Published in final edited form as:
Am J Clin Nutr. 2006 May ; 83(5): 1062–1067.

Small organs with a high metabolic rate explain lower resting energy expenditure in African American than in white adults[2]

Dympna Gallagher, **Jeanine Albu**, **Qing He**, **Stanley Heshka**, **Lawrence Boxt**, **Norman Krasnow**, and **Marinos Elia**
1 *From the Obesity Research Center (DG, QH, and SH) and the Department of Cardiology (NK), St Luke's–Roosevelt Hospital, New York, NY; the Institute of Human Nutrition and the Department of Medicine, College of Physicians and Surgeons, Columbia University, New York, NY (DG, QH, and SH); the Division of Cardiology, North Shore University Hospital, Manhasset, NY (LB); and the Institute of Human Nutrition, University of Southampton, Southampton, United Kingdom (ME).*

Abstract

Background—African Americans have a lower resting energy expenditure (REE) relative to fat-free mass (FFM) than do whites. Whether the composition of FFM at the organ-tissue level differs between African Americans and whites and, if so, whether that difference could account for differences by race in REE are unknown.

Objective—The objectives were to quantify FFM in vivo in women and men at the organ-tissue level and to ascertain whether the mass of specific high-metabolic-rate organs and tissues differs between African Americans and whites and, if so, whether that difference can account for differences in REE.

Design—The study was a cross-sectional evaluation of 64 women (n = 34 African Americans, 30 whites) and 35 men (n = 8 African Americans, 27 whites). Magnetic resonance imaging measures of liver, kidney, heart, spleen, brain, skeletal muscle, and adipose tissue and dual-energy X-ray absorptiometry measures of fat and FFM were acquired. REE was measured by using indirect calorimetry.

Results—The mass of selected high-metabolic-rate organs (sum of liver, heart, spleen, kidneys, and brain) after adjustment for fat, FFM, sex, and age was significantly ($P < 0.001$) smaller in African Americans than in whites (3.1 and 3.4 kg, respectively; $\bar{x} \pm$ SEE difference: 0.30 ± 0.06 kg). In a multiple regression analysis with fat, FFM, sex, age, and race as predictors of REE, the addition of the total mass rendered race nonsignificant.

Conclusions—Racial differences in REE were reduced by >50% and were no longer significant when the mass of specific high-metabolic-rate organs was considered. Differences in FFM composition may be responsible for the reported REE differences.

[2]Supported by National Institutes of Health Grants PO1-DK-42618 (Project 4 and Core C); RR-00645, DK-40414 and P30-DK-26687 and an educational grant from Knoll Pharmaceuticals.

[3] Reprints not available. Address correspondence to D Gallagher, Obesity Research Center, 1090 Amsterdam Avenue, New York, NY 10025. E-mail: dg108@columbia.edu..

DG was involved in study design, data collection, data analysis, and manuscript writing and provided administrative support and supervision. JA was responsible for data collection in studies RR-00645 and DK-40414 on whom additional MRI measures were acquired for inclusion into this study. QH was involved in study coordination for DK-42618 (Project 4). SH was involved in data analysis and manuscript writing. LB analyzed all cardiac MRI data. NK analyzed all echocardiography data. ME provided advice and consultation as a consultant on DK-42618 (Project 4). None of the authors had a personal or financial conflict of interest.

Keywords

Race; ethnicity; African Americans; whites; metabolism; organs; tissues; fat-free mass; resting energy expenditure; magnetic resonance imaging

INTRODUCTION

It is generally accepted that resting energy expenditure (REE) is lower in African American than in white women (1–3), men (4), and children (5–9). After adjustment for differences in body weight or body composition or both, Kushner et al (3), Albu et al (1), and Foster et al (2) found REE to be 6% (160 kcal/d), 3% (120 kcal/d), and 6% (94 kcal/d), respectively, lower in African American than in white women. Because REE is ≈65% of daily energy expenditure, the daily differences in REE observed in these studies (≈100–150 kcal), if not compensated for by a lower intake, may over a prolonged period of time be a contributing factor to the greater incidence of obesity in African American than in white women. But, regardless of the long-term effect on body weight, these observations suggest that race-ethnicity differences in energy requirements may exist.

