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Relationship of Leptin, Resting Metabolic Rate, and Body Composition in PreMenopausal Hispanic and Non-Hispanic White Women

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Abstract

Objective—The purpose was to evaluate the relationships between fasting serum leptin, resting metabolic rate (RMR), and body composition in premenopausal Hispanic and non-Hispanic White (White) women.

Methods—Participants were 67 Hispanic and 43 White women who arrived at the laboratory in a fasted state for measurement of RMR by indirect calorimetry, bone mineral content measured by dual-energy X-ray absorptiometry, and body density measured by hydrodensitometry. Serum leptin levels were determined by EIA.

Results—Multiple regression analysis revealed that body mass and lean body mass were the best predictors of RMR. Leptin was not a significant predictor of RMR.

Conclusion—Further research needs to be done to examine the role of leptin on metabolism, especially in ethnic groups predisposed to development of obesity and related disorders.

Keywords

Hormone; Ethnicity; RMR; Human

INTRODUCTION

The prevalence of overweight and obesity is a public health concern particularly because of the increased risk of cardiovascular disease, diabetes, hypertension, and early mortality. Over one-third of adults (more than 72 million people) are obese (1). Certain racial/ethnic groups are at greater risk for development of overweight and obesity. Among women of at

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least 20 years of age, the National Health and Nutrition Examination Survey (NHANES) 2003–2004 reported non-Hispanic White women as having the lowest prevalence of overweight or obesity (58.0%), non-Hispanic Black women as having the highest (81.6%), and Mexican American women falling in between the two (75.4%) (2). Even though environmental and cultural factors (i.e., availability of fast food, larger portion sizes, and certain types of food) contribute to the obesity epidemic, it is important to examine the physiological mechanisms underlying the development of overweight and obesity.

To successfully regulate the body's energy stores, a balance is needed between energy intake and energy expenditure. Energy homeostasis is maintained by the brain–gut–adipose axis, which is a combination of behavioral, autonomic, and endocrine pathways (3). The identification of the *ob*/*ob* gene in mice and subsequent discovery of the protein leptin has led to significant advances in the understanding of energy homeostasis in humans (3,4).

For most sedentary adults, resting metabolic rate (RMR) is the primary component of daily total energy expenditure; and a low RMR can be predictive of weight gain when an imbalance is created in the energy homeostasis mechanism. The relationship between leptin and resting energy expenditure is unclear. In mice, leptin appears to have a positive effect by increasing energy expenditure (5,6). In humans, the effects of leptin on energy expenditure, in particular RMR, are not well understood. Several studies have suggested that circulating leptin levels are associated with RMR (7–9); whereas other studies have found no association between leptin and RMR (10–14).

It has been well established that women have higher circulating levels of leptin than men (15,16). Additionally, disparities exist among racial/ethnic groups for prevalence of overweight and obesity (1). Understanding the metabolic and hormonal influences on energy expenditure among premenopausal women of different racial groups may help to discern factors that influence disparities in overweight and obesity among women. Therefore, the purpose of this study was to evaluate the relationship between fasting serum leptin levels, RMR, and body composition in premenopausal Hispanic and non-Hispanic White women.

METHODS

Participants

Sixty-seven Hispanic and 43 non-Hispanic White premenopausal women between the ages of 35 and 50 years volunteered for this study. Participants were included if they reported that both parents and three of four grandparents were of the same ancestry (either Hispanic or non-Hispanic White). Prior to data collection, each participant signed an informed consent form approved by the Institutional Review Board of the University of Texas at El Paso. Women were excluded from the study for pregnancy, currently nursing, irregular menstrual cycles, amenorrhea, diagnosed diabetes, thyroid disorders, or if they were on medications known to affect metabolism.

Study Protocol

Each participant reported to the laboratory between 0600 and 0800 h, at least 12 h post-prandial, and following 6–8 h of sleep. Body mass was measured to the nearest 0.01 kg

using a calibrated load cell scale (Tanita Corporation, Tokyo, Japan); height was measured to the nearest 0.1 cm using a stadiometer (Seca Corp., Germany); and BMI (kg/m^2) was calculated. RMR was measured followed by the collection of a fasting blood sample and body composition assessments by dual-energy X-ray absorptiometry (DXA) and hydrostatic densitometry. All laboratory procedures were completed during a single testing session and each woman was asked to void prior to body composition assessments.

