

Estimation of Resting Energy Expenditure Considering Effects of Race and Diabetes Status

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OBJECTIVE— To evaluate the impact of diabetes status and race, in addition to other covariables, on the estimation of resting energy expenditure (REE).

RESEARCH DESIGN AND METHODS— Demographic, anthropometric, and clinical parameters were assessed in 166 adults of varying weights. Subjects were categorized by race (white versus black) and into three subgroups based on glucose tolerance (normoglycemia, impaired glucose tolerance, and type 2 diabetes), termed the diabetes status index (DSI). REE was measured by indirect calorimetry. A multiple regression model was established for optimal prediction of REE based on covariables.

RESULTS— An average decrease in REE of 135 kcal/day independent of all other variables was observed in blacks ($P < 0.001$). DSI was found to be a significant covariable ($P = 0.002$) in predicting REE, which was observed to be higher in diabetic women. Therefore, race and DSI entered the multiple regression equation to predict REE as significant independent variables, together with lean body mass (LBM) and age \times BMI interaction ($P < 0.001$). Overall, REE prediction resulted in an R^2 of 0.79 and a root mean square error of 136 kcal/day. These values indicate that the resultant equations could offer advantages over other key published prediction equations. The equations are: 1) $REE_{\text{female}} = 803.8 + 0.3505 \times \text{age} \times (\text{BMI} - 34.524) - 135.0 \times \text{race} + 15.866 \times \text{LBM} + 50.90 \times \text{DSI}$; and 2) $REE_{\text{male}} = 909.4 + 0.3505 \times \text{age} \times (\text{BMI} - 34.524) - 135.0 \times \text{race} + 15.866 \times \text{LBM} - 9.10 \times \text{DSI}$. The predictive value of the equations did not diminish substantially when fat-free mass estimated by skinfold calipers was substituted for dual-energy X-ray absorptiometry scan measurements of LBM.

CONCLUSIONS— Race and diabetes status are important when predicting REE, coupled with LBM, age, BMI, and sex. Race and DSI have not been considered in equations commonly used to predict REE. Their inclusion could improve individualization of dietary prescriptions for type 2 diabetic subjects and heterogeneous populations.

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Type 2 diabetes and obesity have emerged as leading public health challenges in western societies and constitute an increasing health burden in developing countries. Obesity increases

risk for diabetes in the context of the insulin resistance syndrome, a trait cluster consisting of insulin resistance, obesity, glucose intolerance, upper body fat distribution, hypertension, dyslipidemia, and

dysfibrinolysis (1–4). The insulin resistance syndrome is a major factor conferring increased risk for cardiovascular disease (1–4). Therefore, type 2 diabetes, obesity, and the insulin resistance syndrome are important targets for dietetic intervention. Often, nutrition professionals must estimate energy expenditure when measurements of metabolic rate are unavailable and must consider the potential influence of obesity, glucose intolerance, and diabetes, which could alter energy metabolism. As current dietary recommendations in the treatment of diabetes include care plans based on lifestyle factors and diabetes management goals (5,6), there remains a need for more accurate caloric expenditure assessment to better assist nutrition professionals in developing more individually designed treatments.

Over the past two decades, clinical and metabolic variables have been studied for their impact on resting energy expenditure (REE) and for their value in quantitative estimation of REE. Overwhelming evidence exists that lean body mass (LBM) is the major determinant of REE in any population (7–10), possibly accounting for 65–90% of the variance (8). Prediction of REE based on total body weight may overestimate true caloric needs, and this is underscored by the fact that REE in obese subjects has been shown to be virtually identical to that of nonobese subjects once normalized per LBM (10,11). Thus, equations to assess REE have been developed based on measures of LBM, fat-free mass, and fat mass (12–14). Commonly used equations predicting REE may not be appropriate for obese patients due to their inconsistent body compositions, resulting in variable REEs (13).