Fat-free mass (FFM) is the principal contributor to energy requirements, and total-body FFM is commonly used as a surrogate for metabolically active tissue. However, this practice pools together numerous organs and tissues and gives no consideration to possible racial differences in the composition of FFM. The brain, liver, heart, and kidneys account for ≈60–70% of REE in adults, although the combined weight of those organs is <6% of total body weight (10–12). Skeletal muscle composes 40–50% of total body weight and accounts for only 20–30% of REE (10,11,13).

Compared with whites, African Americans have similar or smaller amounts of total body fat (14) and greater amounts of bone and skeletal muscle mass (15). Even after adjustment for these known differences in body composition, REE is lower in African Americans. Hunter et al (16) found that, after differences in trunk lean mass measured by dual-energy X-ray absorptiometry (DXA) were taken into account, REE differences between African American and white women disappeared, which suggested that the composition of trunk lean mass may differ between races. However, DXA is incapable of distinguishing among the various types of organs and tissues that make up trunk lean mass.

The aims of this study were, first, to quantify several components of FFM in vivo in African American and white women and men at the organ-tissue level by using magnetic resonance imaging [(MRI;) 11] and, second, to ascertain whether the mass of specific organs or tissues differs between races and, if so, whether such differences could account for reported differences in REE. A third, minor aim was to ascertain whether within-race individual variation in the mass of specific organs or tissues helps to account for individual variations in REE. The major hypothesis is that African Americans have a smaller mass of the most metabolically active organs (ie, liver, spleen, kidneys, heart, and brain)—and therefore a lower proportion of body mass as high-metabolic-rate (HMR) tissues—than do whites. If this hypothesis proves to be true, then the previously reported lower REE in African Americans may be explained totally or in part by differences in the composition of the body's FFM compartment.

SUBJECTS AND METHODS

Subjects

Subjects were recruited through advertisements placed in local newspapers or on radio stations and flyers posted in the local community (11). A body mass index (BMI; in kg/m^2) of 17 to

37 was set as a requirement for participation to accommodate MRI scanner limitations. Other inclusion criteria were that participants be ambulatory and nonvigorously exercising and to have no medical condition that could affect the variables under investigation. Each potential subject underwent a medical evaluation that included a physical examination and screening blood tests. Only healthy persons without any diagnosed medical condition and with normal thyroid hormone and cortisol values were enrolled. Race was ascertained on the basis of a subject's self-report that all 4 grandparents were either African American or white.

Written informed consent was obtained from all subjects. The study was approved by the Institutional Review Board of St Luke's–Roosevelt Hospital.

Body-composition measures

Body weight was measured to the nearest 0.1 kg by using a scale (Weight Tronix, New York, NY), and height was measured to the nearest 0.5 cm by using a stadiometer (Holtain, Crosswell, United Kingdom).

Tissues and organs—Liver, kidney, and spleen images were produced by using an axial T1-weighted spin echo sequence with 5-mm slice thickness, no interslice gap, and a 40 × 40-cm^2 field of view with a matrix of 256 × 192 and 2 as the number of excitations (NEX). Approximately 40 slices were acquired from the space reaching from the diaphragm to the base of the kidneys.

