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Lack of Seasonal Differences in Basal Metabolic Rate in Humans – a Cross-Sectional Study

Pimjai Anthanont^a, James A. Levine^{a,b}, Shelly K. McCrady-Spitzer^a, and Michael D. Jensen^a

^aEndocrine Research Unit, Mayo Clinic, 200 1st St SW, Rochester, MN 55905 USA

Abstract

Some studies indicate that basal metabolic rate is greater winter than the summer, suggesting a role for brown fat in human thermogenesis. We examined whether there are clinically meaningful differences in basal metabolic rate under thermoneutral conditions between winter and summer months in inhabitants of Rochester, Minnesota. We collated data from 220 research volunteers studied in the winter (December 1 – February 28) and 214 volunteers studied in the summer (June 1 – August 31), 1995–2012. Basal metabolic rate was measured by indirect calorimetry and body composition by dual-energy X-ray absorptiometry. The effect of season on basal metabolic rate was tested using multivariate regression analysis with basal metabolic rate as the dependent variable and fat free mass, fat mass, age, sex, and season as the independent variables. The groups were comparable with respect to age, body mass index, fat mass and fat free mass. There was no significant difference in basal metabolic rate between winter and summer groups (1667 ± 322 vs 1669 ± 330 kcal/day). Both winter and summer basal metabolic rates were strongly predicted by fat free mass (Pearson's $r=0.75$ and $r=0.77$, respectively, $p<0.0001$). Using multiple linear regression analysis, basal metabolic rate was significantly, independently predicted by fat free mass, fat mass, age and sex, but not season. We conclude that the lack of seasonal variation of thermoneutral basal metabolic rate between winter and summer suggests that modern, Western populations do not engage thermogenically detectable brown fat activity during periods of living in a cold climate.

Keywords

Indirect calorimetry; body composition; brown fat

Introduction

Basal metabolic rate (BMR) is by definition measured in the postabsorptive state and under thermoneutral conditions. It is the amount of energy expended for homeostatic processes in absence of digestion and physical activity. Fat-free mass (FFM) is the strongest determinant of between-individual variability of BMR during weight-stable periods [1–3]. The effect of season/environmental temperature on the variation of BMR is subject to debate. Some investigators, using a longitudinal study design, have found that humans have a greater

Corresponding Author: Michael D. Jensen, Mayo Clinic, Endocrine Research Unit, 200 1st St SW, Rm 5-194 Joseph, Rochester, MN 55905, 507-255-6515 (tel), 507-255-4828 (fax), jensen@mayo.edu.

^bPresent Address: Endocrinology, Mayo Clinic, 13400 E Shea Blvd, Scottsdale, AZ 85259 USA

resting energy expenditure (REE) in winter than the summer [4–7], although it is not clear that the measures were always done under thermoneutral conditions or that body composition was assessed using robust techniques. Others investigators, using similar study designs, have not found seasonal differences in BMR [7,8].

Humans respond to cold by increasing energy expenditure to produce extra heat – a process termed cold-induced thermogenesis, which is proposed to consist of shivering and non-shivering thermogenesis (NST). One of the most interesting mechanisms of NST is via mitochondrial uncoupling protein 1 (UCP1) in brown adipose tissue (BAT), resulting in heat rather than ATP production in response to respiration [9]. Furthermore, chronic cold stimulation elicits hyperplastic process in classical BAT depots [10,11].

Several studies have investigated the effect of seasonality on BAT prevalence rates. BAT, as detected by positron emission tomography (PET) scans conducted under clinical conditions, is more readily detected in winter than in summer [12–19]. Others note that BMR is associated with BAT activity during cold exposure [20,21]. The observations that BAT is more readily detected in clinical PET scans in winter than summer and the reports of greater BMR in winter than summer suggests the former may be responsible for the latter.

Before pursuing an etiological study regarding the BAT/BMR hypothesis, we wished to assess whether BMR is greater in winter compared with summer in a typical Western population that experiences wide variations in outdoor temperature. If BMR is greater in the winter, this would be consistent with a chronic, cold-induced BAT adaptation that could be potentially important with regards to body fat control.

The aim of this study was to determine whether there are detectable, seasonal differences in BMR in residents of Rochester, Minnesota. From the weather record in Rochester, the coldest months are December-February, with average temperatures below -7°C . The warmest months are June-August, with July temperatures averaging 22°C . We hypothesize that inhabitants would have a greater BMR adjusted for body composition in the winter than in the summer.

Subjects and Methods

We extracted BMR, body composition, demographic and laboratory data from our electronic databases for volunteers studied in a variety of institutional review board (IRB)-approved protocols at Mayo Clinic, Rochester MN between 1995 and 2012. We compiled all data from volunteers whose studies were in the winter months (December 1 – February 28) or summer months (June 1 – August 31). The criteria for enrollment for each of the studies were such that the volunteers must healthy, weight stable, and non-smokers. Our research protocols systematically excluded volunteers who regularly take medications that have sympathomimetic activity or who have thyroid disease, unless they are rendered euthyroid as documented by normal TSH. All study protocols were approved by the Mayo Clinic IRB, and all participants must be provided informed written consent.

