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No association between resting metabolic rate or respiratory exchange ratio and subsequent changes in body mass and fatness: $5\frac{1}{2}$ year follow-up of the Québec Family Study

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Objective: To investigate the relationships between resting metabolic rate (RMR) and respiratory exchange ratio (RER) and subsequent changes in body size and fatness.
Design: Prospective longitudinal observational study.
Participants: A sample of 147 participants (76 males, 71 females) 18–68 y of age were followed for approximately $5\frac{1}{2}$ y.
Measures: At baseline, post-absorptive RMR and RER were determined by indirect calorimetry and adjusted for the effects of age, body mass and subcutaneous fatness using regression procedures. Indicators of body size and fatness included body mass, waist circumference, and the sum of six skinfolds. Changes in these indicators (delta scores) were adjusted for age and length of the follow-up period using regression.
Results: Correlations between baseline RMR, RER and subsequent changes in the indicators of body fatness were uniformly low and not significant (range −0.05–0.16). Further, Cox proportional hazards regression analyses indicated that neither RMR nor RER were significant predictors of gains in body mass, waist circumference, or the sum of six skinfolds.
Conclusions: There is no association between RMR or RER and changes in indicators of body size and fatness over $5\frac{1}{2}$ y of follow-up in this sample.
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Introduction

Obesity is a complex disorder that results from a chronic energy imbalance where intake exceeds expenditure. However, there is no consensus as to whether obesity is primarily the result of excess energy intake, reduced energy expenditure, or a combination of the two. Resting metabolic rate (RMR) and respiratory exchange ratio (RER) have been identified as two potential metabolic predictors of body weight gain (Ravussin & Gautier, 1999). RMR is a significant component (50–70%) of total daily energy expenditure (Ravussin & Bogardus, 1989); thus, its relationship to body fatness or changes in body composition over time is of great interest. Nutrient oxidation rates, evaluated by whole body RER, may also be important in the development of obesity, as it has been suggested that greater relative fat oxidation rates may be protective against weight gain (Zurlo *et al*, 1990).

There are inconsistent results in regard to RMR and weight gain in adults. Among the Pima Indians, low RMR was significantly associated with increases in body mass over a 4 y follow-up period (Ravussin *et al*, 1988). However, a study from the Baltimore Longitudinal Study on Aging found no relationship between RMR and weight gain over a 10 y follow-up period in men (Seidell *et al*, 1992). Similarly, Marra and colleagues (Marra *et al*, 1998) found no relationship between RMR and weight gain over 3 y in a sample of non-obese women from Italy. Although small differences in RMR are difficult to detect, they may lead to the development of obesity over the long term. A recent meta-analysis of 12 studies demonstrated that RMR was approximately 3–5% lower in formerly obese subjects compared with controls (Astrup *et al*, 1999). This is obviously not the same as predicting weight gain over time in a free-living population. Nonetheless, it supports the notion that a low RMR adjusted for body mass and body fatness may play a small but statistically significant

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role in weight gain, although the effect is at the margin of the reproducibility of the measurement and day-to-day variation in metabolic rate.

The results of studies relating RER to weight changes are more consistent than those for RMR. A higher RER was a weak but significant predictor of weight gain in samples of Pima Indians (Zurlo *et al*, 1990), non-obese men (Seidell *et al*, 1992), and non-obese women (Marra *et al*, 1998). Thus, based on a limited number of studies, the relationship between RER and weight gain appears to be more robust than that for RMR; however, further research is needed to clarify the strength of the relationship.

The purpose of this study was to examine the relationship between baseline RMR and RER and subsequent changes in body mass and indicators of body fatness. To this end, 147 participants in the Québec Family Study were followed prospectively for an average of $5\frac{1}{2}$ y.