Resting Metabolic Rate Assessment

For the measurement of RMR, participants arrived at the laboratory in the morning, having refrained from alcohol and caffeine consumption, and physical activity for 24 h prior to the test. Participants were fitted with a sealed facemask (Hans-Rudolph Inc., Kansas City, MO, USA) connected to a large two-way non-rebreathing valve (Hans-Rudolph Inc., Kansas City, MO, USA), placed in a comfortable reclined position and allowed to rest for 20 min. Expired gases were then collected for 30 min and analyzed for the fractional concentration of oxygen and carbon dioxide using an automated metabolic measurement system (TrueMax 2400, Consentius Technologies, Sandy, UT, USA). The final 20 min of the metabolic test were averaged and recorded as RMR.

Immediately prior to each metabolic test, the flowmeter was calibrated using a 3 L calibration syringe (Han-Rudolph Inc., Kansas City, MO, USA) and the gas analyzers were calibrated using a two-point calibration method with certified gases (16% O_2, 4% CO_2). Metabolic gas volumes were derived by the Fick equation and energy expenditure (kcal) was calculated as (17):

$$[\text{kcal}] = \text{VO}_2\,(\text{L}/\,\text{min}) \times 4.825(\text{kcal}/\text{L}\,\text{O}_2)]$$

Body Composition Assessment

The body composition of each woman was assessed using DXA and hydrostatic densitometry. For each woman, bone mineral density, bone mineral content, and body composition (DXA-BF) were assessed using DXA (Lunar DPX-NT, GE Lunar Corp., Madison, WI, USA). All measurements were performed in accordance with manufacturer's specifications. Women were asked to remove all jewelry and other accessories, and were measured in a standard set of gym shorts and T-shirts that were provided by the investigators. A quality assurance test, which calibrates and verifies the correct operation of the densitometer, was performed at the start of each testing day to examine the functionality, accuracy, and precision of the system. The coefficient of variance (CV%) for the DXA system used was 0.23% based on 254 quality assurance test procedures and control measurements.

Body density (D_b) of each woman was determined by hydrostatic densitometry (HW). A two-point calibration technique was used to calibrate the electronic strain gauge scale and the gain was set using a 4 kg lead weight. Underwater weight was recorded while completely submerged and at maximal exhalation. Women were asked to perform the submersion procedure a minimum of five trials and the heaviest three values within 100 g were averaged and used for the determination of D_b. Body density was corrected for

measured residual lung volume and gastrointestinal gas (0.1 L), and body composition (BF) was calculated using a three-component model ($HW_{3\text{-}C}$) (Lohman, 1984). Total body bone mineral content determined from DXA was multiplied by the constant 1.25 to estimate total body mineral content (18) for use with the three-compartment model equation.

Immediately prior to the hydrostatic weighing procedure, residual lung volume was determined using the modified O_2 dilution method described by (Wilmore, 1969).

Blood Sample Analysis

A fasting blood sample was collected by venipuncture from an antecubital vein into a blank serum vacuum tube for each participant. The blood sample was allowed to clot, centrifuged for 20 min, and serum aliquots were separated into cryule vials (Wheaton, Millville, NJ, USA) and frozen at −80°C until analyzed for leptin. Serum total leptin concentration (ng/mL) was determined by enzyme immunoassay (LDN, Nordhorn, Germany) and absorbance was assessed using a microtiterplate reader (SpectraMAX 190, Molecular Devices, Sunnyvale, CA, USA). The sensitivity of the leptin assay was 0.2 ng/mL. For low (5 ng/mL), medium (10 ng/mL), and high (21 ng/mL) controls, the inter-assay reproducibility (CV%) was 3.6, 8.6, and 7.8%, respectively, and the intra-assay reproducibility (CV%) was 5.4, 4.3, and 4.1%, respectively. All samples were measured in duplicate and the average of the two measures was recorded as the leptin concentration.

Serum triglycerides (TG) were assessed using an automated analyzer (Cholestech LDX, Hayward, CA, USA). The calibration of the instrument was verified prior to each use, using two calibrator standards of known concentration.

