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SHORT REPORT

A low resting metabolic rate is associated with metabolic syndrome

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KEYWORDS
Obesity;
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Summary
Background & aims: The metabolic syndrome is associated with central accumulation of fat. Previous studies showed that some obese subjects are characterized by a sparing energy metabolism. The aim of this study was to investigate whether obese subjects with metabolic syndrome have a lower resting metabolic rate than obese subjects without metabolic syndrome.
Methods: Forty obese subjects were divided into three groups according to the presence of metabolic syndrome and type 2 diabetes; 15 non-obese healthy control subjects were also enrolled. Body composition (bio-impedance analysis) and resting metabolic rate (indirect calorimetry) were performed.
Results: The group with metabolic syndrome exhibited a significantly lower resting metabolic rate adjusted for fat-free mass with respect to the control group and the obese group without metabolic syndrome (respectively: 108 ± 3 vs. 118 ± 3, $p < 0.01$ and 123 ± 3 kJ/kg fat-free mass 24 h, $p < 0.01$; mean $\pm$ sem). The obese group with metabolic syndrome and type 2 diabetes (T2D) had a not different adjusted resting metabolic rate (114 ± 6 kJ/kg fat-free mass 24 h) with respect to other groups.
Conclusions: An energy sparing condition seems to characterize non-diabetic obese subjects with metabolic syndrome.

Abbreviations: BIA, bioimpedance analysis; BMI, body mass index; FFM, fat-free mass; FM, fat mass; HOMA-IR, homeostasis model assessment of insulin resistance; MS, metabolic syndrome; RMR, resting metabolic rate; RQ, respiratory quotient; T2D, type 2 diabetes

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Introduction

The metabolic syndrome is invariably associated with overweight or frank obesity with central distribution of body fat. Evidences have been accumulated that a sparing energy metabolism, probably due to genetic factors, characterizes some obese subjects.[1] Resting metabolic rate (RMR) is the main component of daily energy expenditure, accounting for about 70% of total daily energy expenditure in most adult sedentary individuals and it is a predictive factor for the development of future body weight gain.[2,3] A recent study showed also that RMR variability was associated with some genetic traits that overlapped regions previously linked to the metabolic syndrome; furthermore, a significant association between RMR and metabolic syndrome was observed.[4] Therefore, subjects predisposed to the metabolic syndrome might also be characterized by a lower energy expenditure that facilitate body weight gain. In the present study we investigated the RMR in different groups of obese subjects distinguished on the basis of the presence or less of the metabolic syndrome with or without diabetes.

Materials and methods

Forty obese subjects (24 males, 16 females; range of age: 30–60 years; range of BMI: 30–39.9 kg/m^2) were recruited among the obesity outpatients seen at the Department of Internal Medicine and Cardiovascular and Kidney Diseases of the University of Palermo. They were divided into three groups according to the presence (MS+) or less (MS−) of the metabolic syndrome, with or without type 2 diabetes (T2D). A group of 15 (9 males and 6 females) normal weight subjects was also recruited as control. Both diabetes and hypertension (when these conditions occurred) were diagnosed for the first time when subjects were enrolled in the study. All participants in the study had their body weight stable for at least the last 3 months and nobody habitually assumed any drug. Before starting the study, body weight was registered in two occasions with a 7–10 days interval, subjects whose body weight changed ± 0.5 kg were excluded from the study. Metabolic syndrome was defined according to the diagnostic criteria of the NCEP-ATPIII, T2D according to the criteria of the American Diabetes Association. The physical and clinical characteristics of the studied groups are reported in Table 1. The study protocol was approved by the ethical committee of our institute; before taking part in the study all subjects were informed of its aims and methods and gave their voluntary written consent. Each patient was tested in the morning after about 10 h of overnight fasting. A blood venous sample was obtained to determine chemical and C-peptide (radioimmunoassay) data; an oral glucose tolerance test was performed in all subjects and blood concentrations of glucose and insulin (radioimmunoassay) were measured before and 2 h after the assumption of a 75 g glucose load. The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated according to Matthews et al.[5] Fat mass (FM, % body weight) and fat-free mass (FFM, kg) were estimated as previously described[6] by means of bioelectrical impedance analysis (BIA; BIA-103, RJL, Detroit, USA/Akern Florence, Italy). Body circumferences were measured at the umbilicus (waist circumference) and at the most prominent buttock level (hip circumference). The RMR and the fasting respiratory quotient (RQ; VCO_2/VO_2: an indirect measurement of the mixture of carbohydrate and lipid oxidation) were obtained by means of the indirect calorimetry method. A ventilated hood system of indirect calorimetry was employed (2900 MMC, Sensormedics; Yorba Linda, CA, USA/Bilthoven, The Netherlands). Respiratory gas exchanges were continuously measured for about 1 h, data were obtained from at least 30 min of stable measurements. The RMR was expressed both in absolute value (kJ/24 h) and adjusted for FFM size (kJ/kg-FFM 24 h). Alternatively, due to the positive intercept in the relationship between RMR and FFM, a regression-based approach was considered in order to adjust RMR for FFM size.[7]

All data are expressed as mean $\pm$ sem. The statistical comparisons between the groups were calculated by means

Table 1 Main characteristics of control subjects and obese groups defined according to the presence (+) or less (−) of metabolic syndrome (MS) and type 2 diabetes (T2D) (mean $\pm$ sem).

	Control	MS−	MS+	MS+/T2D
M/f	9/6	9/6	8/5	7/5
Age (years)	39 ± 2	42 ± 3	42 ± 4	46 ± 3^{b}
Body weight (kg)	64.8 ± 2.2	87.4 ± 3.2^{c}	$100.3 \pm 4.3^{c,d}$	99.5 ± 6.0^{c}
BMI (kg/m^2)	23.5 ± 0.5	31.4 ± 0.7^{c}	$35.0 \pm 0.9^{c,d}$	$35.3 \pm 1.9^{c,d}$
Fat mass (%)	23.6 ± 1.8	33.9 ± 2.5^{c}	33.2 ± 1.9^{b}	33.1 ± 2.3^{b}
Waist circumference (cm)	85 ± 2	94 ± 4^{a}	$116 \pm 2^{c,d}$	$119 \pm 4^{c,d}$
Systolic blood pressure (mmHg)	105 ± 2	122 ± 8	139 ± 10^{a}	151 ± 7^{a}
Diastolic blood pressure (mmHg)	72 ± 1	81 ± 5	86 ± 4^{a}	91 ± 5^{c}
Blood concentration of:				
Total cholesterol (mmol/l)	3.76 ± 0.23	4.38 ± 0.21^{a}	5.31 ± 0.52^{a}	$5.39 \pm 0.36^{b,d}$
HDL cholesterol (mmol/l)	1.24 ± 0.21	1.17 ± 0.10	$0.82 \pm 0.08^{a,d}$	$0.96 \pm 0.16^{a,d}$
Triglycerides (mmol/l)	0.96 ± 0.14	0.99 ± 0.11	$1.77 \pm 0.42^{a,d}$	$2.43 \pm 0.50^{b,e,f}$

Significance level vs. control group: $^{a}p<0.05$; $^{b}p<0.01$; $^{c}p<0.001$.
Significance level vs. MS− group: $^{d}p<0.05$; $^{e}p<0.01$.
Significance level vs. MS+ group: $^{f}p<0.05$.