

# Effects of Gender, Ethnicity, Body Composition, and Fat Distribution on Serum Leptin Concentrations in Children\*

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## ABSTRACT

The Ob protein leptin has been shown to be closely correlated with measures of body fat in humans and animals. Studies have suggested that there are both gender and ethnic differences in serum leptin concentrations, even after controlling for total and relative body fat and body mass index. We hypothesized that gender and ethnic differences in serum leptin concentrations are due to differences in both body composition and body fat distribution. We measured fasting serum leptin concentration, body composition (fat mass and fat-free mass by dual energy x-ray absorptiometry), and body fat distribution (intraabdominal and sc abdominal adipose tissue by computed tomography) in 74 prepubertal boys and girls (43 African-Americans

and 31 Caucasians). Our results showed that gender differences in serum leptin concentrations could not be fully explained by differences in body mass index, total fat mass, or relative body composition. However, when serum leptin concentrations were adjusted for differences in relative body composition (fat mass and fat-free mass) and body fat distribution (sc and intraabdominal adipose tissue), gender no longer had an independent effect on the serum leptin concentration. Serum leptin concentrations were not influenced by ethnicity. Thus, when comparing group differences in serum leptin concentrations, it is necessary to adequately control for group differences in body composition and fat distribution. (*J Clin Endocrinol Metab* 82: 2148–2152, 1997)

THE *ob* gene product, leptin, is a hormone secreted by adipose tissue (1). The circulating concentration of leptin is closely and positively correlated with body fat in humans (1, 2) and mice (2, 3). Using *in vivo* arteriovenous balance techniques, Klein *et al.* (4) have shown that leptin production by sc adipose tissue is positively related to the percent body fat. However, at any given level of percent body fat, there is a fairly large variation in the circulating concentration of leptin (1, 2). The cause of this variation is unknown.

One potential source of variation in serum leptin concentration is gender. Sexual dimorphism in serum leptin concentrations have been reported (5–8). The gender difference remains significant after adjusting for body mass index (BMI), total fat mass, and percent body fat. Rosenbaum *et al.* (6) found differences in leptin concentrations (adjusted for total fat mass) between men and postmenopausal women, suggesting that the gender difference is not due solely to differences in sex hormones.

A second potential source of variation in serum leptin concentrations is body fat distribution, as the level of *ob* messenger ribonucleic acid expression has been shown to vary with anatomical site (9–11). Thus, gender and ethnic

differences in fat distribution could potentially lead to variations in the concentration of serum leptin, independent of total or relative fat mass. Previous studies have produced mixed results concerning the effect of fat distribution on serum leptin concentrations (5, 7, 12). In Western Samoan women, waist circumference, but not waist to hip ratio, was related to the serum leptin concentration after controlling for BMI (5). In contrast, Rosenbaum *et al.* (6) and Hickey *et al.* (7) failed to find an effect of waist to hip ratio or waist circumference on the serum leptin concentration, independent of fat mass. However, it is important to point out that the use of these anthropometric estimates of fat distribution may not adequately assess true differences in fat distribution (13–15) and, therefore, may have limited usefulness.

The purpose of this study was to determine the effects of body fat and body fat distribution on gender and ethnic differences in circulating leptin concentrations in prepubertal, African-American, and Caucasian children. Our study differs from previous ones in that we have accurate measures of body composition (dual energy x-ray absorptiometry) and fat distribution [computed tomography (CT)]. Our results suggest that the sexual dimorphism in serum leptin concentrations may be explained by gender differences in body composition and fat distribution. We found no significant effects of ethnicity on the serum leptin concentration.

## Subjects and Methods

### Subjects

We studied 43 prepubertal African-American (23 girls and 20 boys) and 31 Caucasian (8 girls and 23 boys) children. Children were recruited by newspaper and radio advertisements and by word of mouth. Subjects

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were screened by a medical history evaluation and were ineligible if they were taking medications known to affect body composition or physical activity (e.g. prednisone, ritalin, or GH), if they were diagnosed with syndromes known to affect body composition and/or fat distribution (e.g. Cushing's syndrome, Down's syndrome, type I diabetes, or hypothyroidism), or they had experienced any major illness since birth. Pubertal status (Tanner stage) was determined by physician evaluation of both breast and pubic hair in females (16) and genitalia in males (17). This study was approved by the institutional review board at the University of Alabama at Birmingham. The parents of all participants provided informed consent before testing commenced.