Statistical Analysis

Statistical analyses were conducted using the software package SPSS v13.0 (SPSS, Inc., Chicago, IL, USA). The normal distribution of variables was checked with histograms and Kolmogorov–Smirnov's test. Preliminary analyses for violations of the assumption of normality, linearity, and homoscedasticity revealed that lean body mass (LBM), serum leptin, and serum TG were not normally distributed. Log_{10} and square root transformation of the leptin data did not reflect normality ($p = 0.001$ and $p = 0.015$, respectively); therefore, leptin data were transformed using the equation: $\sqrt{\text{leptin (ng/mL)}+0.375}$ $(p=0.078)$ (19). A log_{10} transformation was used to transform both LBM ($p = 0.060$) and TG ($p = 0.191$) data to reflect normality.

Descriptive data were compared between groups using an independent samples *t*-test. Pearson's and partial correlation coefficients were calculated and multiple stepwise linear regression was used to examine the relationships between RMR and leptin, TG, total body mass (TBM), percentage body fat (%BF), fat mass (FM), and LBM; and to identify those variables with the greatest predictive influence on RMR. *Z*-scores were calculated from Pearson's correlation coefficients to test the statistical significance of the difference between correlation coefficients. A z_{obs} score greater than 1.96 or less than −1.96 indicated a statistically significant difference between correlation coefficients. Collinearity between independent variables was assessed using variance inflation factor and a value above 10.0

indicated multicollinearity. Potential outliers were identified by Mahalanobis distances with a critical value of 20.82. Significance was set at an alpha level of <0.05.

RESULTS

Descriptive Statistics

An independent samples *t*-test was conducted to compare age, height, TBM, BMI, RMR, DXA %BF, $HW_{3\text{-}C}$ %BF, FM, LBM, TG, and total leptin for Hispanic and non-Hispanic White women (Table 1). There were no differences between the two groups for age, TBM, RMR, FM, LBM, or TG ($p > 0.05$). The Hispanic women of this study were of significantly less stature ($p = 0.001$); had a greater BMI ($p = 0.015$); had a greater %BF measured by both DXA ($p = 0.021$) and $HW_{3\text{-}C}$ ($p = 0.034$); and had significantly greater leptin levels ($p = 0.003$) than non-Hispanic White women. When controlled for %BF, leptin levels were not different between Hispanic and non-Hispanic White women ($p = 0.104$).

Correlation Analyses

Pearson correlation coefficients exhibited moderate to high correlations between most of the variables (Table 2). When groups were analyzed individually by ethnicity, most of these relationships remained in Hispanic women, but some were lost in non-Hispanic White women.

For Hispanic women, there was a strong significant correlation between RMR and TBM ($r = 0.77$, $p < 0.001$), RMR and LBM ($r = 0.70$, $p < 0.001$), RMR and FM ($r = 0.66$, $p < 0.001$), and RMR and %BF ($r = 0.59$, $p < 0.001$). Additionally, a strong correlation between leptin and FM ($r = 0.61$, $p < 0.001$), and leptin and %BF ($r = 0.54$, $p < 0.001$) were observed for Hispanic women. A lesser, but still significant moderate correlation was observed for Hispanic women between RMR and leptin ($r = 0.47$, $p < 0.001$) (Table 2).

For non-Hispanic White women, there was a strong correlation between RMR and LBM ($r = 0.76$, $p < 0.001$) and a strong correlation between RMR and TBM ($r = 0.67$, $p < 0.001$). A moderate correlation was observed between RMR and FM ($r = 0.43$, $p < 0.001$) for non-Hispanic White women. There was no significant correlation between RMR and leptin ($r = 0.07$) or RMR and %BF ($r = 0.11$) for non-Hispanic White women. There was no significant relationship between leptin and FM ($r = 0.16$) or %BF ($r = 0.04$); however, leptin and LBM had a moderate correlation ($r = 0.30$, $p < 0.05$) for non-Hispanic White women (Table 2).