Data collection

Data on age, sex, height, weight, body mass index (BMI) were collected. Where available, we also compiled data from measures of fasting plasma glucose, insulin, epinephrine, and norepinephrine concentrations.

Basal metabolic rate

Basal metabolic rate was always measured in the postabsorptive state using calorimetry with ventilated hood (DeltaTrac Metabolic Monitor, Sensor Medics, Yorba Linda, CA). The calibration of these metabolic carts was standardized as previously reported [22]. In brief, the metabolic carts were calibrated each morning and underwent extra quality control including monthly pressure and gas calibrations together with biannual alcohol burn test calibrations. The test-retest difference is <3% for duplicate measures of VO_2 for adults in the same environment on sequential days using our instruments. In addition, each day we first check the calibration with a known gas mixture and if the variance from the known is >1.25% the instrument is reset. We also measure VO_2 and CO_2 monthly in one of our personnel; if the O_2 consumption rate is >20 ml/min different from average, we re-measure that person with another instrument to test for biological versus instrument issues.

All volunteers were admitted to the Mayo General Clinical Research Center (GCRC) in the evening prior to the study day and consumed their evening meal at the same, standardized time (1800 h). They then spent the night in the GCRC in order to assure the indirect calorimetry was performed before rising out of bed the next morning and before any food consumption. The BMR was performed typically between 0700 and 0800 h.

Body composition

Total body fat and fat-free mass were measured using dual-energy x-ray absorptiometry (DXA). All of the DXA measures were performed using Lunar/GE equipment (Madison, Wisconsin). To assure consistency over time and between instruments we employ 4 independent calibration phantoms composed of a range of known fat and non-fat content (courtesy of Hormel Institute, Austin, MN). Each instrument is calibrated to the phantoms such that we can identify any discrepancies between DXA predicted and known percent fat. The DXA-reported body fat was corrected for any calibration errors in the instrument as assessed by the phantoms. When new DXA instruments are purchased they are tested under an IRB approved protocol so that volunteers and the meat block phantoms are scanned on both the old and new DXA instruments. Software updates are cross-validated by analyzing existing scan data with both old and new versions to correct for variations that might be introduced by software modifications. This allowed us to maintain consistent body composition measures over long periods of time and with different instruments.

Analytical Methods

Plasma glucose was measured by a glucose oxidase method. Fasting insulin concentrations were measured using chemiluminescent sandwich assays (Sanofi Diagnostics Pasteur, Chaska, MN). Plasma epinephrine and norepinephrine concentrations were measured using HPLC with electrochemical detection [23].

Statistical analysis

Values are given as mean $\pm$ SD and median (IQR). To assess the differences between volunteers studied in the winter months and those in the summer months, Student's *t* test was used for normally distributed variables, the Wilcoxon rank sum test was used for non-normally distributed variables, and the χ^2 test was used for proportions. Univariate relationships were evaluated by Pearson's correlation. For predictors of BMR, we used univariate and multivariate regression analysis with BMR as the dependent variable and FFM, fat mass, age, sex, and season as the independent variables. Statistical analyses were performed with JMP version 10.0.0. The sequential R square was performed by SAS version 9.2. A two-tailed *P* value of less than 0.05 was considered to indicate statistical significance. We estimated that we were able to measure the small effect size (Cohen's $f^2 = 0.03$) with the use of a two-sided alpha level of 0.05 and a power of 80% by using our sample size of > 400 subjects. We also assessed statistical power using "G*Power" [24] to estimate how many subjects would need to be studied in a longitudinal study design (body composition and BMR both in winter and summer) to provide statistical power equivalent to our cross-sectional comparison with over 200 subjects in each cohort. Assuming the body composition variables predict BMR at least as well as the population correlation ($r = 0.84$) when repeated measures are performed, we estimated that the study of 64 subjects in a paired study design would provide the same statistical power to detect an effect of season as our cross-sectional study design. The only study we found that directly studied more than 64 subjects both in winter and summer [7] reported statistically significant 6% increase in BMR only in the subgroup of 43 young adults.

Results

Subject characteristics (Table 1)

A total of 232 (53%) men and 202 (47%) women were included in the analysis. Data was available from 220 and 214 volunteers who had their BMR measured in winter or summer, respectively. The 2 groups were comparable with respect to age, BMI, fat mass and FFM. There was no difference in BMR between winter and summer groups (1667 ± 322 vs 1669 ± 330 kcal/day). For the subjects with biochemical data available, we found no differences in plasma concentrations of glucose, insulin or catecholamines between winter and summer groups.