For brain volumes, 2 protocols were used during the course of the study: an axial orientation for data collected before 2001 and a coronal orientation for data collected after 2001. Approximately 29 brain images acquired by using the axial protocol were produced by using a body coil with a fast-spin echo T2-weighted sequence with 5-mm contiguous axial images and a 40 × 40-cm^2 field of view with a matrix of 256 × 256 and NEX of 1. For the coronal protocol, after the performance of a sagittal scout (localizer) to identify the anterior commissure–posterior commissure plane (1 min), a transaxial T1-weighted sequence with 1.5-mm slice thickness was acquired in a coronal plane orthogonal to the AC-PC plane over the whole brain with the following parameters: 3-dimensional spoiled gradient–recalled acquisition in the steady state); repetition time (TR) of 34 ms; time to echo (TE) of 5 ms; flip angle of 45°; slice thickness of 1.5 mm and zero gap; 124 slices; field of view, 22 × 16 cm; and a 256 × 192 matrix, reformatted to 256 × 256, which produced a voxel size of 1.5 × 0.9 × 0.9 mm. Acquisition time was 11 min. For the purpose of merging the brain data, a cross-calibration was carried out by using a sample of subjects in whom both protocols were performed. All axially derived volumes were converted to coronal volumes.

SLICEOMATIC image analysis software (version 4.2; Tomovision, Montreal, CA) was used to analyze all images on a personal computer workstation (Gateway, Madison, WI). MRI volume estimates were converted to mass by using the assumed density for each tissue and organ (liver, kidneys, and spleen: 1.05 kg/L; heart and brain: 1.03 kg/L). In our laboratory, the CV for the same scans read by 2 analysts for MRI-derived kidneys, liver, and spleen volumes in adults is 1.6%, 4.3%, and 15.7%, respectively.

During the course of the study, 2 protocols for measuring heart mass were used to derive left ventricular mass (LVM): echocardiography for data collected before 2001 and gated MRI of the heart for data collected since that time. During echocardiography, LVM was evaluated by using a two-dimensionally guided M-mode echocardiogram (Hewlett-Packard 1500, Boise, ID) interfaced with a strip chart recorder, a two-dimensional video recorder, and either a 2.5- or 3.5-MHz probe as described previously (11,17,18). LVM was multiplied by a factor of 1.50 to obtain a value for total heart mass (19). For a gated MRI of the heart, subjects were studied by using a dedicated phased-array cardiac coil (GE Medical Systems, Milwaukee, WI). The

heart was examined in a short axis section by using a k-space–segmented, single-breath-hold, gradient reversal technique (flip angle = 15°; TR = 8.9 ms; TE = 4.7 ms; slice thickness = 8 mm; interslice gap = 0). The entire ventricular mass was examined in a series of 8 to 12 acquisitions. At each anatomic location, images were obtained at 20 phases of the cardiac cycle, beginning 10 msec after the electrocardiographic R wave. For each anatomic level, the first image (obtained 10 msec after the R-wave) and the image with the smallest cavitary area were identified as the end-diastolic and end-systolic images, respectively. The epicardial and endocardial left ventricular borders of each end-diastolic and end-systolic image was measured by using a planimeter with an imager-based analysis program (MASS ANALYSIS, version 4.0.1; GE Medical Systems, Milwaukee, WI). Myocardial volume was obtained by summing the product of the myocardial slice area (epicardial area minus endocardial area) and the slice thickness over the entire heart. Myocardial mass was obtained by multiplying this volume by the specific gravity of the myocardium—ie, 1.05 g/cc. A single radiologist (LB), blind to the hypothesis, analyzed all cases. In a series of 10 normal subjects, the mean intraobserver variability for estimating left ventricular mass was 5.13 ± 2.9%; the interobserver variability was 9.01 ± 1.65% (20). All MRI studies were carried out without prior sedation while subjects were in a postabsorptive state. Although no cross-calibration study was performed, the statistical modeling described below could detect no significant differences in LVM that was attributable to the method of measurement.

Fat and fat-free mass—Total body fat and FFM (body weight − total body fat) were measured with a whole-body DXA scanner, model DPX (software version 3.6Y) or model DPXL (software version 4.7E; both: GE Lunar, Madison, WI). The between-measurement technical errors for total body fat and FFM in the same subject are 3.4% and 1.2%, respectively (21). The daily quality-control and calibration measures practiced in the DXA laboratory were described previously (22).

