



PAPER

Low physical performance in obese adolescent boys with metabolic syndrome

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OBJECTIVE: To assess cardiorespiratory exercise function in obese children with and without metabolic syndrome (MS).

DESIGN: Comparing three groups of subjects with different cardiovascular risk profiles.

SUBJECTS: Twenty-two MS (body weight (mean ± s.d.) 97.3 ± 15.3 kg; age (mean ± s.d.) 14.2 ± 1.9 y), 17 obese (82.6 ± 15.7 kg; 14.2 ± 2.6 y) and 29 normal weight control (64.3 ± 8.5 kg; 15.3 ± 1.0 y) boys.

MEASUREMENTS: Exercise duration (ED), resting heart rate (HR₀), peak heart rate (HR_{peak}), physical working capacity at 170 beat/min (PWC-170), peak oxygen consumption (VO_{2peak}) and the lactic acidosis threshold (LAT) were determined on treadmill, using a continuous ramp protocol.

RESULTS: ED (MS (mean ± s.d.); 655 ± 86 s; obese 703 ± 64 s; control 750 ± 0 s) in absolute value and PWC-170 normalised for body weight (139 ± 40 w; 177 ± 40 w; 211 ± 40 w) were significantly shorter and lower in the MS group, as compared to obese and control groups ($P < 0.05$). VO_{2peak} (2.2 ± 0.4 l/min; 2.4 ± 0.5 l/min; 2.9 ± 0.4 l/min) and LAT (1.3 ± 0.4 l/min; 1.5 ± 0.4 l/min; 1.8 ± 0.4 l/min) normalised for body weight, were significantly shorter and lower in the MS group, as compared to control group ($P < 0.05$). HR₀ was significantly higher ($P < 0.05$) in MS group than in obese and control groups (88 ± 12 bpm; obese 78 ± 10 bpm; 73 ± 10 bpm).

CONCLUSION: Cardiorespiratory exercise performance capacity in MS boys are reduced. It still remains to be elucidated whether the metabolic alterations or the decreased physical activity is responsible for the observed reduction in cardiorespiratory performance.

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Keywords: children; cardiorespiratory exercise capacity; metabolic syndrome

Introduction

The high incidence and aggregation of cardiovascular risk factors, including hyperinsulinaemia, systemic hypertension, elevated serum cholesterol and triglyceride levels, as well as reduced high-density lipoprotein cholesterol level and glucose intolerance, have been well demonstrated in obese individuals.^{1–4} The clustering of these risk factors, called metabolic syndrome (MS), have also been shown in both children and adults.^{3,5} The cardiovascular risk factors tend to track into adulthood when they are left untreated. It is well known that sedentary lifestyle, obesity, decreased physical fitness and cardiovascular risk factors are interre-

lated.^{6,7} Physical performance of obese children is generally decreased, particularly in activities requiring lifting of the body.⁸ Considerable controversy exists as to whether this decreased exercise capacity is due to increased weight *per se*, to a lack of physical activity or to the metabolic consequences of fatness.^{4,9} Our previous results¹⁰ suggested that hyperinsulinaemia might play a role in decreased physical fitness. The aim of the present study was to compare the cardiorespiratory response to exercise of control children and of obese children with and without MS.

Patients and methods

Patients and sampling

In all, 180 obese children (103 males, 77 females), referred to the Obesity Clinic of the Department of Paediatrics, University of Pécs were included into the study after the exclusion of endocrinological disorders, or obesity syndromes.³ An institutionally approved informed consent was obtained

