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Significant features of basal and maximal energy expenditure parameters in obese adults

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Aim: To compare body composition, resting metabolic rate, and maximal aerobic capacity parameters in obese adults and healthy controls, as well as to investigate the associations among these parameters.

Materials and methods: A total of 120 participants, 60 obese participants (30 male, 30 female) with body mass indexes (BMIs) over 30 kg/m^2 and 60 healthy controls (30 male, 30 female) with BMIs of 18–25 kg/m^2, were included in the study. BMI was calculated as body weight divided by the square of the height (kg/m^2). Body fat percentage, total body fat, and lean body mass were established with bioelectric impedance analysis. Resting metabolic rates (RMRs) were determined with indirect calorimeter. Maximal aerobic capacity was estimated with the Astrand exercise protocol.

Results: RMRs (kcal/day) were significantly higher in male and female obese individuals than in the controls. VO_2max (L/min) levels were not significantly different between obese and control individuals of either sex. However, RMR per kilogram of body weight and VO_2max expressed in milliliters per kilogram of body weight were significantly lower in male and female obese adults compared to the controls. BMI, body fat percentage, and total body fat were positively correlated with RMR (kcal/day), but negatively correlated with RMR [(kcal/day)/kg] and VO_2max (mL/kg/min) in both sexes.

Conclusion: We suggest that resting and maximal energy expenditure per kilogram of body weight are impaired in obese adults. Both low resting metabolic rate and weak maximal aerobic capacity are related to excess body fat in obese subjects.

Key words: Obesity, resting metabolic rate, maximal aerobic capacity, exercise, body fat, anthropometry

1. Introduction

Obesity has emerged as one of the most serious public health concerns of the industrial age, and associated morbidity and mortality have been increasing. Obesity has been considered a disease by the World Health Organization since 1997. The global increase in obesity has raised interest in the complex causes of excessive weight gain. Any long-term changes in body weight are caused by a persistent energy imbalance. Simply, obesity can be seen as the consequence of a sustained increase in energy intake relative to energy expenditure. It is characterized by the accumulation of excess body fat and can be conceptualized as the physical manifestation of chronic energy excess (1).

While the rising obesity epidemic has greatly increased interest in the problem on the part of health professionals, the causes behind the epidemic are still poorly understood. Although evidence suggests that genetics plays an important role in body weight regulation, the rapid increase in obesity rates does not seem to be caused by significant genetic changes within populations (2). In today's world the obesogenic environment is conducive to the consumption of energy and unfavorable to the expenditure of energy. Modern computer-dependent, sleep-deprived, physically inactive humans live chronically stressed lives in a society of food abundance (1).

Energy expenditure consists of 3 major components: basal metabolic rate, the thermic effect of food, and physical activity (3). In sedentary humans the resting metabolic rate (RMR) is 80% of total energy expenditure, and energy expenditure due to physical activity is less than RMR; RMR is the main driver of the relation between weight and total energy expenditure. Increased physical activity increases total energy expenditure and is associated with increased muscle mass and decreased adiposity. It is essential in weight loss management to understand and evaluate the

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entire aspect of obesity, including energy expenditure and body composition parameters.

The aims of this study were to compare body composition, resting metabolic rate, and maximal aerobic capacity parameters between obese adults and normal-weight controls, as well as to investigate the associations among these parameters.

2. Materials and methods

2.1. Study design

This study was designed as a comparative association analysis between obese adults and normal-weight controls regarding RMR and maximal aerobic capacity parameters. Because of their biological differences, the analysis of the data was planned separately in men and women. The obese individuals and the controls were pre-selected from voluntary individuals who came to our exercise physiology laboratory to learn their body mass indexes (BMIs), body fat percentages, and physical fitness status. Inclusion and exclusion criteria were determined, and suitable subjects were enrolled in the study.

