

A longitudinal study of resting energy expenditure relative to body composition during puberty in African American and white children¹⁻³

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See corresponding editorial on page 149.

ABSTRACT

Background: Body composition and resting energy expenditure (REE) have not been examined longitudinally during puberty.

Objective: The purpose of this longitudinal study was to examine the influence of pubertal maturation on REE relative to body composition in African American and white children.

Design: The study included 92 white and 64 African American children (mean age at baseline: 8.3 and 7.9 y, respectively) from Birmingham, AL. The children had 2–5 annual measurements of fat mass (FM), lean mass (LM), and REE. The Tanner stages of the children ranged from 1 to 5. Mixed-model repeated-measures analyses were used to test the change in REE relative to body composition with increasing Tanner stage among ethnic and sex groups.

Results: LM increased from Tanner stage 1 to subsequent stages. FM relative to LM decreased from Tanner stage 1 to stages 3, 4, and 5 but not from stage 1 to stage 2. The African American children had relatively higher limb LM and lower trunk LM than did the white children. REE declined with Tanner stage after adjustment for ethnicity, sex, FM, and LM. This decline was significant from Tanner stage 1 to stages 3, 4, and 5 but not to Tanner stage 2. After adjustment for age, Tanner stage, FM, and LM or LM distribution, REE was significantly higher in white than in African American children (by ≈ 250 kJ/d).

Conclusion: In a large sample of children at various Tanner stages, we found an ethnic difference in REE after adjustment for age, Tanner stage, FM, and LM that was not explained by the difference in LM distribution. *Am J Clin Nutr* 2001;73:308–15.

KEY WORDS Body composition, resting energy expenditure, lean mass distribution, puberty, African American children, white children, Tanner stage

INTRODUCTION

Puberty is a period of development that is marked by maturation of secondary sexual characteristics and dramatic changes in hormone concentrations, body composition, and energy partitioning (1). Growth patterns change from a stable and linear pattern in childhood to a pattern of accelerating velocity in puberty (2, 3). Because of the dynamic changes in body composition (4), energy metabolism may also change during pubertal maturation. Absolute basal and resting energy expenditure (REE) increase during puber-

tal development because of the increase in both fat mass (FM) and lean mass (LM) (5). However, it is not known how REE changes with pubertal maturation and how it differs by ethnicity and sex relative to changes in body composition. Morrison et al (6) showed a negative effect of maturation stage on REE after adjustment for body size in early pubertal girls. In contrast, the results of other cross-sectional studies (4, 7) do not support this conclusion.

One explanation for the effect of maturation on REE is the different metabolic contributions of organ and muscle mass during pubertal growth. It was shown that, in an average man, organ mass (brain, liver, heart, and kidney) accounts for two-thirds of metabolic energy, although total organ mass is <6% of body weight (8). The remaining REE is accounted for mainly by muscle, which comprises 40–50% of total body weight. The metabolic rate of the organs is 15–25 times that of muscle. The contribution of organs to REE (60%) is 2.4 times higher than that of muscle (20–25%) per unit mass (8, 9). A recent study concluded that organ size contributes significantly to REE in young adults (10).

African Americans are reported to have a significantly higher total bone mineral-free limb lean mass (LLM) than do whites after adjustment for height, body weight, age, and sex (11). In addition, we showed recently that the ethnic difference in REE disappeared when we adjusted for trunk lean mass (TLM) but not for LLM (12). Thus, we hypothesized that African Americans have a higher bone mineral-free muscle mass and a lower organ mass relative to weight, a distribution that may contribute to a lower REE than in whites.

Our earlier studies in prepubertal children did not show an ethnic difference in REE after adjustment for body composition and

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hormonal indexes of maturation (13, 14). Because African American children begin puberty earlier than do white children (6, 15, 16), the influence of maturation on body composition and REE may be apparent at a younger age in African Americans. To our knowledge, no longitudinal studies have examined changes in body composition and REE in African American and white boys and girls. Therefore, the first objective of this study was to examine changes in FM, LM, and REE with Tanner stage in African American and white boys and girls. The second objective was to examine the ethnic difference in REE after adjustment for body composition and maturation. The third objective was to determine whether there was a difference in LM distribution in African American children compared with white children and whether this difference explained any observed ethnic difference in REE. The hypotheses were: 1) LM increases during maturation but REE relative to body composition decreases; 2) African American children have a lower REE after adjustment for pubertal stage and body composition than do white children; 3) African American children have a relatively higher LLM and lower TLM than do white children, which explains the lower REE; and 4) REE does not differ by ethnicity after adjustment for the distribution of LM.

SUBJECTS AND METHODS

The sample comprised 92 white children (28 boys and 64 girls; mean age at baseline: 8.3 y) and 64 African American children (26 boys and 38 girls; mean age at baseline: 7.9 y) from Birmingham, AL. The children participated in a longitudinal study of how intraabdominal fat influences disease risk factors in children. The children were recruited through newspaper advertisements and by word of mouth. Ethnicity was defined on the basis of the self-ascribed ethnicity status of the children's parents and grandparents. The eligibility criteria were 1) age >4 y, 2) absence of medications known to affect body composition or physical activity (eg, prednisone, methylphenidate, and growth hormone), 3) absence of previous diagnosis of diseases known to affect body composition, fat distribution, or both (eg, Cushing syndrome, Down syndrome, type 1 diabetes, and hypothyroidism), and 4) absence of diagnosis of any major illnesses since birth. We reported prepubertal energy expenditure (13), body composition (17), and aerobic fitness (18) data for these children previously.

