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Original Article

The association between resting metabolic rate and metabolic syndrome May Be mediated by adipokines in overweight and obese women

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ABSTRACT

Aims: Adipokines play a major role in developing metabolic syndrome (MetS), and it has been found that there is a significant relationship between MetS and resting metabolic rate (RMR) in obese people. The present study aimed to investigate the mediatory effect of adipokines on the RMR-MetS relationship.
Methods: This cross-sectional study included 263 obese and overweight women, mean BMI 33.28 (4.93) kg/m, and mean age 39.02 (11.60) who were assessed for RMR using indirect calorimetry. Moreover, using the body composition analyzer the Body composition was measured. Also, Enzyme-linked Immunosorbent Assay (ELISA) test provided a quantitative measurement of biochemical parameters.
Results: The results indicated that women in low RMR group had higher fat mass ($P < 0.0001$), FFM ($P = 0.002$), weight ($P = 0.006$), BMI ($P < 0.0001$), age ($P = 0.01$), and hs-CRP ($P = 0.001$). The results did not confirm any significant mediating roles for RBP4 ($P = 0.051$, $\beta = -0.28$) and Vaspin ($P = 0.06$, $\beta = 0.32$) in the RMR-MetS relationship. Additionally, after a binary regression test, Omentin-1 showed a significant mediating role ($P = 0.25$, $\beta = 0.04$) as an interrelated agent to RMR and MetS.
Conclusion: As this study shows, Omentin-1 was found to play a significant mediating role as a mediatory agent in relationship between RMR and MetS.

1. Introduction

Metabolic syndrome (MetS) represents a cluster of cardiovascular risk factors which main underlying physio-pathological mechanism is thought to be insulin resistance [1]. It affects about one-third of the world's adult population [2]. The prevalence of MetS has increased in males from 8 to 24.2% and in females from 7 to 46.5% [3]. A number of studies have proved that the risks of developing CVD (cardiovascular disease) [3], diabetes [4], and mortality are increased by the presence of MetS [3]. In fact, Metabolic syndrome is a common syndrome that is associated with being overweight or obese, and with the central distribution of body fat [5,6].

Obesity seems to be caused mainly by a combination of a genetic predisposition and lifestyle factors, characterized by physical inactivity and an excessive intake of energy-dense, high-fat foods [6]. Also, body mass and adiposity or weight gain are related to low metabolic rates and [7] thus resting metabolic rate (RMR) can be considered as a predictive factor for the development of future body weight gains [6]. Associations have been found showing that metabolic syndrome is related to being overweight or obese (in particular central adiposity), and also significantly related to lower RMR [7]. Therefore, people predisposed to metabolic syndrome might also be susceptible to lower energy expenditure, which increases the progress of body weight gain. RMR is a major component of daily energy expenditure, accounting for 65–75% of total energy expenditure [7]. Several factors can affect RMR, including

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sex, age, weight, fat free mass (FFM), body fat and body fat distribution [8].

Adipokines are released by adipose tissue and play a paramount role in developing cardiovascular disease [9], insulin resistance [10], and metabolic disorders [11]. Previous studies reported that there is a linkbetween RMR and certain adipokines, including leptin [12], adiponectin [13], Vaspin [14], and retinol binding protein 4 (RBP4) [7]. On the other hand, in an epidemiological study, the relationship between visceral fat mass and increased metabolic risk has been explored for adipokines, which are predominantly expressed and secreted from visceral adipose tissue [14]. Several adipocytokines, such as Omentin-1, Visfatin, Nesfatin and Chemerin, have been newly discovered, and the relationship between blood concentrations and obesity related to metabolic diseases have been widely studied [14]. The present study aimed to investigate the mediatory effect of adipokines on the RMR-MetS relationship.