Energy expenditure

Subjects reported to the lab in the morning after an overnight fast, and REE was measured by using either the in-house–built Columbia respiratory chamber-indirect calorimeter (23) or a metabolic cart (DeltaTrac Monitor; Sensormedics, Yorba Linda, CA). Under thermoneutral conditions, subjects rested comfortably on a bed, and a plastic transparent ventilated hood was placed over their head for 40–60 min. Magnetopneumatic oxygen (Magnos 4G) and carbon dioxide (Magnos 3G) analyzers (both: Hartmann & Braun, Frankfurt, Germany) were used to analyze the rates of oxygen consumption and carbon dioxide production; the data displayed were then stored by the online computer system. For both the chamber and the cart, gas exchange results were evaluated during the stable measurement phase (10–20 min) and converted to REE (kcal/d) by using the formula of Weir (24). Standard gas calibrations were performed on both instruments before each subject was tested. For a standard alcohol phantom, gas concentration measurements are reproducible to within 0.8% (chamber) and 2.6% (cart).

Statistical analysis

Descriptive subject data are expressed as means ± SDs. Student's *t* test was used to compare unadjusted baseline characteristics. General linear models were used to determine the contribution of different variables in accounting for the summed mass of high metabolic rate organs—ie, Total OM—and REE. The dependent variables were log transformed when necessary for linear modeling because of heteroscedasticity and nonnormality of the residual; however, the estimates of the effects of race and REE from the transformed models differed little from those from the untransformed models (<8%), and the untransformed models tended to be more conservative in significance levels. To aid interpretability, the coefficients for the untransformed models are presented. Possible biasing effects of the 2 LVM measurement methods were explored by fitting regression models with LVM as the dependent variable and

with height, weight, FFM, age, and LVM measurement method as independent variables within each sex. Estimates of measurement method effect were < 10 g for both groups and did not approach significance. Data were analyzed by using SPSS for WINDOWS (version 12.0; SPSS, Chicago, IL). Statistical significance was set at $P < 0.05$ (2-tailed *t* test).

RESULTS

Body-composition characteristics

Unadjusted mean (±SD) subject characteristics are shown in Table 1. Because the African American women were significantly ($P = 0.002$) heavier than the white women and because the African American men were significantly ($P = 0.049$) younger than the white men, the unadjusted group means for all other variables were not compared. Also shown in Table 1 are the unadjusted mean values for Total OM and REE by sex for African Americans and whites. Differences in between-group baseline characteristics are taken into account in subsequent analyses by including those variables in the linear models.

Organ-tissue modeling

General linear models were used to examine the associations between Total OM (kg) and several sets of anthropometric or body-composition variables (Table 2). Model 1, a basic model for predicting Total OM from weight, height, sex, and age, showed that sex, weight, age, and height were significant predictors ($R^2 = 0.69$, SEE ≈ 0.30 kg). Model 2 confirmed the significant contribution of race/ethnicity to the regression of Total OM ($P < 0.001$, R^2 0.73, SEE ≈ 0.27 kg). According to the parameters of this regression equation, after adjustment for age, height, and weight, men had 0.31 kg more measured HMR organ tissue than did women, and whites had 0.25 kg more than did African Americans. In Model 3, another basic model for predicting Total OM from fat, FFM (replacing weight and height), sex, and age, showed that all except sex were significantly related to Total OM ($R^2 = 0.69$, SEE ≈0.30 kg). The addition of race/ethnicity to Model 4 again confirmed its contribution to predicting Total OM ($P < 0.001$, R^2 0.75, SEE ≈0.26 kg). Model 4 results indicate that whites had 0.30 kg more HMR organs than did African Americans (3.4 and 3.1 kg, respectively), after adjustment for differences in fat, FFM, sex, and age.