The nature, purpose, and possible risks of the study were explained carefully to the parents before consent was obtained. The study was approved by the Institutional Review Board at the University of Alabama at Birmingham, and the parents provided informed consent before testing began. All measurements were performed at the General Clinical Research Center or in The Department of Nutrition Sciences at the University of Alabama at Birmingham between 1994 and 1999.

General outline of protocol

The children were admitted to the General Clinical Research Center in the late afternoon for an overnight visit. Anthropometric measurements were obtained on the children's arrival. The Tanner stage of each child was determined by a pediatrician on the basis of breast stage and pubic hair development in girls (19) and genitalia development in boys (20). Dinner was served at \approx 1700. An evening snack was allowed, and after 2000 only water and energy-free, noncaffeinated beverages were permitted until after the morning testing. The next morning when the subjects were awakened at 0500, resting metabolic rate was assessed by

indirect calorimetry with use of a Deltatrac Metabolic Monitor (Sensormedics Corp, Yorba Linda, CA). The subjects were allowed to urinate if necessary. If the subjects urinated, they were asked to rest for 15 min before the resting metabolic rate measurement was taken. During testing, all subjects were instructed to lie as still as possible and to remain awake. An adult-size canopy hood was used to collect the expired air. After a 10-min equilibration period, data on oxygen consumption and carbon dioxide production were collected continuously for 20 min. REE was calculated with use of the equation of de Weir (21).

Two weeks later, the children arrived at the Department of Nutrition Sciences at 0700 in the fasted state. Total and regional body compositions were measured by dual-energy X-ray absorptiometry (DXA) using a Lunar DPX-L densitometer (Lunar Corp, Madison, WI) validated previously in the pediatric body weight range (22, 23). The subjects were scanned in light clothing while lying flat on their backs with their arms by their sides. DXA scans were performed and analyzed with use of pediatric software (version 1.5e), as described previously (22, 23). On the day of each test, the densitometer was calibrated by using the procedures provided by the manufacturer. Each subject's height without shoes was measured with a stadiometer. Weight was measured on an electronic scale while the subjects wore light clothing.

The participants returned annually for repeat measurements of body composition and REE; an identical testing protocol was used on each visit (except for 30% of the girls who were not examined in year 2). The initial visit date was between 1994 and 1999. In this analysis, the number of measurements for each child ranged from 2 to 5 (\bar{x} : 3.7) and the total follow-up duration for each child ranged from 1 to 4 y (\bar{x} : 2.7 y).

Statistics

Means and SDs were calculated for each visit for age, weight, height, body mass index, FM, LM, and REE. Analysis of variance was used to determine ethnic and sex differences in these variables in each visit-year. Mixed-model repeated-measures analyses (PROC MIXED; SAS Institute, Inc, Cary, NC) were used to examine relations between body composition and REE with increasing age and Tanner stage among ethnic and sex groups. In contrast with conventionally used general linear models for repeated-measures analysis, PROC MIXED can provide valid SEs and efficient statistical tests for repeated-measures analysis (24, 25). Continuous variables can be used to determine within-subjects effects in PROC MIXED. In addition, PROC MIXED does not require all subjects to have the same number of visits or measurements. All available data are used instead of eliminating subjects with missing data.

FM and LM were examined across age and Tanner stage, with adjustment for ethnicity and sex with these simplified models. Note that each model has an intercept and error term.

$$\text{FM} = \text{age} + \text{Tanner stage} + \text{ethnicity} + \text{sex} \quad (1)$$

$$\text{Relative FM} = \text{age} + \text{Tanner stage} + \text{ethnicity} + \text{sex} + \text{LM} \quad (2)$$

$$\text{LM} = \text{age} + \text{Tanner stage} + \text{ethnicity} + \text{sex} \quad (3)$$

Absolute and relative LLM and TLM were examined by using the following models:

$$\text{LLM (absolute)} = \text{age} + \text{Tanner stage} + \text{ethnicity} + \text{sex} \quad (4)$$

$$\text{LLM (relative)} = \text{age} + \text{Tanner stage} + \text{ethnicity} + \text{sex} + \text{TLM} \quad (5)$$

$$\text{TLM (absolute)} = \text{age} + \text{Tanner stage} + \text{ethnicity} + \text{sex} \quad (6)$$



TABLE 1
Distribution of participants in 5 y

Visit-year	White		African American	
	boys	girls	boys	girls
1	28	64	26	38
2	25	40	22	31
3	21	61	22	32
4	16	37	20	25
5	0	21	16	21

$$\text{TLM (relative)} = \text{age} + \text{Tanner stage} + \text{ethnicity} + \text{sex} + \text{LLM} \quad (7)$$

Relative LLM and TLM were obtained by adjusting each for the other to take body size into account. REE was examined with use of the following model:

$$\text{REE} = \text{age} + \text{Tanner stage} + \text{ethnicity} + \text{sex} + \text{FM} + \text{LM} \quad (8)$$