An examination of the probability that the difference in correlation coefficients was because of a true difference between ethnic groups and not sampling error revealed that leptin explained significantly more of the variance in TBM ($z_{obs} = -2.69$), RMR ($z_{obs} = -2.16$), %BF ($z_{obs} = -2.74$), and FM ($z_{obs} = -2.74$) for Hispanic women than for non-Hispanic White women.

Partial correlation analysis was used to explain the relationship between leptin and RMR while controlling for age, %BF, FM, and LBM. Preliminary analyses were done to ensure no violation of the assumptions of normality, linearity, and homoscedasticity. There was a moderate, positive, partial correlation between leptin and RMR when controlling for age ($r =$

0.34, $p < 0.001$) and a lesser partial correlation for age and %BF ($r = 0.26$, $p = 0.007$), with higher levels of leptin being associated with higher values for RMR. Further inclusion of LBM ($r = -0.05$, $p = 0.597$) and FM ($r = 0.09$, $p = 0.378$) revealed a nonsignificant association between leptin and RMR. An inspection of the zero-order correlation ($r = 0.36$) suggested that controlling for age and %BF had little impact on the relationship between leptin and RMR.

Multiple Regression Analysis

To account for and explain part of the variance within the variables, a multiple stepwise regression was created (Table 3). Overall, the results of the regression analysis indicated that TBM and LBM were the best predictors of RMR ($R^2 = 0.60$, $p < 0.001$). TBM (beta = 0.47) made the strongest unique contribution to explaining RMR, when all other variables in the model were controlled. When Hispanic women were analyzed separately from non-Hispanic White women, multiple regression analysis still determined TBM and LBM as the best predictors of RMR ($R^2 = 0.62$, $p < 0.001$), and TBM (beta = 0.56) made the strongest individual contribution to this model. For non-Hispanic White women, LBM was the best predictor of RMR ($R^2 = 0.57$, $p < 0.001$). Leptin did not make a significant contribution to the prediction of RMR for all participants ($p = 0.60$), Hispanic women ($p = 0.89$), or non-Hispanic White women ($p = 0.12$).

DISCUSSION

The purpose of this study was to evaluate the relationship between fasting serum total leptin levels, RMR, and body composition in premenopausal Hispanic and non-Hispanic White women. The main finding of this study was that for these premenopausal women, RMR was better predicted by TBM and LBM, rather than the hormonal influence of leptin. For Hispanic women, these predictors remain the same (TBM and LBM); however, LBM was the best predictor of RMR in non-Hispanic White women. There was a significant association between leptin and RMR for Hispanic women even after controlling for %BF. There was no significant relationship between leptin and RMR for these non-Hispanic White women.

Leptin and Body Fat

The obese mutation (*ob*), discovered in 1950, is a recessive trait that results in obesity and hyperphagia when the carrier [mouse] is homozygous for the gene (20). Forty-four years following the discovery of the *ob* gene, the *ob* gene was cloned, sequenced, and the encoded protein was subsequently named leptin (after the Greek word *leptos*, meaning “thin”) (4,5). When *ob/ob* mice were administered recombinant leptin, there was a decrease in body fatness (5). When diabetic (*db/db*) mice (which similar to *ob/ob* mice are morbidly obese and demonstrate hyperphagia) were administered leptin, there was no change in body fatness or eating behavior (5). This led to the belief that *ob/ob* mice lacked the hormone leptin, whereas *db/db* mice appeared to be resistant to the effects of leptin.

This belief that leptin deficiency was a cause of obesity led to a series of studies in humans. However, it was found that leptin deficiency in humans is extremely rare and that the

influence of leptin on obesity in humans is most likely a result of some form of leptin resistance (15,21). It is well understood that leptin in humans is highly correlated with measures of body fat, and leptin levels are higher in women compared to men regardless of body fat percentage. Leptin values were significantly greater in our Hispanic women compared to our non-Hispanic White women. Percentage body fat values were also significantly greater for the Hispanic women. These results parallel national data for obesity prevalence rates (1) and also leptin values collected through NHANES (22). Data from NHANES III reported leptin values being greatest in non-Hispanic black women, followed by Mexican American women, whereas non-Hispanic White women had the lowest reported leptin values (22). These patterns observed for leptin between ethnic groups are similar to patterns of overweight and obesity in women. Non-Hispanic Black women have the greatest incidence for overweight and obesity, non-Hispanic Whites have the lowest rate, and Hispanic women lie in between the two (1). Our study of premenopausal women found that both FM and %BF were positively correlated with leptin values for Hispanic women, but not for non-Hispanic White women. Our leptin values also showed a weak, but significant correlation with circulating TG when both groups were combined and for Hispanic women separately. Within our data set however, we found no significant predictors of leptin concentrations for either Hispanic or non-Hispanic women (data not shown).