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from children and their parents. We considered children as obese if their body weight exceeded the expected weight for height by more than 20% and body fat content (BF) was higher than 25% in males and 30% in females.^{11,12} Weight and height were measured by standard beam scale and Holtain stadiometer, respectively. Body composition was estimated according to the method of Parizkova and Roth from the sum of five skinfolds (biceps, triceps, subscapular, suprailiac and calf) as measured by Holtain caliper.¹² Blood pressure was measured in each subject at least three times on three separate days by the same observer using mercury-gravity manometer with proper cuff size, according to the method recommended by the Second Task Force on Blood Pressure Control in Children.¹³ If the average of the three blood pressure values was above the 95th percentile for age and sex, 24 h ambulatory blood pressure monitoring (ABPM) was performed. Children with mean ABPM values exceeding the 95th percentile value for height and sex were considered hypertensive.¹⁴ Oral glucose tolerance tests (75 g anhydrous glucose) were carried out after an overnight fast and blood samples were taken at 0, 30, 60, 90, 120 and 180 min for the determination of blood glucose, serum insulin and lipid levels. Blood glucose was measured by glucose oxidase method.¹⁵ Serum cholesterol, triglyceride and HDL-cholesterol levels were determined by enzymatic method using Boehringer kits.^{16–18} Serum total cholesterol and triglyceride values were considered abnormal if their concentration was higher than 5.2 and 1.5 mmol/l respectively.¹⁹ HDL-cholesterol value was considered abnormal if its concentration was lower than 0.9 mmol/l.¹⁹ If any of the lipid levels was abnormal, the child was considered dyslipidaemic. Plasma immunoreactive insulin levels were measured with commercially available radioimmunoassay kits from the Institute of Isotopes of the Hungarian Academy of Sciences. The upper limit of normal fasting plasma insulin (mean + 2 s.d. of 100 non-obese children) was 18.7 μ U/ml. Impaired glucose tolerance was defined according to the WHO 1985 definition.²⁰

After assessing the cardiovascular risk factors in our cohort of 180 obese children, 22 boys with multiple cardiovascular risk factors (MS) and 17 boys free of any cardiovascular risk factor (obese) were included into the study. Healthy boys with normal weight matched for age served as controls (control; $n = 29$). The anthropometric parameters of these groups are shown in Table 1. MS was defined as the simultaneous occurrence of obesity, hyperinsulinaemia, hypertension and both or at least one of the impaired glucose tolerance and dyslipidemia. To evaluate the association between the physical fitness level and multiple cardiovascular risk factors, a multistage test—involving an incremental treadmill test—was performed.

Exercise testing procedure

After arrival to the laboratory, the subjects rested for 30 min. The exercise test was performed on a treadmill (EOS-Sprint,

Table 1 Anthropometric data of patients (mean \pm s.d.)

	MS ($n = 22$)	Obese ($n = 17$)	Control ($n = 29$)
Age (y)	14.16 \pm 1.88	14.15 \pm 2.58	15.25 \pm 1.03
Body weight (kg)	97.29 \pm 15.3* [†]	82.57 \pm 15.68 [‡]	64.27 \pm 8.50
BMI (kg/m ²)	34.05 \pm 3.37* [†]	29.31 \pm 3.80 [‡]	20.52 \pm 2.59
W/H	0.89 \pm 0.05*	0.85 \pm 0.05 [‡]	0.79 \pm 0.04
LBM (kg)	70.24 \pm 11.7* [†]	61.26 \pm 11.08 [‡]	52.97 \pm 6.00
BF (kg)	27.04 \pm 4.25* [†]	21.31 \pm 4.95 [‡]	11.29 \pm 2.92

* $P < 0.05$ MS vs control. [†] $P < 0.05$ MS vs obese. [‡] $P < 0.05$ obese vs control.

BMI, body mass index; W/H, waist-to-hip ratio; LBM, lean body mass; BF, body fat.