Because of a highly significant correlation between LLM and TLM ($r = 0.94$, $P < 0.0001$), a variable LIMTRK was created on the basis of the principal-component method to describe LM distribution:

$$\text{LIMTRK} = 0.707 \times \text{LLM} + 0.707 \times \text{TLM} \quad (9)$$

LIMTRK contained all the information on both LLM and TLM and eliminated multicollinearity caused by the significant correlation between the 2. The contribution of LM distribution to REE was then determined as follows:

$$\text{REE} = \text{age} + \text{Tanner stage} + \text{ethnicity} + \text{sex} + \text{FM} + \text{LIMTRK} \quad (10)$$

Fixed class variables in these models were visit-year, subject identifier, ethnicity, sex, and Tanner stage. Random variables included age, FM, LM, LLM, and TLM. Because the changes from one Tanner stage to the next may not be linear, we recoded 5 Tanner stages into 3 dummy variables. Also, Tanner stages 4 and 5 were combined because the sample was small. These dummy variables were defined by using Tanner stage 1 as the reference group. Therefore, the parameter estimates represented the difference in body composition and REE between Tanner stages 1 and 2, Tanner stages 1 and 3, and Tanner stages 1 and 4 or 5. *F* tests were used to determine the overall fixed effects of the models. Overall least-squares means \pm SEMs were reported for each ethnic-sex subgroup. All statistical analyses were conducted by using SAS (version 7.00; SAS Institute Inc). The level of significance was $P < 0.05$ for all tests.

RESULTS

Subject characteristics

The distribution of the participants over the 5 y of observations is presented in **Table 1**. In the first visit-year, there were 156 participants. The number of years of follow-up ranged from 2 to 5. During the second year, 30% of the girls were not evaluated. The distribution of Tanner stage in each year is shown in **Table 2**. Tanner stage was not examined in 4 cases. During the first visit-year, most of the children were prepubertal.

The characteristics of the participants in ≤ 5 visit-years are shown in **Table 3**. There was no significant ethnic or sex difference in age or height for any year. African American children

were heavier than were white children during the third visit-year ($P = 0.03$). Body-composition data in ≤ 5 visit-years are shown in **Table 4**. There was no significant ethnic or sex difference in FM during 5 visit-years. The white children had significantly lower LM than did the African American children during the third and fourth visit-years. The boys had significantly higher LM and REE than did the girls during the first and second years. African American children had a higher LLM than did white children ($P < 0.01$) and boys had a higher LLM than did girls only during the first visit-year. There was no significant ethnic difference in TLM during any visit-year. Boys had a higher TLM than did girls only during the first 2 visit-years. Absolute REE was not significantly different between African American and white children in any year. Boys had a higher REE than girls only during the first 2 visit-years.

Body composition and resting energy expenditure by Tanner stage

The differences in FM, LM, LLM, TLM, and REE between Tanner stage 1 and subsequent Tanner stages are shown in **Table 5**. Absolute FM did not vary significantly by Tanner stage after adjustment for age, ethnicity, and sex. FM adjusted for LM decreased significantly between Tanner stages 1 and 3, 1 and 4, and 1 and 5 ($P < 0.05$) but not between stages 1 and 2. LM increased significantly from Tanner stage 1 to subsequent Tanner stages ($P < 0.005$). LLM adjusted for TLM increased significantly from Tanner stage 1 to 3 but not from stage 1 to stages 2, 4, or 5 after adjustment for age, ethnicity, and sex. TLM adjusted for LLM increased significantly between Tanner stages 1 and 3, 1 and 4, and 1 and 5 ($P < 0.05$) but not between stages 1 and 2. The change in REE from Tanner stage 1 to stage 2 was not significant. The decrease in REE between Tanner stages 1 and 3 (315 kJ/d; $P = 0.01$) and between stages 1 and 4 (935 kJ/d; $P < 0.0001$) was significant after adjustment for age, ethnicity, sex, FM, and LM.

Body composition and resting energy expenditure in ethnic and sex subgroups

Body composition and REE in ethnic-sex subgroups are shown in **Table 6**. There was no ethnic or sex difference in FM after adjustment for age, Tanner stage, and LM. There were significant ethnic and sex differences in LM after adjustment for age and Tanner stage ($P = 0.01$ and $P = 0.0002$, respectively). Body composition, adjusted for age and Tanner stage, in ethnic and sex subgroups is shown in **Figure 1**. On average, LM was higher in African American than in white children (by 1.4 kg; $P = 0.01$) and in boys than in girls (by 2.1 kg; $P = 0.0002$). Relative LLM was higher in African American than in white children (by 1.6 kg; $P < 0.0001$) and in boys than in girls (by 1.0 kg; $P = 0.0005$). Relative TLM was higher in white than in African

TABLE 2
Frequency of various Tanner stages of the participants in 5 y¹

Visit-year	Tanner stage			
	1	2	3	4 and 5
1	145	9	1	1
2	89	21	6	1
3	60	57	14	4
4	26	44	22	5
5	10	13	16	18

¹Four subjects were not evaluated for Tanner stage.