### Protocol

Children were admitted to the General Clinical Research Center in the late afternoon for an overnight visit. Upon arrival, anthropometric measurements were obtained, and dinner was served at approximately 1700 h. An evening snack was allowed as long as it was consumed before 2000 h. After 2000 h, only water and noncaloric, noncaffeinated beverages were allowed until after the morning testing. Between 1850–1950 h, a single slice CT scan was taken at the level of the umbilicus. The following morning, blood was collected for hormone analyses. Two weeks later, the children arrived at the Energy Metabolism Research Unit at 0700 h in the fasted state, and body composition was determined by dual energy x-ray absorptiometry (DXA).

### Fat distribution and body composition

Subcutaneous abdominal adipose tissue (SAAT) and intraabdominal adipose tissue (IAAT) were measured by CT scanning with a HiLight/Advantage Scanner (General Electric, Milwaukee, WI) as previously described (15). A single slice scan (5 mm) of the abdomen was performed at the level of the umbilicus and analyzed for cross-sectional area of adipose using the density contour program. CT data are presented as the cross-sectional area of tissue (square centimeters) with the Hounsfield units for adipose tissue as  $-190$  to  $-30$ . We have shown the test-retest reliability for IAAT to be 1.7% (18). All scans were analyzed by the same investigator (T.R.N.). The total body radiation dose to each subject was approximately 0.26 rad. This total body radiation dose is less than that received from a standard chest x-ray.

Body composition [fat mass and fat-free mass (FFM)] was measured by DXA using a Lunar DPX-L densitometer (Lunar Radiation Corp., Madison, WI). We have previously validated the use of DXA against

carcass analysis in a pig model that encompassed the pediatric weight range (19). Subjects were scanned in light clothing while lying flat on their backs with arms at their sides. DXA scans were performed and analyzed with pediatric software version 1.5e.

### Serum leptin concentrations

Serum leptin was determined using serum from the fasted children with a RIA kit (Linco Research, St. Charles, MO). All serum samples (100  $\mu$ L) were analyzed in duplicate in a single assay. The intraassay coefficient of variation was 4.2% at 64% bound (2.61 ng/mL) and 3.5% at 28% bound (14.9 ng/mL).

### Statistical methods

Because the data for some variables were not normally distributed, they were  $\log_{10}$  transformed to increase the normality of their distribution. In these cases, the values shown are the back-transformed means (geometric mean). Differences in subject characteristics were determined by two-way ANOVA, with gender and ethnicity as class variables (the interaction of gender and ethnicity was included in the ANOVA). Simple correlations (Pearson product moment) were assessed for serum leptin concentration and measures of fat. To determine the independent effects of variables on the serum leptin concentration, multiple linear regression analyses were performed (general linear models procedure). All statistics were performed using SAS for Windows (version 6.10, SAS Institute, Cary, NC). Data were considered significant if  $P < 0.05$ .

## Results

Descriptive statistics are shown in Table 1. There were no significant effects of gender or ethnicity on age, weight, BMI, or absolute values for total fat, SAAT, or IAAT. Percent body fat, serum leptin, and FFM were all significantly affected by gender; girls had greater body fat and leptin concentrations and lesser FFM than boys.

In all groups, BMI, fat (percentage and total), SAAT, and IAAT were strongly and positively correlated to the serum leptin concentration (Table 2). FFM was correlated to the