Erich JAEGER GmbH&CoKG, Würzburg, Germany), according to a multistage protocol. The protocol involved 3 min of lying on the belt, 3 min sitting on a chair and standing 3 min on the treadmill. After these initial phases the belt speed and the inclination were increased every 30 s, such that the estimated work rate increased in a linear fashion until the predicted maximum load (W/kg) was reached. We used Jone's prediction in determining the predicted maximum exercise capacity,²¹ using the age, sex, weight and height. At least one bipolar chest ECG lead was continuously monitored throughout the test, and the beat to beat R-R intervals were registered. Blood pressure was measured each minute by auscultation. Respiratory variables were measured by means of a Jaeger EOS-Sprint exercise metabolic measurement system. The metabolic system consist of: a highly linear pneumotach including pressure transducer, amplifier and digital integrator with temperature compensation; a highly accurate gas analysators for O₂ and CO₂; an automatic calibration system; and the barometric pressure transducer and temperature sensor. The O₂ and the CO₂ concentrations were determined from the mixed expired air and the volume of the expired air was measured using a pneumotachograph. The subjects breathed through a tightly fitting face mask and a non-rebreathing respiratory valve into the pneumotachograph and a mixing bag. The air from the mixing bag was continuously sampled by the gas analyser, which was previously calibrated with known gas mixtures. The pneumotachograph was calibrated with a 21 syringe prior to each test.

Exercise duration (ED), resting heart rate (HR₀), peak heart rate (HR_{peak}), physical working capacity at 170 beat/min (PWC-170), peak oxygen consumption (VO_{2peak}) and the lactic acidosis threshold (LAT) were determined. LAT was determined by the V-slope method.²²

Statistical analysis

Means, standard deviations were calculated with standard methods. Statistical significance of the means was analysed with analysis of variance (ANOVA), and the statistical significance was tested by Scheffe *post hoc* test. Variables were normalised for body weight, using body weight as covariant.

Table 2 Cardiovascular risk factors values in patients (mean ± s.d.)

	MS (n = 22)	Obese (n = 17)	Control (n = 29)
Insulin (μU/ml)	26.22 ± 16.07*†	11.93 ± 5.69	13.20 ± 6.20
Cholesterol (mmol/l)	4.51 ± 0.99*	4.01 ± 0.62	3.62 ± 0.63
Triglyceride (mmol/l)	1.62 ± 0.89*	1.39 ± 0.54	1.02 ± 0.49
Systolic blood pressure (mmHg)	143.63 ± 18.65*	135.88 ± 20.48‡	123.79 ± 9.32
Diastolic blood pressure (mmHg)	82.95 ± 9.71*	78.82 ± 8.39	73.62 ± 7.42

*P < 0.05 MS vs control. †P < 0.05 MS vs obese. ‡P < 0.05 obese vs control.

Results

Boys with MS had a significantly higher body weight (BW), lean body mass (LBM) and body fat (BF) compared with obese and control groups. Obese boys also had significantly higher body weight, LBM and BF (Table 1). Since there were significant differences in BW, LBM and BF between the three groups, variables of the physical fitness were normalised for BW.

Serum insulin was significantly higher in MS group as compared to obese and control groups. Serum total cholesterol, triglyceride and blood pressure values were significantly higher in MS group, as compared to control group. In the obese group only the systolic blood pressure was significantly higher than in the control group (Table 2).

Obese children with or without MS, demonstrated a significantly shorter ED than did normal controls. In the MS group markedly shorter ED was observed as compared to the obese group (Figure 1).

HR₀ was significantly higher in obese groups than in controls. This difference was more pronounced in MS group. However, there was no difference in the peak heart rate between MS and obese groups. HR_{peak} on the other hand, was significantly higher in obese children with MS as compared to controls. The peak heart rate response of obese children with no MS did not differ from that of controls (Figure 2).