TABLE 3
Characteristics of the participants during the study¹

	White boys	White girls	African American boys	African American girls
Age (y)				
Year 1	8.5 ± 1.8 (5.6–12.0)	8.3 ± 1.3 (4.9–11.0)	7.7 ± 1.5 (5.1–10.2)	8.0 ± 1.9 (4.6–12.1)
Year 2	9.6 ± 1.7 (6.7–13.0)	9.3 ± 1.5 (5.8–12.3)	8.8 ± 1.5 (6.1–11.2)	9.0 ± 2.0 (5.7–13.0)
Year 3	10.2 ± 1.6 (7.6–13.0)	10.3 ± 1.4 (7.0–13.5)	10.0 ± 1.5 (7.1–12.2)	10.1 ± 1.9 (6.5–14.1)
Year 4	11.1 ± 1.5 (8.6–13.3)	11.1 ± 1.4 (8.0–14.5)	10.6 ± 1.7 (8.1–13.3)	10.9 ± 1.8 (7.5–13.0)
Year 5		12.1 ± 1.4 (9.5–14.2)	11.5 ± 1.6 (9.1–13.8)	12.1 ± 1.8 (8.5–14.1)
Weight (kg)				
Year 1	33.3 ± 10.8 (16.2–61.7)	30.9 ± 9.8 (16.0–64.8)	34.9 ± 13.1 (19.9–67.1)	32.6 ± 12.2 (14.0–71.7)
Year 2	38.2 ± 13.5 (17.4–66.9)	34.2 ± 11.7 (16.5–77.1)	40.0 ± 16.1 (21.3–74.0)	37.8 ± 14.3 (16.0–74.7)
Year 3	40.0 ± 14.3 (19.5–78.0)	49.4 ± 14.2 (18.7–85.8)	47.6 ± 17.4 (28.0–83.3)	45.2 ± 15.2 (16.5–73.7)
Year 4	42.4 ± 12.9 (21.3–64.2)	43.5 ± 14.8 (19.6–102.8)	49.3 ± 19.8 (26.7–93.0)	48.9 ± 16.5 (18.5–85.6)
Year 5		48.5 ± 13.0 (25.6–69.9)	54.3 ± 20.8 (27.5–104.1)	55.7 ± 19.5 (20.6–100.5)
Height (cm)				
Year 1	131.8 ± 12.6 (110.0–160.0)	129.8 ± 9.6 (104.0–152.5)	131.6 ± 10.5 (113.0–153.0)	129.3 ± 14.1 (101.5–163.8)
Year 2	139.1 ± 13.0 (114.3–166.3)	135.6 ± 10.1 (109.0–159.3)	136.3 ± 11.6 (119.0–157.0)	137.1 ± 14.1 (109.0–162.5)
Year 3	142.9 ± 12.1 (119.5–170.0)	142.5 ± 9.5 (114.0–166.0)	144.5 ± 9.5 (129.3–161.0)	144.4 ± 13.6 (115.0–164.0)
Year 4	147.8 ± 11.6 (124.5–165.5)	147.9 ± 10.5 (120.0–174.0)	147.1 ± 10.6 (132.0–167.3)	149.4 ± 12.8 (119.4–166.0)
Year 5		152.0 ± 11.5 (125.0–169.9)	153.8 ± 11.3 (136.3–173.5)	155.8 ± 11.3 (123.5–171.0)

¹ $\bar{x} \pm SD$; range in parentheses. Weight was obtained from dual-energy X-ray absorptiometry. The number of visits ranged from 2 to 5 in white girls and in African American boys and girls, and from 2 to 4 in white boys.

²Significant effect of ethnicity, $P = 0.03$ (ANOVA).

