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Short communication

Is resting metabolic rate different between men and women?

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A low resting metabolic rate (RMR) has been proposed as a possible cause for the increased body fat commonly seen in women compared with men. Absolute RMR is higher in men, but whether RMR adjusted for lean body mass (LBM) remains higher is unresolved. The objective of the present study was to determine whether RMR adjusted for various body composition factors differed between healthy adult men and women. Thirty men (28·3 ± 8·0 years, BMI 23·7 ± 2·1 kg/m^2) and twenty-eight women (28·7 ± 6·9 years, BMI 22·2 ± 1·9 kg/m^2) were included in the analyses. RMR was measured by open-circuit indirect calorimetry for 60 min. Extracellular water (ECW) was measured by corrected Br^- space and total body water (TBW) by ^2H dilution. LBM was estimated as TBW/0·732. Intracellular water (ICW) was calculated as TBW−ECW, and body cell mass (BCM) as ICW/0·732. Men were heavier and had higher BMI, LBM, BCM and ECW, but less fat mass. Absolute RMR was higher in men than women (7280 ± 844 *v.* 5485 ± 537 kJ/d, $P<0·0001$). This difference became non-significant when RMR was adjusted for LBM by ANCOVA (6536 ± 630 *v.* 6282 ± 641 kJ/d, $P=0·2191$), but remained significant when adjusted for BCM (6680 ± 744 *v.* 6128 ± 756 kJ/d, $P=0·0249$). Fat mass explained a significant amount of variation in RMR in women (r^2 0·28, $P=0·0038$), but not in men (r^2 0·03, $P=0·3301$). The relationships between body fat and the various subcompartments of BCM and RMR require further elucidation.

Resting metabolic rate: Body composition: Lean body mass: Fat mass

Resting metabolic rate (RMR), the largest component of total daily energy expenditure, plays a significant role in the regulation of energy balance; a low RMR is predictive of weight gain (Ravussin *et al.* 1988). Factors influencing RMR include age (Molnar & Schutz, 1997), nervous system activity (Poehlman *et al.* 1997), ethnicity (Foster *et al.* 1997), genetics (Bogardus *et al.* 1986) and, perhaps most importantly, body composition (Jensen *et al.* 1988; Welle & Nair, 1990; Dionne *et al.* 1999; Weyer *et al.* 1999).

More specifically, lean body mass (LBM) has been found to be the single best determinant of RMR in both men and women (Cunningham, 1980; Arciero *et al.* 1993); the relationship between LBM and RMR has therefore been investigated to explain differing rates of weight gain in various subgroups. For example, the higher prevalence of obesity in women than men (World Health Organization, 2000; National Institutes of Health, 1998) coupled with the role of RMR in maintaining energy balance has led to the suggestion that the propensity to gain weight is largely due to a lower RMR in women (Arciero *et al.* 1993). Absolute RMR is higher in men, but whether this difference persists once RMR is adjusted for LBM remains controversial. Using the two-compartment whole body model of body composition (Heymsfield *et al.* 1997), some groups have reported LBM-adjusted RMR to be higher in men (Ferraro *et al.* 1992; Arciero *et al.* 1993; Goran *et al.* 1994; Molnar & Schutz, 1997; Poehlman *et al.* 1997), whereas others have not (Ravussin *et al.* 1986; Owen, 1988; Mifflin *et al.* 1990; Klausen *et al.* 1997; McCrory *et al.* 1998).

These contradictory findings may be explained, in part, by the heterogeneity of the LBM compartment, which contains both extracellular mass (skeleton, cartilage, connective tissue, lymph and plasma) and body cell mass (BCM; skeletal muscle and organs). This further subdivision into the cellular model of body composition

Abbreviations: BCM, body cell mass; CBS, corrected Br^- space; ECW, extracellular water; FM, fat mass; ICW, intracellular water; LBM, lean body mass; RMR, resting metabolic rate; TBW, total body water.