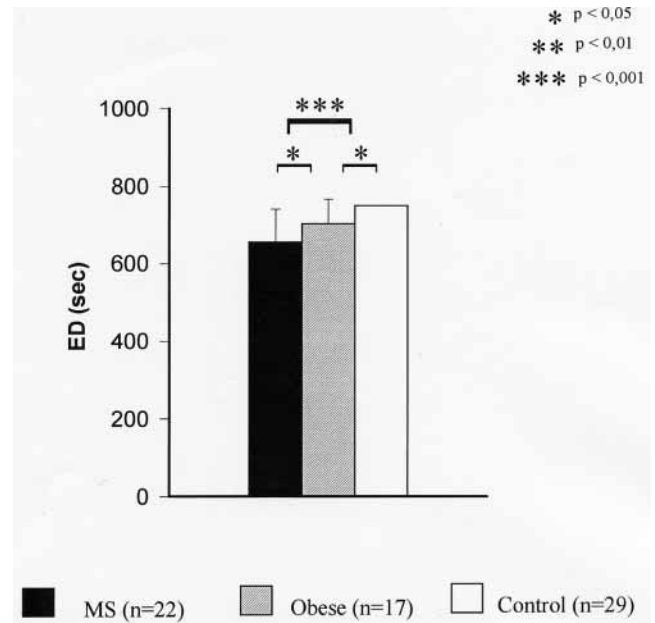


Figure 1 Endurance time (ED).

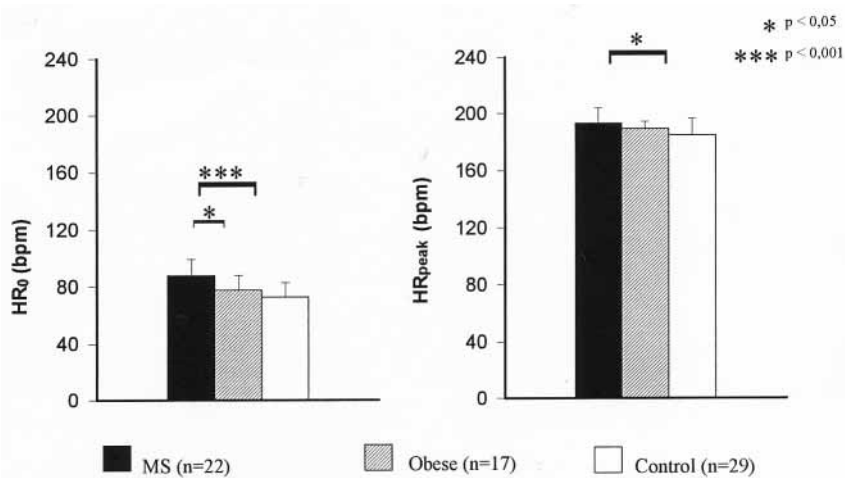


Figure 2 Resting (HR₀) and peak (HR_{peak}) heart rate.

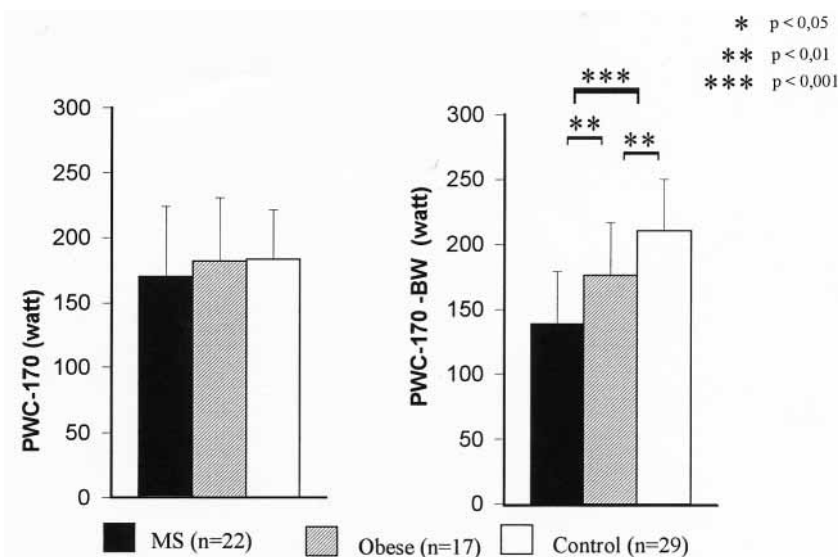


Figure 3 Physical working capacity (PWC-170), and PWC-170 normalised for body weight (PWC-170-BW).

