activity checked, 0.5 h each for 2 activities checked). When a 1-h time block between awakening in the morning and going to sleep at night was left blank, the activities sit, stand, walk, and play were each assigned 0.25 h. The time spent sleeping, sitting, standing, walking, and playing for each day was then calculated. The school days and the weekend days were averaged separately and weighted accordingly to derive an average number of hours per day in each activity. For a day to be included in either the school day or weekend day average, only 2 h of missing data was allowed. Only activity diaries with at least 4 school days and 1 weekend day of data were considered valid. Valid activity diaries were missing for 1 girl at baseline and 7 girls at year 5 (3 girls did not return their activity diaries, and 4 girls did not have a year 5 visit). The time spent sitting and standing was summed to provide a measure of sedentary time. Time spent walking was considered moderate activity, and time spent playing was considered vigorous activity. The time spent in moderate and vigorous activity were summed to provide nonsedentary time.

Other variables

Weight was measured in the morning, while subjects were in a fasted state, by using a digital scale (Seca, Hamburg, Germany) that was accurate to 0.1 kg. Height (without shoes) was also measured at this time by using a wall-mounted stadiometer that was accurate to 0.1 cm. We used the Centers for Disease Control and Prevention 2000 growth charts (28) to calculate percentiles of body mass index (BMI; in kg/m²) for age based on each girl’s measured height and weight at each visit. Race or ethnicity (ie, white, black, Hispanic, Asian, and other) was based on self-report on a questionnaire completed at study entry; 19 girls (68%) identified themselves as white, 5 as black, 1 as Hispanic, 2 as Asian, and 1 as other. For all analyses, race or ethnicity was further categorized into a dichotomous variable (black or nonblack).

Tanner staging (29) of breast development was assessed by either a study physician or a female coinvestigator at each visit up until menarche. The girls were instructed to call the study personnel when they had their first period. Some girls reported their date of menarche during one of their annual follow-up visits. At these visits, girls were asked if they had started their period during the preceding year; if the answer was yes, the girl was asked to recall the date.

Early in the study, the heights and weights of the biological parents of each girl were collected either by self-report or by measurements taken at MIT (in normal clothing, without shoes). Only 3 girls were missing data necessary to classify parental weight status. Among the remaining 25 girls, only 1 had self-reported rather than measured data. Parental overweight was defined as a BMI ≥ 25 (30). Girls were classified as having 2 normal-weight biological parents (NWP) or at least 1 overweight biological parent (OWP).

Statistical analysis

All statistical analyses were performed by using SAS software (version 8.1; SAS Institute, Cary, NC). Mean (±SD) age, height, weight, BMI-for-age percentile, percentage body fat, FM, and FFM were calculated for each visit. Mixed-model repeated-measures analyses (using PROC MIXED in SAS) were performed to evaluate the changes with age in TEE, RMR, and AEE both before and after adjustment for key covariates; covariates considered for inclusion in each model were race, parental overweight, pubertal status, FFM, and FM (for RMR only). Because of our earlier observation of a higher absolute RMR at menarche (± 6 mo) than at 4 y after menarche (31), we assessed the influence of puberty on EE, with pubertal status expressed in terms of the timing of each visit relative to menarche. For each visit, a girl’s pubertal status was retrospectively classified as >1 y before menarche, 1 y before menarche to 6 mo after menarche, or >6 mo after menarche. Because the exact timing and duration of the proposed elevation in RMR are unclear, we evaluated 2 additional pubertal status variables: \(1) >1 \text{ y before menarche}, \text{within 1 y of menarche, and } >1 \text{ y after menarche}\) and \(2) >1.5 \text{ y before menarche, 1.5 y before menarche to 6 mo after menarche, and } >6 \text{ mo after menarche}\). The last of the 3 proposed pubertal status variables performed better overall in cross-sectional analyses as assessed by a comparison of each model’s adjusted \(r^2\) value and the \(P\) value of its pubertal status variable. Hence, we selected this variable for consideration in the mixed models. We
also alternatively evaluated time until menarche as a continuous, time-varying covariate.