Resting Metabolic Rate and Body Composition

It is well understood that overweight and obese individuals have higher absolute RMR values than lean individuals and that women have lower RMR values compared to men (23). However, when RMR is adjusted for LBM, obese individuals have similar RMR values to lean individuals. A low RMR may be predictive of weight gain. For this group of women, there was no significant difference in absolute values of RMR between Hispanic and non-Hispanic White women. There was also no significant difference in LBM between the two groups. This suggests that the difference in %BF between the two groups is not related to a lower RMR in the Hispanic women. To the authors' knowledge, differences in RMR between Hispanic and non-Hispanic White premenopausal women has not been previously studied. Research examining RMR between non-Hispanic White women and non-Hispanic Black women has suggested that non-Hispanic Black women have lower RMR values, potentially contributing to their increased rates of overweight and obesity (24–26). Further research examining the effect of ethnicity/race on RMR between Hispanics and non-Hispanic Whites is needed.

LBM is considered to be a major determinant of RMR (27). However, LBM is not a homogeneous compartment; it is composed of "metabolically fast" lean tissue (i.e., liver, heart, kidneys, and gut) and "metabolically slow" lean tissue (i.e., muscle) (28). The individual rates of energy expenditure are very different between skeletal muscle and organ mass (29). While muscle mass comprises approximately 40% of adult body weight, it contributes to only about 20% of RMR; conversely, the human brain and liver (~4% of body weight) contribute 40–45% of RMR (28). It is possible that the difference in organ weights between individuals may account for differences in RMR. The best predictors of RMR in these premenopausal women were TBM and LBM. This was expected, because the major

determinant of RMR is LBM and is justified by the high correlation that is seen between RMR and TBM, and RMR and LBM for these women.

Leptin and RMR

The relationship between leptin and RMR has been studied previously with disparate results. In *ob*/*ob* mice, infusion of leptin has corresponded to an increase in RMR and physical activity levels (5,30); however, this association was not seen in *db/db* or wild-type mice (5,30,31).

Human studies have seen similar inconsistent results. Leptin and RMR have been found to be inversely related in obese men and women (9), and positively related in sedentary African American and Caucasian women (32). Two human studies that have found a relationship between leptin and RMR have examined bound leptin and free leptin concentrations. Free leptin is positively correlated with FM, and negatively correlated with RMR; bound leptin however appears to be positively correlated with RMR (7,8). Additionally, there appears to be a saturation effect of free leptin in cerebrospinal fluid that is not seen with bound leptin concentrations in cerebrospinal fluid (7). Most studies have found no relationship between leptin and resting metabolism. Free leptin concentration and RMR do not seem to be influenced by age (12,13), gender (10,12), or weight loss (33). Results from the current study indicate that leptin was positively correlated with RMR for Hispanic, but not non-Hispanic White women. However, when examining the best predictors of RMR, leptin was not a significant contributor. Additionally, this study measured total leptin concentrations as opposed to bound leptin concentration. This may partially account for our leptin concentrations not being significant predictors of RMR.

CONCLUSIONS

Our data indicate that the best predictors of RMR in premenopausal women are TBM and LBM. Leptin values are greater in Hispanic premenopausal women than in non-Hispanic White women (Table 2). This is most likely because of the increased body fat percentage of the Hispanic women. However, even after controlling for body fat, leptin was positively correlated with RMR in Hispanic women, though it was not considered a significant predictor of RMR.

Further research examining how bound leptin may regulate energy expenditure and potential methods of upregulating the transport of leptin across the blood–brain barrier may give insight to the physiological workings of the hormone in humans. It is also possible that leptin is just one of a complex series of hormones that contribute to regulation of appetite and energy expenditure.