Modeling of resting energy expenditure

Given the finding that Total OM differed significantly between whites and African Americans, we used general linear models to investigate whether differences in the size of Total OM could account for observed race/ethnic differences in REE (Table 3). Model 1, a basic model for predicting REE from fat, FFM, sex, and age, showed that all variables were significant predictors of REE [$P < 0.005$, except sex ($P = 0.023$); $R^2 = 0.68$, SEE ≈ 150 kcal/d). The addition of race group in Model 2 confirmed the significant role of race in REE ($P = 0.002$, R^2 0.71, SEE ≈ 142 kcal/d), and the coefficient indicated that REE was ≈ 103 kcal/d lower in African Americans than in whites (1342 and 1445 kcal/d, respectively). In Model 3, a variable representing the sum of measured HMR organ masses, Total OM was added to Model 2, to ascertain whether information about the organ mass could account for the REE differences and render the race variable redundant. The Total OM variable made a significant ($P < 0.001$) contribution to the model, accounted for an additional 4% of the variance in REE, and reduced the SEE to 132 kcal/d. After the addition of Total OM, the remaining contribution of race, ≈ 40 kcal/d, was no longer significant ($P = 0.25$).

Similar regressions were carried out to evaluate the contribution of individual variation in Total OM to individual variation in REE, within each race/ethnicity group. In the African American group, fat, FFM, age and sex accounted for 65% of the variability in REE; the addition of Total OM increased the adjusted R^2 to 70% ($P = 0.01$). In the whites, fat, FFM, age, and sex accounted

for 74% of the variability; the addition of Total OM increased the adjusted R^2 to 76% (P = 0.013)

DISCUSSION

The primary purpose of this study was to ascertain whether the previously reported racial differences in REE could be explained by a lower combined volume of selected HMR organs (ie, liver, kidney, spleen, heart and brain) in African Americans than in whites. The first significant finding is that, after adjustment for relevant confounding variables, the mass of the HMR organs measured in this study is smaller in the African Americans than in the whites. The second major finding is that, when the mass of this HMR compartment is included as a variable in the prediction of REE, more than half of the observed difference in REE previously attributed to race is explained, and no significant difference in REE between African Americans and whites in this sample remains. As may be expected, variability in the mass of HMR organs also explained a portion of the observed within-race REE differences—specifically, an additional 5% and 2% of the variance in African Americans and whites, respectively. These findings highlight the influential role that subtle differences in phenotype may play in investigations of an important biological phenomenon such as REE.

Multiple regression analyses were conducted to explore the relations between REE and various independent variables. The addition of information about HMR organ mass to a regression model that already included FFM as an explanatory variable added significantly to the model and rendered the race term nonsignificant. The implication, therefore, is that African Americans may have a significantly smaller proportion of FFM as specific HMR organs than do whites, and this possibility, when considered in the prediction of REE from total FFM, helps explain the many previous reports of lower REE in African Americans than in whites.

Although the remaining 40 kcal/d racial difference in REE was not significant in this study, it is plausible that a larger study with more complete and precise quantification of HMR organs will find a difference of this size to be significant. What may account for the remaining race effect? One possibility is that not all HMR organs were measured in this study, and the unmeasured organs may be smaller in African Americans than in whites. Another possibility is that racial differences may exist in the specific metabolic rates of organs and tissues, but that has not yet been investigated.

Quantifying the mass of specific organs and tissues in vivo is possible with the use of imaging techniques, including MRI (11,25,26). Many previous investigations relating REE and body composition were hindered by the inability of the measurement techniques to quantify body composition at the organ-tissue level. The protocol described here is by no means exhaustive, and the selected HMR organs were chosen in part because of their ease of quantification.

These findings expand our knowledge of racial differences in body composition. It has been known for some time that, in comparison with whites, African Americans have significantly greater bone mass and skeletal muscle mass (15) and similar fat mass (14) but a different fat distribution pattern. We now add to this list of body-composition differences a smaller mass of a group of HMR organs (ie, liver, kidney, spleen, heart and brain) that we were able to measure in African American men and women. The clinical implications of these body-composition differences remain to be explored.