Because RMR adjusted for body composition appears to be lower in black than in white girls (2, 25, 32–34), we evaluated a race × visit interaction term to assess any racial differences in the changes that occur in TEE and its components with age. In addition, we tested a parental overweight × visit interaction term in all adjusted analyses because we previously observed in another subgroup of girls a significant parental overweight × visit interaction in RMR evaluated before menarche (baseline), at menarche, and 4 y after menarche (31). We also determined the significance of changes with age in absolute FFM, FM, PAL, and time spent sleeping, being sedentary, and in moderate and vigorous activity. Time was modeled as a categorical variable (ie, visit) in all analyses to allow comparisons between the visits. Final models in which time was included as either a categorical or continuous variable were compared by using the maximum log-likelihood ratio test to determine whether the data were consistent with linearity. The covariance structure was retested after each change to a given model by using the log-likelihood ratio test for nested models and Akaike’s information criterion (35) for nonnested models. Tukey’s honestly significant differences were used to evaluate the differences across age. Results were considered significant if the observed P value was < 0.05.

RESULTS

At baseline, 19 (68%) of the 28 girls were classified as Tanner stage 1, and the remaining 9 girls were pubertal, although nonmenarcheal. Mean age, height, weight, BMI-for-age percentile, percentage body fat, FM, and FFM at each visit are shown in Table 1. Significant increases in both FFM and FM were observed from ≈10 to ≈12 to ≈15 y of age; FFM increased from 25.3 to 32.3 to 42.0 kg, and FM increased from 8.4 to 13.1 to 16.4 kg, respectively (P < 0.0001 for FFM and P < 0.001 for FM for the differences between visits). Between the year 5 visit and the visit 4 y after menarche (≈15 y), mean FFM increased significantly (P < 0.01), by ≈1.4 kg, and mean FM appeared to increase by ≈0.8 kg (P = 0.32). Mean (±SD) age at menarche for this subcohort was 12.5 ± 0.9 y.

Of the 25 girls with data on parental weight status, 10 had 2 NWP (NWP girls), and 15 had at least 1 OWP (OWP girls). Among the 3 girls missing data on parental overweight, 1 was white and 2 were black. One NWP girl who dropped out of the study before she experienced menarche was missing data on menarcheal age and was not included in models considering pubertal status.

Mean absolute TEE increased significantly at each age, rising from 8176 to 9355 to 10 364 kJ/d at ≈10, ≈12, and ≈15 y, respectively (Figure 1). Overall, FFM (P < 0.0001; direct association), race (P < 0.0001; lower in blacks), and pubertal status (P < 0.001; inverse association) were significant predictors of TEE, and therefore they were included in the final model. In addition, the parental overweight × visit interaction term was significant (P < 0.001). The adjusted means of TEE for each parental weight group at each visit are shown in Figure 2. Although TEE adjusted for FFM, race, and pubertal status appeared to increase with age in the NWP girls and to decrease with age in the OWP girls, within each parental weight group, the differences in adjusted TEE between the visits were not significant. The results did not differ significantly between the models in which time was a categorical or continuous variable.

![Figure 1](https://academic.oup.com/ajcn/article-abstract/81/5/1102/4649583)

**Figure 1.** Absolute total energy expenditure of 28 girls at baseline, year 2, and year 5, corresponding to ≈10, ≈12, and ≈15 y of age, respectively. Significance was assessed by mixed-model repeated-measures analysis with Tukey’s honestly significant differences used in the comparisons of the means. Bars with different superscript letters are significantly different from one another (P < 0.0001). Error bars represent the 95% CI (1), n = 24 at year 5.
Adjusted \( \bar{x} \) total energy expenditure of 9 girls with 2 normal-weight parents (NWP) and 15 girls with \( \geq 1 \) overweight parent (OWP) at baseline, year 2, and year 5, corresponding to \( \approx 10, \approx 12, \) and \( \approx 15 \) y of age, respectively. Adjusted means of a significant parental overweight \( \times \) visit interaction term are presented from a mixed-model repeated-measures analysis containing fat-free mass, race (black or nonblack), pubertal status (> 1.5 y before menarche, 1.5 y before menarche to 6 mo after menarche, or 6 mo after menarche), parental overweight (NWP or OWP), and visit (\( P < 0.001 \)). Tukey’s honestly significant differences were used in the comparisons of the means. Bars with different superscript letters represent the 95% CI. No significant differences in adjusted TEE were observed between visits. Error bars represent the 95% CI. \( n = 13 \) in the OWP group at year 5.