Race may also significantly influence REE. Several studies have documented a difference in REE by race (15), particularly in black women compared with white women (16–18). Although Weyer et al. (19) found no differences in REE or fat oxidation when comparing Pima Indi-

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Abbreviations: DEXA, dual-energy X-ray absorptiometry; DSI, diabetes status index; IGT, impaired glucose tolerance; LBM, lean body mass; REE, resting energy expenditure; RMSE, root mean square error; UWW, underwater weighing; WHO, World Health Organization.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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Table 1—Clinical characteristics and REE rates in white and black men and women with normoglycemia, IGT, and type 2 diabetes

Subgroup	n	Age (years)	BMI (kg/m ²)	LBM (kg)	REE (kcal/day)	REE (kcal · kg LBM ⁻¹ · day ⁻¹)
Whites						
Normoglycemia						
All	76	36 ± 11	27.4 ± 5.7	51.8 ± 12.1	1,581 ± 319	30.9 ± 3.4
Men	33	31 ± 9	26.8 ± 4.5	62.6 ± 8.1	1,836 ± 262	29.4 ± 3.0
Women	43	39 ± 11	27.9 ± 6.5	43.5 ± 6.8	1,386 ± 199	32.1 ± 3.3
IGT						
All	17	39 ± 11	27.8 ± 6.5	53.6 ± 11.5	1,649 ± 250	31.4 ± 4.7
Men	11	37 ± 13	29.0 ± 6.7	59.8 ± 9.0	1,769 ± 209	29.9 ± 3.2
Women	6	41 ± 6	25.6 ± 5.9	42.2 ± 4.7	1,430 ± 152	34.3 ± 5.8
Type 2 diabetes						
All	26	46 ± 10	27.8 ± 4.9	52.8 ± 10.7	1,630 ± 256	31.5 ± 5.3
Men	9	49 ± 8	26.6 ± 4.6	63.1 ± 7.2	1,739 ± 241	27.5 ± 2.1
Women	17	44 ± 11	28.4 ± 5.0	47.3 ± 7.8	1,572 ± 251	33.5 ± 5.4
Blacks						
Normoglycemia						
All	21	37 ± 8	27.3 ± 5.1	53.7 ± 12.0	1,491 ± 261	28.2 ± 3.5
Men	9	32 ± 8	26.2 ± 3.6	65.6 ± 6.0	1,695 ± 111	26.0 ± 2.2
Women	12	41 ± 6	28.2 ± 6.0	44.8 ± 5.7	1,339 ± 236	29.9 ± 3.3
IGT						
All	5	30 ± 7	36.1 ± 10.9	59.8 ± 11.7	1,650 ± 258	27.9 ± 2.5
Men	3	32 ± 9	35.2 ± 12.8	63.6 ± 9.0	1,686 ± 171	26.7 ± 2.6
Women	2	27 ± 3	37.5 ± 12.0	54.2 ± 16.8	1,595 ± 445	29.6 ± 0.9
Type 2 diabetes						
All	21	47 ± 7	31.4 ± 5.7	52.3 ± 11.6	1,541 ± 270	29.8 ± 3.3
Men	5	48 ± 7	30.1 ± 3.1	69.4 ± 9.7	1,842 ± 260	26.6 ± 1.2
Women	16	46 ± 7	31.8 ± 6.3	47.0 ± 5.2	1,447 ± 200	30.8 ± 3.0

Data are means ± SD.

ans with whites, significantly lower values for REE and fat oxidation have been observed (20–22) in blacks. In addition, conditions including critical illnesses, thermal injury, and diabetes (23–25) also affect metabolic rate and require more specific equations to predict energy requirements when calorimetry is unavailable. Despite evidence that diabetes and race can influence REE corrected for LBM, no predictive equation estimating REE has been developed to address these issues in sample populations (26–28). No evidence has been published on racial differences among patients who have type 2 diabetes, even though blacks are twice as likely to develop diabetes compared with whites (29). Therefore, it is important to develop tools that predict REE in patients with diabetes and in blacks as a guide to dietary prescriptions.

Because existing equations for estimating REE have not optimally considered the effects of type 2 diabetes and race, we had two goals in this work: 1) to assess the effects of diabetes status (i.e.,

normoglycemia, impaired glucose tolerance, and type 2 diabetes) and race on REE and to develop a predictive equation incorporating these parameters; and 2) to compare the accuracy of key published equations for predicting REE.

