Published in final edited form as:
Metabolism. 2012 February ; 61(2): 237–241. doi:10.1016/j.metabol.2011.06.019.

Influence of Resting Energy Expenditure on Blood Pressure is Independent of Body Mass and a Marker of Sympathetic Tone

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Abstract

Introduction—two recent examinations reported a strong association between blood pressure (BP) and resting energy expenditure (REE), independent of body mass and body composition. Both reports postulate that neuro-humoral processes that contribute to variation in REE may partly mediate the body mass effect on BP. Therefore, we examined the relationship of REE and BP in 108 asymptomatic women to (a) confirm previous findings in a novel population, and (b) to examine the impact of a marker of sympathetic tone on this relationship, as this was indicated as a potentially salient intermediary in previous reports.

Methods—all testing was performed during a 4-day admission to the General Clinical Research Center. Resting energy expenditure was measured by indirect calorimetry; body composition was determined by DEXA; and 24-hour fractionated urinary norepinephrine was determined by HPLC.

Results—multiple linear regression revealed REE as a significant predictor of SBP ($\beta = 0.30$, $P = 0.04$), independent of race ($\beta = 0.28$, $P = 0.01$), age ($\beta = -0.02$, $P = 0.80$), height ($\beta = -0.38$, $P = 0.08$), fat mass ($\beta = 0.22$, $P = 0.20$), fat free mass ($\beta = 0.08$, $P = 0.65$), and 24-hour fractionated urinary norepinephrine ($\beta = 0.06$, $P = 0.57$); and the same model using DBP as the dependent variable approached significance ($\beta = 0.24$, $P = 0.09$).

Discussion—this study affirms previous findings that REE may be a potential mediator in resting BP, independent of many well-cited factors and, additionally, a marker of sympathetic tone.

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ClinicalTrials.gov identifier: NCT00067873

Conflict of Interest: No financial conflicts exist.

Author Contributions:
Study concept and design: Brock, Hunter
Acquisition of data: Brock, Hunter
Drafting of the manuscript: Brock, Hunter, Tompkins, Fisher
Critical revision of the manuscript for important intellectual content: Brock, Hunter, Tompkins, Fisher
Statistical expertise: Hunter, Brock
Administrative, technical, or material support: Brock, Hunter
Study Supervision: Hunter

Keywords

Weight Loss; Hypertension; Obesity; Metabolism; Body Fat

Introduction

The association between body mass and blood pressure (BP) has been widely reported, and obesity is considered a major risk factor for hypertension (HTN) [1–3]. Despite considerable scientific effort, the underlying causal mechanisms remain to be clearly elucidated, partly due to the widespread impact of obesity (or the process of becoming obese) on endocrine, physiologic, and metabolic function [4]. Furthermore, the temporal relationship between BP and weight gain, weight loss, and/or (re) distribution of weight remains controversial, and complicating matters further, HTN does not present uniformly with obesity and is commonly observed in lean individuals. Notwithstanding these challenges, several potential underlying mechanisms (i.e. ion exchange, sympathetic tone, insulin sensitivity, oxidative stress, etc.) have been identified as mediators of BP and are thought to impact BP before appreciable weight gain occurs [5–8]. Recently, resting energy expenditure (REE), a sum measurement of all metabolic processes at rest, has been reported to be strongly associated with BP, independent of age, race, and anthropometric measures [9–11]. Although REE and body mass have a strong linear relationship, body size and/or composition do not account for all of the variation in REE, and therefore, the remaining variation in REE is likely attributable to inter-individual disparities in several underlying neuro-humoral systems that may directly impact BP, as others have reported [3–6, 9, 12–14].

