Int J Clin Exp Med 2016;9(6):12375-12383
www.ijcem.com /ISSN:1940-5901/IJCEM0021503

Original Article

Basal metabolic rate is correlated with blood pressure among young population

Tao Chen[1,2*], Xiaofang Chu[4*], Yongkai Zhu[1*], Yiting Lan[1], Hailong Zhao[1], Huifeng Zhu[3], Chong Shen[1]

[1]Department of Epidemiology and Biostatistics, School of Public Health, Nanjing Medical University, Nanjing 211166, China; [2]Division of Health and Social Care Research, King's College London, London SE1 1UL, UK; [3]Pathology Pharmacology Teaching and Research Group, Nanjing Health School of Jiangsu Union Technical Institute, Nanjing 210038, China; [4]Department of Health Inspection and Quarantine, School of Public Health, Nanjing Medical University, Nanjing 211166, China. [*]Equal contributors.

Received December 9, 2015; Accepted March 19, 2016; Epub June 15, 2016; Published June 30, 2016

Abstract: Backgrounds: Our study was to confirm the positive relationship of predicted basal metabolic rate (BMR) and blood pressure (BP) and to determine if BMR was still predictive, irrespective of demographic and anthropometric variables in young Chinese population. Methods: BP measurements were performed by trained workers through a standardized method using Omron HE: 7071 electronic sphygmomanometer. BMR values were calculated by a given formula of Omron HBF-306 Body Fat Monitor. Results: The mean systolic blood pressures (SBP) and diastolic blood pressures (DBP) as well as pulse pressure (PP) increased with increasing BMR deciles (SBP by 20 mmHg, DBP by 4 mmHg and PP by 15 mmHg from the 1st decile to the 5th decile, P_{trends}<0.001 for all). Pearson correlation analyses indicated that BMR was positively correlated with SBP (r=0.601, P<0.001), DBP (r=0.243, P<0.001) and PP (r=0.588, P<0.001). After adjusting with potential confounding factors, the regression coefficients indicated that every 1 Kcal/day higher BMR level was associated with an increase of 0.021 mmHg SBP (P<0.001), 0.008 mmHg DBP (P<0.001) and 0.013 mmHg PP (P<0.001). This did not alter substantially after further adjustment for waist circumference. In addition, we also found that the relationship between BMR and BP did not materially change, particularly for SBP and PP, after using a series of equations for BMR calculation. Conclusions: The study showed that the predicted BMR might be a good metabolic surrogate to predict the development of high blood pressure considering its simplicity and feasibility in clinical practices.

Keywords: Blood pressure, basal metabolic rate, hypertension

Introduction

High blood pressure (BP) is considered as an independent risk factor of cardiovascular diseases [1]. It was suggested that in East Asian populations, diastolic blood pressure (DBP) values of 80 mmHg or more could explain more than 50% of deaths due to strokes and almost 25% of deaths associated with coronary heart disease [2]. In China, hypertension was responsible for 24.6% of all-cause mortality and 64.0% of the deaths related to cardiovascular diseases in 2010 [3].

Many factors contribute to the variations of BP, including age, gender, smoking, drinking, metabolism and heredity [4, 5]. Previous studies have proved that obesity is strongly correlated with blood pressure [6-8]. A study in Israel found that body mass index (BMI) in adolescents was significantly associated with systolic blood pressure (SBP) and DBP in both genders and in both the normal weight and overweight groups [6].

The exact mechanisms that contribute to elevated BP in people with obesity are not clear. Some studies reported that obesity-related hypertension were associated with abnormalities of metabolism, including abnormal levels of insulin, leptin and cytokines, renin-angiotensin-aldosterone system activation, and stimulation of sympathetic nervous system [9, 10]. Furthermore, studies showed that people with a high sensitivity of energy imbalance were less prone to gain weight [11] and another study revealed that obesity could be prevented by increasing energy expenditure [12]. Therefore,

several studies have investigated the interrelationship to discover whether basal metabolic rate (BMR), measured by open/close-circuit metabolic analyzer, may potentially mediate resting BP and results revealed that BMR was a significant determinant of SBP and DBP even after adjustment for many well-cited factors such as body size, fat mass and physical activity [13, 14].

Apart from the direct measured BMR, several predictive equations were widely used because of its simplicity and feasibility in most time [15]. However, few studies were performed to testify whether the positive relationship between predictive BMR and SBP, DBP and pulse pressure (PP) still exist among young population. Therefore the purpose of the present study was two-fold. The first was to confirm the previous findings in young Chinese population even using various predictive formulations. The second was to determine if BMR was predictive of BP, irrespective of demographic and anthropometric variables.