TABLE 4
Body composition and resting energy expenditure (REE) during the study¹

Variable	White boys	White girls	African American boys	African American girls
FM (kg)				
Year 1	9.8 ± 6.2 (2.6–24.0)	9.8 ± 5.9 (2.9–32.6)	10.6 ± 8.2 (3.2–31.7)	10.5 ± 6.8 (2.7–29.2)
Year 2	10.7 ± 8.3 (1.9–26.3)	9.9 ± 7.2 (1.5–33.8)	12.0 ± 10.9 (1.7–37.4)	11.4 ± 8.1 (3.1–31.3)
Year 3	10.7 ± 7.8 (2.1–31.0)	12.8 ± 9.1 (2.3–44.7)	15.2 ± 11.9 (2.7–39.2)	14.4 ± 9.0 (2.2–31.5)
Year 4	11.2 ± 8.0 (2.8–29.8)	13.4 ± 9.4 (2.3–48.8)	14.4 ± 13.3 (2.3–46.6)	15.3 ± 10.4 (2.9–38.4)
Year 5		14.8 ± 11.7 (1.8–48.6)	14.8 ± 13.2 (1.8–48.6)	18.2 ± 13.1 (3.3–46.9)
LM (kg)				
Year 1 ²	21.1 ± 5.0 (10.6–40.2)	20.1 ± 4.1 (12.5–34.4)	22.9 ± 5.1 (14.6–34.5)	20.8 ± 5.8 (10.6–40.2)
Year 2 ³	26.1 ± 6.0 (14.8–38.9)	23.1 ± 4.8 (14.4–41.3)	26.5 ± 5.5 (17.1–35.3)	24.9 ± 7.0 (12.1–40.4)
Year 3 ⁴	27.8 ± 6.9 (16.7–45.3)	26.2 ± 5.7 (15.8–45.5)	30.7 ± 6.0 (22.5–42.9)	29.0 ± 7.2 (13.6–42.2)
Year 4 ⁵	29.6 ± 6.9 (17.7–42.4)	28.6 ± 6.4 (16.6–51.3)	33.0 ± 8.0 (23.2–49.2)	31.5 ± 7.4 (14.7–44.8)
Year 5		31.9 ± 5.6 (20.3–42.6)	37.4 ± 9.8 (24.5–56.7)	35.8 ± 7.7 (16.4–51.7)
LLM (kg)				
Year 1 ^{6,7}	9.8 ± 3.0 (4.4–17.1)	8.6 ± 2.1 (4.7–15.7)	10.8 ± 2.7 (6.2–16.0)	9.9 ± 3.2 (4.1–20.8)
Year 2 ⁸	11.7 ± 3.4 (5.3–19.0)	10.2 ± 2.6 (5.2–19.4)	12.4 ± 3.0 (7.7–17.2)	12.0 ± 3.9 (4.8–20.6)
Year 3 ⁹	12.8 ± 4.1 (6.2–23.1)	11.9 ± 2.9 (6.2–20.2)	14.8 ± 3.0 (10.1–20.1)	14.1 ± 4.0 (5.3–21.1)
Year 4 ¹⁰	13.9 ± 3.7 (6.9–20.4)	13.1 ± 3.1 (6.6–22.5)	16.1 ± 3.9 (10.6–25.0)	15.3 ± 4.0 (6.1–21.6)
Year 5 ⁴		14.7 ± 2.8 (8.6–20.2)	18.6 ± 4.8 (11.2–26.2)	17.9 ± 4.0 (7.0–24.8)
TLM (kg)				
Year 1 ¹¹	10.2 ± 2.3 (5.8–15.9)	9.4 ± 2.0 (5.8–16.7)	10.0 ± 2.6 (6.6–17.6)	9.0 ± 2.6 (4.6–17.1)
Year 2 ¹¹	12.0 ± 2.7 (7.1–17.7)	10.7 ± 2.3 (6.7–19.5)	11.7 ± 2.6 (7.0–16.0)	10.9 ± 3.1 (5.4–17.1)
Year 3	12.5 ± 2.8 (7.8–19.7)	12.0 ± 2.9 (7.1–22.7)	13.7 ± 3.3 (10.0–22.0)	12.7 ± 3.1 (6.0–18.2)
Year 4	13.3 ± 3.1 (8.2–19.7)	13.0 ± 3.3 (7.6–26.1)	14.5 ± 4.2 (9.6–23.3)	13.9 ± 3.5 (6.4–21.2)
Year 5		14.6 ± 2.8 (9.0–19.6)	16.1 ± 5.1 (10.3–27.0)	15.6 ± 3.6 (7.1–24.4)
REE (kJ/d)				
Year 1 ¹²	5590 ± 1092 (3879–8088)	5038 ± 674 (3502–7895)	5561 ± 1176 (3925–8937)	5017 ± 828 (3502–7435)
Year 2 ¹³	5996 ± 912 (4619–7736)	5301 ± 916 (3402–8577)	5895 ± 1209 (3636–8242)	5397 ± 971 (3611–6916)
Year 3	6050 ± 975 (4648–9146)	5803 ± 1050 (3594–9171)	6222 ± 1226 (4820–9916)	5891 ± 1004 (3740–8021)
Year 4	6092 ± 1618 (4297–8431)	5937 ± 1084 (3376–8958)	5891 ± 1092 (4912–9037)	5761 ± 1046 (3410–7954)
Year 5		6259 ± 1297 (4297–9125)	6289 ± 1477 (4050–9435)	6167 ± 1293 (3925–8121)

¹ $\bar{x} \pm SD$; range in parentheses. FM, fat mass; LM, lean mass; LLM, leg lean mass; TLM, trunk lean mass.

^{2,3,7,11–13}Significant sex effect (ANOVA): ² $P = 0.01$, ³ $P = 0.04$, ⁷ $P = 0.02$, ¹¹ $P = 0.03$, ¹² $P = 0.001$, ¹³ $P = 0.002$.

^{4–6,8–10}Significant ethnicity effect (ANOVA): ⁴ $P = 0.01$, ⁵ $P = 0.03$, ⁶ $P = 0.006$, ⁸ $P = 0.02$, ⁹ $P = 0.001$, ¹⁰ $P = 0.003$.



TABLE 5
Changes in FM, LM, LLM, TLM, and REE from Tanner stage 1 to subsequent Tanner stages¹

	Change	P
Absolute FM (kg)		
Stage 2	-0.1 ± 0.3	NS
Stage 3	-0.5 ± 0.5	NS
Stages 4 and 5	-1.0 ± 0.9	NS
Relative FM (kg) ²		
Stage 2	-0.3 ± 0.3	NS
Stage 3	-1.7 ± 0.6	0.003
Stages 4 and 5	-2.3 ± 1.0	0.02
LM (kg) ³		
Stage 2	0.6 ± 0.2	0.001
Stage 3	3.0 ± 0.3	0.003
Stages 4 and 5	3.2 ± 0.6	<0.0001
LLM (kg) ⁴		
Stage 2	0.2 ± 0.1	NS
Stage 3	0.4 ± 0.2	0.03
Stages 4 and 5	0.4 ± 0.3	NS
TLM (kg) ⁵		
Stage 2	0.1 ± 0.1	NS
Stage 3	0.4 ± 0.2	0.006
Stages 4 and 5	0.5 ± 0.3	0.04
REE (kJ/d) ⁶		
Stage 2	-76.1 ± 72	NS
Stage 3	-315 ± 121	0.01
Stages 4 and 5	-935 ± 195	<0.0001

¹ $\bar{x} \pm$ SEM. Tanner stage 1 was the reference group. FM, fat mass; LM, lean mass; LLM, leg lean mass; TLM, trunk lean mass; REE, resting energy expenditure; NS, not significant.