**TABLE 1.** Descriptive statistics

	African-American boys (n = 20)	African-American girls (n = 23)	Caucasian boys (n = 23)	Caucasian girls (n = 8)	Effects <sup>a</sup>
Age (yr)	7.6 $\pm$ 1.6 (5.1–10.2)	7.3 $\pm$ 1.7 (4.6–10.0)	8.2 $\pm$ 1.5 (5.6–10.9)	7.6 $\pm$ 1.4 (5.2–9.2)	NS
Wt (kg) <sup>b</sup>	31.4 $\pm$ 13.3 (19.8–68.4)	29.0 $\pm$ 13.3 (14.1–57.9)	29.4 $\pm$ 7.7 (20.3–45.1)	30.9 $\pm$ 5.5 (23.3–41.5)	NS
BMI (kg/m <sup>2</sup> )	19.0 $\pm$ 5.6 (11.8–35.7)	18.6 $\pm$ 4.2 (12.8–28.0)	17.9 $\pm$ 3.1 (13.9–25.5)	18.8 $\pm$ 2.7 (17.0–24.1)	NS
Fat (%)	25 $\pm$ 5 (14–48)	31 $\pm$ 9 (18–47)	25 $\pm$ 10 (11–44)	28 $\pm$ 7 (18–38)	Gender <sup>c</sup>
FFM (kg) <sup>b</sup>	21.8 $\pm$ 6.21 (14.6–34.5)	18.3 $\pm$ 5.4 (10.6–29.1)	20.6 $\pm$ 3.8 (14.5–25.9)	19.9 $\pm$ 2.4 (17.2–24.2)	Gender <sup>c</sup>
Fat (kg) <sup>b</sup>	7.5 $\pm$ 7.0 (3.7–31.7)	8.6 $\pm$ 7.9 (2.7–26.2)	6.9 $\pm$ 5.2 (2.6–19.8)	8.5 $\pm$ 4.2 (4.2–15.3)	NS
SAAT (cm <sup>2</sup> ) <sup>b</sup>	42 $\pm$ 83 (8–462)	59 $\pm$ 77 (16–273)	56 $\pm$ 72 (14–230)	84 $\pm$ 70 (36–181)	NS
IAAT (cm <sup>2</sup> ) <sup>b</sup>	22 $\pm$ 23 (8–114)	20 $\pm$ 17 (7–54)	24 $\pm$ 20 (7–65)	32 $\pm$ 23 (12–77)	NS
Leptin (ng/mL) <sup>b</sup>	4.8 $\pm$ 5.6 (1.8–25.9)	8.5 $\pm$ 9.9 (2.2–39.8)	5.1 $\pm$ 5.6 (1.5–23.2)	8.0 $\pm$ 6.1 (3.6–16.6)	Gender <sup>d</sup>

Values are the mean  $\pm$  SE, with the data range in parentheses.

<sup>a</sup> Effects from a two-way ANOVA, with gender and ethnicity as class variables (the interaction of gender and ethnicity was included in the model).

<sup>b</sup> Data for these variables were transformed ( $\log_{10}$ ) before statistical analysis. Data presented are the back-transformed means (geometric mean).

<sup>c</sup>  $P < 0.05$ .

<sup>d</sup>  $P < 0.01$ .

**TABLE 2.** Correlation analyses of body composition variables on  $\log_{10}$  leptin concentration

	African-American boys (n = 20)	African-American girls (n = 23)	Caucasian boys (n = 23)	Caucasian girls (n = 8)
BMI (kg/m <sup>2</sup> )	0.80 <sup>a</sup>	0.86 <sup>a</sup>	0.82 <sup>a</sup>	0.85 <sup>b</sup>
Fat (%)	0.88 <sup>a</sup>	0.92 <sup>a</sup>	0.87 <sup>a</sup>	0.93 <sup>a</sup>
FFM (kg) <sup>c</sup>	0.54 <sup>b</sup>	0.74 <sup>a</sup>	0.16	0.57
Fat (kg) <sup>c</sup>	0.88 <sup>a</sup>	0.90 <sup>a</sup>	0.85 <sup>a</sup>	0.93 <sup>a</sup>
SAAT (cm <sup>2</sup> ) <sup>c</sup>	0.90 <sup>a</sup>	0.86 <sup>a</sup>	0.79 <sup>a</sup>	0.80 <sup>d</sup>
IAAT (cm <sup>2</sup> ) <sup>c</sup>	0.83 <sup>a</sup>	0.80 <sup>a</sup>	0.62 <sup>cd</sup>	0.72 <sup>b</sup>

<sup>a</sup>  $P < 0.001$ .<sup>b</sup>  $P < 0.05$ .<sup>c</sup> Data for these variables were transformed ( $\log_{10}$ ) before statistical analysis.<sup>d</sup>  $P < 0.01$ .**TABLE 3.** Multiple linear regression analyses with leptin ( $\log_{10}$  ng/mL) as the dependent variable