2. Subjects

This cross-sectional study included 263 obese and overweight women, mean BMI 33.28 (4.93) kg/m, and mean age 39.02 (11.60) years. For all participants, body weight (BW) and body height (BH) were measured to determine the BMI, expressed as BMI = BW (kg)/BH (m^2) (24). This study was carried out from October 2014 to January 2015. This study was approved by the local ethics committee of Tehran University of Medical Sciences. The participants were selected based on pre-defined inclusion criteria: 1) 25 ≤ BMI obesity and overweight; 2) absence of any acute or chronic inflammatory disease; 3) no medical history of hypertension; and 4) no alcohol or drug abuse. Any participants with a history of any condition affecting inflammatory markers were also excluded from the study, such as: known cardiovascular diseases, thyroid diseases, malignancies, current smoking, diabetes mellitus, sustained hypertension, heart failure, acute or chronic infections, and hepatic or renal diseases. All participants signed a written informed consent prior to initiation of any clinical screening procedures [15].

3. Materials and methods

Body weight was measured with a calibrated digital scale (SECA, Vogel & Halke, Hamburg, Germany) to the nearest 0.1 kg after shoes, coats, and sweaters had been removed. The measured weight was subtracted by 0.5–1.0 kg to determine the weight without any remaining. Body height was measured to the nearest 0.5 cm by a height measurement device integrated into the scale. Waist and hip circumferences were also measured to the nearest 1.0 cm using a non-elastic tape. Moreover, body composition was determined using a bioelectrical impedance method (BIA 101, RJL Systems, Data-Input, Frankfurt, Germany). Finally, FFM and FM were calculated using the equation of Deurenberg et al.

3.1. Complete body composition analysis

Body composition was assessed by the use of Body Composition Analyzer BC418MA – Tanita (United Kingdom). This equipment is designed to send out a very weak electric current to measure the impedance (electrical resistance) of the body. Therefore, in principle, subjects were barefoot when they were assessed by this device. Moreover, since impedance fluctuates in accordance with the distribution of the body fluid, we followed all of the following instructions for an accurate measurement. To prevent a possible discrepancy in the measured values, taking measurements after vigorous exercise was avoided and the researchers waited until the subject was sufficiently rested. As changes in body-water distribution and body temperature can have a major impact on measurements, they were performed in the morning in a fasting condition (always urinating before taking measurements, etc.) to get a more accurate result of the measurements every single time. The device calculates body mass index (BMI), percentage of fat mass (FM%), fat free mass and fat free mass size (FFM kg), visceral fat, and predicts muscle mass on the basis of data obtained by dual-energy x-ray absorptiometry (DXA) using bioelectrical impedance analysis (BIA). The specific device shows separate body composition mass for the right arm, the left arm, the trunk, the right leg and the left leg. Various body composition components were reported in the current study, including fat percent, fat mass, free fat mass and visceral fat [15].

3.2. Biochemical, serum cytokines concentration assay

Serum hypersensitive C-reactive protein (hs-CRP) was measured by of the use of an immunoturbidimetric assay (high-sensitivity assay, Hitachi 902). In order to measure circulating adipokines, the serum of samples was used. Serum concentrations of all adipokines were measured in triplicate, and 10 replicates per enzyme-linked immunosorbent assay (ELISA) plate were used as internal quality controls. RBP4 in serum samples was measured by competitive ELISA (Adipo-Gen, Seoul, Korea), and the inter- and intra-assay variabilities were 4.2 and 4.5%, respectively. Serum Omentin-1 was measured using an ELISA kit (Enzo Life Sciences; sensitivity 0.4 ng/ml, reference range 0.5–32 ng/ml, inter-assay variability 4.61%, intra-assay variability 5.2%). Vaspin (human) was measured by ELISA Kit (Enzo Life Sciences; sensitivity 0.01 ng/ml, inter-assay variability 5.8%) [16]. Fasting serum glucose was measured by the GOD/PAP method, and triglyceride levels were measured by the GPO–PAP method. The Enzymatic Endpoint method was used for measuring total cholesterol levels, and direct high-density lipoprotein-cholesterol was measured using enzymatic clearance assay. Randox laboratories kit (Hitachi 902) was used for all measurements [17].