²Adjusted for age, ethnicity, sex, and LM.

³Adjusted for age, ethnicity, and sex.

⁴Adjusted for age, ethnicity, sex, and TLM.

⁵Adjusted for age, ethnicity, sex, and LLM.

⁶Adjusted for age, ethnicity, sex, FM, and LM.

American children (by 1.0 kg; $P = 0.0005$). There were significant ethnic and sex differences for REE after adjustment for age, Tanner stage, FM, and LM. Adjusted REE was higher in white than in African American children (by 172 kJ/d; $P = 0.001$) and

in boys than in girls (by 264 kJ/d; $P = 0.0001$). REE adjusted for FM, LLM, and TLM (expressed as LIMTRK) showed the same pattern as did the model adjusted for FM and LM.

Body composition and resting energy expenditure in ethnic and sex subgroups during puberty

The sex-specific changes in FM and LM during puberty are shown in **Figure 2**. The change in relative FM from Tanner stage 1 to stages 4 and 5 was significantly different between boys and girls ($P < 0.005$). The decrease in FM from Tanner stage 1 to stages 4 and 5 was 5.4 kg in boys and 0.5 kg in girls. The increase in LM from Tanner stage 1 to 2 and from 1 to 4 and 5 was significantly different between boys and girls ($P = 0.01$).

The increase in LM from Tanner stage 1 to 2 was not significant in boys but was 1.1 kg in girls ($P < 0.05$). The increase in LM from Tanner stage 1 to stages 4 and 5 was 5.0 kg in boys and 3.5 kg in girls. There was no ethnic difference in the change in FM and LM from Tanner stage 1 to subsequent Tanner stages.

The decrease in REE was significantly different between boys and girls between Tanner stages 1 and 2 ($P < 0.0001$) but not between stage 1 and other stages after adjustment for age, ethnicity, FM, and LM. There was no significant ethnic difference in the change in REE from Tanner stage 1 to subsequent Tanner stages. The ethnicity-specific change in REE with adjustment for body composition, sex, and Tanner stage are shown in **Figure 3**. There was not an interaction between ethnicity and Tanner stage.

DISCUSSION

This is the first longitudinal analysis to examine changes in REE and body composition, particularly the distribution of LM, in African American and white boys and girls during puberty. In this cohort, maturation was negatively related to REE independent of FM and LM. African American children had a lower REE than did white children after adjustment for age, Tanner stage, sex, FM, and LM. This ethnic difference was not explained by LM distribution.

In this study, we observed a positive relation between pubertal stage and LM, but not relative FM, after adjustment for age, ethnicity, and sex. LM increased in both boys and girls with

TABLE 6
FM, LM, LLM, TLM, and REE in ethnic and sex subgroups¹

	White boys	White girls	African American boys	African American girls
Absolute FM (kg) ²	12.2 ± 1.2	11.2 ± 1.0	12.7 ± 1.3	12.9 ± 1.1
Adjusted FM (kg) ³	10.5 ± 1.2	10.4 ± 1.0	10.6 ± 1.2	11.7 ± 1.1
LM (kg) ^{2,4,5}	30.0 ± 0.8	28.4 ± 0.6	32.1 ± 0.8	29.4 ± 0.7
Absolute LLM (kg) ^{2,6,7}	13.9 ± 0.4	13.1 ± 0.3	15.9 ± 0.4	14.5 ± 0.4
Relative LLM (kg) ⁶⁻⁸	12.5 ± 0.3	11.9 ± 0.2	14.0 ± 0.2	13.6 ± 0.2
Absolute TLM (kg) ^{2,9}	13.8 ± 0.4	13.2 ± 0.3	14.0 ± 0.4	12.8 ± 0.4
Relative TLM (kg) ^{6,10}	12.1 ± 0.1	12.2 ± 0.1	11.4 ± 0.1	11.5 ± 0.1
REE (kJ/d) ^{4,11,12}	5330 ± 159	5054 ± 138	5138 ± 159	4887 ± 130
REE (kJ/d) ¹²⁻¹⁴	5347 ± 155	5063 ± 138	5146 ± 159	4883 ± 130

¹ $\bar{x} \pm$ SEM. FM, fat mass; LM, lean mass; LLM, leg lean mass; TLM, trunk lean mass; REE, resting energy expenditure.

²Adjusted for age and Tanner stage.

³Adjusted for age, Tanner stage, and LM.

^{4,6,14}Significant ethnicity effect: ⁴ $P = 0.01$, ⁶ $P < 0.001$, ¹⁴ $P = 0.003$.