2.2. Subjects

The study protocol was approved by the local ethical committee of clinical research, and all patients and controls participated voluntarily with written informed consent. Inclusion criteria included not actively participating in sports and having no risk associated with exercise. Exclusion criteria included major musculoskeletal problems; use of drugs affecting resting metabolic rate; acute infection; dehydration; cardiovascular, respiratory, and metabolic disorders; and other systemic diseases.

A total of 120 participants, 60 obese adults (30 male, 30 female) with BMIs over 30 kg/m^2 and 60 controls (30 male, 30 female) with BMIs of 18–25 kg/m^2 were included in the study.

2.3. Anthropometric parameters

Height was measured using an inflexible steel meter stick while subjects stood with heels, back, and shoulders against a wall with feet together and head on the Frankfort plane (4). The body weight measurements were taken, after subjects removed their outerwear and shoes, with calibrated measurement devices (5). BMI was calculated as body weight divided by the square of the height (kg/m^2).

2.4. Body composition

Body composition parameters (body fat %, total body fat, and lean body mass) were determined by a bioelectrical impedance analysis (BIA) system (Bodystat 1500, Bodystat Ltd., Douglas, Isle of Man, UK). The basic premise of BIA procedure is that the volume of fat-free tissue in the body will be proportional to the electrical conductivity of the body (6). Cautions were given before taking the measurements as follows (6–9): subjects were instructed to avoid eating or drinking within 4 h, using diuretics within 7 days, exercising strenuously within 24 h, and consuming alcohol within 48 h before the test procedure. Moreover, the subjects urinated completely within 30 min before the test and used limited diuretic agents (caffeine, chocolate, etc.). Metal objects were removed from the body, and measurements were performed at room temperature. Exclusion criteria were conditions that change the body fluid–electrolyte balance such as dehydration and menstruation. Impedance was measured between the right wrist and right ankle using a tetrapolar electrode system. The subjects lay supine with arms separated from the body, and with legs not touching each other. Signal electrodes were positioned in the middle of the dorsal surfaces of the hands and feet proximal to the metacarpophalangeal and metatarsophalangeal joints. Detecting electrodes were more proximally positioned at the ankle and the wrist. An excitation current of 500 μA at 50 kHz was applied to the distal electrodes, and the voltage was detected by proximal electrodes. The data were analyzed using the manufacturer's software, and body fat percentage, total body fat, and lean body mass were determined for each subject.

2.5. Resting metabolic rate

RMR was measured using an indirect calorimeter (Quark b^2, Cosmed, Rome, Italy) with a computerized metabolic card, which analyzed oxygen consumption and carbon dioxide production (10). The device was calibrated prior to each test. The subjects were instructed to avoid food intake for 12 h and not to perform exercise for 24 h before the test. The tests were performed during the same hours of the day (0830–1030). After resting for 15 min, the measurements were obtained from the subjects in a silent, lightless laboratory at room temperature. The subjects were asked to put on a face mask, lay in a supine position, and not to move their arms or legs during the test.

2.6. Maximal aerobic capacity

Before the exercise test, the risk of exercise for the subjects was assessed according to the American College of Sports Medicine criteria (11). The exercise test was performed with appropriate equipment and physician supervision (6). The maximum volume of oxygen consumed to produce energy (maximal aerobic capacity or VO_2max) was estimated by the Astrand test protocol, a valid submaximal exercise test for estimating VO_2max (12). The subjects were taken into the laboratory in appropriate clothes.

The subjects were instructed to avoid food intake 2 h before the test and not consume beverages or foods containing caffeine or alcohol (13). The Astrand test was performed on a computerized cycle ergometer (Monark 839E, Monark Exercise AB, Sweden); optimal seat height was adjusted for each subject, and heart rate was monitored with a chest belt telemetry system (Polar, Monark Exercise

AB, Sweden). Initial workload was selected according to sex and conditioning level (11). The subjects were asked to perform a 6-min submaximal exercise test by maintaining a pedaling cadence of 50 revolutions/min and reaching a steady state heart rate (within 6 beats/min) at the 5th and 6th min of the test. The VO_2max was estimated from heart rate and workload by Astrand test using a nomogram and age-correcting factor (6). Maximal aerobic capacity (aerobic exercise capacity or cardiorespiratory fitness) was expressed in liters per minute VO_2max (L/min) and milliliters per kilogram of body weight VO_2max (mL/kg/min).