Methods

Study population

A cross-sectional study was conducted in a medical university and data of questionnaire were collected by uniformly trained surveyors. In total, 802 students in grade one were chosen and recruited. The participants came from different majors, including Clinical Medicine, Medicine, Health Law, Optometry and Ophthalmology, Oral Medicine, English, Clinical Medicine, Rehabilitation Medicine, Laboratory Medical Science, Nursing, Preventive Medicine, Hygienic Detection and Analysis, and Biostatistics.

Measurement and definition of BP

Three separate times of BP measurements were performed by uniformly trained survey or through a standardized procedure using Omron HE: 7071 electronic sphygmomanometer (OMRON (Dalian) CO., LTD. China). Participants were required to avoid to do intense sports and fast in one hour before measurements. Besides, they were asked whether they had taken any medicines which could affect blood pressure. After a 5 minute resting, participants were asked to sit in a relaxed and standard posture and taken measurements. All participants were needed to measure BP for three times. SBP, DBP and heart rate were recorded. The mean values of three measurements were used in the statistical analyses.

Measurement of risk factors

Age, gender, waist circumference (WC), BMI and family history of hypertension (FHH) were considered as risk factors of hypertension. Relevant data were recorded on their individual questionnaires. Height and weight were measured when participants took off heavy clothes and shoes. Besides, subjects were asked to keep an erect posture with their eyes looking at the front horizontally. Electronic weight meter and meter ruler should been calibrated before every measurement. BMI was calculated as the weight in kilograms divided by the square of the height in meters. WC was measured on bare skin as the narrowest circumference between the lower costal margin and the iliac crest in centimeters. FHH was recorded on the condition that at least one immediate family relative was recalled as hypertension.

BMR determination

BMR was obtained from a hand-to-hand bioelectric impedance analysis instrument (Omron HBF-306, OMRON (Dalian) CO., LTD. China). All procedures were performed according to the manufacturer's instructions. The subjects did not exercise or consume caffeine or alcohol prior to the measurement. Having rested quietly for at least two hours after dinner, participants took measurements of BMR in a standardized position under a relaxed state. Anthropometric data like height and weight as well as gender and age information were filled into the instrument and the device was held while both arms were stretched horizontally in front of the body. The equation used by this instrument to calculate BMR is based on Dietary Reference intakes for Japanese and indicated as follows: Male: 18.6*weight+347; Female: 18.3*weight+272 (age range: 18-29 years old).

Statistical methods

Data was input with Epi Data 3.1 software package. Continuous data with normal distribution were reported as mean (± standard devia-

Table 1. Characteristics of enrolled participants

Measure	Female (n=548)	Male (n=226)	Total (n=774)
Age (years)	19.9±0.7	20.1±0.9	19.9±0.8
Height (cm)	161.2±5.6	173.8±7.2	164.9±8.4*
Weight (kg)	54.9±7.6	66.7±10.2	58.3±10.0*
BMI (kg/m^2)	21.0±2.6	22.0±3.2	21.3±2.8*
WC (cm)	73.5±6.7	79.2±8.1	75.2±7.6*
SBP (mmHg)	110.3±9.0	126.1±12.1	114.9±12.3*
DBP (mmHg)	67.8±6.8	70.1±7.7	68.5±7.1*
PP (mmHg)	42.5±6.4	56.0±9.6	46.4±9.7*
BMR (Kcal/day)	1277.0±143.4	1590.7±214.7	1368.6±219.9*
FHH (n, %)	392 (71.53%)	134 (59.29%)	526 (67.96%)*

Differences between female and male were statistically significant at *P<0.001. Abbreviations: BMI, Body mass index; WC, Waist circumference; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PP, Pulse pressure; BMR, Basal metabolic rate; FHH, Family history of hypertension.

tion). Categorical data were shown as number (percentage). Categorical variables were compared using chi-square test and continuous variables were compared using the independent sample *t* test.

Pearson correlation analysis was used to test the correlation of age, height, weight, BMI, WC, BMR, SBP, DBP and PP. Multiple linear regressions were performed to evaluate the effect of BMR on BP by adjusting for age, gender, and FHH and then additionally adjusting for WC.

In order to examine the consistency of the relationship between BMR and BP (SBP, DBP and PP), we further calculated the value of BMR and built multiple linear regression models by the following algorithms: Harris and Benedict Equation (HBE) [16], Mifflin-St Jeor Equation (MSJE) [17], LIU equation (LIUE) [18], Kleiber's Law (KL) [19] and Owen Equations (OE) [20, 21]. Statistical analyses were conducted by Stata 12.0 software package. Differences were regarded statistically significant at P<0.05.

Results

Characteristics of participants

Overall, 744 (548 females, 226 males) students finished the survey questionnaire and accepted physical examination with a response rate of 92.8%. The average age of participants was 19.9±0.8 years old.