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(Heymsfield *et al.* 1997) is not trivial, as BCM is responsible for all of the O_2 consumption, CO_2 production and work performed by the body (Moore, 1980) and can therefore be considered the metabolic 'furnace'. Adjusting RMR for LBM makes the fundamental assumption that BCM maintains a constant relationship with the larger LBM compartment, both within and between subjects. This is now recognized not to be the case (Weinsier *et al.* 1992; Gallagher *et al.* 1996).

Therefore, the objective of the current study was to determine whether RMR adjusted for various body composition factors including weight, LBM, BCM and fat mass (FM) would be significantly different between healthy adult men and women.

Methods

Subjects

Fifty-eight apparently healthy adult men and women were recruited from the University of Toronto and Ryerson University in Toronto, Canada. None had a history of diabetes, Crohn's disease, hyper- or hypothyroidism, or renal or heart disease. Females were in the self-reported follicular phase of menses. The study was approved by the Research Ethics Board of The Hospital for Sick Children, Toronto, Canada.

Procedures

Studies were carried out during a 1 d visit to the Clinical Investigation Unit of The Hospital for Sick Children. Subjects arrived after a 12 h fast and gave informed consent. All measurements were conducted with subjects wearing only a hospital gown and undergarments. Weight was measured to the nearest 0·1 kg on a beam balance scale (Detecto model; Cardinal Scales, Web City, MO, USA) and height to the nearest 0·1 cm with a stadiometer (Holtain Ltd., Crymmych, UK). After a baseline blood sample (15 ml), each subject was given an oral dose of water labelled with ^2H_2O for the measurement of total body water (TBW) and NaBr for the measurement of extracellular water (ECW). The dosages were as follows: 0·25 g 99·9 atom percent ^2H_2O (CDN Isotopes, Pointe-Claire, Quebec, Canada)/kg estimated TBW (assuming 60 % of body weight is TBW) and 1·0 ml 30 % NaBr (Fisher Scientific, Nepean, Ontario, Canada)/kg body weight. The container was then rinsed with approximately 30 ml deionized water, which the subject subsequently drank to ensure that the entire dose was consumed. A plateau blood sample (15 ml) was obtained 3 h after administration of the ^2H_2O and NaBr (Schoeller *et al.* 1980; Vaisman *et al.* 1987); subjects remained fasted during the equilibration period. Blood samples were spun in a refrigerated centrifuge (Beckman J6B Centrifuge, Beckman Coulter Inc., Fullteron, CA, USA) at 1200 g for 10 min and plasma samples were stored at −20°C until analysed.

Body composition

Plasma samples were analysed for their ^2H_2O content using an isotope ratio mass spectrometer (CF-IRMS, Model ANCA GSL; Europa Scientific Inc., Crewe, UK), following equilibration with H_2 gas (Scrimgeour *et al.* 1993). TBW was calculated as

$$[((\text{Dose} \times 99{\cdot}9)/20) \times \text{APE} \times (18{\cdot}02/1000)]/1{\cdot}04,$$

where Dose is dose of ^2H_2O in g, 99·9 is the atom percent of ^2H_2O, 20 is the molecular weight of ^2H_2O, APE is atom percent excess ($AP_{plateau} - AP_{baseline}$), 18·02 is the molecular weight of unlabelled water, 1000 converts moles to litres and 1·04 is the correction for H_2 dilution space. The intra-assay CV for a standard 200 ppm ^2H solution was 0·11 % and for a 300 ppm solution was 0·06 %. The intraindividual CV for plasma ^2H atom percent was 0·45 %.

ECW was estimated as corrected Br^- space (CBS) from plasma samples by the Br^- dilution technique (Vaisman *et al.* 1987). Br^- concentration in the ECW space was determined from a 0·05 ml plasma sample by neutron activation of the stable ^{79}Br to ^{80}Br (Jervis *et al.* 1977) and using the following equation:

$$\text{CBS} = (\text{Br dose}/\text{plasma Br enrichment at } 3\,\text{h}) \times 0{\cdot}90 \times 0{\cdot}95 \times 0{\cdot}94,$$

where 0·90 is the correction for non-extracellular Br^- distribution, 0·95 is the Donnan equilibrium factor, and 0·94 is the correction for water in the plasma. The intra-assay CV for a standard 0·2 % Br solution was 0·65 % and the intraindividual CV for plasma Br^- concentration was 6·05 %. LBM in kg was estimated as TBW (l)/0·732, based on a fat-free tissue hydration constant of 73·2 % (Pace & Rathbun, 1945). Intracellular water (ICW) in litres was calculated as TBW − ECW, and BCM in kg was calculated as ICW/0·732. FM in kg was calculated as body weight − LBM.