Mean absolute RMR was significantly lower at age \( \approx 10 \) y (5226 kJ/d) than at age \( \approx 12 \) y (5929 kJ/d), age \( \approx 15 \) y (5853 kJ/d), and at 4 y after menarche (\( \bar{x} \) age: 16.6 y; 5820 kJ/d) (Figure 3). Although the differences in mean RMR between the visits at year 2, year 5, and 4 y after menarche were not significant, the observation that mean absolute RMR at year 2 was 76 kJ/d higher than at year 5, despite the average accumulation of an additional 9.7 kg FFM and 3.3 kg FM between the 2 visits, is unexpected. Overall, FFM (\( P < 0.0001 \); direct association) and race (\( P < 0.01 \); lower in blacks) were significant predictors of RMR, whereas FM was marginally significant (\( P = 0.05 \)); all 3 variables were included in the final model. In addition, the parental overweight \( \times \) visit interaction term was significant (\( P < 0.001 \)). The adjusted means of RMR for each parental weight group at each visit are shown in Figure 4. Among the NWP girls, mean adjusted RMR was 5766, 5983, 5054, and 4674 kJ/d at \( \approx 10, \approx 12, \approx 15 \), and 16.6 (\( \bar{x} \)) y of age, respectively. Mean adjusted RMR was significantly higher at year 2 than at year 5 (\( P < 0.001 \)) and at 4 y after menarche (\( P < 0.0001 \)), although the increase of 218 kJ/d from baseline to year 2 was not significant. Adjusted RMR at baseline was also significantly higher than at 4 y after menarche (\( P < 0.01 \)), but it did not differ significantly from that at year 5 (\( P = 0.09 \)). In the OWP girls, mean adjusted RMR at baseline (6171 kJ/d) and year 2 (5870 kJ/d) did not differ significantly (\( P = 0.47 \)), yet both were significantly higher (\( P < 0.0001 \) for each) than adjusted RMR at year 5 (4745 kJ/d) and at 4 y after menarche (4682 kJ/d).

Absolute AEE increased significantly from 2134 to 2489 to 3502 kJ/d from \( \approx 10 \) to \( \approx 12 \) to \( \approx 15 \) y of age, respectively (\( P < 0.05 \) for all) (Figure 5). Overall, FFM (\( P < 0.0001 \); direct association), race (\( P = 0.04 \); lower in blacks), and pubertal status (\( P = 0.02 \); inverse association) were significant predictors of AEE and therefore are included in the final model. Visit was not significant (\( P = 0.51 \)) in the model that included pubertal status; mean adjusted AEE was 2548, 2402, and 2607 kJ/d at \( \approx 10, \approx 12, \) and \( \approx 15 \) y of age. The pattern of change in adjusted AEE with age...
Absolute PAL was significantly higher ($P < 0.0001$) at $\approx 15$ y of age (1.77) than at $\approx 10$ and $\approx 12$ y of age (both 1.57; Figure 6). The pattern of change in PAL across the 3 visits did not differ significantly between the NWP and OWP girls. Overall, FFM was a significant predictor of PAL ($\beta = 0.013$, $P = 0.004$), as was weight when evaluated in separate models ($\beta = 0.006$, $P = 0.04$).