Descriptive statistics for anthropometric, blood pressure, and metabolic data for the total sample and different genders were presented in **Table 1**. Generally, Males had significantly higher height, weight, BMI, WC, SBP, DBP, PP and BMR than females. The percent of participants with family history of hypertension in females was 71.53%, which was higher than that in males (59.29%). All differences of parameters except age between female and male were statistically significant at P<0.05.

Mean SBP has increased along with the increasing BMR deciles (from 106.3±7.5 mmHg in the 1st decile to 126.6±12.0 mmHg in the 5th decile, P for trend <0.001) and mean DBP has increased along with increasing BMR deciles (from 66.2±6.3 mmHg in the 1st decile to 70.7±7.9 mmHg in the 5th decile, P for trend <0.001). The same pattern persisted in mean PP (from 40.1±5.7 mmHg in the 1st decile to 55.9±10.2 mmHg in the 5th decile, P for trend <0.001). Data was shown in **Table 2**. Supplementary analysis by the gender did not find materially change as seen from total population (Tables S1 and S2). From the above results in tables, we could not find a threshold where the association became significant.

Correlation analysis

BMR was positively correlated with height (r=0.646, P<0.001), weight (r=0.963, P<0.001), BMI (r=0.741, P<0.001), WC (r=0.771, P<0.001), SBP (r=0.601, P<0.001), DBP (r=0.243, P<0.001) and PP (r=0.588, P<0.001) in whole study population (**Table 3**). Further partial correlation analysis by adjustment for age showed the consistent results. BMR still had a strong association with height (r=0.645, P<0.001), weight (r=0.963, P<0.001), BMI (r=0.752, P<0.001), WC (r=0.771, P<0.001), SBP (r=0.601, P<0.001), DBP (r=0.244, P<0.001) and PP (r=0.588, P<0.001).

In males, BMR increased with the augmentation of height (r=0.246, P<0.001), weight (r=0.932, P<0.001), BMI (r=0.844, P<0.001), WC (r=0.788, P<0.001), SBP (r=0.369, P<0.001), DBP (r=0.139, P=0.037) and PP (r=0.353, P<0.01). In females, positive correlations were also observed between BMR and height (r=

Table 2. Distribution of BP and BMR data

Deciles of BMR	Mean BMR (Range) (Kcal/day)	Number of valid subjects	SBP (mmHg)	DBP (mmHg)	PP (mmHg)
1	1124.6±46.5 (953-1187)	155	106.3±7.5	66.2±6.3	40.1±5.7
2	1233.4±26.9 (1191-1279)	155	109.7±8.3	67.0±6.8	42.7±6.1
3	1326.2±29.7 (1279-1385)	154	112.0±9.9	68.3±6.7	43.8±6.7
4	1451.4±43.6 (1385-1530)	155	119.8±11.2	70.2±6.8	49.6±9.5
5	1706.9±186.5 (1531-2988)	155	126.6±12.0	70.7±7.9	55.9±10.2
	P for trend		<0.001	<0.001	<0.001

Abbreviations: BP, Blood pressure; BMR, Basal metabolic rate; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PP, Pulse pressure.

Table 3. Correlation matrix for age, Anthropometric, metabolic and BP data

	Age (years)	Height (cm)	Weight (kg)	BMI (kg/m^2)	WC (cm)	SBP (mmHg)	DBP (mmHg)	PP (mmHg)	BMR (Kcal/day)
Age (years)	1.000	0.053	0.022	-0.022	0.026	0.016	-0.046	0.055	0.031
Height (cm)		1.000	0.610*	0.090**	0.360*	0.475*	0.124*	0.515*	0.646*
Weight (kg)			1.000	0.814*	0.816*	0.572*	0.247*	0.548*	0.963*
BMI (kg/m^2)				1.000	0.780*	0.382*	0.246*	0.306*	0.741*
WC (cm)					1.000	0.413*	0.222*	0.363*	0.771*
SBP (mmHg)						1.000	0.623*	0.817*	0.601*
DBP (mmHg)							1.000	0.058	0.243*
PP (mmHg)								1.000	0.588*
BMR (Kcal/day)									1.000

Significant difference at *p<0.001, **p<0.05. Abbreviations: BMI, Body mass index; WC, Waist circumference; BP, Blood pressure; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PP, Pulse pressure; BMR, Basal metabolic rate.

0.458, P<0.001), weight (r=0.982, P<0.001), BMI (r=0.859, P<0.001), WC (r=0.763, P<0.001), SBP (r=0.353, P<0.001), DBP (r=0.235, P<0.001) and PP (r=0.250, P<0.001). Data of males and females were listed in Tables S3 and S4), respectively.