Methods

Sample

The Québec Family Study (QFS) is a longitudinal prospective study designed to investigate genetic and environmental effects on various physical and biochemical traits, particularly obesity and its co-morbidities. Participants were originally recruited from the Greater Québec City area through the local media (television, radio, newspapers, etc). The overall study design has been discussed elsewhere (Bouchard, 1996). The present sample consists of 147 participants (76 males, 71 females) 18 – 68 y of age who were measured at baseline in 1992 – 1997 and were followed for an average of $5\frac{1}{2}$ y. The QFS is an observational study and no intervention was imposed upon the participants. Measurements of RMR and RER were made at baseline, while measurements of body size and fatness were made both at baseline and at follow-up. All procedures were approved by the Medical Ethics Committee at Laval University, and informed consent was obtained from all participants.

Metabolic measurements

RMR and RER were determined from indirect calorimetry measurements from the last 10 min of a 30 min period using a ventilated hood and an open-circuit system. Measurements were made early in the morning while participants were in a fasted state (≈12 h), and participants were asked to refrain from exercise on the day before the tests. Gas samples were assayed with a zirconia cell O_2 analyser (Amatek CD-3A, Thermox instruments Division, Pittsburgh) and an infrared CO_2 analyser (Amatek S-3A). Analysers were calibrated before each test using gases of known percentages of O_2 and CO_2. RER is simply the ratio of CO_2 produced to O_2 consumed, while RMR was converted from measurements of O_2 and RER using the formula of Weir (1999):

$$\text{RMR} = [(\text{RER} \times 1.1) + 3.9)] \times \text{litres } O_2$$

Measurements of RMR in our laboratory have been shown to be quite reliable. The intra-class correlation for measurements made on 13 participants performed twice within 2 weeks was 0.88, with a coefficient of variation (CV) of 5.6% (Bouchard, 1985). A 7 day dietary recall was used to estimate daily energy intake of kilocalories and macronutrients. Percentage of dietary energy from fat was used as a covariate in the analyses for RER.

Anthropometry

Stature and body mass were measured without shoes and wearing only shorts and a T-shirt, while waist circumference was measured with a flexible tape to the nearest mm. Skinfolds at the triceps, biceps, medial calf, subscapular, suprailiac and abdominal sites were taken on the left side of the body with a Harpenden caliper to the nearest 0.5 mm following the recommendations of the International Biological Programme (Weiner & Lourie, 1969) and were summed to provide an index of subcutaneous adiposity (sum of skinfolds). The reproducibility of the body composition measurements was quite good, with intra-class correlations of 1.0 for body mass (CV = 0.7%) and ranging from 0.94 to 0.98 (CV = 10 – 19%) for the individual skinfolds in a sample of 61 participants measured twice within 2 weeks (Bouchard, 1985).

Statistical analyses

Given that age, sex and body composition are major determinants of RMR (Ravussin & Bogardus, 1989; Bouchard *et al*, 1989) it was adjusted for the effects of age, age^2, age^3, mass and sum of skinfolds by sex using regression procedures. RER was similarly adjusted and all adjusted values were retained for further analysis. The changes (deltas) in body mass, waist circumference, and the sum of skinfolds were adjusted for the effects of age, age^2, age^3 and length of the follow-up period using regression procedures.

The relationships between RMR, RER and subsequent changes in body mass and fatness were investigated using Pearson correlations and Cox proportional hazard regressions. Pearson correlations were calculated between adjusted RMR and RER and the changes in the indicators of body size and fatness. Although the correlational analyses accounted for the length of the follow-up period, a basic assumption is that the changes over time in the population follow a normal distribution. However, it is difficult to assess this criteria (normal distribution in response) due to differing lengths of the follow-up period. Thus, the use of a more robust method of analysing survival data is preferred. Cox proportional hazards regression is a semi-parametric method that does not require distributional assumptions to be made (Cox, 1972). Changes in the indicators of body fatness that were approximately equal to the upper quartile cut-off (of changes) were used for the study of gains in body size and fatness. The cut-off values used to define an event (a threshold for weight gain or change in fatness) were 5.6 kg for body mass, 6.0 cm for waist circumference, and 27 mm for the sum of skinfolds. For the Cox regressions, variables were not adjusted by regression procedures as in the correlational analyses, rather covariates were added into the model as required. In order to control for initial weight status, the Cox regressions were also performed separately by BMI category (BMI < 25 kg/m^2 and BMI ≥ 25 kg/m^2).