RESEARCH DESIGN AND METHODS

The protocol was approved by the institutional review board, and written informed consent was obtained from every subject. Data were collected on 96 women and 70 men (total n = 166), aged 18–64 years. All subjects were recruited in a similar manner; that is, subjects were sequentially entered into the protocol as they responded to ads placed in the local metropolitan newspaper. This study group was comprised of 117 whites and 49 blacks (5 Hispanic volunteers were classified as whites for study purposes). Table 1 characterizes the number, age, BMI, and LBM of the study population stratified by sex, race, and diabetes status (normoglycemia, impaired glucose tolerance, and type 2 dia-

betes). In normoglycemic individuals, the mean waist-to-hip ratio and body fat percentage by dual-energy X-ray absorptiometry (DEXA) were 0.90 ± 0.02 and 20.3 ± 0.8% in white men, 0.86 ± 0.01 and 19.8 ± 0.5% in black men, 0.78 ± 0.02 and 37.3 ± 1.1% in white women, and 0.81 ± 0.03 and 35.8 ± 1.4% in black women, respectively. In patients with type 2 diabetes, the waist-to-hip ratio and body fat percentage were 0.95 ± 0.02 and 20.8 ± 0.1.9% in white men, 0.95 ± 0.01 and 23.6 ± 1.7% in black men, 0.87 ± 0.02 and 35.3 ± 1.5% in white women, and 0.90 ± 0.03 and 39.2 ± 1.6% in black women, respectively. Among nondiabetic subjects, whites had lower systolic (117 ± 1 vs. 124 ± 3 mmHg) and diastolic (66 ± 1 vs. 70 ± 2 mmHg) blood pressure than blacks (P < 0.05). The whites with type 2 diabetes had similar mean HbA_{1c} values compared with their black counterparts (7.9 ± 0.1 vs. 8.1 ± 0.2%; P = NS). All subjects were chemically euthyroid and without renal, hepatic, or cardiac disease.

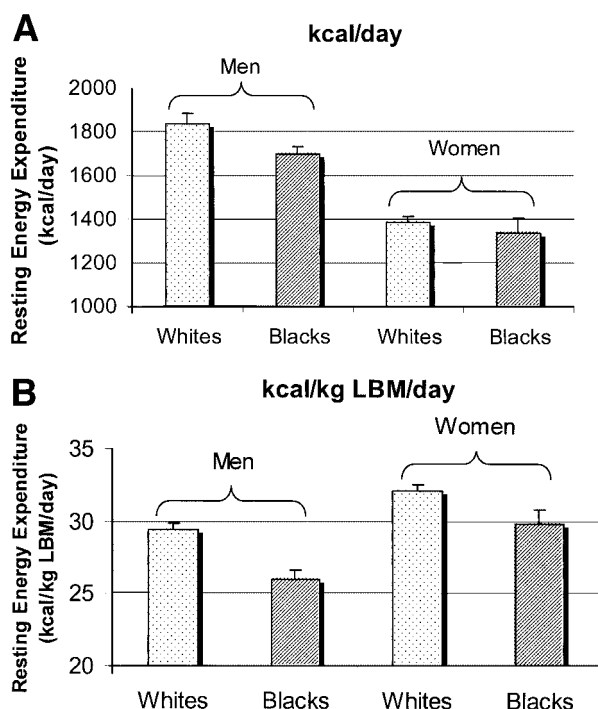


Figure 1—The effects of race and sex on rates of REE. REE was measured by indirect calorimetry in white and black men and women with normal glucose tolerance. REE data were expressed as kilocalories per day (A) and were normalized per kilogram LBM (B) as determined by DEXA. The height of each bar represents the mean \pm SD.

The patients with type 2 diabetes had been treated with diet, metformin, sulfonylureas, and/or insulin, but were withdrawn from therapy before study. One to 2 weeks were allowed for medication washout if patients were poorly controlled, with $HbA_{1c} \geq 9\%$, and 2–3 weeks if patients had an initial $HbA_{1c} < 9\%$. No subjects were on any other medications known to affect carbohydrate or lipid-lowering metabolism.

Before and during the study, subjects were equilibrated on an isocaloric diet ($28\text{--}32 \text{ kcal} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$) consisting of 50% carbohydrates, 30% fat, and 20% protein. Subjects were instructed to maintain usual activity levels, and no subjects engaged in regular exercise. Oral glucose tolerance tests were performed per the National Diabetes Data Group (30) to determine diabetes status, and individuals were categorized into subgroups of normoglycemia, impaired glucose tolerance (IGT), and type 2 diabetes. No subjects had gestational diabetes, type 1 diabetes, or atypical diabetes, and all possessed a fasting serum C-peptide level $>1.2 \text{ ng/ml}$, measured using a chemiluminescent immunoassay (Immulite 2000; Diagnostic Products, Los Angeles, CA). Plasma

glucose was measured by the glucose oxidase method with a glucose analyzer (YSI 2300; YSI, Yellow Springs, OH). Clinical characteristics of the study subjects are shown in Table 1.