Resting metabolic rate

RMR was measured during the ^2H_2O and NaBr equilibration period by continuous open-circuit indirect calorimetry (2900 Energy Expenditure Unit or Vmax Series, both Sensormedics, Yorba Linda, CA, USA) in a thermoneutral environment. Instruments were calibrated prior to measurement in each subject against standard mixed reference gases (4 % CO_2, 16 % O_2 and the balance N_2). Expired air was collected by means of a ventilated hood for 60 min; after a 20 min rest period, the last 40 min of steady-state data were used in calculations. During the measurement period subjects remained supine, were instructed not to talk or fidget, and watched television to reduce boredom and to prevent sleeping. Data for each subject were carefully reviewed; those minutes during which the subject may have moved, laughed, spoken or fallen asleep were deleted. The percentage of predicted RMR (Schofield, 1985) was calculated as $(RMR_{measured}/RMR_{predicted}) \times 100$. External validity of each instrument was tested regularly for the duration of the study by oxidation of 5 ml (3·94 g) ethyl alcohol. For the V_{max} and 2900 units, respectively, the CV between expected and observed CO_2 production was 0·26 and 1·23 %, O_2 consumption was 2·07 and 1·52 % and respiratory quotient was 2·35 and 1·91 %.

Table 1. Age and body composition of fifty-eight apparently healthy adult men and women
(Mean values with their range and standard deviations)

	Men (*n* 30)			Women (*n* 28)			
	Mean	Range	SD	Mean	Range	SD	*P* value
Age (years)	28·3	(19–55)	8·0	28·7	(20–46)	6·9	0·8478
Weight (kg)	74·1	(55·6–98·0)	9·5	59·4	(41·8–77·1)	8·0	<0·0001
Height (cm)	176·6	(165·6–202·4)	8·2	163·3	(144·1–178·2)	7·9	<0·0001
BMI$^{(kg/m^2)}$	23·7	(19·3–27·6)	2·1	22·2	(19·5–27·0)	1·9	0·0053
TBW (l)	43·8	(34·3–57·9)	5·6	30·6	(22·3–38·4)	3·6	<0·0001
TBW (% body weight)	59·2	(54·4–68·2)	3·6	51·9	(44·7–58·0)	4·2	<0·0001
ECW (l)	16·4	(11·4–23·5)	2·7	12·5	(7·4–17·9)	2·4	<0·0001
ECW (% body weight)	22·2	(18·9–27·8)	2·2	21·1	(14·7–33·7)	3·8	0·2127
FM (kg)	14·3	(4·9–22·5)	4·4	17·5	(10·2–27·0)	5·0	<0·0001
FM (% body weight)	19·1	(6·8–25·6)	4·8	29·1	(20·7–39·0)	5·7	<0·0001
LBM (kg)	59·8	(47·0–79·1)	7·6	41·9	(30·4–52·4)	5·0	<0·0001
LBM (% body weight)	80·9	(74·4–93·2)	4·8	70·9	(61·0–79·2)	5·7	<0·0001
BCM (kg)	37·4	(29·3–50·9)	5·2	24·8	(15·8–31·4)	4·2	<0·0001
BCM (% body weight)	50·6	(41·4–61·1)	4·7	42·0	(28·4–53·8)	6·3	<0·0001
BCM:LBM ratio	0·62	(0·52–0·68)	0·04	0·59	(0·42–0·71)	0·07	0·0241

TBW, total body water = [((Dose $^2H_2O \times 99·9)/20) \times$ APE, atom percent excess $\times$ (18·02/1000)]/1·04; ECW, extracellular water = (Br dose/(-plasma Br enrichment at 3 h)) $\times$ 0·9 $\times$ 0·95 $\times$ 0·94; FM, fat mass = body weight − lean body mass (LBM); LBM = TBW/0·732; BCM, body cell mass = (TBW − ECW)/0·732.