Results

Table 1 presents descriptive characteristics of the participants at baseline. Values are presented as means and standard deviations. The average age was 38.7 ± 15.2 y (range 18.4 – 67.7 y) in males and 38.7 ± 13.8 y (range 19.6 – 59.5 y) in females, while the average values for BMI were 24.9 ± 3.6 kg/m^2 (range 17.4 – 34.3 kg/m^2) in males and 22.8 ± 3.2 kg/m^2 (range 17.9 – 32.8 kg/m^2) in

Table 1 Descriptive characteristics of sample at baseline

	Males			*Females*		
	n	*M*	*s.d.*	*n*	*M*	*s.d.*
Age (y)	76	38.7	15.2	71	38.7	13.8
Stature (cm)	76	172.0	6.6	71	159.5	6.0
Body mass (kg)	76	73.8	10.9	71	57.9	7.1
Body mass index (kg/m^2)	76	24.9	3.6	71	22.8	3.2
Waist circumference (cm)	76	87.3	11.1	70	72.3	7.3
Sum of skinfolds (mm)	75	74.4	29.7	71	98.9	36.0
RMR (kJ/min)	76	4.60	0.4	71	3.77	0.4
RER	76	0.82	0.04	71	0.82	0.04

RMR, resting metabolic rate; RER, respiratory exchange ratio.

females. Mean values for RMR and RER were 4.60 ± 0.4 kJ/min and 0.82 ± 0.04 in males and 3.77 ± 0.4 kJ/min and 0.82 ± 0.04 in females, respectively. Table 2 presents the average changes during the follow-up period along with the associated ranges. The average follow-up period was 5.7 y in both males and females (Table 2), ranging from 2.8 to 7.7 y. Average gains in body mass were 2.8 kg in males and 3.5 kg in females, while the sum of six skinfolds increased by 11.2 mm in males and 19.3 mm in females.

Table 3 presents correlations between baseline RMR and RER and changes in indicators of body fatness during the approximately $5\frac{1}{2}$ y follow-up period. Results are presented for three different adjustment strategies: (1) adjusted for

Table 2 Changes in indicators of body fatness during the approximately $5\frac{1}{2}$ y follow-up period

	Males					*Females*				
	n	*M*	*s.d.*	*Min*	*Max*	*n*	*M*	*s.d.*	*Min*	*Max*
Follow-up (y)	76	5.7	0.7	2.8	7.7	71	5.7	0.8	3.0	7.6
Body mass (kg)	76	2.8	4.7	−12.5	14.5	71	3.5	4.4	−7.7	16.8
Waist circumference (cm)	76	2.5	4.8	−14.4	14.8	70	3.9	5.0	−7.8	18.5
Sum of skinfolds (mm)	75	11.2	17.6	−39.0	62.0	71	19.3	25.1	−36.9	87.7

Table 3 Correlations between resting metabolic rate, respiratory exchange ratio and changes in indicators of body fatness

	RMR[a]	*RMR*[b]	*RMR*[c]	*RER*[a]	*RER*[b]	*RER*[d]
Males						
Δ Body mass	0.10	0.12	0.11	0.00	0.00	0.01
Δ Waist circumference	0.11	0.03	0.08	−0.05	−0.04	−0.04
Δ Sum of skinfolds	0.04	−0.01	−0.03	−0.01	−0.01	0.00
Females						
Δ Body mass	0.13	0.07	0.02	0.09	0.12	0.11
Δ Waist circumference	0.16	0.07	0.05	0.03	0.09	0.09
Δ Sum of skinfolds	0.13	0.07	0.04	0.08	0.12	0.11

Δ = change (delta) scores adjusted for age and length of the follow-up period by regression.
[a]Adjusted for age.
[b]Adjusted for age and body mass.
[c]Adjusted for age, body mass and sum of skinfolds.
[d]Adjusted for age, body mass, sum of skinfolds and percentage of dietary intake from fat.
No correlations significant at $P < 0.05$.