Predictors of BMR (Tables 2 and 3, and Figure 1)

Consistent with previous reports, FFM was the variable most strongly correlated with BMR ($r=0.75$ and $r=0.77$ for winter and summer groups, respectively, both $p<0.0001$). Fat mass was also positively correlated with BMR in the winter and summer groups ($r=0.30$ and $r=0.26$, respectively). By simple linear regression analysis, FFM explained 56% and 59% of the variance in BMR in winter and summer, respectively. Multiple linear regression analysis confirmed that BMR was significantly, independently predicted by FFM and fat mass, as well as age and sex (Table 3 and Figure 1). However, season was not a significant predictor of BMR. In this model, FFM, fat mass, age and sex explained 70% of the variance in BMR (all $p<0.001$). Fat free mass was the largest contributor to predicting BMR, accounting for ~82% of the final model.

Discussion

Cold exposure is commonly used to enhance BAT detection by PET scan techniques [12,20,25] and can acutely increase REE [20,21]. Furthermore, PET scan detectable BAT is more prevalent in winter than in summer [12–19]. To understand if this cold climate-associated BAT might result in detectable increases in BMR under thermoneutral conditions we utilized existing, carefully collected BMR and body composition data from inhabitants of Rochester, MN, where there are marked seasonal differences in outdoor temperature. Data from over 400 volunteers studied in the Mayo Clinic GCRC under strict protocol conditions between 1995 and 2012 were available for this analysis. Consistent with previous reports, almost 70% of the variance in BMR could be accounted for by FFM, fat mass and age. Despite an average difference in ambient temperatures between winter and summer of ~29°C, there was not a difference in BMR between winter and summer. These findings suggest that, either there is insufficient cold exposure in free living Minnesota adults to stimulate additional BAT activity or that, upon overnight acclimatization to a thermoneutral environment, the additional BAT is inactive.

A recent PET study demonstrated that 2 hours of daily cold exposure for 4 weeks increases human BAT FDG uptake and oxidative metabolism [26]. However, the authors also found that cold-stimulated energy expenditure was not greater after cold acclimation despite the increase in BAT [26]. A potential explanation for this finding is that subclinical skeletal muscle shivering, not BAT, is the major source of the increase in energy expenditure during acute cold exposure [27].

Some investigators have found that humans have a greater BMR or REE in winter than the summer [4–7], whereas others have not found this to be the case [7,8]. Winter REE has been reported to be as much as 6% [7] to 35% [5] greater than summer REE. However, some of studies reporting higher winter REE summer included relatively small numbers of subjects [4,6], did not account for body composition [4,5], did not define the duration of temperature acclimatization before measuring the REE [5,7], or included subjects exposed to more extremes of cold exposure [5]. Studies with small numbers of subjects may have insufficient statistical power to detect a true seasonal effect on REE [8]. In those studies where body composition was not measured [4,5], it is impossible to determine whether changes in BMR were due to changes in body composition. In some studies it appears that REE was measured with insufficient adaptation to thermoneutral conditions [5,7]; in that case subclinical shivering cannot be excluded as the explanation for the greater REE. Recently, Leonard et al. measured BMR in 94 Yakut adults and found no seasonal change in BMR in older adults (>50 years old), but a significant increased winter BMR in young adults [7]. Confounding factors include changes in body composition in young women and possible greater participation in outdoor activities in young men in this study.

In contrast to some of these limitations, we had access to data from a relatively large population living in an environment that is known for extreme cold in the winter and moderate temperatures in the summer. All of our research volunteers undergo robust body composition and BMR measures under standardized conditions. Basal metabolic rate measured using indirect calorimetry is accurate in our hands [22]. We believe that our efforts

to assure consistent BMR and body composition measurements under standardized conditions in large numbers of subjects provides a more definitive answer to the question as to whether seasonal changes in BMR occur in modern times. Although the effect of season in our statistical model was close to being statistically significant ($P = 0.08$), it was likely driven by 3–6 outlying values and could account for only 0.2 % of the explained variance in any case.