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References

1. Ogden, CL.; Carroll, MD.; McDowell, MA.; Flegal, KM. NCHS data brief no 1. Hyattsville, MD: National Center for Health Statistics; 2007. Obesity among adults in the United States – no change since 2003–2004.
2. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. J Am Med Assoc. 2006; 295:1549–1555.
3. Cone, RD.; Low, MJ.; Elmquist, JK.; Cameron, JL. Neuroendocrinology. In: Larson, PR.; Kronenberg, HM.; Helmed, S.; Polonsky, KS., editors. Williams Textbook of Endocrinology. Philadelphia, PA: Saunders; 2003. p. 81-176.
4. Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. Positional cloning of the mouse obese gene and its human homologue. Nature. 1994; 372:425–432. [PubMed: 7984236]
5. Halaas JL, Gajiwala KS, Maffei M, et al. Weight-reducing effects of the plasma protein encoded by the obese gene. Science. 1995; 269(5223):543–546. [PubMed: 7624777]
6. Hamann A, Matthaei S. Regulation of energy balance by leptin. Exp Clin Endocrinol Diabetes. 1996; 104(4):293–300. [PubMed: 8886745]
7. Brabant G, Horn R, von zur Muhlen A, et al. Free and protein bound leptin are distinct and independently controlled factors in energy regulation. Diabetologia. 2000; 43(4):438–442. [PubMed: 10819236]
8. Magni P, Liuzzi A, Ruscica M, et al. Free and bound plasma leptin in normal weight and obese men and women: relationship with body composition, resting energy expenditure, insulin-sensitivity, lipid profile and macronutrient preference. Clin Endocrinol (Oxf). 2005; 62(2):189–196. [PubMed: 15670195]
9. Niskanen L, Haffner S, Karhunen LJ, Turpeinen AK, Miettinen H, Uusitupa MI. Serum leptin in relation to resting energy expenditure and fuel metabolism in obese subjects. Int J Obes Relat Metab Disord. 1997; 21(4):309–313. [PubMed: 9130029]
10. Kennedy A, Gettys TW, Watson P, et al. The metabolic significance of leptin in humans: gender-based differences in relationship to adiposity, insulin sensitivity, and energy expenditure. J Clin Endocrinol Metab. 1997; 82(4):1293–1300. [PubMed: 9100610]
11. McCargar LJ. Leptin response and body regulation in humans. J Clin Biochem Nutr. 1999; 26:77–84.
12. Neuhauser-Berthold M, Herbert BM, Luhrmann PM, et al. Resting metabolic rate, body composition, and serum leptin concentrations in a free-living elderly population. Eur J Endocrinol. 2000; 142(5):486–492. [PubMed: 10802527]
13. Roberts SB, Nicholson M, Staten M, et al. Relationship between circulating leptin and energy expenditure in adult men and women aged 18 years to 81 years. Obes Res. 1997; 5(5):459–463. [PubMed: 9385622]
14. Rosenbaum M, Nicolson M, Hirsch J, Murphy E, Chu F, Leibel RL. Effects of weight change on plasma leptin concentrations and energy expenditure. J Clin Endocrinol Metab. 1997; 82(11): 3647–3654. [PubMed: 9360521]
15. Jequier E, Tappy L. Regulation of body weight in humans. Physiol Rev. 1999; 79(2):451–480. [PubMed: 10221987]
16. Lonnqvist F, Nordfors L, Schalling M. Leptin and its potential role in human obesity. J Intern Med. 1999; 245(6):643–652. [PubMed: 10395194]
17. Knoebel, L. Energy metabolism. In: Selkurt, E., editor. Physiology. Boston, MA: Little-Brown; 1987. p. 535-547.
18. Salamone LM, Fuerst T, Visser M, et al. Measurement of fat mass using DEXA: a validation study in elderly adults. J Appl Physiol. 2000; 89(1):345–352. [PubMed: 10904070]
19. Ott, RL.; Longnecker, M. An Introduction to Statistical Methods and Data Analysis. Pacific Grove, CA: Duxbury; 2001. Inferences about more than two population central values; p. 403-409.