Body fat percentage and LBM were measured by DEXA (Lunar, Madison, WI) as described (31). Fourteen subjects were too large or tall for this scanning device, and their body fat percentage and LBM were determined by underwater weighing (UWW). DEXA measurements of body fat are highly correlated with those from UWW over the age range of our population (32,33). Constant-tension calipers were used to measure skinfold thickness (Lange Caliper; Beta Technology, Santa Cruz, CA) and to estimate fat-free mass according to the method of Pollock et al. (34).

After an overnight fast, REE was measured for 30 min. REEs were determined by computerized, open-circuit, indirect calorimetry (Deltatrac II; SensorMedics, Yorba Linda, CA) as described (35,36). Whole-body oxygen consumption (V_{O_2}) and CO_2 production (V_{CO_2}) were calculated by measuring gradients across the face and the flow rates of air using the Haldane transformation. Energy expendi-

ture was determined from the respiratory quotient value.

Multiple regression analyses were performed using Stata 7.0 software to develop prediction equations of REE. Height and weight were used to calculate BMI; the latter was entered as a regression variable, whereas height and weight were omitted. BMI and LBM were separately assessed as predictor variables for REE. Additionally, diabetes status and race were introduced as possible predictive factors consistent with our hypothesis that diabetes and race are independently related to REE, independent of age, sex, and anthropometric measures (15–20, 26–28,37). For data analyses purposes, a DSI was created to be used in REE prediction models, coded as normal = 0, IGT = 1, and type 2 diabetes = 2. Similarly, sex was coded as female = 0 and male = 1, and race was coded as white = 0 and black = 1. Quadratic functions of the continuous variables were assessed. A binary variable indicating the few UWW-based LBM values was also evaluated. The stepwise procedure was used to assess the significance of the linear terms. The forward selection method was used to assess the significance of the multiple interaction terms, and all interactions involving two variables were examined.

RESULTS— Table 1 and Fig. 1 show mean REE values in men and women stratified by race and DSI. Three salient points emerge. First, as illustrated in Fig. 1, REE values (in kilocalories per day) are higher in normoglycemic men than women, which is consistent with a greater body mass in men. In contrast, REE values normalized per kilogram LBM are higher in women than men ($P < 0.05$). Table 1 indicates that these sex differences are observed in all three categories of glucose tolerance and apply to both whites and blacks. Second, black men ($P < 0.01$) and women ($P < 0.05$) had lower REE values (in both kilocalories per day and kilocalories per kilogram LBM per day) than their white counterparts (Fig. 1), despite the fact that age, BMI, and LBM were similar in comparing race and sex subgroups (Table 1). Lower REE rates in blacks were present in all three categories of glucose tolerance (Table 1). Third, type 2 diabetes was associated with a modest increase in REE/LBM because black men and black women with type 2 diabetes and white women with type 2

Table 2—Coefficients and significance of variables in the multiple regression equation predicting REE

Variable	Coefficient	SE	t	P*	Partial R ²
Age	−12.1	2.0	−6.12	<0.001	0.192
Sex	105.6	43.7	2.42	0.017	0.036
Race	−135.0	24.5	−5.51	<0.001	0.161
Age × BMI	0.351	0.058	6.14	<0.001	0.193
LBM	15.9	1.8	8.88	<0.001	0.333
DSI	50.9	15.8	3.23	0.002	0.062
Sex × DSI	−60.0	26.1	−2.30	0.023	0.032
Constant	803.8	98.3	8.17	<0.001	—

*Two sided.

diabetes tended to have somewhat higher REE/LBM values than their normoglycemic counterparts (Table 1). Fasting glucose was not correlated with resting metabolic rate among nondiabetic or diabetic patients when considered as separate groups; thus, it appears that diabetes status per se is the critical determinant rather than severity of fasting hyperglycemia.