Table 4 Results of Cox regressions for gains in body size or composition, from baseline resting metabolic rate and respiratory exchange ratio

		RMR[a]		*RMR*[b]		*RMR*[c]		*RER*[a]		*RER*[b]		*RER*[d]	
	Cut-off value	χ^2	*P*	χ^2	*P*	χ^2	*P*	χ^2	*P*	χ^2	*P*	χ^2	*P*
Total sample													
Body mass (kg)	5.6	0.37	0.55	0.11	0.74	0.00	0.98	0.00	1.00	0.02	0.88	0.07	0.79
Waist circumference (cm)	6.0	2.25	0.13	0.64	0.42	0.72	0.40	0.54	0.46	0.25	0.62	0.00	0.99
Sum of skinfolds (mm)	27.0	0.01	0.93	0.12	0.73	0.27	0.60	0.69	0.40	0.63	0.43	0.22	0.64
BMI < 25 kg/m^2													
Body mass (kg)	5.6	1.11	0.29	0.00	0.99	0.01	0.93	0.08	0.78	0.13	0.71	0.10	0.76
Waist circumference (cm)	6.0	1.36	0.24	0.21	0.65	0.26	0.61	0.32	0.57	0.00	0.95	0.35	0.56
Sum of skinfolds (mm)	27.0	0.55	0.46	0.07	0.79	0.00	1.00	0.77	0.38	0.13	0.72	0.01	0.91
BMI ≥ 25 kg/m^2													
Body mass (kg)	5.6	0.72	0.39	0.04	0.84	0.00	0.95	0.00	0.96	0.05	0.82	0.27	0.60
Waist circumference (cm)	6.0	1.66	0.20	0.65	0.42	0.61	0.44	1.19	0.28	0.85	0.36	0.38	0.54
Sum of skinfolds (mm)	27.0	1.57	0.21	0.61	0.43	1.65	0.20	0.01	0.91	0.02	0.90	0.24	0.62

[a]Age and sex included as covariates.
[b]Age, sex and body mass included as covariates.
[c]Age, sex, body mass and sum of skinfolds included as covariates.
[d]Age, sex, body mass, sum of skinfolds and percentage of dietary intake from fat included as covariates.

age only; (2) adjusted for age and body mass; and (3) adjusted for age, body mass and sum of skinfolds. There are no apparent patterns in the correlations, as all correlations are low and not significant, ranging from −0.03 to 0.16 for RMR and −0.05 to 0.12 for RER. Table 4 presents the results of the Cox regressions for RMR and RER. Neither RMR nor RER were significant predictors of gains in body mass or indicators of body composition. The results were similar after the sample was stratified into normal weight (BMI < 25 kg/m^2) and overweight (BMI ≥ 25 kg/m^2) categories.

Discussion

The results from the correlational and survival analyses suggest that RMR and RER are not significantly associated with subsequent changes in indicators of body fatness, at least over the short term ($5\frac{1}{2}$ y). These results are not unexpected given the inconsistency in findings from previous studies. Table 5 presents summary information on the relationships between RMR and RER and subsequent weight gain from the available studies. Correlations between RMR and weight gain range from a negative 0.19 ($P = 0.04$) in the Pima Indians (Ravussin *et al*, 1988) to a positive 0.12 ($P < 0.01$) in men from the Baltimore Longitudinal Study on Aging (Seidell *et al*, 1992). Further, correlations in the present study and those from a study of non-obese Italian women were low and not significant (Marra *et al*, 1998). The correlations presented in Table 5 for RER and weight gain follow a more consistent pattern than those for RMR; however, they remain low (range 0.01−0.27).