Statistics

All data were normally distributed and are presented as mean values and standard deviations. Results were considered to be statistically significant at a *P* value of <0·05. The SAS program (version 8.0, SAS Institute Inc., Cary, NC, USA) was used for all computations, using parametric tests. Student's *t* tests were used to compare age, body composition variables and RMR between men and women. Measured and predicted RMR were compared using paired *t* tests. The Pearson correlation coefficient was used to quantify the univariate association between RMR and selected independent variables. This association was further evaluated using the multivariate technique of all possible regressions. RMR was the outcome variable, and possible predictors included age, weight, height, BMI, FM, LBM, TBW, ECW and BCM. Analysis of covariance was used to adjust RMR for various body composition variables if there was no evidence of a significant sex × predictor variable interaction.

Results

Body composition

Physical characteristics of the fifty–eight subjects are shown in Table 1. BCM was strongly associated with LBM in men ($r^2 = 0·81$) but less so in women ($r^2 = 0·56$, both $P<0·0001$) and made up a greater proportion of LBM in men, as indicated by the higher BCM:LBM ratio.

Energy expenditure and its relationship to body composition

Differences in RMR between men and women are shown in Table 2. Absolute RMR was 32·7 % higher in men, but this difference became non-significant when adjusted for LBM using analysis of covariance. The difference in RMR between the sexes persisted when RMR was adjusted for weight and BCM. There was a significant sex × FM interaction ($P=0·0008$) such that a FM-adjusted comparison of RMR was statistically inappropriate. The single best

Table 2. Predicted, absolute and adjusted resting metabolic rate (kJ/d) measured in men and women
(Mean values and standard deviations)

	Men (*n* 30)		Women (*n* 28)		
	Mean	SD	Mean	SD	*P* Value
Predicted RMR*	7414	561	5732	498	<0·0001
Absolute RMR	7280	844	5485	537	<0·0001
RMR (% predicted)	98·2	7·8	95·7	5·8	0·1629
Adjusted† for weight	6854	562	5942	567	<0·0001
Adjusted† for LBM	6536	630	6282	641	0·2191
Adjusted† for BCM	6680	744	6128	756	0·0249

RMR, resting metabolic rate; LBM, lean body mass; BCM, body cell mass.
* From Schofield (1985).
† RMR adjusted by analysis of covariance.

predictor of RMR for the whole sample was LBM (r^2 0·85), followed by BCM (r^2 0·76), weight (r^2 0·74), height (r^2 0·61), ECW (r^2 0·56) and BMI (r^2 0·30), all $P<0·0001$. For men alone, the single best predictor was LBM (r^2 0·65), whereas for women, it was weight (r^2 0·59), both $P<0·0001$. FM did not explain a significant amount of variation in RMR in men (r^2 0·03, $P=0·3301$) nor did the addition of FM to the LBM-containing regression model increase prediction of RMR ($P=0·7393$). FM did explain a significant amount of variation in RMR in women (r^2 0·28, $P=0·0038$). Addition of FM to the LBM-containing regression model increased RMR prediction ($P=0·0104$), and reduced the mean squared error (LBM alone: r^2 0·50, $\sqrt{}$mean squared error = 387; LBM and FM together: r^2 0·62, $\sqrt{}$mean squared error = 345). Age explained a non-significant amount of variation in RMR (r^2 0·001, $P=0·8094$).

Discussion

The major findings of the present study were: (1) RMR is not significantly different between men and women after adjusting for LBM as part of the two-compartment whole-body-level model of body composition; (2) adjusting RMR for BCM as part of the three-compartment cellular-level model did not further correct differences between the sexes.