2.7. Statistical analysis

The data were analyzed using SPSS 16.0 (SPSS, Chicago, IL, USA). Distribution of the group was analyzed with the Kolmogorov–Smirnov test. Differences between groups were determined by t-test. The correlations between the parameters were analyzed with Pearson correlation tests. All parametric results were expressed as mean ± standard deviation for each group. The significance level was determined as $P \leq 0.05$.

3. Results

A total of 63 obese individuals were invited to participate in the study. Three refused to participate because they did not want to perform the exercise test.

The mean values for age, height, body weight, BMI, body fat percentage, total body fat, and lean body mass in the male and female groups are shown in Tables 1 and 2. We found that body weight, BMI, body fat percentage, total body fat, and lean body mass values were significantly higher in male and female obese adults than in the controls.

Tables 3 and 4 show the mean values for RMR and VO_2max parameters in the male and female subjects. RMRs (kcal/day) were significantly higher in male and female obese individuals than in the controls. The mean VO_2max (L/min) values did not differ significantly between the obese and control groups in either sex. However, RMR [(kcal/day)/kg] per kilogram of body weight and VO_2max (mL/kg/min) expressed in milliliters per kilogram of body weight were significantly lower in both male and female obese adults compared to the controls.

Table 1. Mean values of age, height, body weight, BMI, and body fat in male obese and control groups.

	Obese (n = 30)	Control (n = 30)	P value
Age (year)	40.3 ± 10.4	38.2 ± 8.4	0.393
Height (cm)	170.9 ± 5.8	172.4 ± 6.2	0.338
Body weight (kg)	100.8 ± 13.9	68.9 ± 6.9	<0.001
BMI (kg/m^2)	34.4 ± 3.7	23.2 ± 2.3	<0.001
Body fat (%)	31.2 ± 4.2	16.6 ± 4.1	<0.001
Total body fat (kg)	31.9 ± 8.2	11.4 ± 3.0	<0.001
Lean body mass (kg)	68.9 ± 6.6	57.5 ± 6.3	<0.001

BMI: body mass index.

Table 2. Mean values of age, height, body weight, BMI, and body fat in female obese and control groups.

	Obese (n = 30)	Control (n = 30)	P value
Age (year)	36.1 ± 8.0	34.5 ± 8.9	0.467
Height (cm)	157.4 ± 6.3	159.7 ± 4.4	0.110
Body weight (kg)	89.6 ± 15.9	57.0 ± 8.3	<0.001
BMI (kg/m^2)	36.2 ± 6.4	22.4 ± 2.5	<0.001
Body fat (%)	45.2 ± 6.1	29.6 ± 5.4	<0.001
Total body fat (kg)	41.0 ± 12.2	17.2 ± 5.1	<0.001
Lean body mass (kg)	48.6 ± 6.2	40.1 ± 4.0	<0.001

BMI: body mass index.

Table 3. Mean values of resting metabolic rate and maximal aerobic capacity parameters in male obese and control groups.

	Obese (n = 30)	Control (n = 30)	P value
RMR (kcal/day)	1920.6 ± 468.3	1586.8 ± 252.2	0.001
RMR [(kcal/day)/kg]	19.2 ± 4.7	23.1 ± 3.6	0.001
VO_2max (L/min)	2.44 ± 0.44	2.46 ± 0.63	0.880
VO_2max (mL/kg/min)	24.9 ± 5.2	35.7 ± 9.8	<0.001

RMR: resting metabolic rate, VO_2max: maximal aerobic capacity.

Table 4. Mean values of resting metabolic rate and maximal aerobic capacity parameters in female obese and control groups.