In a direct examination of REE and BP, Kunz et al [9] observed significantly higher REEs in obese hypertensives compared to matched obese normotensives (obese HTN's > REE by ~9 %), indicating a potential discrepancy in the presumed causal relationship between obesity and HTN. More recently, two studies have observed a similar significant association in a cross-sectional analysis of a large sample of Siberians, Africans, and African-Americans [10, 11]. Resting energy expenditure in both reports emerged as a robust predictor of systolic and, lesser so, diastolic blood pressure. In multivariate models, Luke et al [10] observed that REE is a highly significant predicting variable of BP, independent of body mass and adiposity, and entirely accounted for the BMI effect on BP in a large sample of normal-weight Africans (Nigeria) and overweight African-Americans (Chicago, U.S.). In congruence with these findings and predicated off of Luke et al's examination, Snodgrass and colleagues [11] observed that basal metabolic rate was strongly correlated with BP, independent of body mass and composition, as well as age, smoking status, ethnicity, and degree of urbanization in a large sample of three different indigenous normal-weight Siberian populations. Both of these reports underscore Kunz et al's findings that the presumed relationship between body weight/composition and BP/HTN may not entirely be a consequence of more body weight/adiposity. In both examinations, variation in sympathetic tone was highlighted as one of several potential underlying mechanisms in the REE/BP relationship that may partially manifest as differences in REE.

Although these two reports have yet to be directly supported in a large clinical sample in the U.S., if confirmed, REE's influence on BP may be a potential novel and under-addressed area of inquiry in the patho-physiological understanding of the link between body mass/composition and BP. Therefore the purpose of the present study was two-fold. The first was to confirm the previous findings in a large clinical sample of African- and European-American women that reside in Birmingham, Alabama. The second was to determine if REE was predictive of BP, independent of similarly examined demographic and anthropometric

variables, as well as 24-hour fractionated norepinephrine, a well-known marker of sympathetic tone.

Methods

Subjects

Fifty-three African-American and 55 European-American normal-weight, normotensive women were included in the present analysis (35 ± 6.3y; 23.8 ± 1.1 kg/m^2; 110.8 ± 8.6/62.1 ± 7.8 mmHg). Full descriptive characteristics are reported in Table 1. Participants were overall healthy, asymptomatic, premenopausal European- and African-American women that reside in Birmingham, AL and recruited to participate in a larger clinical trial designed to examine metabolic factors of body mass/composition. All participants were non-smokers and free of medication that may affect blood pressure and energy expenditure. Because metabolism may be affected by the menstrual cycle, all testing was performed in the follicular phase of the menstrual cycle (within 10 days of menses). Procedures followed were in accordance with the ethical standards of the institution committee on human experimentation. Before participating in the study, all participants provided informed consent for the protocol, which was approved by the Institutional Review Board and Human Services Regulation for Protection of Human Research Subjects.

Study Design

Subjects participating in the study were maintained in a weight-stable state for 4 weeks prior to evaluation in the General Clinical Research Center (GCRC), as a part of the parent study on the role of metabolic factors on obesity. Body weight measurements were made during visits to the GCRC 3 days/week for the first 2 weeks, and 5 days/week for the last 2 weeks. During the final 2-week period, all meals were provided by the GCRC research kitchen to establish dietary consistency across all subjects, as differences in diet quality and composition (i.e. sodium consumption) have been reported to affect blood pressure and energy expenditure [15]. After the 4-week weight and diet stabilization period, subjects were admitted to the GCRC for 4 days, during the follicular phase of the menstrual cycle, and underwent assessment of blood pressure, body size, body composition, and energy expenditure. After all assessments were completed, subjects were discharged from the GCRC.

Anthropometric Assessment

Body height (stretch stature) to the nearest 0.1 cm was assessed using a stadiometer, and body weight to the nearest 5 g was assessed using a digital scale. All measurements were performed while subjects were in a fasted state and immediately after they voided in the morning by trained GCRC staff. Body composition was evaluated using dual-energy X-ray absorptiometry (Lunar DPX-L densitometer; LUNAR Radiation, Madison WI) and GE's Adult Software version 1.33, as previously described [16].

Blood Pressure

Blood pressure was measured with automatic auscultation while lying in the supine position. Readings were taken in the morning after a 12-h fast, and were reported as an average of three successive mornings.