As expected, absolute RMR was significantly higher in men and can be explained primarily by greater LBM in men. When adjusting RMR for body weight, a crude indicator of body composition, the difference between men and women decreased from 32·7 to 15·3 % ($P<0·0001$). When we subdivided the body into the two-compartment model, and adjusted RMR for LBM, the difference in RMR between men and women decreased to 4·0 % ($P=0·2190$). This is consistent with other findings in the literature (Ravussin *et al.* 1986; Owen, 1988; Fukagawa *et al.* 1990; Mifflin *et al.* 1990; Klausen *et al.* 1997; McCrory *et al.* 1998) and indicates that LBM plays an important role in regulating metabolism. Although not statistically significant, the potential clinical significance of this difference should not be overlooked. For example, Ravussin *et al.* (1988) showed that a lower adjusted RMR of 297 kJ/d in a group of fifteen subjects resulted in a weight gain of >10 kg over 4 years.

To explore the effects of LBM on resting metabolism further, we subdivided the body into the three-compartment cellular-level model to isolate the effects of BCM on RMR (Heymsfield *et al.* 1997). Although it did decrease the difference from 32·7 to 9·0 %, adjusting RMR for BCM did not fully correct the difference in RMR between men and women ($P=0·0249$), nor did it reduce the variance. We consider that this may be measurement artifact, as there is no biologically plausible reason to explain why adjusting for the metabolic 'furnace' would not correct differences in RMR between the sexes. This point merits two further comments.

First, body composition variables that reflect metabolically active tissues, such as weight, LBM, TBW and BCM, are inter-related; the importance of measurement technique when comparing body composition-adjusted RMR between different populations becomes evident. In the present study, BCM was calculated by subtraction [(TBW − ECW)/0·732], rather than measured by a direct method such as total body K counting. Therefore, we introduced the potential error of two measurement techniques. At the time of preparation of this manuscript, we could find no other studies comparing BCM-adjusted RMR between healthy, normal to overweight adult men and women. Although total body K-adjusted differences in RMR have been reported (Jensen *et al.* 1988; Welle & Nair, 1990), total body K in these cases was used to measure LBM, not BCM. Therefore, these studies did not address the issue of BCM-adjusted RMR.

Second, there is evidence that even BCM is not a homogeneous compartment; it comprises high-energy-requiring organs such as the liver and brain, and moderate-energy-requiring skeletal muscle (Weinsier *et al.* 1992; Gallagher *et al.* 1996). We studied BCM as a whole in the present study, and so were unable to compare high- *v.* moderate-energy-requiring compartments between men and women. It has been suggested that future studies should focus on the RMR of individual tissues and organs across the human life span (Nelsen *et al.* 1992; Wang *et al.* 2000).

FM explained 28 % of the variation in RMR in the women, but only 3 % in the men. Relationships between FM and energy metabolism have been reported elsewhere (Garby *et al.* 1988; Dionne *et al.* 1999; Weyer *et al.* 1999). Age did not contribute to the variation in RMR. This is consistent with some findings in the literature (Owen, 1988; Klausen *et al.* 1997) but not with others (Fukagawa *et al.* 1990; Paolisso *et al.* 1995). This may reflect the narrow age range of our sample (19–55 years, although only five subjects were aged ≥40 years), rather than a true lack of effect of age on energy metabolism.

There are a few limitations to the present study that bear mentioning. Although all female subjects reported being in the follicular phase of menses, we did not ask them the specific day of the menstrual cycle. Stage of menstrual cycle has been found to affect basal, sleeping and/or resting metabolism in some studies (Solomon *et al.* 1982; Meijer *et al.* 1992), but not all (Piers *et al.* 1995; Diffey *et al.* 1997; Li *et al.* 1999). Similarly, oral contraceptive use may increase RMR by 3–5 % (Diffey *et al.* 1997; Piers *et al.* 1997); we did not ask our female subjects about oral contraceptive use. However, even if the majority of our female subjects had been using oral contraceptives at the time of the study, this would only have closed the metabolic gap between the male and female subjects. Nonetheless, RMR was significantly higher in the males in the present study.

Once controlled for LBM, RMR did not differ significantly between healthy adult men and women. The relationship between the components of BCM and RMR requires further elucidation.

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