	Obese (n = 30)	Control (n = 30)	P value
RMR (kcal/day)	1512.6 ± 434.2	1289.4 ± 412.3	0.046
RMR [(kcal/day)/kg]	17.0 ± 4.2	22.8 ± 7.1	<0.001
VO_2max (L/min)	1.99 ± 0.54	1.85 ± 0.26	0.220
VO_2max (mL/kg/min)	22.8 ± 6.4	32.4 ± 5.1	<0.001

RMR: resting metabolic rate, VO_2max: maximal aerobic capacity.

Tables 5 and 6 list the significant correlations between body fatness (BMI, body fat) and energy expenditure parameters (RMR, VO_2max) in both the male and female obese groups. BMI, body fat percentage, and total body fat were positively correlated with RMR (kcal/day) but negatively correlated with RMR [(kcal/day)/kg] and VO_2max (mL/kg/min) in both sexes.

4. Discussion

Obesity is a chronic metabolic disease associated with atherosclerotic and cardiovascular changes (14). It is common in the population, and the etiology of obesity is complex. Although approximately 50% of adults in many countries are overweight and obese, it is difficult to reduce excessive weight once it becomes established (15). In the long term, scientific studies that investigate different aspects of obesity might provide some useful findings for achieving weight loss. In this study we found that RMR and maximal aerobic capacity were lower in obese individuals than in normal-weight controls, which is in accordance with previous research. In the current study, resting and maximal energy expenditure parameters were investigated together in obese adults and in the controls.

Forman et al. (16) compared the RMR of African-American (n = 25) and Caucasian (n = 22) premenopausal obese women and found that predicted RMR for the

Table 5. Correlations of BMI and body fat parameters in male obese group.

	RMR (kcal/day)		RMR [(kcal/day)/kg]		VO_2max (mL/kg/min)	
	r value	P value	r value	P value	r value	P value
BMI (kg/m^2)	0.464	0.000	–0.491	0.000	–0.618	<0.001
Body fat (%)	0.475	0.000	–0.383	0.003	–0.637	<0.001
Total body fat (kg)	0.515	0.000	–0.426	0.001	–0.615	<0.001

BMI: body mass index, RMR: resting metabolic rate, VO_2max: maximal aerobic capacity.

Table 6. Correlations of BMI and body fat parameters in female obese group.

	RMR (kcal/day)		RMR [(kcal/day)/kg]		VO_2max (mL/kg/min)	
	r value	P value	r value	P value	r value	P value
BMI (kg/m^2)	0.372	0.003	–0.465	0.000	–0.754	<0.001
Body fat (%)	0.284	0.028	–0.498	0.000	–0.760	<0.001
Total body fat (kg)	0.405	0.001	–0.448	0.000	–0.774	<0.001

BMI: body mass index, RMR: resting metabolic rate, VO_2max: maximal aerobic capacity.

African-Americans was the same as the measured RMR, whereas Caucasian women expended about 13% more energy than predicted (16). They suggested that the lower prevalence of obesity in Caucasian women might be due in part to a higher RMR. Ruige et al. (17) studied 457 overweight and obese white patients and found that RMR is the most important predictor of adiponectin, followed successively by insulin resistance, fat mass, age, visceral fat, and fasting triacylglycerol. They speculated that subjects with low RMR, who are theoretically at greater risk of obesity-related disorders, are especially protected by adiponectin (17). Jia et al. (7) studied 109 Chinese adults (52 men and 57 women) and found that resting energy expenditure per kilogram was significantly lower in overweight/obese subjects than in normal-weight subjects. In another study, 124 formerly obese women were compared with 121 women who had never been obese (18), and it was found that the formerly obese subjects had a 3%–5% lower mean relative RMR than the control subjects. The researchers suggested that the difference could be explained by a low RMR being more frequent among the formerly obese subjects than among the control subjects. Whether the cause of low RMR is genetic or acquired, its existence is likely to contribute to the high rate of weight regain in formerly obese persons. Bosy-Westphal et al. (19) studied 45 overweight and obese women who were following a low-calorie diet. They found a decrease of almost 50% in resting energy expenditure after weight loss, which they explained by losses in fat-free mass and fat mass. Similar to the above studies, we found that RMR per kilogram of body weight was significantly lower in both male and female obese adults than in the controls (Tables 3 and 4). However, RMR (kcal/day) was significantly higher in both male and female obese individuals than in the controls (Tables 3 and,4). Due to excessive body fat and much lean body mass, RMRs (kcal/day) were significantly higher in the obese adults compared to the controls. In addition, BMI, body fat percentage, and total body fat were positively correlated with RMR (kcal/day) but negatively correlated with RMR per kilogram of body weight in both male and female groups (Tables 5 and 6). These correlations were mostly at a moderate level. Hallgren et al. (20) found that oxygen consumption expressed per gram of tissue was lower in obese subjects than in tissue from lean subjects. They stated that fat mass has an inconsequential metabolic rate, as supported by evidence that adipose tissue has a low mass-specific rate of energy utilization. It is conceivable that RMR is lower in obese subjects than in same-BMI lean subjects.