3.3. RMR measurements

Resting Metabolic Rate (RMR) was obtained by means of the indirect calorimetric method using Meta Check, (Korr Medical Technologies, Salt Lake City, Utah) recommended by professional nutritionists using a standard protocol that has been described in detail previously. Using the Meta Check mouthpiece, the individual being tested breathes in normal air, then the gas the person exhales is conveyed to the Meta Check through the breathing hose. The Meta Check analyzes the volumetric flow and oxygen concentration of the exhaled gas to determine the amount of oxygen consumed by the body due to metabolism. RMR was measured by indirect calorimetry following an overnight fasting period of 10–12 h. Subjects were required to fast and remain in a resting state for 12 h prior to the test and to abstain from smoking for at least 4 h before the commencement of the procedure, although the ideal interval was 12 h, to ensure the body was in a resting and post-absorptive state. Patients were instructed to rest in a supine position on a mattress for 15 min, then they underwent the measurement for a period of 20 min. However, the first 5 min were not included, and only the last 15 min were used to calculate RMR [15].

3.4. Statistical method

Normal distribution of quantitative data was assured using Kolmogrov-Smirnov. The differences between high and low RMR were assessed by independent-sample T test and re-analyzed by ANCOVA to adjust for the effects of confounders, including age and

BMI. The binary regression model was used to find the mediatory role of adipokines on the correlation between resting metabolic rate and metabolic syndrome. The level of significance was set at a probability of ≤0.05 for all tests. Statistical analysis was performed using SPSS version 22.0 (SPSS, Chicago, IL, US).

4. Results

4.1. Study population characteristics

The mean age, height, weight and BMI of a total of 263 women who were participating in our study were: 39.02 (11.60) years, 159.99 (6.04) cm, 84.75 (13.69) kg and 33.28 (4.93) kg/m^2, respectively (Table 1).

4.2. Characteristics of study population based on RMR categorization

After RMR categorization into two groups, namely high RMR (n = 136) and low RMR (n = 127), significant differences were found between two groups according to age, BMI, weight, fat percentage, fat mass, visceral fat (P < 0.0001). Women in low RMR group had higher fat mass (P < 0.0001), FFM (P = 0.002), weight (P = 0.006), BMI (P < 0.0001), age (P = 0.01), and hs-CRP (P = 0.001). After adjustment for age and BMI, significant differences were found in terms of BMI, weight, fat percentage, fat mass, FFM and Omentin-1 between the groups. However, as shown in Table 2, the significant differences of hs-CRP and WC were lost after adjustment.

4.3. Association between MetS components related to RMR

In the binary regression model, MetS components are considered as dependent factors and RMR regarded as a covariate factor. In the crude model, an inversely significant relationship was found just for MetS (P = 0.05, β = −1.12) as well as marginally significant associations for TG and HDL (P = 0.07, β = −0.88; P = 0.08, β = −1.36; respectively). On the other hand, after using the adjusted model, the significant relationship for MetS was lost (P = 0.07, β = −1.06). However, there were no significant differences between RMR and some MetS components such as AO (abdominal obesity) = WC ≥ 90 cm (male) and 85 cm (female), HDL-C < 1.04 mmol/L, HT (hypertension) = blood pressure ≥130/85 mmHg, FBS ≥6.1 mmol/L (Table 3).

Table 1
Study population characteristics.