Study limitations

Limitations of the present cross-sectional study include the relatively large differences between the races in weight and other characteristics, the long time-span of the data acquisition that resulted in the use of multiple measurement methods and instruments over time, and the

relatively small sample of African American men. An assumed organ/tissue density developed from reference man data (27) was used for each organ and tissue quantified by MRI; it is unclear whether these density values can be accurately applied across race/ethnicity groups and across the entire adult age span. We did not quantify the volume of lungs, gastrointestinal tract, or stomach, all of which could also be characterized as HMR organs, because of the technical challenges posed in quantifying the mass of these and similar organs/ tissues.

Summary

Approximately half of the observed remaining difference in REE between African American and white men and women, after adjustment for age, fat, and FFM, may be explained by differences in the mass of several HMR organs (ie, liver, kidneys, spleen, heart and brain). African Americans may have a significantly smaller proportion of FFM as HMR organs than do whites, which may account for previous reports of lower REE among African Americans. The mass of HMR organs and tissues should be considered in future studies involving the assessment of REE across racial groups and may also contribute to accounting for individual variability in REE.

References

1. Albu J, Shur M, Curi M, Murphy L, Heymsfield S, Pi-Sunyer FX. Resting metabolic rate in obese, premenopausal black women. Am J Clin Nutr 1997;66:531–8. [PubMed: 9280169]
2. Foster GD, Wadden TA, Vogt RA. Resting energy expenditure in obese African American and Caucasian women. Obes Res 1997;5:1–6. [PubMed: 9061709]
3. Kushner RF, Racette SB, Neil K, Schoeller DA. Measurement of physical activity among black and white obese women. Obes Res 1995;3:261S–5S. [PubMed: 8581785]
4. Weyer C, Snitker S, Bogardus C, Ravussin E. Energy metabolism in African Americans: potential risk factors for obesity. Am J Clin Nutr 1999;70:13–20. [PubMed: 10393133]
5. Kaplan AS, Zemel BS, Stallings VA. Differences in resting energy expenditure in prepubertal black children and white children. J Pediatr 1996;129:643–7. [PubMed: 8917227]
6. Morrison JA, Alfaro MP, Khoury P, Thornton BB, Daniels SR. Determinants of resting energy expenditure in young black girls and young white girls. J Pediatr 1996;129:637–42. [PubMed: 8917226]
7. Sun M, Gower BA, Bartolucci AA, Hunter GR, Figueroa-Colon R, Goran MI. A longitudinal study of resting energy expenditure relative to body composition during puberty in African-American and white children. Am J Clin Nutr 2001;73:308–15. [PubMed: 11157329]
8. Wong WW, Butte NF, Ellis KJ, et al. Pubertal African-American girls expend less energy at rest and during physical activity than Caucasian girls. J Clin Endocrinol Metab 1999;84:906–11. [PubMed: 10084569]
9. Yanovski SJ, Reynolds JC, Boyle AJ, Yanovski JA. Resting metabolic rate in African-American and Caucasian girls. Obes Res 1997;5:321–5. [PubMed: 9285838]
10. Elia, M. Organ and tissue contribution to metabolic rate. In: Kinney, JM.; Tucker, HN., editors. Energy metabolism. Tissue determinants and cellular corollaries. New York: Raven Press; 1992. p. 61-77.
11. 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:E249–58. [PubMed: 9688626]
12. Grande, F. Energy expenditure of organs and tissues. In: Kinney, JM., editor. Assessment of energy metabolism in health and disease. Report of the first Ross Conference on Medical Research. Columbus, OH: Ross Laboratories; 1980. p. 88-92.
13. Elia, M. Tissue distribution and energetics in weight loss and undernutrition. In: Kinney, JM.; Tucker, HN., editors. Physiology, stress, and malnutrition. Philadelphia, PA: Raven Press; 1997. p. 383-411.
14. Gallagher D, Visser M, Sepulveda D, Pierson RN, Harris T, Heymsfield SB. How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups? Am J Epidemiol 1996;143:228 –39. [PubMed: 8561156]