^{5,7,9,12}Significant sex effect: ⁵ $P = 0.0002$, ⁷ $P = 0.0005$, ⁹ $P = 0.001$, ¹² $P < 0.0001$.

⁸Adjusted for age, Tanner stage, and TLM.

¹⁰Adjusted for age, Tanner stage, and LLM.

¹¹Adjusted for age, Tanner stage, FM, and LM.

¹³Adjusted for age, Tanner stage, FM, and LIMTRK (a variable created on the basis of the principal-component method and incorporating LLM and TLM).

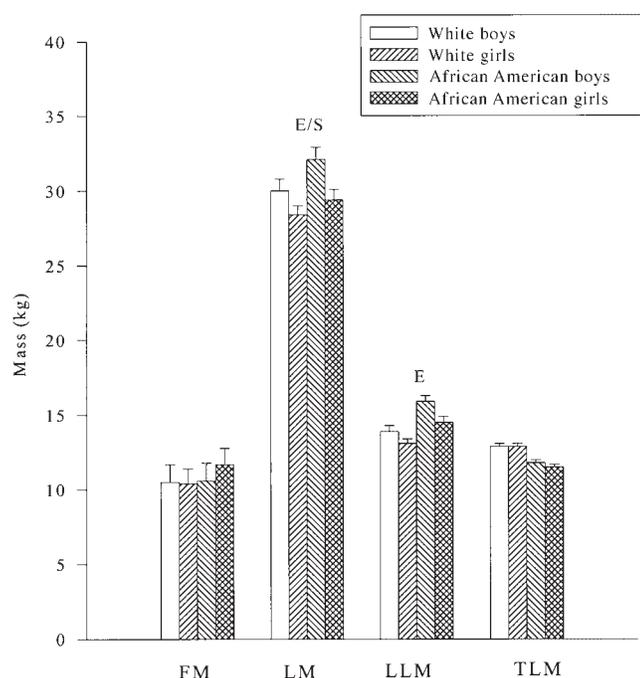


FIGURE 1. Body composition adjusted for age and Tanner stage in African American and white children and adolescents. Tanner stage ranged from 1 to 5. Fat mass (FM) was adjusted for lean mass (LM), limb lean mass (LLM) was adjusted for trunk lean mass (TLM), and TLM was adjusted for LLM. E/S, significantly different by ethnicity and sex. E, significantly different by ethnicity.

increasing Tanner stage. Boys had a later spurt but a longer LM increment than did girls. Relative FM remained constant in girls but decreased in boys after Tanner stage 2. These findings are similar to previous findings based on cross-sectional data (1, 4). The pattern of change in body composition during puberty was studied previously (26–32). In one study (30), children within a given age range who had a higher maturation rate had a higher accumulation of both LM and FM than did children who had a lower maturation rate. Because African American children begin puberty earlier than do white children (6, 15, 16), the influence of maturation on body composition and REE may be apparent at a younger age in African Americans. This pattern may help to explain the higher LM in African American than in white children at a certain age in this study.

REE declined with increasing pubertal stage after adjustment for age, ethnicity, sex, FM, and LM. In the current analysis, the decrease in REE was not significant from Tanner stage 1 to 2 but was significant from Tanner stage 1 to stages 3 and 4. An inverse relation between REE and age was observed previously in 98 early-pubertal girls (6). REE adjusted for LM was the lowest in girls at Tanner stage 3. The difference in REE between stages 3 and 1 was significant in white girls. In African American girls, the difference between stages 2 and 1 was significant. This earlier study indicated that the change in REE during puberty occurred sooner in African Americans than in whites. In another study, Weinsier et al (33) reviewed 31 data sets from 1111 subjects

and found a decrease in REE with increasing age. REE was the highest in preschool children, followed by adolescents, then adults. In contrast with these findings, pubertal stage was not considered an important contributor to REE independent of body composition in several other cross-sectional studies (4, 7, 34). In 83 children and adolescents whose Tanner stage ranged from 1 to 5 (4), REE varied significantly with sex but not with pubertal stage independent of LM. During puberty, skeletal muscle is accumulated more rapidly than is organ mass (21). The decrease in REE, adjusted for LM, with maturation may be explained by the increase in proportion of the relatively less energy-demanding skeletal muscle component of LM (8).

In the current PROC MIXED analysis using longitudinal data from children at various stages of maturation, we observed a lower REE (≈ 250 kJ/d) in African American children than in white children after adjustment for age, Tanner stage, sex, FM, and LM. An ethnic difference in REE was not observed in our cohort when the children were prepubertal (13). The ethnic divergence in the change in REE tended to occur after Tanner stage 2 (Figure 3). However, the interaction term between Tanner stage and ethnicity in the mixed model for REE was not significant. Our data differ from the results of cross-sectional studies in which a lower REE was observed in African Americans than in whites in both prepubertal (6, 35, 36) and pubertal (34) children. The reason for this inconsistency remains unclear but may have been a failure to accurately assess Tanner stage in different ethnic groups that mature at different rates, or subtle differences between physical and physiologic maturation that cannot be easily assessed.

