

Research article

The influence of weight loss on anaerobic threshold in obese women

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Abstract

Obesity is associated with decreased physical activity. The aim of the study was to assess the anaerobic threshold in obese and normal weight women and to analyse the effect of weight-reduction therapy on the determined thresholds. Patients and methods: 42 obese women without concomitant disease (age 30.5 ± 6.9 y; BMI 33.6 ± 3.7 kg·m⁻²) and 19 healthy normal weight women (age 27.6 ± 7.0 y; BMI 21.2 ± 1.9 kg·m⁻²) performed cycle ergometer incremental ramp exercise test up to exhaustion. The test was repeated in 19 obese women after $12.3 \pm 4.2\%$ weight loss. The lactate threshold (LT) and the ventilatory threshold (VT) were determined. Obese women had higher lactate (expressed as oxygen consumption) and ventilator threshold than normal weight women. The lactate threshold was higher than ventilatory one both in obese and normal weight women (1.11 ± 0.21 vs 0.88 ± 0.18 L·min⁻¹, $p < 0.001$; 0.94 ± 0.15 vs 0.79 ± 0.23 L·min⁻¹, $p < 0.01$, respectively). After weight reduction therapy neither the lactate nor the ventilatory threshold changed significantly. The results concluded that; 1. The higher lactate threshold noted in obese women may be related to the increased fat acid usage in metabolism. 2. Both in obese and normal weight women lactate threshold appears at higher oxygen consumption than ventilatory threshold. 3. The obtained weight reduction, without weight normalisation was insufficient to cause significant changes of lactate and ventilatory thresholds in obese women.

Key words: Obesity, anaerobic threshold, lactate threshold, ventilatory threshold.

Introduction

Obesity is caused by imbalance between energy intake and output leading to an accumulation of body fat. Excess fat accumulation combined with decreased physical activity in obese subjects is associated with serious medical consequences and is associated with increased morbidity and mortality (Shepherd A, 2009). Moreover, obesity is related to the frequent occurrence of insulin resistance, which may influence on metabolic processes during exercise (Salvadori et al., 2004).

Anaerobic threshold is an important parameter that reflects endurance capacity, apart from oxygen consumption. Anaerobic threshold could be estimated by direct (measuring lactate concentration) and indirect (based on ventilatory parameters) methods. Lactate threshold is determined as the point at which blood lactate concentration begins to increase above the resting value, which

indicates the initial imbalance point between lactate production and muscular lactate elimination during incremental exercise (Meyer et al. 2005). Unlike the lactate threshold, the ventilatory threshold may not be the result of the rise of serum lactate concentration during exercise as it was assumed previously (Wasserman et al., 1973; 1994).

The lactate threshold depends on many factors, including energetic substrates that are used during exercise. Lactate concentration depends on consumed diet (Langfort, 1996). High carbohydrate diet decreases lipid metabolism, while low carbohydrate diet with high fat stores favours increased utilization of fat and decreases carbohydrate metabolism (Jasson and Kaiser, 1982). Presumably, this explains the divergent data concerning the achieved lactate threshold in relation to diet modifications (Costil et al., 1977; Yoshida, 1986).

Both metabolic and physical consequences of excess body mass in obesity could influence the ventilatory and the lactate thresholds. High concentration of circulating free fatty acids in obesity may influence lactate threshold (Hulens et al., 2001; Rowland, 1991). There are few publications dealing with anaerobic thresholds in obese patients (Colak and Ozcelik, 2004; Hulens et al., 2001; Ozcelik et al., 2006; Rowland, 1991; Salvadori et al., 1991; 1992).

The aim of the present study was to assess the anaerobic threshold using two different methods in obese and normal weight women and to analyse the effect of weight-reduction therapy on the determined thresholds.

Methods

Fifty-nine obese women were enrolled into this study. All subjects were diagnosed with obesity (BMI ≥ 30 kg·m⁻²) without concomitant diseases. Exclusion criteria included evidence of any disease, drug therapy and contraindication for exercise test and smoking. The study protocol was approved by the Ethics Committee of Medical University of Silesia and all subjects gave their informed written consent for participation in the study.

Analysis was only performed in obese women with the duration of exercise exceeding 9 minutes corresponding to the load of 75 W. Finally, the examined group consisted of 42 women (age 30.5 ± 6.9 y; BMI 33.6 ± 3.7 kg·m⁻², Table 1).

All obese women participated in a 3-month weight

Table 1. Characteristics of normal weight and obese women and results of body weight reduction therapy. Data are means (\pm SD).