	Min	Max	Mean	SD
Demography				
Age (years)	17.00	69.00	39.02	11.60
Body Composition				
BMI(kg/m^2)	25.00	47.70	33.28	4.93
Height (cm)	146.00	175.00	159.99	6.04
Weight (Kg)	61.20	121.90	84.75	13.69
Fat percentage %	20.30	52:00	40.38	5.56
Fat Mass (Kg)	16.00	61.90	34.70	9.46
FFM (Kg)	38.40	84.00	50.05	6.38
Trunk Fat(Kg)	1.30	28.50	18.71	4.32
Visceral Fat(Kg)	3.00	17.00	8.83	2.94
Biochemical measurements				
FBS(mmol/L)	71.00	331.00	102.73	33.69
HDL-C (mg/dL)	24.00	97.00	47.80	12.09
TG (mmol/L)	38.00	384.00	125.51	51.77
T-chol (mmol/L)	99.00	270.00	184.58	34.40
LDL-C (mg/dL)	44.00	176:00	102.91	24.36
hs-CRP (mg/L)	0.10	39.30	3.78	5.04
RBP4 (ng/ml)	54.52	65.72	59.00	2.42
Omentin1 (μg/ml)	2.54	70.71	35.01	31.04
RMR measurements				
RMR	504.00	2550.00	1605.1	355.36
RMR/kg	12.53	30.03	19.82	3.07

BMI, Body mass index; FFM, Fat free mass; FBS, fasting blood sugar.
HDL-C, high density lipoprotein cholesterol; TG, triglyceride; Tchol, total cholesterol; LDL-C, low density lipoprotein cholesterol; hs-CRP.
High-sensitivity C-reactive Protein; RBP4, retinol binding protein4.
RMR, Resting metabolic rate.

Table 2
Characteristics of study population based on RMR categorization.

	Low RMR Mean ± SD (n = 127)	High RMR Mean ± SD (n = 136)	0.95% CI	P value	P value**
Demography					
Age (years)	39 ± 12.81	35.46 ± 9.43	−6.25 to −0.82	0.01	0.09
Body Composition					
BMI(kg/m2)	33.46 ± 5.58	30.45 ± 3.64	−4.14 to −1.87	<0.0001*	0.009*
Height (Cm)	160.47 ± 5.22	159.16 ± 5.39	−2.60 to −0.02	0.04*	0.16
Weight (Kg)	85.94 ± 15.15	77.36 ± 10.65	−11.74 to −5.41	<0.0001*	0.006*
Fat percentage %	41.57 ± 4.79	37.62 ± 4.91	−5.12 to −2.77	<0.0001*	0.02*
Fat Mass (Kg)	36.23 ± 9.79	29.44 ± 7.67	−8.91 to −4.66	<0.0001*	0.004*
FFM(Kg)	49.72 ± 6.36	47.77 ± 3.97	−3.22 to −0.67	0.002*	0.03*
Trunk fat (Kg)	18.73 ± 4.03	17.61 ± 3.76	−2.06 to −0.17	0.02*	0.22
Visceral Fat(Kg)	9.06 ± 2.89	6.95 ± 2.21	−2.73 to −1.48	<0.0001*	0.01*
Mets Components					
FBS(m mol/L)	104.42 ± 34.25	100.04 ± 25.91	−11.79 to 2.96	0.24	0.98
HDL (m mol/L)	45.51 ± 10.31	47.2 ± 12.42	−1.09 to 4.47	0.23	0.31
TG (m mol/L)	129.15 ± 42.78	119.58 ± 47.03	−20.51 to 1.37	0.08	0.29
SBP (mmHg)	111.33 ± 13.35	111.08 ± 18.81	−4.23 to 3.73	0.90	0.73
DBP(mmHg)	79.66 ± 12.58	76.76 ± 13.44	−6.06 to 0.26	0.07	0.76
WC (Cm)	99.61 ± 9.41	96.34 ± 7.96	−5.38 to −1.15	0.002*	0.54
Adipokines & Cytokine					
RBP-4 (ng/ml)	59.15 ± 2.31	58.77 ± 2.48	−0.96 to 0.20	0.20	0.13
Omentin-1 (μg/ml)	39.71 ± 6.21	38.15 ± 9.23	−3.48 to 0.36	0.11	0.06
hs-CRP (mg/l)	5.11 ± 6.92	2.91 ± 3.59	−3.52 to −0.87	0.001	0.37

* **Collinear** variable not inter to the ANCOVA analysis, BMI, Body mass index; FFM, Fat free mass.
FBS, fasting blood sugar; HDL-C, high density lipoprotein cholesterol; TG, triglyceride; T-chol, total. cholesterol; LDL-C, low density lipoprotein cholesterol; hs-CRP, High-sensitivity C-reactive Protein.
RBP4, retinol binding protein4; RMR, Resting metabolic rate; **Adjustment for age and BMI, *P < 0.05.