Resting Energy Expenditure

During three consecutive mornings after an overnight stay in the GCRC and a 12-h fast, REE was measured immediately after awakening between the hours of 6 and 7 AM. Subjects were not allowed to sleep during the procedure and measurements were made in a

quiet, softly lit, well-ventilated room. Room temperature was maintained between 22 and 24 °C, and the participant was in a supine position on a hospital bed. After resting for 15 min, REE was measured for 30 min with a computerized, open-circuit, indirect calorimetry system with a ventilated canopy (Delta Trac II; Sensor Medics, Yorba Linda, CA). The last 20 mins of measurement were used for analysis. Oxygen uptake (VO_2) and carbon dioxide production (CO_2) were measured continuously and values were averaged at 1-min intervals. Coefficient of variation for the repeat REE was < 4%. As REE did vary slightly from day to day, the average REE for the three consecutive mornings was considered more reflective of the subjects' normal REE, and thus the value reported.

Sympathetic Tone

High-performance liquid chromatography (HPLC) was use to provide fractionated catecholamine measurements of norepinephrine. During the 4-day admittance to the GCRC, urine specimens were collected for a 24-hour period. The 24-hr urine specimen collection samples were preserved with 6 N HCL, and subsequent 10 ml aliquots were used for HPLC analysis [17].

Statistical Analysis

Descriptive characteristics are reported as means and standard deviations. Simple Pearson correlations were used to identify potential predictors of SBP, DBP, and pulse pressure (PP). Age, ethnicity, height, fat mass (FM), fat-free mass (FFM), norepinephrine, and REE were selected as independent variables for the multivariate regression analyses. Subsequently, the variables of interest were included in multiple linear regression analysis, with SBP, DBP, and PP as dependent variables. All analyses were performed using the Statistical Package for the Social Sciences (SPSS), version 10 (SPSS, Chicago, IL).

Results

Simple correlation analysis revealed previously well documented associations between REE and body size/composition, and as expected, REE was most strongly associated with FFM ($r = 0.58$; $P < 0.01$). Similarly expected was our observed association between REE and ethnicity ($r = -0.32$; $P < 0.01$), as our group has examined this issue extensively and found that African-American women have lower REEs than European-American women [18]. However, this association must be interpreted cautiously because of typically unaccounted for differences in bone density, limb and trunk body composition, and visceral organ mass that may confound REE and body composition comparisons between African- and European-American women [18]. Blood pressure was not associated with REE in simple correlation analysis. This is contrary to Luke and colleagues [10] report and is likely explained by the limited variation of BP in our sample, as Luke et al's sample had, on average, 2 to 3 times greater variation in resting BP. Both pulse pressure (PP) and norepinephrine trended towards significance with REE. The full correlation analysis is presented in Table 2.

Multiple linear regression analysis revealed several well reported predictors of SBP, DBP, and PP (age, ethnicity, height, FM), as well as a more recent, and our primary variable of interest for this report, REE. Resting energy expenditure was a significant predictor of SBP ($\beta = 0.30$, $P = 0.04$), independent of race ($\beta = 0.28$, $P = 0.01$), age ($\beta = -0.02$, $P = 0.80$), height ($\beta = -0.38$, $P = 0.08$), fat mass ($\beta = 0.22$, $P = 0.20$), fat free mass ($\beta = 0.08$, $P = 0.65$), and 24-hour fractionated urinary norepinephrine ($\beta = 0.06$, $P = 0.57$); and the same model using DBP as the dependent variable approached significance ($\beta = 0.24$, $P = 0.09$); whereas, REE was not a significant predictor of PP ($\beta = 0.09$, $P = 0.50$). The full results of the linear regression models are reported in Table 3.

Discussion

Resting energy expenditure has been recently reported to mediate the body mass effect on blood pressure in normal-weight Siberians, normal-weight Africans, and over-weight African-Americans. The current study extends the previous findings, in that REE appears to mediate the body mass effect on BP in a sample of normal-weight African-American and European-American women who reside in Birmingham, AL. Furthermore, the current findings support the strength of this relationship, as it is independent of many of the commonly reported anthropometric/demographic variables and, additionally, a marker of sympathetic tone.