Independent variable	$\beta$	SE( $\beta$ )	$P$ value
Intercept	-0.52	0.14	<0.001
Gender <sup>a</sup>	0.20	0.05	<0.001
Ethnicity <sup>b</sup>	-0.05	0.05	0.31
BMI	0.06	0.01	<0.001
Model $r^2 = 0.66$			
Intercept	-0.33	0.09	<0.001
Gender <sup>a</sup>	0.15	0.04	<0.001
Ethnicity <sup>b</sup>	-0.03	0.04	0.39
Fat (kg) <sup>c</sup>	1.08	0.07	<0.001
Model $r^2 = 0.79$			
Intercept	0.55	0.32	0.09
Gender <sup>a</sup>	0.10	0.04	0.02
Ethnicity <sup>b</sup>	-0.04	0.04	0.33
Fat (kg) <sup>c</sup>	1.26	0.09	<0.001
FFM (kg) <sup>c</sup>	-0.74	0.26	<0.01
Model $r^2 = 0.81$			
Intercept	0.47	0.31	0.13
Gender <sup>a</sup>	0.06	0.04	0.12
Ethnicity <sup>b</sup>	-0.0005	0.04	0.99
Fat (kg) <sup>c</sup>	0.98	0.16	<0.001
FFM (kg) <sup>c</sup>	-0.75	0.25	<0.01
SAAT (cm <sup>2</sup> ) <sup>c</sup>	0.37	0.12	<0.01
IAAT (cm <sup>2</sup> ) <sup>c</sup>	-0.22	0.14	0.12
Model $r^2 = 0.84$			

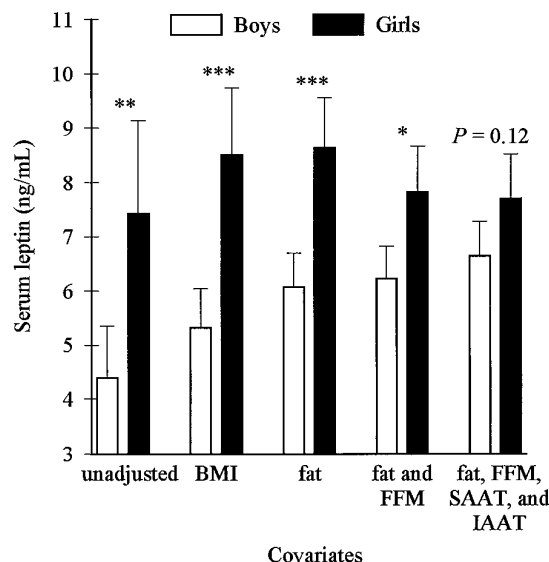
 $\beta$ , Regression coefficient; SE( $\beta$ ), SE of regression coefficient.<sup>a</sup> Where boys = 1 and girls = 2.<sup>b</sup> Where Caucasian = 1 and African Americans = 2.<sup>c</sup> Data were transformed ( $\log_{10}$ ) before analysis.

serum leptin concentration only in African-American children.

Results from multiple linear regression models revealed that gender was associated with serum leptin concentrations after controlling for BMI, total body fat, and relative body fat (fat mass and FFM; Table 3). However, when FFM and fat distribution (IAAT and SAAT) were added to the model, gender was no longer a significant ( $P = 0.12$ ) independent predictor of the serum leptin concentration (Table 3). A summary of adjusted means is shown graphically in Fig. 1. Ethnicity was not an independent predictor of serum leptin concentrations in any of the above models (Tables 3).

### Discussion

This study examined the influence of gender, ethnicity, body composition, and fat distribution on serum leptin con-



**FIG. 1.** Serum leptin concentration (nanograms per mL) by gender in 74 children. Data were analyzed by ANOVA (unadjusted) or by analysis of covariance (ANCOVA) with the listed variables as covariates. Both gender and ethnicity were included as class variables in the models. Data shown are back-transformed least square means for the effect of gender. The serum leptin concentration, total fat, FFM, SAAT, and IAAT were transformed ( $\log_{10}$ ) before analysis. \*,  $P < 0.05$ ; \*\*,  $P < 0.01$ ; \*\*\*,  $P < 0.001$ .

centrations in prepubertal children. We found that the gender difference in serum leptin concentrations could not be fully explained by gender differences in BMI, total fat mass, or relative body composition. However, when serum leptin concentrations were adjusted for differences in relative body composition (fat mass and FFM) and body fat distribution (SAAT and IAAT), gender no longer had an independent effect on the serum leptin concentration. Serum leptin concentrations were not influenced by ethnicity.

Our data agree with those reported by others, in that the sexual dimorphism in serum leptin concentrations persists after controlling for BMI, fat mass, or relative fat mass (5–8, 12, 20, 21). However, when body fat distribution (SAAT and IAAT) was added to the model (in addition to body composition), gender no longer was significantly related to the serum leptin concentration. Our data suggest that the sexual dimorphism in serum leptin concentrations may be due to both relative body composition and fat distribution. To our knowledge, no other study investigating the sexual dimorphism in serum leptin concentrations has controlled for both relative body composition and fat distribution using DXA and CT.