The results of the present study are in direct contrast to those of the study on the Pima Indians that indicated a significant relationship between RMR (Ravussin *et al*, 1988) and subsequent weight gain. A low correlation ($r = -0.19$, $P = 0.04$) between RMR and weight gain was reported, and the cumulative incidence of a 10 kg weight gain over 4 y was highest in the group with the lowest RMR values at baseline. On the other hand, there was no relationship between RMR and weight gain over 10 y in men from the Baltimore Longitudinal Study on Aging; in fact, the correlation between RMR and weight gain was positive both before ($r = 0.33$; $P < 0.001$) and after adjustment for fat-free mass ($r = 0.12$; $P < 0.01$), which is in the opposite direction to what is expected. Finally, a study of non-obese Italian women reported that the correlation between RMR and weight gain over a 3 y follow-up was not significant; however, the correlation coefficients were not presented (Marra *et al*, 1998). Thus, the results of the present and previous studies suggest that if there is a relationship between RMR and weight gain, it is extremely weak.

The present study failed to show a significant relationship between RER and weight gain, unlike three previous studies (Zurlo *et al*, 1990; Seidell *et al*, 1992; Marra *et al*, 1998). Although the correlation of $r = 0.11$ among QFS females was not significant; a correlation of similar magnitude in the Baltimore Longitudinal Study on Aging in men ($r = 0.10$) was significant, due to the large number of subjects. Thus, caution must be used when comparing results from various studies, as sample sizes largely determine the 'statistical' significance of correlations. RER was a significant predictor of weight gain in a sample of 111 Pima Indians (Zurlo *et al*, 1990). Pimas with an RER at the 90th percentile were 2.5 times more likely to gain ≥ 5 kg than those at the 5th percentile for RER. In men with BMI values less than 25 kg/m^2, but not in those with BMI values greater than 25 kg/m^2, RER predicted minimum gains in body weight of between 5 and 15 kg (Seidell *et al*, 1992). Additionally, RER was related to weight changes in non-obese women (BMI < 30 kg/m^2): mean RER values were 0.91 in women gaining more than 3 kg over 3 y *vs* 0.84 in the remaining women ($P = 0.047$) (Marra *et al*, 1998).

The studies that have shown the strongest evidence for effects of RMR and RER on weight gain have been those done on Pima Indians. These studies have been well controlled and have used sophisticated statistical analyses. However, there are several differences between the samples of the Pima studies and the present study which may account for differences in the results. In the Pima study, 15 out of 126 (11.9%) participants gained 10 kg or more over a maximum of 4 y of follow-up (Ravussin *et al*, 1988), while in the present study, 10 out of 147 participants (6.8%) gained at least 10 kg over the $5\frac{1}{2}$ y follow-up. The age range was 18−41 y (mean 26 y) in the Pima, with an average body mass at baseline of 92.5 kg (49.0−178.1 kg). In the present study, the age range was 18−68 y (mean 39 y), with an average body mass of 66.1 kg (39.8−101.8 kg). Thus, the Pimas were younger and considerably heavier at baseline than the current sample from Québec. These, and potential ethnic differences in patterns of weight gain, may partially account for the discrepant results between the Pima studies and those using other samples.

Differences among the results from the various studies may also be attributable to methodological differences. For

Table 5 Pearson correlations between resting metabolic rate and respiratory exchange ratio and weight changes from available studies

Sample	*Sex*	*n*	*r*	*P*	*Reference*
Resting metabolic rate					
Pima	M/F	126	−0.19	0.04	Ravussin *et al*, 1988
Baltimore Longitudinal Study on Aging	M	775	0.12	< 0.01	Seidell *et al*, 1992
Québec Family Study	M	76	0.11	ns	Present study
Québec Family Study	F	71	0.02	ns	Present study
Non-obese Italian women	F	58	—[a]	ns	Marra *et al*, 1998
Respiratory exchange ratio					
Pima	M/F	111	0.27	< 0.01	Zurlo *et al*, 1990
Baltimore Longitudinal Study on Aging	M	775	0.10	< 0.01	Seidell *et al*, 1992
Québec Family Study	M	76	0.01	ns	Present study
Québec Family Study	F	71	0.11	ns	Present study
Non-obese Italian women	F	58	0.26	< 0.05	Marra *et al*, 1998

[a]Correlation was reported only as being not significant.