15. Ortiz O, Russell M, Daley TL, et al. Differences in skeletal muscle and bone mineral mass between black and white females and their relevance to estimates of body composition. Am J Clin Nutr 1992;55:8–13. [PubMed: 1728823]

16. Hunter GR, Weinsier RL, Darnell BE, Zuckerman PA, Goran MI. Racial differences in energy expenditure and aerobic fitness in premenopausal women. Am J Clin Nutr 2000;71:500–6. [PubMed: 10648264]

17. Jahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantification in M-mode echocardiography: results of a survey of echocardiographic measurements. Circulation 1978;58:1072–83. [PubMed: 709763]

18. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. Circulation 1977;55:613–8. [PubMed: 138494]

19. Jones RS. Weight of the heart and its chambers in hypertensive cardiovascular disease with and without failure. Circulation 1953;7:357–69. [PubMed: 13033063]

20. Katz J, Whang J, Boxt LM, Barst RJ. Estimation of right ventricular mass in normal subjects and in patients with primary pulmonary hypertension by nuclear magnetic resonance imaging. J Am Coll Cardiol 1993;21:1475–81. [PubMed: 8473659]

21. Gallagher D, Kovera AJ, Clay-Williams G, et al. Weight loss in post-menopausal women: no adverse alterations in body composition and protein metabolism. Am J Physiol Endocrinol Metab 2000;279:124–31.

22. Song M, Ruts E, Kim J, Janumala I, Heymsfield S, Gallagher D. Sarcopenia and increased muscle adipose tissue infiltration in elderly African American women. Am J Clin Nutr 2004;79:874–80. [PubMed: 15113728]

23. Heymsfield SB, Allison DB, Pi-Sunyer FX, Sun Y. Columbia respiratory chamber indirect calorimeter: a new approach to air modeling. Med Biol Eng Comput 1994;32:406–10. [PubMed: 7967805]

24. Weir JB. New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol (Lond) 1949;109:1–9. [PubMed: 15394301]

25. Gallagher D, Allen A, Wang Z, Heymsfield SB, Krasnow N. Smaller organ tissue mass in the elderly fails to explain lower resting metabolic rate. Ann N Y Acad Sci 2000;904:449–55. [PubMed: 10865788]

26. McNeill G, Foster MA, Love J, Antfang V. Liver and kidney volume and their relationship to metabolic rate at rest. Proc Nutr Soc 1995;54:151A. [PubMed: 7568249](abstract)

27. Snyder, WS.; Cook, MJ.; Nasset, ES.; Karhaussen, LR.; Howells, GP.; Tipton, IH. Report of the Task Group on Reference Man. Oxford, UK: Pergamon Press; 1984.

TABLE 1

Subject characteristics[1]

	African American women (n = 34)	White women (n = 30)	African American men (n = 8)	White men (n = 27)
Age (y)	51.0 ± 23.1 (21–88)[2]	46.4 ± 17.8 (21–86)	34.3 ± 18.2 (19–68)	50.8 ± 20.6 (22–84)[3]
Body weight (kg)	71.7 ± 15.0[4]	61.6 ± 8.9	83.5 ± 10.7	78.0 ± 11.1
Height (m)	1.63 ± 0.06	1.63 ± 0.06	1.78 ± 0.06	1.77 ± 0.08
BMI (kg/m^2)	27.1 ± 5.1	23.3 ± 3.5	26.4 ± 3.2	24.9 ± 3.1
Fat by DXA (kg)	25.4 ± 10.7	19.2 ± 7.3	14.1 ± 4.8	15.1 ± 8.2
FFM by DXA (kg)	46.3 ± 6.3	42.5 ± 3.9	69.3 ± 8.5	62.9 ± 7.1
Total OM by MRI (kg)	2.99 ± 0.37	3.09 ± 0.34	3.68 ± 0.57	3.76 ± 0.49
REE (kcal/d)	1265 ± 210	1264 ± 182	1594 ± 219	1624 ± 221

[1] All values are $\bar{x}$ ± SD. DXA, dual-energy X-ray absorptiometry; FFM, fat-free mass; Total OM, sum of individual organ (liver, kidney, spleen, heart, and brain) masses by magnetic resonance imaging (MRI).