As mentioned above, differences in energy expenditure between organ and skeletal muscle may help to explain the decline in REE with maturation. It has been suggested that TLM

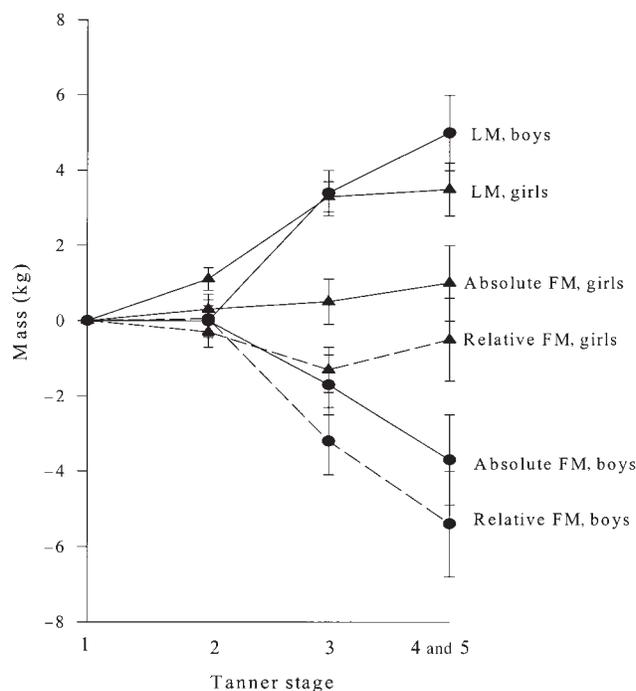


FIGURE 2. Sex-specific changes in body composition during puberty, adjusted for age and ethnicity. LM, lean mass; FM, fat mass; relative FM, FM adjusted for LM.

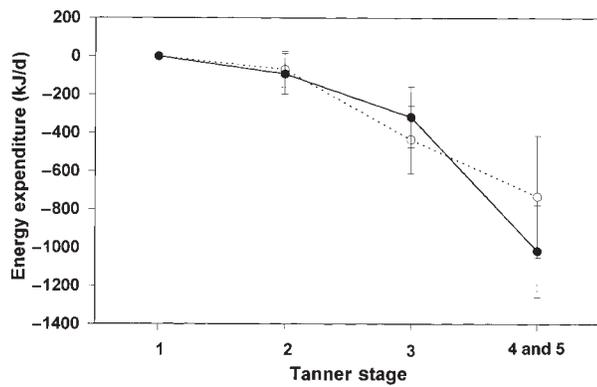


FIGURE 3. Ethnic-specific changes in REE during puberty, relative to Tanner stage 1, in African Americans (●, —) and whites (○, ····), adjusted for age, sex, and body composition.

and LLM serve as surrogates for mass of organs and muscles, respectively, that are not easily measured (37, 38). LLM is the main component of total lean mass and the sum of skeletal muscle in arms and legs as defined in DXA scan analysis. TLM, because of the organ component, may contribute more to REE than does LLM (11). Previous studies in adults had conflicting results regarding the contribution of LM distribution to REE (9, 10, 37, 39–41). By analyzing data from cadavers, Garby et al (40) showed that organ weights contributed significantly to REE after adjustment for body weight or LM. Other studies showed that, although composition of LM itself was a determinant of REE, an independent contribution of organ weight was not found, or was found to be weak, after LM was taken into consideration (37, 41).

In the present study, African American children had relatively higher LLM and lower TLM than did white children. Both relative LLM and relative TLM contributed significantly to REE. We chose to create a new variable by using principal-component analysis that incorporated information on both LLM and TLM and avoided multicollinearity between the 2. This new variable allowed us to examine the relative contribution of LLM and TLM to REE. The addition of relative LM distribution did not eliminate the significant ethnic contribution to REE. A recent study from our laboratory in premenopausal women showed that a racial difference in REE persisted after adjustment for LLM but disappeared after adjustment for TLM (12). The limitation of that study was that LLM and TLM are only crude surrogates of muscle and organ mass. In addition, we were not able to separate the 2 components and examine independent contributions of each. Further studies are needed to investigate the actual mass of organ and muscle and the relative contribution of each to REE.

If future research confirms a lower REE in African Americans than in whites during pubertal development, the lower metabolism may be related to the development of obesity in African Americans. Previous longitudinal studies from our laboratory showed that REE does not predict fat gain in white prepubertal children over a 5-y period (42) or in African American children (43) over 3–5 y. Whether a lower REE predisposes African Americans to a higher obesity rate needs to be examined further in children at later Tanner stages when REE starts to decline. Other environmental factors, such as food intake and physical activity, may be more significant with regard to the overall regulation of energy balance. In a recent study by Luke et al (44), African men

and women living in Nigeria had an identical REE to African Americans living in Chicago, despite large differences in body composition (the Nigerian subjects had more lean mass and less fat mass). One important concern with the present study and other studies of children is the definition of maturation stage. Studies reviewed in this article used different staging criteria. One limitation of our study was that we did not use bone age, an important determinant of maturation that may improve the ability to gauge maturation. Another limitation of our study was the small sample in Tanner stages 4 and 5, especially white children.

In conclusion, the results of this study indicate a negative relation between pubertal maturation and REE after adjustment for FM and LM. African American children, on average, had a lower REE than did white children (by 250 kJ/d) after adjustment for FM, LM, and pubertal stage. African American children had relatively more LLM and less TLM than did white children. The ethnic difference in REE was not explained by the relative distribution of LLM and TLM. 

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