We performed multiple regression analysis to develop an optimal equation for predicting REE. From the analyses in Table 1, we anticipated that predictive equations would take into account race and diabetes status. The results of the multiple regression analyses are shown in Table 2, which lists the multiple independent predictors for REE entering the regression equation on the basis of statistical significance, as well as partial R² values. When included in the regression, BMI and age × BMI were each significant when the other effect was excluded. However, when both appeared in the model, age × BMI remained significant (P = 0.002) but not BMI (P = 0.182). Only age × BMI is retained, as described in Table 2. Even with age × BMI in the regression equation, LBM retained an independent and additive predictive value. In fact, LBM was the most significant single predictor of REE, with nearly 16 kcal added for every incremental kilogram of LBM. Importantly, both race and diabetes status could independently and significantly explain variation in REE, even with age, sex, age × BMI, and LBM in the regression equation. Significant quadratic effects were not detected, and other interactions were not statistically significant. The UWW indicator was not significant (P = 0.99); thus, use of this method to assess body composition in a small number of individuals who could not be stud-

ied using DEXA was not problematic. The value of R² for the overall regression equation was 0.79, the root mean square error (RMSE) was 136.0 kcal/day, and the conditional variances of REE did not vary significantly (P = 0.16).

Most previous publications present equations for predicting REE in adults in a sex-stratified format. Although sex in the context of Table 2 is not the most significant variable, we expressed sex-specific equations in accordance with common practice: 1) REE_{female} = 803.8 + 0.3505 × age × (BMI − 34.524) − 135.0 × race + 15.866 × LBM (kg) + 50.90 × DSI; and 2) REE_{male} = 909.4 + 0.3505 × age × (BMI − 34.524) − 135.0 × race + 15.866 × LBM (kg) − 9.10 × DSI.

A remarkable aspect of these equations is the inclusion of a term for diabetes status (P = 0.002). Furthermore, the coefficient for diabetes status differs significantly between women and men (P = 0.023), and this reflects the sex × DSI interaction term appearing in Table 2 before sex stratification. It is possible to compute such equation pairs separately by sex for normal, IGT, and type 2 diabetic subjects by introducing a second DSI variable (yielding six equations in all), but the statistical advantage was deemed not significant (P = 0.50) and therefore not presented.

Our model also explicitly incorporates the race variable into the REE predictive equations, unlike those developed in previous studies (Fig. 2). Quantitatively, black racial identification was independently associated with a decrease in predicted REE of 135 ± 24 kcal/day compared with whites.

In various clinical scenarios, health care professionals may not have access to

DEXA scan measurements of LBM for use in the predictive equations and may need to rely on caliper measurements of body fat to calculate fat-free mass (34). Therefore, we assessed the adequacy of the equations using fat-free mass values derived from skinfold caliper measurements. The value of R² for the overall regression equation was 0.76, and the RMSE was 138.7 kcal/day, indicating that the predictive value of the equations did not deteriorate substantially.

CONCLUSIONS—Based on our multiple regression analyses, diabetes status (normal, IGT, and type 2 diabetes) and race (white and black) independently contribute to variability for REE, and equations used to predict REE can be optimized by inclusion of these variables. Regarding diabetes status, there was a sex × DSI interaction relevant to REE prediction, such that diabetes status is an important predictor of REE in women. The inclusion of race was of particular importance because blacks averaged 135 kcal lower daily REE than whites, independent of other factors that may be related to race, such as BMI, LBM, and DSI. If race was excluded from the analyses, the R² value would decrease from 0.79 to 0.75. Notably, no interactions involving race were significant in the model. Based on these data, we recommend that diabetes status, race, and LBM all be considered in REE estimation.