20. Ingalls AM, Dickie MM, Snell GD. Obese, a new mutation in the house mouse. J Hered. 1950; 41(12):317–318. [PubMed: 14824537]
21. Considine RV, Considine EL, Williams CJ, et al. Mutation screening and identification of a sequence variation in the human ob gene coding region. Biochem Biophys Res Commun. 1996; 220(3):735–739. [PubMed: 8607834]
22. Ruhl CE, Everhart JE. Leptin concentrations in the United States: relations with demographic and anthropometric measures. Am J Clin Nutr. 2001; 74(3):295–301. [PubMed: 11522551]
23. Ferraro R, Lillioja S, Fontvieille AM, Rising R, Bogardus C, Ravussin E. Lower sedentary metabolic rate in women compared with men. J Clin Invest. 1992; 90(3):780–784. [PubMed: 1522233]
24. Albu J, Shur M, Curi M, Murphy L, Heymsfield SB, Pi-Sunyer FX. Resting metabolic rate in obese, premenopausal black women. Am J Clin Nutr. 1997; 66(3):531–538. [PubMed: 9280169]
25. Forman JN, Miller WC, Szymanski LM, Fernhall B. Differences in resting metabolic rates of inactive obese African-American and Caucasian women. Int J Obes Relat Metab Disord. 1998; 22(3):215–221. [PubMed: 9539188]
26. Jakicic JM, Wing RR. Differences in resting energy expenditure in African-American vs Caucasian overweight females. Int J Obes Relat Metab Disord. 1998; 22(3):236–242. [PubMed: 9539192]
27. Muller MJ, Bosy-Westphal A, Kutzner D, Heller M. Metabolically active components of fat-free mass and resting energy expenditure in humans: recent lessons from imaging technologies. Obes Rev. 2002; 3(2):113–122. [PubMed: 12120418]
28. Henry CJ. Mechanisms of changes in basal metabolism during ageing. Eur J Clin Nutr. 2000; 54(Suppl 3):S77–S91. [PubMed: 11041079]
29. Gallagher D, Belmonte D, Deurenberg P, et al. Organ-tissue mass measurement allows modeling of REE and metabolically active tissue mass. Am J Physiol. 1998; 275(2 Pt 1):E249–E258. [PubMed: 9688626]
30. Pelleymounter MA, Cullen MJ, Baker MB, et al. Effects of the obese gene product on body weight regulation in ob/ob mice. Science. 1995; 269(5223):540–543. [PubMed: 7624776]
31. Campfield LA, Smith FJ, Guisez Y, Devos R, Burn P. Recombinant mouse OB protein: evidence for a peripheral signal linking adiposity and central neural networks. Science. 1995; 269(5223): 546–549. [PubMed: 7624778]
32. Nies MA, Chen KY, Van der Wal JS. Energy expenditure, body composition, and biochemical indicators in healthy community women. Int J Food Sci Nutr. 2003; 55(3):237–247. [PubMed: 15223601]
33. Filozof CM, Murua C, Sanchez MP, et al. Low plasma leptin concentration and low rates of fat oxidation in weight-stable post-obese subjects. Obes Res. 2000; 8(3):205–210. [PubMed: 10832762]
34. Lohman TG, Slaughter MH, Boileau RA, Bunt J, Lussier L. Bone mineral measurements and their relation to bone density in children, youths, and adults. Human Biology. 1984; 56(4):667–679. [PubMed: 6530219]
35. Wilmore JH. A simplified method for determination of residual lung volumes. J Appl Physiol. 1969; 27(1):96–100. [PubMed: 5786976]

TABLE 1

Descriptive Characteristics of Premenopausal Hispanic and Non-Hispanic White Women (Mean ± SE)