Epidemiological reports have consistently demonstrated a myriad of chronic disease outcomes with advancing BMI [9, 18–21]. This has often led to a working hypothesis that obesity, per se, is the primary causal feature or risk factor for metabolic aberrations and overt chronic disease conditions with down-stream consequences on metabolic processes. The present analysis along with other reports would suggest that alterations in metabolic and neuro-humoral processes may precede, act in concert with, or be entirely independent of body weight [9–14]. Although the inherent difficulty of parsing out the causal features and the temporal relationship between indentified actors and averse health outcomes remains very challenging, the present confirmation that REE is associated with BP, independent of body weight, in normal-weight, healthy women confirms that the association between BMI and BP may be considerably confounded by REE. As Kunz et al [9] and Luke et al [10] eluded to in previous reports, REE is most likely serving as a proxy for other correlated neuro-humoral processes and/or systemic oxidative stress that are direct actors on BP, and body weight in of itself is not a primary determinant of BP, but may serve as an exacerbating factor during the process of adding additional body weight, primarily adipose tissue, as a result of positive energy balance. Additionally, variation in brown adipose tissue quantity and activity may be an underlying contributing factor that affects metabolic rate, thermogenesis, and sympathetic tone [22]

The role of catecholamines, insulin, and leptin are all considered primary neurohormonal factors in obesity-related HTN. Although methodological complications are still present and reports are mixed about the relationship between epinephrine and norepinephrine with body weight, Ward et al [23] in the Normative Aging Study found that in 752 non-diabetic men urinary norepinephrine excretion levels were strongly related to BP. In a similar study Masuo et al [24] reported in a cross-sectional sample of 724 Japanese men that urinary norepinephrine was an independent predictor of BP. Our finding in the current study that urinary norepinephrine is not a predictor of BP should be interpreted cautiously. In both of the previous reports, the participants had a wide range of BMIs, ranging from normal to obese; whereas, our cohort were all in the normal BMI category, with a small range of BMIs. According to the Landsberg [25] hypothesis of obesity, elevated SNS activity is a compensatory mechanism in response to obesity that acts to increase metabolic rate to offset the positive energy balance and prevent further weight gain. As a result of this, the well known effects of elevated SNS activity on the heart, kidneys, and vasculature are unfortunately impacted as well and have a deleterious consequence on BP. In our cohort, due to the normal BP and BMI, although urinary norepinephrine was not predictive of BP, this does not necessarily provide evidence that SNS activity is not an important contributing factor to obesity-related elevations in BP or overt HTN, as previous reports have linked SNS over-activity to higher HR and metabolic factors such as hyperinsulinemia, insulin resistance, hyperleptinemia, and obesity-related HTN [26].

In summary, this report extends previous findings that REE may be an important mediator of BP. Body mass accounts for the preponderance of the inter-individual variation in REE, and

the remaining variation in REE is likely due to other metabolic processes that may serve as intermediaries in the REE/BP association. The collective findings from our report along with other recent findings provide compelling evidence that the BMI-independent variation in REE is capturing an underlying metabolic process that is likely a pathway involved in obesity-related HTN. Although, the current report offers little insight into the underlying mechanisms, the proxy features of REE on the BP/HTN relationship clearly warrant further investigation, as this may be an important addition to the conceptual framework of obesity-related HTN.

Acknowledgments

Funding: This research was in part supported by National Institutes of Health grants T32-HL007457-25, R01 DK 49779, General Clinical Research Center grant M01 RR-00032, and Clinical Nutrition Research Unit grant P30-DK 56336.