Parameters	Normal weight (n=19)	Obese (n=42)	Obese (n=19)		significance before and after
			Before	After	
Body mass (BM) [kg]	57.4 (6.7)	92.1 (12.1) ***	92.7 (12.7) ***	81.7 (17.5) ***	.001
BMI [kg·m ⁻²]	21.2 (1.9)	33.6 (3.7) ***	33.8 (4.0) ***	29.8 (4.2) ***	.001
Fat mass [kg]	15.1 (4.0)	39.7 (8.7) ***	40.1 (9.2) ***	30.9 (8.6) ***	.001
Fat mass [%BM]	25.6 (4.8)	42.7 (3.9) ***	43.0 (3.9) ***	37.2 (4.6) ***	.001
Fat free mass (FFM) [kg]	42.5 (4.4)	52.4 (4.5) ***	52.5 (4.5) ***	50.8 (4.8) ***	.001
Fat free mass (FFM) [%BM]	74.4 (4.7)	57.3 (3.9) ***	57.0 (3.9) ***	62.7 (4.6) ***	.001
LA rest [mmol/L]	1.47 \pm 0.19)	1.84 (.51) ***	1.93 (.44) ***	1.47 (.37)	.01
RQ at rest	.84 (.05)	.77 (.08) ***	.78 (.09) *	.79 (.08) *	NS
RQ at VT	.82 (.08)	.78 (.08) **	.77 (.08) *	.78 (.06) *	NS

Statistical significance vs Normal Weight group *** p < 0.001; ** p < 0.01; * p < 0.05

reduction therapy that consisted of: a 1000-1200 kcal/day balanced diet and recommended regular physical exercises (no less than three times a week for about 30 minutes including: walking, cycling and swimming; 60-70% of maximal heart rate - HRmax). During a 3-month complex group weight loss therapy, every second week all patients participated in counselling with a physician, a dietician (one hour) and one hour psychotherapy (cognitive and behavioural methods were used). Each patient was asked to note in a diary ingested food and the type and duration of physical activity. Before a complex therapy they ate 35 % of fat, 19% of protein and 45% carbohydrates. During therapy diet contained 19% of fat, 25% of protein and 56% of carbohydrates.

After the body weight reduction program, 26 women from the study group were re-examined (mean age 31.6 ± 7.4 years; body weight 83.8 ± 13.9 kg; BMI 29.4 ± 6.5 kg·m⁻²). During the re-examination seven patients stopped the incremental test prematurely and were excluded. Finally, 19 women were included into the analysis. The initial anthropometric parameters and age of this group did not differ significantly from those of all obese participants (Table 1).

Additionally, we examined 26 normal weight women, 19 of them were included into the study as a control group (Table 1). The reason for the high dropout rate both of obese and normal weight women was the early discontinuation of the test due to fatigue and pain of knee-joints.

Body mass was measured using Tanita, height by wall-mounted stadiometer. BMI was calculated as weight in kilograms divided by height in meter squared (kg·m⁻²). Obesity and normal weight were diagnosed according to WHO criteria (BMI ≥ 30 kg·m⁻² and < 25 kg·m⁻², respectively).

Body composition was measured using bioelectrical impedance method from hand to foot using Bodystat 1500 body composition analyzer. Subjects were investigated without shoes and in bathing suits in the morning, in thermal comfort. After weight reduction therapy all measurements were repeated.

Three days diet composition was analysed before tests. Diet of normal weight subjects contained approximately 20% of fat, 25% of proteins and 55% of carbohydrates. Description of the diet of obese subjects was mentioned above. A three months history of physical activity was obtained for each study subject. 19% obese women

reported very low, 24% low, 52% medium and only 5% high physical activity, while 6% of normal weight reported very low, 10% low, 79% medium and 5% high physical activity. In the analysis of physical activity the type of occupation were taken into consideration. It has to be stressed that self-reported food intake is usually lowered, but physical activity is raised by obese subjects.

ECG was performed in each subject before the exercise test. The incremental cycle ergometer (Monark) exercise test was performed after overnight fasting. The exercise test started with 25 W load, and then its workload was increased by 25 W every 3 minutes up to the moment of exhaustion. The termination of the test was followed by a 5-minute period in a sitting position on cycle ergometer. Oxygen uptake (VO₂max) and metabolic responses were measured using the START 2000 (MES) metabolic system which analyses respiratory gases on a breath-by-breath basis. The values were averaged per minute and the final values derived from the last minute of each load (when the blood samples were withdrawn). Prior to each test analysers were calibrated using gas calibration (5% CO₂, 12% O₂ and balance N₂ and the volume calibration) by standardized syringe.

During the rest and at the end of each load, blood samples were taken from the finger for assessment of blood lactate concentration (LA). Blood LA concentration was measured enzymatically using commercial kits (Boehringer GmbH, Mannheim, Germany). Δ LA was calculated as a difference between the lactate concentration at each level during the test and the resting value. Lactate threshold (aerobic threshold, LT) was expressed as the oxygen consumption and was determined by regression analysis of the two-segment model plot of log[LA] versus log [VO₂] as described by Beaver et al. (1985). The LA by VO₂ data were divided into two segments fitted with linear regressions. If a significant departure from linearity was found, the data point demarcating the segments with the largest slope difference was designated as the lactate threshold. Figure 1 shows the LT and VT determinations for one patient.

The ventilatory threshold was determined by individual breaking point on VE/VO₂ by the oxygen consumption curves using the complete breath by breath data. The threshold was located where the VE/VO₂ curve inflected upward (Ekkekakis et al., 2008; Ohuchi et al., 1996, Figure 1). All thresholds were also determined in relation to body weight and fat free mass (FFM).