Table 3
Association of RMR and MetS and its component.

	Crude model			Model 1		
	β±SE	0.95% CI	*P*-value*	β±SE	0.95% CI	**P-value
MetS	−1.12 ± 0.59	0.1−1.04	0.05*	−1.06 ± 0.6	0.1−1.12	0.07
AO	−0.08 ± 0.99	0.13−6.37	0.93	0.02 ± 1.01	0.14−7.43	0.97
TG	−0.88 ± 0.49	0.15−1.09	0.07	−0.86 ± 0.5	0.15−1.12	0.08
HDL	1.36 ± 0.78	0.05−1.18	0.08	1.41 ± 0.79	0.05−1.15	0.07
HT	0.24 ± 0.86	0.23−6.89	0.77	1.09 ± 1.1	0.34−26	0.32
FBS	024 ± 0.86	0.23 -o6.89	0.77	0.44 ± 0.90	0.26−9.11	0.62

HDL-C, high density lipoprotein cholesterol; FBS, Fasting blood sugar; TG, triglyceride; HT, hypertension; Mets, Metabolic syndrome; AO; abdominal obesity.
P < 0.05. P–value from Binary Regression Model; Mets as dependent variable and RMR as covariate P–value after adjustment for age **

Table 4
Mediatory role of Adipokines on RMR & MetS relationship.

RBP4				Omentin-1			Vaspin		
	SE±β	0.95% CI	*P*-value*	SE±β	0.95% CI	*P*-value*	SE±β	0.95% CI	*P*-value*
Mets	0.14±-0.28	0.56 to 1.00	**0.051**	0.04 ± 0.03	0.96 to1.12	0.25	0.32 ± 0.17	0.97 to1.95	**0.06**

P-value from Binary Regression Model Mets as dependent variable and RMR as covariate at the first step, the next step for testing mediatory role of adipokines we enter the adipokines levels as covariate.
RMR, Resting metabolic rate; Mets, Metabolic syndrome; RBP4, Retinol binding protein4.

4.4. Modulatory functions of adipokines on the RMR-MetS relationship

The binary regression model was used to evaluate the mediatory role of adipokines on the correlation between resting metabolic rate and metabolic syndrome. The results did not indicate any significant mediating role of RBP4 ($P = 0.051$, $\beta = -0.28$) and Vaspin ($P = 0.06$, $\beta = 0.32$) in relation to RMR and MetS. However, a significant mediating role of Omentin-1 ($P = 0.25$, $\beta = 0.04$) was found as a interrelated agent to RMR and MetS (Table 4). By adding the adipokines levels as new covariates in the regression model, the significant association between MetS and RMR disappeared. Therefore, the mediatory role of adpokines on the measured association was indicated. Based on these results, shown in Table 4, the significant association between RMR and MetS disappeared with all three adipokines. However, the strongest association was observed for Omentin-1.

5. Discussion

This study set out to determine the mediatory effect of adipokines, RMR, and MetS in patients with overweight and obesity. The major finding of this study was that there is a association between some of adipokines such as Omentin-1, Vaspin and RBP4 as mediating factors in the RMR-MetS relationship among obese people. These results confirmed that lower levels of RMR were strongly associated with higher weight, body mass index (BMI), fat percentage, fat mass, visceral fat, and lower fat free mass (FFM). As reported by other authors, a low RMR for a given body size has been considered as a risk factor for weight gain [18], and this condition is associated with a propensity toward excess adiposity and resistance to weight loss [19]. The results of one study indicated that both greater amounts of lean mass (LM) and fat mass (FM) had the strongest relationships with RMR in both men and women [20]. Although, the relationship between greater FFM and obesity is marked, the detection of RMR impairment is difficult [21].