Table 3 Original LAT and VO_{2peak} values, and those normalised for body weight (LAT-BW, VO_{2peak} -BW) (mean \pm s.d.)

	MS (n = 22)	Obese (n = 17)	Control (n = 29)
LAT (l/min)	1.53 \pm 0.42	1.53 \pm 0.48	1.61 \pm 0.28
LAT-BW (l/min)	1.33 \pm 0.37*	1.50 \pm 0.37 [†]	1.78 \pm 0.37
VO_{2peak} (l/min)	2.70 \pm 0.60	2.51 \pm 0.74	2.47 \pm 0.39
VO_{2peak} -BW (l/min)	2.19 \pm 0.42*	2.43 \pm 0.45 [†]	2.91 \pm 0.43

* $P < 0.05$ MS vs control. [†] $P < 0.05$ obese vs control.

Absolute values of PWC-170 were not different in the three groups; however, when PWC-170 was normalised for body weight, it was significantly lower in obese as compared to controls and further decreased in obese children with MS (Figure 3).

The VO_{2peak} and the LAT were also significantly lower in the obese groups when normalised for the body weight (Table 3).

Discussion

The relationship between physical performance and obesity, on the one hand, and physical performance and atherosclerotic risk factors, on the other hand, have been studied by several authors, with conflicting results.

Obesity may be associated with a decrement in exercise performance, particularly at maximal work levels. Davies *et al*⁹ found that during maximal exercise there was a marked decrement in exercise performance in obese females as compared with controls. During maximal performance the absolute VO_{2max} was the same in obese and nonobese

subjects but for a given body weight or lean body mass VO_{2max} was significantly reduced. During light exercise when oxygen intake for a given work output was standardised for body weight it was shown that obese patients exercised within the normal range of aerobic energy expenditure. Zanconato *et al*²³ performed maximal exercise testing on 23 obese children aged 9–14 y, who had lower endurance time and VO_{2max}/kg values than the controls, but their absolute VO_{2max} values were not significantly different from the controls.

There are data indicating that hyperinsulinaemia, which is ubiquitously associated with obesity, might have a direct or indirect effect on the cardiovascular system and, consequently, on exercise performance. Hyperinsulinaemic obese children had significantly lower physical working capacity than the non-hyperinsulinaemic ones, in spite of their similar anthropometric characteristics and lipid profiles.¹⁰

The majority of obese children, especially those with MS, had resting tachycardia, which can be explained by elevated sympathetic nervous system activity in response to hyperinsulinaemia. In our earlier investigations we could detect increased norepinephrine levels in obese children with hypertension and hyperinsulinaemia.²⁴ While in some cases a decreased activity of the sympathetic nervous system is emphasized in the aetiology of obesity, some data suggest that overfeeding and hyperinsulinaemia stimulates the sympathetic nervous system.^{25–27} In addition, it has been demonstrated that an increase in brain insulin reduces neuropeptide Y and its gene expression in the arcuate nucleus²⁸ which results in the stimulation of the sympathetic nervous system.²⁹

Fripp *et al*⁶ demonstrated a correlation between physical fitness and risk factors for atherosclerosis in male adolescent population. They also found that higher levels of fitness were

associated with better risk profiles (decreased body mass index, lower systolic and diastolic blood pressure and triglyceride levels, and higher high-density lipoprotein levels). The multiple linear regression analysis demonstrated that body mass index accounted for much of the variation in fitness parameters.

Our results demonstrated clearly that children with MS had significantly lower physical performance as measured by ED and body weight corrected PWC-170, VO_{2peak} and LAT values than obese children without metabolic disturbances. The question, whether the metabolic alterations or the decreased physical activity are responsible for the poor physical performance in children with MS, cannot be answered at present, and further investigations are warranted.

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