Linear regression analysis

Multiple linear regression analysis was used to reveal the potential effects of BMR on BP (SBP, DBP and PP). The regression coefficients indicated that every 1 Kcal/day higher BMR level was associated with an increase of 0.021 mmHg SBP (P<0.001), 0.008 mmHg DBP (P<0.001) and 0.013 mmHg PP (P<0.001). Further adjustment for WC, the effect still presented statistical significance (0.021 mmHg SBP, P<0.001; 0.005 mmHg DBP, P=0.030 and 0.016 mmHg PP, P<0.001). Data were listed in **Table 4**.

Compared with other factors, the standardized regression coefficient of BMR indicated that BMR was the most powerful predictor of SBP (standardized β=0.371), DBP (standardized β=0.157) and PP (standardized β=0.358).

Analysis of the association between BMR and BP indicated that BMR accounted for 36.1%, 5.9% and 34.6% of the variance in SBP, DBP and PP, respectively, while combining with other factors (age, gender, WC and FHH) explained 42.7% for SBP, 6.6% for DBP and 45.8% for PP.

Additionally, we testified the positive finding of BMR on BP with five equations. The predicted values of BMR with these equations (HBE, MSJE, LIUE, KL and OE) were 1558.5±141.8, 1401.8±196.0, 1402.1±198.1, 1473.7±187.9 and 1297.2±183.7, respectively. Compared with the measured BMR value 1368.6±219.9, most of the calculated values were larger than measured values except that of OE. Multiple linear models were also built as before (**Table 5**). The regression coefficients for the relation between BMR and SBP were 0.032, 0.028, 0.028, 0.026 and 0.055, respectively. All of the

Table 4. Multiple linear regression models using SBP, DBP and PP as dependent variables, respectively

Variables	SBP			DBP			PP		
	Coeff (SE)	*P*	Standardized β	Coeff (SE)	*P*	Standardized β	Coeff (SE)	*P*	Standardized β
Model 1									
Age	-0.561 (0.446)	0.209	-0.035	-0.482 (0.330)	0.145	-0.051	-0.079 (0.341)	0.816	-0.006
BMR	0.021 (0.002)	<0.001	0.38	0.008 (0.001)	<0.001	0.245	0.013 (0.002)	<0.001	0.304
Gender (female vs. male)	-9.286 (0.989)	<0.001	-0.343	0.009 (0.731)	0.99	0.001	-9.295 (0.755)	<0.001	-0.438
FHH (yes vs. no)	-0.305 (0.729)	0.675	-0.012	-0.415 (0.539)	0.442	-0.027	0.109 (0.556)	0.845	0.005
Constant	113.158 (10.147)	<0.001		67.730 (7.501)	<0.001		45.427 (7.747)	<0.001	
Model R^2	0.427			0.062			0.457		
Model 2									
Age	-0.566 (0.447)	0.206	-0.035	-0.508 (0.330)	0.124	-0.054	-0.057 (0.341)	0.867	-0.005
BMR	0.021 (0.003)	<0.001	0.371	0.005 (0.002)	0.03	0.157	0.016 (0.002)	<0.001	0.358
Gender (female vs. male)	-9.357 (1.046)	<0.001	-0.345	-0.389 (0.772)	0.614	-0.025	-8.967 (0.798)	<0.001	-0.422
WC	0.015 (0.074)	0.835	0.009	0.087 (0.055)	0.111	0.093	-0.072 (0.056)	0.203	-0.057
FHH (yes vs. no)	-0.295 (0.731)	0.687	-0.011	-0.354 (0.539)	0.512	-0.023	0.059 (0.557)	0.915	0.003
Constant	112.894 (10.233)	<0.001		66.235 (7.552)	<0.001		46.659 (7.804)	<0.001	
Model R^2	0.427			0.066			0.458		

Abbreviations: BMR, Body metabolic rate; WC, Waist circumference; FHH, Family history of hypertension; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PP, Pulse pressure. Model 1: Adjusting age, gender, and FHH. Model 2: Additionally adjusting WC.

Table 5. Regression coefficients (standardized regression coefficients) of BMR for SBP, DBP and PP‡

Equation	SBP	DBP	PP
HBE	0.032 (0.365)*	0.005 (0.096)	0.027 (0.396)*
MSJE	0.028 (0.447)*	0.005 (0.142)	0.023 (0.466)*
LIUE	0.028 (0.448)*	0.006 (0.161)	0.022 (0.453)*
KL	0.026 (0.401)*	0.006 (0.163)**	0.020 (0.392)*
OE	0.055 (0.823)*	0.010 (0.261)	0.045 (0.857)*

Regression coefficient for the relationship between BMR and SBP, DBP and PP was statistical significant at *p<0.001, **p<0.05. Abbreviations: HBE, Harris and Benedict Equation; MSJE, Mifflin-St Jeor Equation; LIUE, LIU equation; KL, Kleiber's Law; OE, Owen Equations; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PP, Pulse pressure; BMR, Basal metabolic rate. ‡Adjusting for age, gender, WC and FHH.

regression coefficients were larger than that in original model (0.021). The comparable results existed in other two models using DBP and PP as dependent factors. Moreover, BMR was still the most powerful predictor by the standardized regression coefficient in comparison with other factors and the magnitude did not materially vary.