In this study, only MetS, triglyceride (TG), and high-density lipoprotein cholesterol (HDL) showed marginal links with RMR, in both the adjusted and unadjusted groups. Although no statistically significant differences were found between the two groups at the levels of MetS component fasting blood sugar (FBS), HDL, and TG (after adjustment), individuals with low RMR displayed greater HDL levels than expected whichcontrasts with studies reporting obese individuals with glucose intolerance have a higher RMR compared to subjects with glucose tolerance [22]. Obesity is associated with elevated oxidative stress and a proliferated product of pro-inflammatory cytokines [23]. Additionally, chronic inflammation induces insulin dysfunction [24], and MetS components could be increased by it.

Waist circumference (WC), as a main determinant of MetS, showed an inverse association with RMR. This is in agreement with studies which have investigated the high frequency of obesity in women with Polycystic Ovarian Syndrome (PCOS), related to lower energy expenditure, RMR, and/or thermic response to standard meals in these women [20]. The current study's results did not confirm a correlation between RMR and blood pressure, although in other studies, people with high blood pressure (BP) had a higher RMR than those with normal BP.

There are several studies which have indicated adipokine signals correlating with energy balance and metabolic syndrome, and have suggested possible links between RMR and serum RBP4, leptin, adiponectin, Nesfatin-1 and Vaspin. This study approved the correlation of Omentin-1 and RMR. It seems that Omentin-1, by inhibition of tumor necrosis factor (TNF-α), can decrease leptin levels and the effect on energy expenditure and RMR [25]. The current results could not confirm that RBP4 has a correlation with RMR. Retinol-binding protein 4 is one of the adipokines involved in the development of insulin resistance. One finding showed reduced RMR/kg in higher RBP4 concentrations, therefore, a negative relationship was found between RBP4 and RMR/kg in the obese group [7].

This study's results confirmed that Vaspin is marginally linked to MetS. Other studies also showed that the serum Vaspin concentration was negatively correlated with age, waist and hip circumference (HC), systolic and diastolic blood pressure, and duration of diabetes. Tan et al. found that women with Polycystic Ovarian Syndrome had higher serum and adipose tissue Vaspin levels, and detected significant positive associations between circulating Vaspin and Vaspin levels in adipose tissue with BMI and Waist-Hip Ratios. Jian W. et al. showed that serum Vaspin concentration was significantly correlated with BMI, Waist-Hip Ratio (WHR) and the homeostasis model assessment of insulin resistance in T2DM patients [26].

Additionally, Vaspin had a mediatory effect between visceral fat and fat mass, which is associated with RMR in obese participants

[27]. The association between RBP4 and MetS was confirmed in this study. Other population studies have demonstrated an association between serum RBP4 and the severity of atherosclerosis, and the risk of cardiovascular events and type 2 diabetes [28].

The current study confirmed Omentin-1's stronger mediating role than Vaspin's and RBP4's, in relation to RMR and MetS. Previous studies have shown a significant relationship between RMR criteria and circulation Omentin-1 levels [29]. On the other hand, Shibata N. et al. found a negative association between circulating Omentin-1 concentration and with metabolic syndrome factors [30]. The main limitation of the present study was the relatively small number of participants and the lack of men participants. To the best of the researchers' knowledge, the strength of this study is that it is the first study evaluating the association between metabolic syndrome (MetS) and resting metabolic rate (RMR) through the mediation of adipokines. As mentioned above, it seems that, although this study cannot absolutely confirm the relationships between certain adipokines and RMR or MetS, it does demonstrate that adipokines can have a mediatory effect on RMR and MetS. Other studies, therefore, are needed with much larger samples and wider age ranges.

Conflicts of interest

No potential conflicts of interests relevant to this article were reported.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.dsx.2018.10.012.

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