DeLany et al. (21) studied 114 African-American and Caucasian girls and boys, and they found that total daily energy expenditure and RMR were significantly higher in obese children, as a result of their greater fat-free mass and body fat, than in lean children. However, activity-related energy expenditure did not differ significantly between the obese and lean children. Average total daily energy expenditure did not change over 2 years, but RMR increased significantly, and activity-related energy expenditure decreased significantly (21). They claimed that a decrease in physical activity over 2 years might have contributed to the risk of obesity. Faintuch et al. (22) found that aerobic capacity was more markedly diminished in seriously obese bariatric candidates. In another study (23), VO_2max (mL/kg/min) was significantly lower in male and female obese adults compared to normal-weight controls, but VO_2max (L/min) was not. In addition, VO_2max (mL/kg/min) was negatively correlated with BMI and body fat percentage (23). Watanabe et al. (24) studied junior high school children and found significant correlations between VO_2max (mL/kg/min) and body fat percentage in both boys (r = –0.742) and girls (r = –0.843). They suggest that obesity accentuates exercise intolerance and low aerobic capacity. Ostojic et al. (25) found a negative correlation between body fat and VO_2max in elementary school children (754 boys and 367 girls). Similar to the above studies, we found that VO_2max (mL/kg/min) levels were significantly lower in both male and female obese adults compared to controls, but VO_2max (L/min) levels were not (Tables 3 and 4). Due to excess BMI in the obese group, VO_2max (L/min) was not significantly different between the obese and control subjects of either sex. In addition, BMI, body fat percentage, and total body fat were

negatively correlated at a strong level with VO_2max (mL/kg/min) in both male and female groups (Tables 5 and 6). Low maximal aerobic capacity indicates physical inactivity (23). The population statistics of most countries in the world indicate that industrialization and computerization are associated with an increase in sedentariness and, more recently, with a significant shift from healthy weight to overweight (26). Fat gain is the result of chronic exposure to an obesogenic lifestyle and should be seen as an adaptation that ultimately facilitates the body's energy storage in order to re-establish a new homeostatic state (1). An active lifestyle can affect energy balance and body fat in obese individuals, and physical activity has been traditionally considered as a strategy to burn calories (26). However, to reach this outcome, aerobic exercise should be performed regularly.

Mitochondria play central roles in ATP production and energy expenditure. Nutrient excess leads to mitochondrial dysfunction, which in turn leads to obesity-related pathologies, in part due to the harmful effects of reactive oxygen species (27). Ritov et al. (28) found that mitochondrial enzymatic oxidative capacity (total activity of NADH oxidase) was reduced significantly in obese and type 2 diabetic individuals. We think that due to mitochondrial dysfunction, energy expenditures are low in both resting and maximal physical activity conditions in obese adults compared with normal-weight controls.

In conclusion, we suggest that resting and maximal energy expenditure per kilogram of body weight are impaired in obese adults. Both low resting metabolic rate and weak maximal aerobic capacity are related to excess body fat in obese individuals.

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