example, in the Baltimore Longitudinal Study on Aging (Seidell *et al*, 1992), the study from Italy (Marra *et al*, 1998), and the present study, RMR and RER were measured in a post-absorptive state, while measurements of RMR and RER in the Pima were based on 24 h measurements in a respiratory chamber (Zurlo *et al*, 1990; Ravussin *et al*, 1988). Further, with the exception of the studies on the Pima, body composition was not assessed by underwater weighing. Subcutaneous skinfolds were used in the present study and the Baltimore Longitudinal Study on Aging (Seidell *et al*, 1992). Body composition was also not incorporated in the analyses by Marra and colleagues (Marra *et al*, 1998). Thus, the adjusted phenotypes used to represent RMR and RER among the studies are not directly comparable.

In summary, the present study has demonstrated no relationship between RMR or RER and subsequent changes in body weight or fatness. These results are not unexpected given the inconsistent results obtained in previous studies. Thus, the available evidence suggests that even if there is an association between RMR or RER and weight gain, it is extremely weak. Given the world-wide increases in the prevalence of obesity we are now experiencing, more studies in humans relating metabolic parameters to changes in body fatness using longitudinal designs are warranted.

References

Astrup A, Gotzsche PC, van de Werken K, Ranneries C, Toubro S, Raben A & Buemann B (1999): Meta-analysis of resting metabolic rate in formerly obese subjects. *Am. J. Clin. Nutr.* **69**, 1117–1122.

Bouchard C (1985): Reproducibility of body-composition and adipose-tissue measurements in humans. In: *Body-Composition Assessments in Youth and Adults, Report of the Sixth Ross Conference on Medical Research*, ed. AF Roche, pp 9–14. Columbus, OH: Ross Laboratories.

Bouchard C, Tremblay A, Nadeau A, Després J-P, Thériault G, Boulay MR, Lortie G, Leblanc C & Fournier G (1989): Genetic effect in resting and exercise metabolic rates. *Metabolism* **38**, 364–370.

Bouchard C (1996): Genetic epidemiology, association, and sib-pair linkage: Results from the Québec Family Study. In: *Molecular and Genetic Aspects of Obesity, Pennington Center Nutrition Series*, Vol 5, ed. GA Bray & DH Ryan, pp 470–481. Baton Rouge, LA: Louisiana State University Press.

Cox DR (1972): Regression models and life-tables. *J. R. Stat. Soc. Ser. B* **34**, 187–220.

Marra M, Scalfi L, Covino A, Esposito-Del Puente A & Contaldo F (1998): Fasting respiratory quotient as a predictor of weight changes in non-obese women. *Int. J. Obes.* **22**, 601–603.

Ravussin E, Lillioja S, Knowler WC, Christin L, Freymond D, Abbott WGH, Boyce V, Howard BV & Bogardus C (1988): Reduced rate of energy expenditure as a risk factor for body-weight gain. *New Engl. J. Med.* **318**, 467–472.

Ravussin E & Bogardus C (1989): Relationship of genetics, age and physical fitness to daily energy expenditure and fuel utilization. *Am. J. Clin. Nutr.* **49**, 968–975.

Ravussin E & Gautier JF (1999): Metabolic predictors of weight gain. *Int. J. Obes.* **23**, 37–41.

Seidell JC, Muller DC, Sorkin JD & Andres R (1992): Fasting respiratory exchange ratio and resting metabolic rate as predictors of weight gain: The Baltimore Longitudinal Study on Aging. *Int. J. Obes.* **16**, 667–674.

Weiner JS & Lourie JA (1969): *Human Biology: a Guide to Field Methods*. Oxford: Blackwell Scientific.

Weir JB (1949): New method for calculating metabolic rate with special reference to protein metabolism. *J. Physiol.* **109**, 1–9.

Zurlo F, Lillioja S, Esposito-Del Puente A, Nyomba Br L, Raz I, Saad MF, Swinburn BA, Knowler WC, Bogardus C & Ravussin E (1990): Low ratio of fat to carbohydrate oxidation as predictor of weight gain: study of 24-h RQ. *Am. J. Physiol.* **259**, E650–E657.