Discussion

As a measurement of least energy used by bodies of warm-blooded animals when they are under a relaxed, awake and fasting state [22], BMR can mirror the interaction between extrinsic and intrinsic environment [23]. Generally, there are two main methods to determine the value of calories that we need in a given day: one is called indirect calorimetry (relatively accurate, but more difficult), which uses expired gases to calculate the amounts and types of fuel being utilized; the other one is to use several different formulas (less accurate, but much easier). In this study, we evaluated the association between predicted BMR and BP in a young population (mean age, 19.9 years). We had shown that predicted BMR was significantly associated with SBP and DBP as well as PP. This relationship persisted after adjusting WC, an important factor with BP.

The main findings of this study are in congruence with the findings of previous researches. Previous study found that resting energy expenditure was positively connected with SBP independent of height, resting heart rate and body composition among overweight African- and European-American women with 34.8±6.3 years old [24], and the relation persisted across 4.5 years [25]. Differently, unless patients were regarded as obesity with a high BMI value (>30 kg/m^2), normotensive and hypertensive individuals presented no obvious difference in resting metabolic rate [26]. In this study, the results indicated that BMR was an independent risk factor of high BP, after adjustment for age, gender, WC, FHH and it was the most powerful predictor to BP elevation, especially for SBP and PP. Therefore, the remaining variation in blood pressure which cannot be explained by WC may have something to do with metabolic processes. Furthermore, by comparing predicted BMR values, which were reported to be suitable for different people [27-29] with those determined by indirect calorimetry, our result revealed that predicted BMR might be a good metabolic surrogate to monitor BP variations due to its simplicity and feasibility in clinical practices.

Nowadays, the mechanism contributed to the relation between BMR and BP is nebulous. Several possible mechanisms may be responsible. First, the blood pressure-raising systems played an important role in hypertension and over 50% of cases of high blood pressure could be classified as neurogenic essential hypertension [30]. Stimulated renal sympathetic nervous system (RSNS) links to upregulating BMR, which resulted in increased arterial pressure in essential hypertensive patients, particularly in young hypertensive persons [31]. Second, thyroid hormones were reported to mediate the association between BMR and blood pressure. Studies have found thyroid hormone levels (T3 [triiodothyronine] and T4 [thyroxine]) are closely related to BMR through their direct effects on rates of oxidative metabolism in most tissues [32], which in turn precipitates changes in cardiovascular function and blood pressure regulation [33]. Third, the relation between BMR and BP may be partly explained by heritable regulation. A loss-of-function mutation of the gene encoding the melanocortin4 receptor (MC4R) was reported to be associated with a low risk of high blood pressure [34], while genetic variation in Neuropeptide Y interacting with Y_1 receptor (NPY1R) could lead to dramatic increase of DBP [35]. Finally, developmental

factors could relate BMR to BP. Congenital undernutrition might lead to elevated food intake at early postnatal age [36] and a high-fat diet may increase renal sympathetic activity, as well as blood pressure [37]. Combined with the correlation results of BMR and BP, these findings supported that the elevated BMR was strongly relevant to the augmentation of BP, which may provide valuable orientation for future research.

Generally, this study has the following potential limitations. First, as a cross-sectional survey, the subjects were the young medical college students with a similar age, low obesity (1.4%) and unhealthy lifestyle rate (smoking: 0.6%; drinking: 0.8%), in a specific region. These results may be different from older population as well as the population in different countries, although a positive relationship between resting metabolic rate and SBP was reported to be independent of age and race [24]. Second, gender difference may cause potential stratum effect nonetheless previous study among Indigenous Siberians indicated that gender had no obvious influence on the association between BMR and BP [14]. In this study, we adjusted for age, gender, WC and FHH by multiple regression models and that would help to control potential bias in the effect of BMR on BP for whole population. Third, there might be residual confounding factors which can affect BP, such as mental stress [38], physical exercise, lifestyles [39] and so on.

In conclusion, the study showed that predicted BMR had a strong relationship with BP (SBP, DBP and PP), especially SBP and PP. The predicted BMR might be a good metabolic surrogate to predict the development of high blood pressure considering its simplicity and feasibility in clinical practices. Further replication of predicted BMR and BP would be warranted in a large sample and prospective study to testify which equations will be more suitable to cardiovascular risk stratification.