After testing all possible pairwise interactions, only one interaction showed statistical significance, namely age × BMI. When this interaction was included in the model, age remained significant and BMI did not; therefore, BMI by itself was removed in the interest of model simplicity. When the BMI sample mean, 28.25 kg/m², was introduced into the equations, the coefficient for the age variable became −2.199. The negative sign is in agreement with other key equations, such as the Harris Benedict equation (38), the 1985 revised World Health Organization (WHO) equation (7), and other commonly used equations described in Fig. 2 (discussed below), but the magnitude of the coefficient is smaller. We believe this is a consequence of our inclusion of the LBM variable. Moreover, it is also worth noting that, for obese subjects, such a conditional age coefficient becomes positive. However, because LBM and age × BMI likely experience a surrogate partial

Equation	Reported R ² Value	RMSE value	Population Description
Current equation Female: $803.8 + 0.3505 \cdot \text{Age} \cdot (\text{BMI} - 34.524) - 135.0 \cdot \text{Race} + 15.866 \cdot \text{LBM (kg)} + 50.90 \cdot \text{DSI}$ Male: $909.4 + 0.3505 \cdot \text{Age} \cdot (\text{BMI} - 34.524) - 135.0 \cdot \text{Race} + 15.866 \cdot \text{LBM (kg)} - 9.10 \cdot \text{DSI}$	Overall R ² = 0.79	136.0	n=166; Lean and obese; white and black; Normal, impaired diabetic glucose tolerance
Harris, Benedict (1919) Male: $66.5 + 13.75(\text{wt in kg}) + 5.9(\text{Ht in cm}) - 6.76(\text{age})$ Female: $655.1 + 9.6(\text{wt in kg}) + 1.86(\text{Ht in cm}) - 4.68(\text{age})$	Male R ² = 0.75 Female R ² = 0.53 (original publication)	160.1	n=239; subjects skewed towards young, white, lean
World Health Organization (1985) Male: 18-30 yrs $(15.4 \times \text{wt in kg}) - (27 \times \text{height in m}) + 717$ 31-60 yrs $(11.3 \times \text{wt in kg}) + (16 \times \text{ht in m}) + 901$ >60 yrs $(8.8 \times \text{wt in kg}) + 1128 \times \text{ht in m} - 1071$ Female: 18-30 yrs $(13.3 \times \text{wt in kg}) + (334 \times \text{ht in m}) + 35$ 31-60 yrs $(8.7 \times \text{wt in kg}) - (25 \times \text{ht in m}) + 865$ >60 yrs $(9.2 \times \text{wt in kg}) + (637 \times \text{ht in m}) - 302$	Overall R ² = 0.59 18-30 male R ² = 0.42 31-60 male R ² = 0.36 <60 male R ² = 0.71 18-30 female R ² = 0.53 31-60 female R ² = .49 >60 female R ² = 0.67	168.3	n=11,000; Many ethnic groups and broad BMI range
Mifflin, St. Jeor (1990) Male: $(10 \times \text{wt}) + (6.25 \text{ ht}) - (5 \times \text{age}) + 5$ Female: $(10 \times \text{wt}) + (6.25 \times \text{ht}) - (5 \times \text{age}) - 161$	Overall R ² = 0.71	160.3	n=498; Lean and obese; Racial distribution unclear
BMI equation: Harrington et al (1997) Male: $(\text{BMI} \times 28.15) - (\text{age} \times 6.44) + 1290$ Female: $(\text{BMI} \times 28.15) - (\text{age} \times 6.44) + 905$	Overall R ² = 0.62	177.0	Same population as Mifflin, St. Jeor (1990)
Cunningham (1991) 370 + 21.6(FFM)	R ² not stated; equation derived from 8 other studies	163.2	n=1483; Lean and obese; Different ethnic groups
Ferraro/Ravussin (1992) 671 + 14.6 (FFM in kg) + 7.3(FM in kg) - 3.2(age) + 120 for males	Overall R ² = 0.82	155.0	n=342; Lean and obese; white/Pima Indian
Owen (1988) 186 + 23.6(FFM)	R ² = 0.71	163.2	n=104; Lean and obese; Different ethnic groups
Weigle (1988) 245 + 20.4(FFM)	R ² = 0.92	163.2	n=28; Obese and previously obese; white

Figure 2—Comparison of the equations that predict REE in study populations. RMSE is calculated for each equation using current data. It estimates variability from both biological and statistical sources around an REE prediction for any given individual. FFM, fat-free mass; FM, fat mass.

inclusion in earlier models through correlation with other predictors, caution should be exercised in comparing current coefficients for age, sex, and BMI with previously published equations. Despite the high degree of significance for age \times BMI ($P < 10^{-8}$) in the multiple regression equation, LBM exerted effects independent of age \times BMI and remained the strongest predictive variable for prediction of REE ($t = 8.88$, degrees of freedom [df] = 158, $P < 10^{-15}$).