[2] Range in parentheses (all such values).

[3] Significantly different from American men, $P = 0.049$ (Student's t test).

[4] Significantly different from white women, $P = 0.002$ (Student's t test).

TABLE 2

Organ mass regression models[1]

Model and variable	Regression coefficient		*P*	R^2
	Estimate	SE		
Model 1: Total OM = age + height + weight + sex				0.69
Intercept	0.927	0.810	0.046	
Age	−0.008	0.002	< 0.001	
Height	1.05	0.506	0.041	
Weight	0.012	0.003	< 0.001	
Sex	0.39	0.093	< 0.001	
Model 2: Total OM = age + height + weight + sex + race				0.73
Intercept	1.223	0.756	0.109	
Age	−0.008	0.001	< 0.001	
Height	0.801	0.473	0.094	
Weight	0.016	0.003	< 0.001	
Sex	0.305	0.089	0.001	
Race	−0.251	0.062	< 0.001	
Model 3: Total OM = age + fat + FFM + sex				0.69
Intercept	2.122	0.280	< 0.001	
Age	−0.007	0.002	< 0.001	
Fat	0.008	0.004	0.029	
FFM	0.025	0.006	< 0.001	
Sex	0.260	0.138	0.063	
Model 4: Total OM = age + fat + FFM + sex + race				0.75
Intercept	1.848	0.032	< 0.001	
Age	−0.007	0.001	< 0.001	
Fat	0.010	0.003	0.004	
FFM	0.033	0.005	< 0.001	
Sex	0.025	0.132	0.853	
Race	−0.301	0.060	< 0.001	

[1] Total OM, the sum of individual organ (ie, liver, kidney, spleen, heart, and brain) masses by magnetic resonance imaging; FFM, fat-free mass. In the models, sex is represented as women, 0 and men, 1; race is represented as white, 0 and African American, 1. FFM was measured by using dual-energy X-ray absorptiometry.

TABLE 3

Resting energy expenditure (REE)–body-composition regression models

Model and variable	Regression coefficient		*P*	R^2
	Estimate	SE		
Model 1: REE = fat + FFM + age + sex				0.68
Intercept	804.7	141.1	< 0.001	
Fat	5.53	1.82	0.003	
FFM	11.4	2.84	< 0.001	
Age	−3.50	0.83	< 0.001	
Sex	161.8	69.8	0.023	
Model 2: REE = fat + FFM + age + sex + race				0.71
Intercept	710.7	138.1	< 0.001	
Fat	6.10	1.75	0.001	
FFM	14.1	2.84	< 0.001	
Age	−3.21	0.80	< 0.001	
Sex	81.1	71.4	0.259	
Race	−102.9	32.6	0.002	
Model 3: REE = fat + FFM + age + sex + race + Total OM				0.75
Intercept	321.2	159.6	0.047	
Fat	4.06	1.69	0.018	
FFM	7.22	3.13	0.023	
Age	−1.81	0.82	0.030	
Sex	76.0	66.1	0.253	
Race	−39.5	34.0	0.248	
Total OM	210.8	51.8	< 0.001	

[1] REE, resting energy expenditure (kcal · kg^{-1} · d^{-1}); FFM, fat-free mass (measured by using dual-energy X-ray absorptiometry; Total OM, the sum of individual organ (ie, liver, kidney, spleen, heart, and brain; in kg) masses as measured by magnetic resonance imaging. In the models, sex is represented as women, 0 and men, 1; race is represented as white, 0 and African American, 1.