Variable	Overall	Hispanic	Non-Hispanic White
N	110 (100.0%)	67 (60.9%)	43 (39.1%)
Age (years)	43.0 ± 0.43	42.8 ± 0.56	43.4 ± 0.64
Height (cm)	163.1 ± 0.57	161.5 ± 0.63*	165.5 ± 0.98
TBM (kg)	68.5 ± 1.35	69.7 ± 1.84	66.6 ± 1.90
BMI (kg/m^2)	25.8 ± 0.49	26.7 ± 0.67*	24.3 ± 0.62
RMR (kcal/day)	1,479.9 ± 18.76	1,492.6 ± 24.82	1,460.1 ± 28.52
DXA %BF	40.2 ± 0.80	41.6 ± 1.01*	37.9 ± 1.23
$HW_{3\text{-}C}$ %BF	36.4 ± 0.79	37.8 ± 1.05*	34.4 ± 1.13
FM (kg)	25.8 ± 0.98	27.7 ± 1.36	23.5 ± 1.31
LBM (kg)	40.1 ± 0.57	39.8 ± 0.72	40.6 ± 0.92
TG (mg/dL)[a]	115.7 ± 6.91	118.2 ± 8.51	111.6 ± 11.86
Leptin (ng/dL)	7.0 ± 0.65	8.3 ± 0.95*	4.8 ± 0.65

TBM, total body mass; BMI, body mass index; RMR, resting metabolic rate; DXA, dual-energy X-ray absorptiometry; $HW_{3\text{-}C}$, three-component hydrostatic densitometry; %BF, percentage body fat; FM, fat mass; LBM, lean body mass; TG, triglycerides.

[a]For triglycerides only: overall $N = 100$; for Hispanics, $N = 62$; for non-Hispanic Whites, $N = 38$.

*Significantly different from non-Hispanic White ($p < 0.05$).

TABLE 2

Pearson's and Spearman's Correlation Coefficients for Tested Variables in Hispanic and Non-Hispanic Whites

	TBM	BMI	RMR	%BF	FM	LBM	TG
Overall							
TBM							
BMI	0.933†						
RMR	0.735†	0.662†					
%BF	0.687†	0.743†	0.323†				
FM	0.925†	0.920†	0.586†	0.902†			
LBM	0.728†	0.602†	0.707†	0.023	0.414†		
TG[a]	0.457†	0.449†	0.281†	0.419†	0.457†	0.268†	
Leptin	0.536†	0.570†	0.356†	0.418†	0.510†	0.362†	0.260†
Hispanic							
TBM							
BMI	0.952†						
RMR	0.766†	0.737†					
%BF	0.715†	0.770†	0.586†				
FM	0.899†	0.817†	0.655†	0.904†			
LBM	0.728†	0.648†	0.695†	0.079	0.459†		
TG[a]	0.457†	0.465†	0.255*	0.486†	0.492†	0.218	
Leptin	0.536†	0.623†	0.471†	0.536†	0.612†	0.435†	0.372†
Non-Hispanic White							
TBM							
BMI	0.908†						
RMR	0.665†	0.505†					
%BF	0.614†	0.639†	0.107				
FM	0.899†	0.862†	0.429†	0.890†			
LBM	0.755†	0.635†	0.755†	−0.037	0.395†		

	TBM	BMI	RMR	%BF	FM	LBM	TG
TG[a]	0.449†	0.451†	0.313	0.288	0.385*	0.362*	
Leptin	0.256	0.330*	0.074	0.044	0.158	0.302*	0.007

TBM, total body mass; RMR, resting metabolic rate; %BF, percent body fat ($HW_{3\text{-}C}$); FM, fat mass; LBM, lean body mass; TG, triglycerides.

[a] For triglycerides only: overall, $N = 100$; for Hispanics, $N = 62$; for non-Hispanic Whites, $N = 38$.

* $p < 0.05$;

† $p < 0.01$ (two-tailed).

TABLE 3

Stepwise Multiple Regression Analysis Examining Predictors of Resting Metabolic Rate in Premenopausal Hispanic and Non-Hispanic White Women

Dependent variable	Step number	Predicting variable	Beta	R^2	*p*-Value
Overall					
RMR	1	TBM	0.47	0.10	<0.001
	2	LBM	0.37	0.06	<0.001
Hispanic					
RMR	1	TBM	0.56	0.06	<0.001
	2	LBM	0.28	0.04	0.004
Caucasian					
RMR	1	LBM	0.75	0.57	<0.001

Variables entered in the model: (1) dependent variable, resting metabolic rate (RMR); (2) predicting variables, leptin, total body mass (TBM), percent body fat ($HW_{3\text{-}C}$), fat-free mass, and lean body mass (LBM).

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