In the current studies, we used DEXA scans to quantify LBM, which excludes bone mass as well as fat mass from the total body mass. DEXA scans may not be available in nonresearch settings; however, the predictive value of the REE equations was only minimally diminished when caliper-derived measurements of fat-free mass were substituted for DEXA scan measurements of LBM. Therefore, health care professionals trained in the

use of skinfold calipers to assess body fat percentage can apply these equations in common clinical situations when DEXA scan data are not available. This is consistent with the observation that LBM is a very important factor, even with BMI accounted for in the regression equation. Other methods to assess LBM, such as estimation equations (38), creatinine clearance, or portable bioelectric impedance, were not tested in the current study.

Figure 2 describes eight key equations we chose to compare for their prediction of REE in our sample population of white and black subjects with variable body weights and glucose tolerances. The first three equations, the Harris Benedict (39), the 1985 revised WHO (7), and the Mifflin-St. Jeor (40), were selected because of their familiarity and common use in clinical practice. All include weight, height, age, and sex as variables. For validation, the Harris Benedict equation has

been studied in healthy, mostly lean, white subjects, the WHO equation in a large population with assorted ethnicities and BMIs, and the Mifflin-St. Jeor equation in lean and obese subjects of uncertain racial distribution (7,39,40). A fourth equation, termed the BMI equation in Fig. 2, was developed using BMI as one of its variables (41) from the same population used for the Mifflin-St. Jeor equation. The BMI equation is worth exploring because BMI is easily obtained and is a widely recognized and recommended tool to assess adiposity (42). We also studied equations developed by Cunningham (8), Ferraro and Ravussin (9), and Owen (43) because these studies used large sample sizes and included multiracial groups with a wide range of adiposity. Finally, an equation by Weigle et al. (44) was studied for its inclusion of obese and previously obese subjects (Fig. 2). Although the Harris Benedict, Mifflin-St. Jeor, WHO, and BMI

equations are still used for practicality and the Cunningham, Ferraro and Ravussin, Owen, and Weigle equations are compared for their accuracy when body composition is known, none utilize reference to diabetes status or race affiliation.

The R^2 correlation coefficients of all eight equations were calculated and compared in Fig. 2. All equations were found to be predictive of REE, with R^2 values ranging from 0.59 to 0.92 in the original publications. The Ferraro and Ravussin ($R^2 = 0.82$) and Weigle ($R^2 = 0.92$) equations were reported to have higher R^2 values than our equations' observed value of 0.79. Notably, these studies involved more homogeneous sample populations. The study by Weigle et al. (44) included 42 obese and previously obese whites, of whom 7 were the same subjects longitudinally compared at baseline versus a 21.5% weight loss. Ferraro and Ravussin studied 342 nondiabetic whites and Pima Indians, and Native American status was found to not affect REE when compared with whites. Predictably, had these populations included diabetic or black individuals, the predictive value of the equations would be reduced since these variables were not considered. The more widely used equations, such as the Harris-Benedict, WHO, Mifflin-St. Jeor, and the BMI formula, appeared to be the least predictive, based on R^2 values. In comparing the utility of the various equations, it is important to consider that each equation will be biased because the authors' equations are those that best fitted their own data. Figure 2 also illustrates RMSE values, which reflect the biological variability or confidence limits around an REE prediction for any given individual. When used to analyze our current dataset, the previously published equations were found to have RMSE values of 155 to 177, which are considerably higher than the value of 136 using the current equation. Therefore, our equation can predict REE with improved confidence, despite the fact that our sample population reflects variable glucose tolerances, adiposity levels, and races.

The current data support the clinical utility and application of the proposed predictive equations in two ways. First, the R^2 and RMSE values of the equations were comparable or superior to other equations in common use and were only minimally diminished when caliper-derived measurements of fat-free mass

were substituted for DEXA scan measurements of LBM. Second, the current equations are unique in accounting for the effects of race and diabetes status. Ignoring these variables could potentially lead to suboptimal clinical outcomes. For example, other commonly used equations will tend to overestimate REE in blacks, giving lower mean REE rates in this racial group compared with whites. If this is not accounted for, weight loss in blacks given prescribed hypocaloric diets will be less than the predicted result, delaying or preventing the achievement of desired therapeutic goals.

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