Mean values by age for the time spent sleeping, being sedentary, and in moderate and vigorous activity, taken from the activity diary, are shown in Figure 7. On average, time spent sleeping declined significantly, from 10.8 h/d at $\approx 10$ y of age to 9.7 h/d by $\approx 15$ y of age ($P < 0.0001$). Sedentary time increased significantly, by $\approx 2$ h/d, from $\approx 10$ to $\approx 15$ y of age ($P < 0.001$). Time spent in moderate and in vigorous activity appeared to decline over the same interval, although the differences were smaller and not significant. When the hours spent in moderate and in vigorous activity were summed to reflect nonsedentary time, the observed differences by age still were not significant. Mean nonsedentary time was 3.7, 3.3, and 3.0 h/d at $\approx 10$, $\approx 12$, and $\approx 15$ y of age, respectively ($P = 0.15$ for time modeled as categorical and $P = 0.06$ for time modeled as continuous). For each of the activity diary variables, the results with categorical time (ie, visit) did not differ significantly from those with time modeled as a continuous variable.

**DISCUSSION**

Our study measured longitudinal changes in EE during adolescence, a period that is believed to be critical in the development of obesity. Longitudinal studies of EE are rare, particularly in adolescents, because of the high costs associated with repeated measures of EE and the challenge of retaining adolescents in longitudinal studies. Therefore, our data are unique. However,
our findings must be viewed cautiously because of the sample size and the complexity of our adjusted analyses.

Only a few longitudinal studies have published data on changes in TEE, RMR, or AEE (or all) in children (1–6, 31). One study was restricted to boys (4). Two other studies looked exclusively at changes in metabolic rate: Sun et al (2) found an inverse relation between Tanner stage and adjusted RMR, whereas in a previous study, we (31) found results consistent with the current study. In a study whose results were also consistent with the current findings, mean TEE adjusted for FFM in a study of 8 girls did not differ significantly at $\approx 10.4$ and $\approx 12.8$ y of age (3). Two-year follow-up data from the Baton Rouge Children’s Study on changes in TEE, RMR, and AEE adjusted for race and obesity status were presented (6). Changes in absolute AEE or EE adjusted for changes in body size or body composition were not reported. A study of Pima Indian children found that mean absolute TEE and AEE increased by 60% and 150%, respectively, between ages 5 and 10 y (5). In contrast, in a study by Goran et al (1), mean absolute TEE increased in 11 girls from age 5.5 to 6.5 y and then declined significantly, by a mean of 866 kJ/d, by age 9.5 y. This decline was attributed to a 50% reduction in AEE, which was hypothesized to be an energy-conserving mechanism in girls just before puberty (1). In the current study, however, we did not observe a decline in either absolute TEE or AEE with age; nor do our results suggest a decline in AEE just before puberty. Mean absolute AEE increased from 2276 to 2481 kJ/d from baseline to year 2 in the 16 girls who became pubertal during this period.

Except for our earlier publication on RMR (31), none of the aforementioned studies on longitudinal EE, to our knowledge, considered the potential influence of parental overweight. In the current study, we found that changes with age in adjusted RMR and TEE, but not AEE, differed according to parental weight status. These findings suggest that the observed influence of parental overweight on TEE is driven by genetic influences on RMR. However, because of the complexity of the adjusted TEE and RMR analyses in the small sample size in our study, these findings regarding parental overweight should be viewed as hypothesis-generating observations that require confirmation in other study populations.

We found that mean absolute RMR at $\approx 12$ y of age did not differ significantly from mean RMR at $\approx 15$ y of age, despite significant increases during the interim in both FFM, the major determinant of RMR (36), and FM, an independent contributor to RMR (25, 34, 37, 38). This observation is consistent with our earlier findings in 44 girls of a significantly higher mean absolute RMR at menarche ($\pm 6$ mo) than at 4 y after menarche (31). Among the girls in the current study, one-third were within $\pm 6$ mo of menarche, and all but 2 were pubertal but nonmenarchal; 7 girls were excluded in both studies. Therefore, the findings of the current study coupled with those published earlier (31) suggest that the observed elevation in RMR is not specific to menarche but most likely begins in midpuberty and persists through menarche. The lack of significance of the pubertal status variable in the adjusted RMR model may reflect the sparseness of RMR measures around menarche as well as the study’s limited power. Plausible mechanisms for the proposed elevation in RMR are discussed elsewhere (31).