A previous study in children (20) found a significant effect of gender on the serum leptin concentration after controlling for BMI and SAAT. Our results concur; when we controlled for differences in BMI and SAAT, gender remained significant ( $P < 0.01$ ; data not shown). However, BMI may not be a good measure of body composition. For instance, at any given BMI, women tend to have more body fat than men (22). Moreover, when we control for BMI in our data set, girls have significantly greater percent relative body fat than boys ( $P < 0.001$ ; data not shown).

Because of the inherent problems with using ratios (per-

cent fat) to normalize data (23, 24), we chose to enter both fat mass and FFM into our regression models to control for differences in body composition. When entered together, both fat mass and FFM were independent predictors, albeit in different directions, of the serum leptin concentration. As has been shown, the serum leptin concentration is positively associated with fat mass. However, when FFM is entered into the model with fat mass, FFM is negatively related to serum leptin concentrations. These results suggest that at a given fat mass, a person with a greater FFM would have a lower serum leptin concentration than a person with a lesser FFM, if all else is equal. This finding implies that FFM or some factor related to FFM or relative body composition plays a role in the regulation of leptin production.

We found that fat distribution was a contributor to the variations in serum leptin concentrations independent of body composition. Thus, at any given body composition, individuals with relatively greater fat mass partitioned to the sc areas would be expected to have greater serum leptin concentrations. As premenopausal women, relative to men, tend to partition fat to the sc spaces (25), they would be expected to have greater serum leptin concentrations than men at any given body composition. This relationship may explain the persistence of the sexual dimorphism in serum leptin concentrations after controlling for body composition and the lack of a gender effect when both body composition and fat distribution are considered.

Similarly, gender differences in serum leptin may also be explained by the sexual dimorphism in IAAT. Men tend to deposit relatively more fat in the intraabdominal space than do women (25), and leptin production from IAAT does not appear to be related to body fatness in an independent manner. Our study shows that although IAAT was positively correlated with the serum leptin concentration, the relationship was not independent of total fat mass ( $P = 0.89$ ; data not show) or of body composition and SAAT, a result similar to that reported by Dua *et al.* (26). In addition, Montague *et al.* (11) showed that leptin messenger ribonucleic acid expression from intraabdominal adipocytes does not correlate with BMI in either men or women. Thus, fat distribution appears to have a profound effect on the serum leptin concentration; if all else is equal, individuals with fat distributed in the intraabdominal area (male distribution?) would be expected to have lower serum leptin levels than individuals with fat distributed to the sc area (female distribution?).

We found no effect of ethnicity (African-American *vs.* Caucasian) on the serum leptin concentration. Two previous studies likewise failed to find ethnic differences in serum leptin concentrations once body fat was taken into account. Hassink *et al.* (21) found no difference in serum leptin concentrations when comparing Caucasian to African-American and Hispanic children (the latter two groups were collapsed into one group due to sample size) after controlling for BMI and tricep skinfold. Similarly, Dagoga-Jack *et al.* (27) found no difference in leptin levels between Caucasian and African-American adults after controlling for BMI.

In contrast to the above reports and the present study, Nicklas *et al.* (28) reported differences in serum leptin concentrations between African-American and Caucasian obese postmenopausal women. The lower leptin concentrations in

African-American women persisted after controlling for total fat mass. Although the two groups of women did not differ with respect to fat mass, the African-American women did have significantly more lean tissue mass. Based on our multiple regression model (fat mass was a positive, whereas FFM was a negative, predictor of the leptin concentration), the African-American women in their study would be predicted to have lower serum leptin concentrations than the Caucasians. Therefore, the ethnic difference in serum leptin concentration in the study by Nicklas *et al.* (28) may be explained by differences in body composition.

In conclusion, our data show that serum leptin concentrations in children are highly correlated with measures of body fat, and that gender differences in serum leptin concentrations may be due to differences in body composition and body fat distribution. Future studies examining the role of gender in serum leptin concentrations should consider both relative body composition (fat *vs.* FFM) and fat distribution (intraabdominal *vs.* sc). Additionally, the inclusion of both fat and FFM in our regression models showed that FFM had a negative effect on the serum leptin concentration, suggesting that FFM or something closely correlated with FFM may have a negative feedback effect on serum leptin concentrations. Our data also showed that the effect of body fat distribution on the serum leptin concentration is independent of body composition. Lastly, we found no evidence for an effect of ethnicity on the serum leptin concentration in our sample of children.

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