Key to Abbreviations

BP	Blood Pressure
HTN	Hypertension
REE	Resting Energy Expenditure
GCRC	General Clinical Research Center
HPLC	High Performance Liquid Chromatography
DEXA	Dual Energy X-Ray Absorptiomery
FM	Fat Mass
FFM	Fat Free Mass

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Table 1

Descriptive Statistics (N = 108)

Characteristics	Mean ± SD
Age (y)	34.8 ± 6.3
Ethnicity (% African-American)	49.2
Height (cm)	165.8 ± 6.4
Body Weight (kg)	65.3 ± 6.4
BMI (kg/m^2)	23.8 ± 1.1
Fat Mass (kg)	22.2 ± 4.3
Fat Free Mass (kg)	43.9 ± 3.9
Mean Systolic Blood Pressure (mm Hg)	110.8 ± 8.6
Mean Diastolic Blood Pressure (mm Hg)	62.1 ± 7.8
Pulse Pressure (mm Hg)	48.7 ± 6.4
Resting Energy Expenditure (kcal/d)	1290.9 ± 141.1
24-Hour Fractionated Norepinephrine (μg)	35.4 ± 14.2

Table 2

Pearson Correlation Matrix for Resting Energy Expenditure, Body Composition, and Blood Pressure (N = 108).

	REE (kcal/d)	Age (y)	Ethnicity	Body Mass (kg)	Fat Mass (kg)	Fat-Free Mass (kg)	Height (cm)	BMI (kg/m^2)	SBP (mm Hg)	DBP (mm Hg)	Pulse Press (mm Hg)	24-Hour Norepi (μg)
REE	--	−0.14	−0.32**	0.56**	0.30**	0.58**	0.51**	0.33**	0.13	0.01	0.18 (0.05)	0.18 (0.06)
Age		--	−0.14	0.03	0.03	−0.04	−0.01	0.03	−0.05	0.16	−0.26**	0.25*
Ethnicity			--	−0.02	0.15	0.14	−0.02	0.04	0.18 (0.06)	0.23*	−0.3	0.08
Body Weight				--	0.80**	0.74**	0.90**	0.21*	0.06	−0.05	0.15	0.29**
FM					--	0.18	0.72**	0.48**	0.02	−0.06	0.10	0.30**
FFM						--	0.70**	0.47**	0.08	−0.01	0.12	0.14
Height							--	0.21*	−0.01	−0.04	0.04	0.29**
BMI								--	0.17	−0.01	0.25**	0.13
SBP									--	0.70**	0.50**	0.10
DBP										--	−0.26**	0.11
PP											--	0.00
24-hour Norepi												--

* = < 0.05;

** = < 0.01

Metabolism. Author manuscript; available in PMC 2013 February 1.

Table 3

Regression Models for the Relation Between Blood Pressure and Resting Energy Expenditure (N = 108)

Model	B	R^2	β	P
Systolic Blood Pressure		0.12		0.09
Intercept	153			
Resting Energy Expenditure (kcal/d)	0.01		0.30	0.04
Age (y)	−0.04		−0.03	0.80
Ethnicity	4.84		0.28	0.01
Height (cm)	−0.51		−0.38	0.08
Fat Mass (kg)	0.43		0.22	0.20
Fat-Free Mass (kg)	0.18		0.08	0.65
24-Hour Fractionated Norepinephrine (μg)	0.04		0.06	0.57
Diastolic Blood Pressure		0.11		0.12
Intercept	52.9			
Resting Energy Expenditure (kcal/d)	0.03		0.24	0.09
Age (y)	0.21		0.17	0.1
Ethnicity	4.85		0.31	0.006
Height (cm)	0.05		0.00	0.99
Fat Mass (kg)	−0.13		−0.08	0.66
Fat-Free Mass (kg)	−0.34		−0.08	0.34
24-Hour Fractionated Norepinephrine (μg)	0.03		0.04	0.70
Pulse Pressure		0.15		0.02
Intercept	101.6			
Resting Energy Expenditure (kcal/d)	0.00		0.09	0.5
Age (y)	−0.26		−0.26	0.009
Ethnicity	−0.15		−0.12	0.91
Height (cm)	−0.52		−0.53	0.01
Fat Mass (kg)	0.57		0.39	0.02
Fat-Free Mass (kg)	0.55		0.37	0.056
24-Hour Fractionated Norepinephrine (μg)	0.02		0.04	0.7