Acknowledgements

We thank Lulu Ren, Zhaoqi Zhang, Jiaqian Feng, Lei Zhang for preparation of the project. We also appreciate Yuchao Li, Yinjia Deng, Huiwen Qian, Xinxin Xu, Zhuoyi Shen, Hui Huang, Xue Wang, Fan Zhao, Congcong Zhang, Dongxin Jiang, Yixuan Li, Yingru Lin, Lujue Liu, Rui Dai, Xiaoqiang Sun, Shuying Guo, Jiali Jin, Rong Wu, Suhong Chen, Xiaomin Lu, Wen Zhang, Xinming Chu, Wenwen Yuan, Wencheng Zhou for their assistance with field survey. Finally, we want to express our gratitude to all subjects in this study. This work was supported by the key project of College Students Practice Innovation Training of Jiangsu Province (201310312006Z), the Research Project of Health Vocational and Technical Education of Jiangsu Province (JZ201312), the National Natural Science Foundation of China (No. 81273165) and the Priority Academic Program for the Development of Jiangsu Higher Education Institutions (Public Health and Preventive Medicine). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Disclosure of conflict of interest

None.

Address correspondence to: Dr. Chong Shen, Department of Epidemiology and Biostatistics, School of Public Health, Nanjing Medical University, 101 Longmian Avenue, Jiangning, Nanjing 211166, China. Tel: +86 25 86868443; Fax: +86 25 8652-7613; E-mail: sc@njmu.edu.cn

References

[1] Bloomfield GS, Wang TY, Boulware LE, Califf RM, Hernandez AF, Velazquez EJ, Peterson ED, Li JS. Implementation of Management Strategies for Diabetes and Hypertension: From Local to Global Health in Cardiovascular Diseases. Glob Heart 2015; 10: 31-38.

[2] Rodgers A, Lawes C, Macmahon S. Reducing the global burden of blood pressure-related cardiovascular disease. J Hypertens Suppl 2000; 18: S3-S6.

[3] Liu M, Li Y, Liu S, Wang W, Zhou M. [Burden on blood-pressure-related diseases among the Chinese population, in 2010]. Zhonghua Liu Xing Bing Xue Za Zhi 2014; 35: 680-683.

[4] Hypertension editors' picks: obesity-associated hypertension. Hypertension 2015; 65: e10-e16.

[5] Kunes J, Kadlecova M, Vaneckova I, Zicha J. Critical developmental periods in the pathogenesis of hypertension. Physiol Res 2012; 61 Suppl 1: S9-S17.

[6] Chorin E, Hassidim A, Hartal M, Havakuk O, Flint N, Ziv-Baran T, Arbel Y. Trends in Adolescents Obesity and the Association between BMI and Blood Pressure: A Cross-Sectional Study in 714,922 Healthy Teenagers. Am J Hypertens 2015; 28:1157-63.