We assessed age-related changes in physical activity by using PAL, AEE adjusted for FFM, and the time spent in activity as recorded in an activity diary. The observed increase in PAL suggests an increase in physical activity in midadolescence. In contrast, the changes with age in AEE adjusted for FFM and in sedentary time both suggest that the girls in our study became less active with age. Westerterp (39) showed that the fraction of the day spent in activities of moderate intensity significantly predicts PAL, whereas no relation was found between PAL and the time spent in high-intensity activity, presumably because of its relatively short duration. If this observation in adults is broadly applicable, then TEE and PAL likely are more influenced by the interaction between the relative proportions of time spent in low- and moderate-intensity activity than by the time spent in vigorous activity (39). Therefore, the shift that we observed in the proportions of time spent sleeping (a decrease) and being sedentary (an increase) might explain the increased PAL at age 15 y because there is a higher energy cost associated with being sedentary (metabolic equivalent [MET]: 1.1–1.9 (40, 41)) than with sleeping (MET: $\approx 0.9$). In that scenario, however, one would expect adjusted AEE to rise along with PAL, but that is contrary to our findings.

In a meta-analysis of data from 17 doubly labeled water studies conducted in children, Hoos et al (42) attributed the age-related increases in PAL to increases in body weight. Their conclusion was based on their findings of a positive association between age and PAL and of no association between age and AEE/kg body wt. A similar conclusion was reached by Ekelund et al (18) on the basis of their cross-sectional findings that PAL and absolute AEE were significantly higher and that AEE/kg FFM and body movement, as measured with an accelerometer, were significantly lower in adolescents than in children. Our previous findings of a positive influence of body weight on the MET values of walking (43) indirectly support the notion that body weight influences PAL, because both PAL and MET share the assumption that dividing EE by RMR removes the influence of weight. In addition, in separate models estimated in the current study, both weight and FFM were significantly related to the changes in PAL with age.

In addition to the small sample size, several potential limitations of our study are noteworthy. First, because our sample was predominantly white and middle-class, the age-related changes we observed in TEE and physical activity may not be generalizable to other groups. Second, we assumed that the pattern of change in the various components of EE did not differ between black and nonblack girls. Although we found no race $\times$ visit interaction for adjusted TEE, AEE, or RMR, our sample provided limited power. Third, we obtained only a single measure of TEE at each age. A single measure may not represent habitual EE if the doubly labeled water measurement is performed during a relatively low or high period of activity (44). The within-subject variation in doubly labeled water measurements attributed to analytic and inherent biological variation is estimated at 8% (44). Fourth, we cannot rule out the possibility that qualitative changes in reporting accuracy may influence the age-related changes observed in time spent in activity. Children’s accuracy in self-reporting activity may improve with age (45) or, alternatively, may decline with age if children no longer seek help from their parents or become less enthusiastic and hence less diligent in their recording as adolescents.

In conclusion, our data show a discrepancy in age-related changes in physical activity between PAL and both AEE adjusted for FFM and time spent in activity. PAL may be influenced by body weight. Until the most valid measure of age-related changes in physical activity is identified, the role that physical activity plays in the development of childhood obesity will remain uncertain.
LONGITUDINAL CHANGES IN ENERGY EXPENDITURE

1109

The authors gratefully acknowledge the girls who participated in this study and the staff at the General Clinical Research Center at the Massachusetts Institute of Technology for their assistance.

In addition to making intellectual contributions to the manuscript, LGB and WHD designed the study and collected data, AM and GED provided statistical advice, and JLS collected data, performed the oxygen isotope analyses and the statistical analyses, and wrote the manuscript. None of the authors had any personal or financial conflicts of interest.

REFERENCES