Admittedly, there are some limitations to our study. If we had studied the same individuals in winter and summer this might have been a more powerful test of the hypothesis. However, the absolute requirement for body composition measures for both studies in order to adjust BMR does introduce more sources of error than merely the BMR measure itself. We estimated that the study of 64 volunteers in a paired study design would provide the same statistical power as our cross-sectional study including 434 subjects. Few of the studies we found studied 64 or more subjects with a longitudinal study design. Because we don't have information regarding the duration of our volunteers' exposures to outdoor temperatures we cannot know whether there is sufficient durations of cold exposure, even in Minnesota winters, to activate brown fat. We also didn't account for variability in BMR that may occur with the menstrual cycle in women. Approximately 10% of women can have variations in their BMR of $> 6\%$ related to the menstrual cycle [28]. We included 202 women, of whom 155 were under the age of 50, and thus potentially cycling. This would suggest that ~ 15 women out of 202 may have had more menstrual cycle-related variability in BMR than we assumed for our power calculations. Because our total population included more than 400 adults, the issue of menstrual cycle-related variability in BMR likely did not materially reduce our statistical power. Likewise, over 95% of our participants over the time interval we included were Caucasian. These results cannot be extrapolated to African-Americans, who tend to have lower BMR's after adjusting for body composition [29]. It is possible that 24 hour energy expenditure could be affected by season, although a small study of UK adults did not find an effect [7,8]. In essence, this is an experiment of nature, testing whether adults in a Western environment exposed to months of a very cold climate have higher BMR's than the same population enjoying a moderate, summer climate. Anecdotally, however, radiologists at Mayo Rochester detect BAT during diagnostic PET scans much more often in winter than in summer, suggesting there is enough cold exposure in our current environment to stimulate BAT, even under the thermoneutral conditions of our diagnostic nuclear medicine scans. Although we don't have information regarding our volunteers' habitual physical activity, previous investigators have found that intra-individual variations of BMR cannot be explained by habitual non-exercise physical activity [8], or fitness [30–32], after differences in body composition are accounted for.

Conclusions

In summary, we found no seasonal differences in BMR between adults in the same community exposed to cold winters and moderate summers. These data suggest that, to the extent brown fat contributes to thermogenesis in humans living in a Western environment, it is insignificant under thermoneutral conditions despite living for months in a cold climate.

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Table 1

Baseline characteristics, body composition, basal metabolic rate, and plasma concentrations of fasting glucose, insulin, and catecholamine in healthy research volunteers in winter and summer.

	Winter	Summer	p value
N	220	214	
Male sex-no (%)	112 (50.9)	120 (56.1)	0.28
Age (years)	42 ± 18	39 ± 17	0.10
Height (cm)	171.9 ± 9.9	172.8 ± 9.8	0.32
Weight (kg)	80.6 ± 18.1	81.5 ± 16.9	0.60
BMI (kg/m ²)	27.3 ± 5.4	27.2 ± 4.9	0.99
Fat mass (kg)	25.8 ± 12.9	25.4 ± 11.8	0.70
FFM (kg)	54.6 ± 12.2	55.9 ± 12.6	0.27
BMR (kcal/day)	1667 ± 322	1669 ± 330	0.94
Fasting glucose (mmol/l)	5.0 ± 0.5 (N=162)	5.0 ± 0.4 (N=168)	0.38
Insulin (pmol/l)	30.4 (21.5, 47.4) (N=192)	30.6 (21.5, 50.3) (N=190)	0.87
Epinephrine (pg/ml)	21 (15, 26) (N=111)	20 (14, 32) (N=120)	0.97
Norepinephrine (pg/ml)	131(109, 166) (N=69)	122 (97, 160) (N=91)	0.35
Total cholesterol (mmol/l)	4.50 ± 0.87 (N=158)	4.48 ± 1.00 (N=177)	0.82
Triglycerides (mmol/l)	1.40 (0.87, 1.56) (N=158)	1.17 (0.82, 1.65) (N=177)	0.45
HDL (mmol/l)	1.25 ± 0.40 (N=157)	1.23 ± 0.51 (N=178)	0.62
LDL (mmol/l)	2.69 ± 0.77 (N=155)	2.65 ± 0.85 (N=169)	0.66

Values are mean $\pm$ SD.

BMI, body mass index; BMR, basal metabolic rate; FFM, fat free mass; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 2

Pearson's correlation of fat free mass, fat mass, sex, and basal metabolic rate in winter and summer.

	Correlation coefficient	
	Winter	Summer
FFM	0.747 [†]	0.771 [†]
Fat mass	0.292 [†]	0.259 [*]
Sex	0.563 [†]	0.568 [†]

^{*}
p<0.001

[†]
p<0.0001

FFM, fat free mass.

Table 3

Multiple linear regression analysis with basal metabolic rate as a dependent variable and fat free mass, fat mass, age, sex, and season as independent variables (model $R^2= 0.70$).

	Regression coefficient	95% CI	Sequential partial R^2	p value
FFM (kg)	15.3	12.6 – 18.1	0.575	<0.0001
Fat mass (kg)	10.1	8.4 – 11.8	0.078	<0.0001
Age (year)	–4.0	–5.0 to –2.9	0.033	<0.0001
Sex *	126.1	55.9 – 196.3	0.009	0.0005
Season **	30.6	–3.6 – 64.7	0.002	0.08
y-Intercept	641	518 – 765		<0.0001

*
For sex: male =1 and female =0.

**
For season: winter =1 and summer =0.

CI, confidence interval; FFM, fat free mass.