[7] Rodrigues Barbosa A, Balduino Munaretti D, Da Silva Coqueiro R, Ferreti Borgatto A. Anthropometric indexes of obesity and hypertension in elderly from Cuba and Barbados. J Nutr Health Aging 2011; 15: 17-21.
[8] Chen T, Li W, Wang Y, Xu T, Hu B, Chen W, Zhu M. Body mass index and hypertension among Chinese governmental and institutional employees in Beijing. Angiology 2012; 63: 337-342.
[9] Vaneckova I, Maletinska L, Behuliak M, Nagelova V, Zicha J, Kunes J. Obesity-related hypertension: possible pathophysiological mechanisms. J Endocrinol 2014; 223: R63-R78.
[10] Grassi G. Sympathetic neural activity in hypertension and related diseases. Am J Hypertens 2010; 23: 1052-1060.
[11] Cornier MA, Mcfadden KL, Thomas EA, Bechtell JL, Bessesen DH, Tregellas JR. Propensity to Obesity Impacts the Neuronal Response to Energy Imbalance. Front Behav Neurosci 2015; 9: 52.
[12] Niwa M, Numaguchi Y, Ishii M, Kuwahata T, Kondo M, Shibata R, Miyata K, Oike Y, Murohara T. IRAP deficiency attenuates diet-induced obesity in mice through increased energy expenditure. Biochem Biophys Res Commun 2015; 457: 12-18.
[13] Luke A, Adeyemo A, Kramer H, Forrester T, Cooper RS. Association between blood pressure and resting energy expenditure independent of body size. Hypertension 2004; 43: 555-560.
[14] Snodgrass JJ, Leonard WR, Sorensen MV, Tarskaia LA, Mosher MJ. The influence of basal metabolic rate on blood pressure among indigenous Siberians. Am J Phys Anthropol 2008; 137: 145-155.
[15] Henry CJ. Basal metabolic rate studies in humans: measurement and development of new equations. Public Health Nutr 2005; 8: 1133-1152.
[16] Roza AM, Shizgal HM. The Harris Benedict equation reevaluated: resting energy requirements and the body cell mass. Am J Clin Nutr 1984; 40: 168-182.
[17] Mifflin MD, St Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr 1990; 51: 241-247.
[18] Liu HY, Lu YF, Chen WJ. Predictive equations for basal metabolic rate in Chinese adults: a cross-validation study. J Am Diet Assoc 1995; 95: 1403-1408.
[19] Smil V. Laying down the law. Nature 2000; 403: 597.
[20] Owen OE, Holup JL, D'Alessio DA, Craig ES, Polansky M, Smalley KJ, Kavle EC, Bushman MC, Owen LR, Mozzoli MA, Et A. A reappraisal of the caloric requirements of men. Am J Clin Nutr 1987; 46: 875-885.
[21] Owen OE, Kavle E, Owen RS, Polansky M, Caprio S, Mozzoli MA, Kendrick ZV, Bushman MC, Boden G. A reappraisal of caloric requirements in healthy women. Am J Clin Nutr 1986; 44: 1-19.
[22] Mcnab BK. On the utility of uniformity in the definition of basal rate of metabolism. Physiol Zool 1997; 70: 718-720.
[23] Parati G, Ochoa JE, Lombardi C, Bilo G. Assessment and management of blood-pressure variability. Nat Rev Cardiol 2013; 10: 143-155.
[24] Brock DW, Tompkins CL, Fisher G, Hunter GR. Influence of resting energy expenditure on blood pressure is independent of body mass and a marker of sympathetic tone. Metabolism 2012; 61: 237-241.
[25] Sriram N, Hunter GR, Fisher G, Brock DW. Resting energy expenditure and systolic blood pressure relationships in women across 4.5 years. J Clin Hypertens (Greenwich) 2014; 16: 172-176.
[26] Kunz I, Schorr U, Klaus S, Sharma AM. Resting metabolic rate and substrate use in obesity hypertension. Hypertension 2000; 36: 26-32.
[27] Frankenfield DC, Rowe WA, Smith JS, Cooney RN. Validation of several established equations for resting metabolic rate in obese and nonobese people. J Am Diet Assoc 2003; 103: 1152-1159.
[28] Rao ZY, Wu XT, Liang BM, Wang MY, Hu W. Comparison of five equations for estimating resting energy expenditure in Chinese young, normal weight healthy adults. Eur J Med Res 2012; 17: 26.
[29] Reidlinger DP, Willis JM, Whelan K. Resting metabolic rate and anthropometry in older people: a comparison of measured and calculated values. J Hum Nutr Diet 2015; 28: 72-84.
[30] Parati G, Esler M. The human sympathetic nervous system: its relevance in hypertension and heart failure. Eur Heart J 2012; 33: 1058-1066.
[31] Johns EJ, Kopp UC, Dibona GF. Neural control of renal function. Compr Physiol 2011; 1: 731-767.
[32] Fazio S, Palmieri EA, Lombardi G, Biondi B. Effects of thyroid hormone on the cardiovascular system. Recent Prog Horm Res 2004; 59: 31-50.
[33] Danzi S, Klein I. Thyroid hormone and blood pressure regulation. Curr Hypertens Rep. 2003; 5: 513-520.
[34] Greenfield JR, Miller JW, Keogh JM, Henning E, Satterwhite JH, Cameron GS, Astruc B, Mayer JP, Brage S, See TC, Lomas DJ, O'Rahilly S, Farooqi IS. Modulation of blood pressure by

central melanocortinergic pathways. N Engl J Med 2009; 360: 44-52.

[35] Wang L, Rao F, Zhang K, Mahata M, Rodriguez-Flores JL, Fung MM, Waalen J, Cockburn MG, Hamilton BA, Mahata SK, O'Connor DT. Neuropeptide Y(1) Receptor NPY1R discovery of naturally occurring human genetic variants governing gene expression in cella as well as pleiotropic effects on autonomic activity and blood pressure in vivo. J Am Coll Cardiol 2009; 54: 944-954.

[36] Vickers MH, Breier BH, Cutfield WS, Hofman PL, Gluckman PD. Fetal origins of hyperphagia, obesity, and hypertension and postnatal amplification by hypercaloric nutrition. Am J Physiol Endocrinol Metab 2000; 279: E83-E87.

[37] Lim K, Burke SL, Head GA. Obesity-related hypertension and the role of insulin and leptin in high-fat-fed rabbits. Hypertension 2013; 61: 628-634.

[38] Van Woudenberg M, Shin J, Bernard M, Syme C, Abrahamowicz M, Leonard G, Perron M, Richer L, Veillette S, Gaudet D, Paus T, Pausova Z. CYP17A1 and Blood Pressure Reactivity to Stress in Adolescence. Int J Hypertens 2015; 2015: 734586.

[39] Sfrantzis KD, How JM, Sartor DM. Implications of diet modification on sympathoinhibitory mechanisms and hypertension in obesity. Auton Neurosci 2015; 189:25-30.

Table S1. Distribution of anthropometric, BP and BMR data for 226 males

Deciles of BMR	Mean BMR (Range) (Kcal/day)	Number of valid subjects	SBP (mmHg)	DBP (mmHg)	PP (mmHg)
1	1359.6±55.5 (1221-1432)	45	119.5±10.6	69.9±6.6	49.6±7.1
2	1469.4±24.4 (1433-1508)	45	127.3±11.5	70.8±7.1	56.5±9.7
3	1550.4±24.2 (1511-1601)	46	122.8±12.1	68.6±8.1	54.2±7.8
4	1665.2±41.5 (1601-1727)	45	128.5±9.3	69.6±7.2	58.9±8.2
5	1909.6±220.7 (1727-2988)	45	132.5±12.6	71.8±8.9	60.7±11.0

Abbreviations: BP, Blood pressure; BMR, Basal metabolic rate; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PP, Pulse pressure.

Table S2. Distribution of anthropometric, BP and BMR data for 548 females

Deciles of BMR	Mean BMR (Range) (Kcal/day)	Number of valid subjects	SBP (mmHg)	DBP (mmHg)	PP (mmHg)
1	1104.8±40.7 (953-1158)	110	106.2±7.6	66.2±6.3	40.0±5.7
2	1193.0±19.3 (1158-1224)	109	108.4±8.1	66.6±6.5	41.8±6.2
3	1259.4±20.7 (1224-1293)	110	110.1±8.1	67.6±7.0	42.5±5.7
4	1332.3±25.6 (1293-1385)	109	110.8±9.9	67.7±6.7	43.1±6.6
5	1495.2±108.2 (1385-1865)	110	115.8±8.7	70.9±6.5	45.0±6.8

Abbreviations: BP, Blood pressure; BMR, Basal metabolic rate; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PP, Pulse pressure.

Table S3. Correlation matrix for age, Anthropometric, metabolic and BP data for 226 males

	Age (years)	Height (cm)	Weight (kg)	BMI (kg/m^2)	WC (cm)	SBP (mmHg)	DBP (mmHg)	PP (mmHg)	BMR (Kcal/day)
Age (years)	1.000	-0.026	-0.011	-0.014	0.071	-0.065	-0.032	-0.056	-0.011
Height (cm)		1.000	0.269*	-0.086	0.089	0.148***	0.003	0.183**	0.246*
Weight (kg)			1.000	0.900*	0.842*	0.400*	0.143***	0.388*	0.932*
BMI (kg/m^2)				1.000	0.849*	0.365*	0.180**	0.315*	0.844*
WC (cm)					1.000	0.264*	0.149***	0.213**	0.788*
SBP (mmHg)						1.000	0.605*	0.773*	0.369*
DBP (mmHg)							1.000	-0.037	0.139***
PP (mmHg)								1.000	0.353*
BMR (Kcal/day)									1.000

Significant difference at *P<0.001, **P<0.01 and ***P<0.05. Abbreviations: BMI, Body mass index; WC, Waist circumference; BP, Blood pressure; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PP, Pulse pressure; BMR, Basal metabolic rate.

Table S4. Correlation matrix for age, Anthropometric, metabolic and BP data for 548 females

	Age (years)	Height (cm)	Weight (kg)	BMI (kg/m^2)	WC (cm)	SBP (mmHg)	DBP (mmHg)	PP (mmHg)	BMR (Kcal/day)
Age (years)	1.000	-0.045	-0.075	-0.060	-0.069	-0.063	-0.083	-0.002	-0.096***
Height (cm)		1.000	0.481*	0.015	0.245*	0.115**	0.047	0.112**	0.458*
Weight (kg)			1.000	0.858*	0.770*	0.361*	0.236*	0.261*	0.982*
BMI (kg/m^2)				1.000	0.741*	0.358*	0.257*	0.234*	0.859*
WC (cm)					1.000	0.292*	0.205*	0.196*	0.763*
SBP (mmHg)						1.000	0.709*	0.663*	0.353*
DBP (mmHg)							1.000	-0.058	0.235*
PP (mmHg)								1.000	0.250*
BMR (Kcal/day)									1.000

Significant difference at *P<0.001, **P<0.01 and ***P<0.05. Abbreviations: BMI, Body mass index; WC, Waist circumference; BP, Blood pressure; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; PP, Pulse pressure; BMR, Basal metabolic rate.