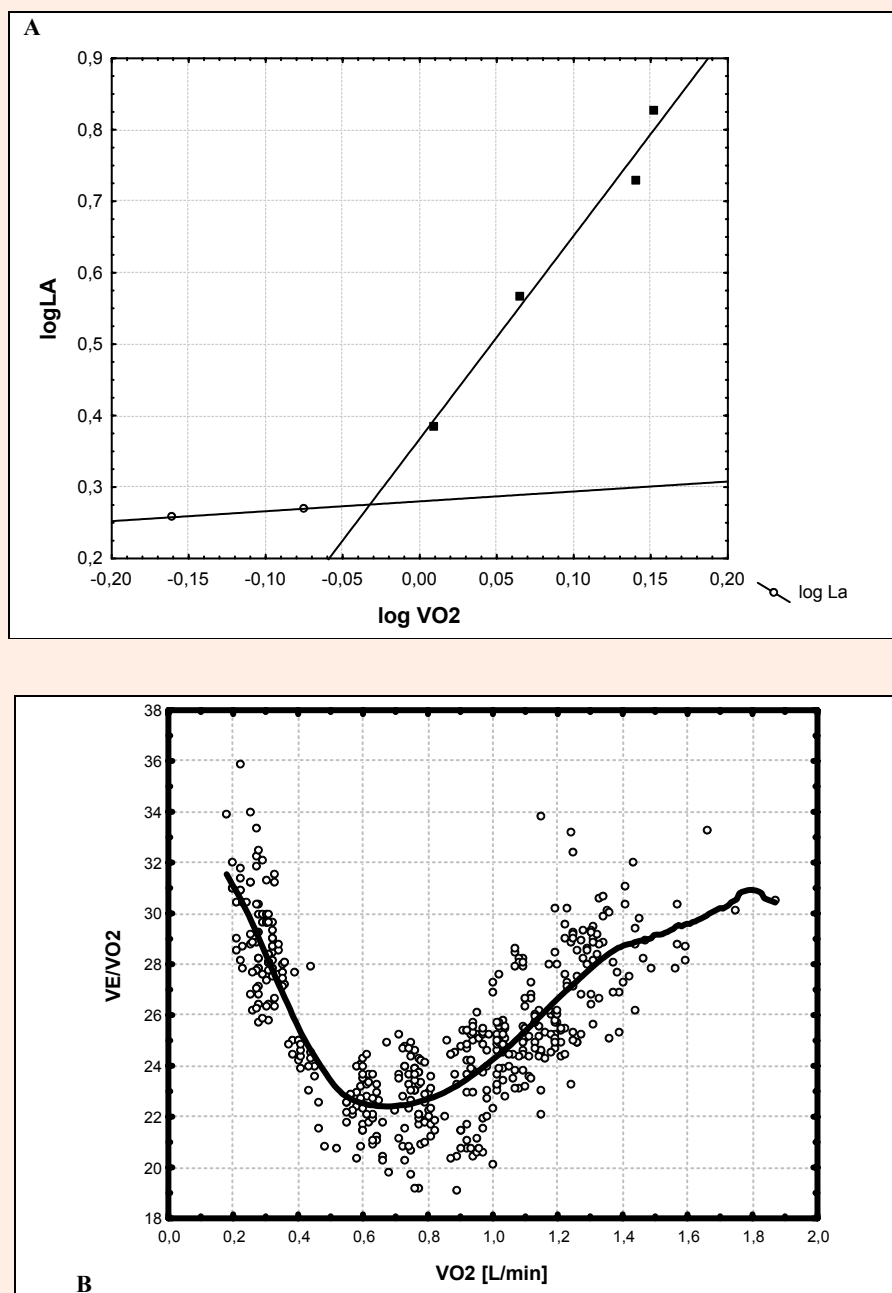


Figure 1. Method of determination of the lactate (A) and ventilatory (B) thresholds for one subject. VO_2 – oxygen consumption; VE/VO_2 – ventilatory equivalent ratio for oxygen; LA : blood lactate concentration.

All values are expressed as means \pm standard deviation. All analyses were performed using the Statistica 6.0. Normality of distribution was analysed using the Kolmogorow-Smirnow test. U Mann-Whitney pair-wise comparison for independent samples and Wilcoxon pair-wise comparison for dependent samples were used. Correlation coefficients were calculated according to Spearman. P values <0.05 were considered as statistically significant.

Results

Comparison of obese and lean women

At rest, blood lactate concentration was significantly higher, and respiratory quotient (RQ) was significantly

lower in obese women than in the control group. During exercise blood lactate concentration and RQ were increasing after 50 W load, with significantly lower values in obese subjects (Figure 2 and 3). Oxygen consumption at rest and during the exercise was higher in obese than normal weight women (Table 2), but from 75 W the differences were losing significance. The VE and VE/VCO_2 data were presented in Table 3.

The lactate threshold (LT) expressed as the amount of oxygen consumption were significantly higher in obese women than in the control group. However, the lactate thresholds in relation to body weight and fat free mass were lower in the obese (Table 4). A similar pattern was observed for ventilatory thresholds (Table 4).

Both in obese and normal weight women the

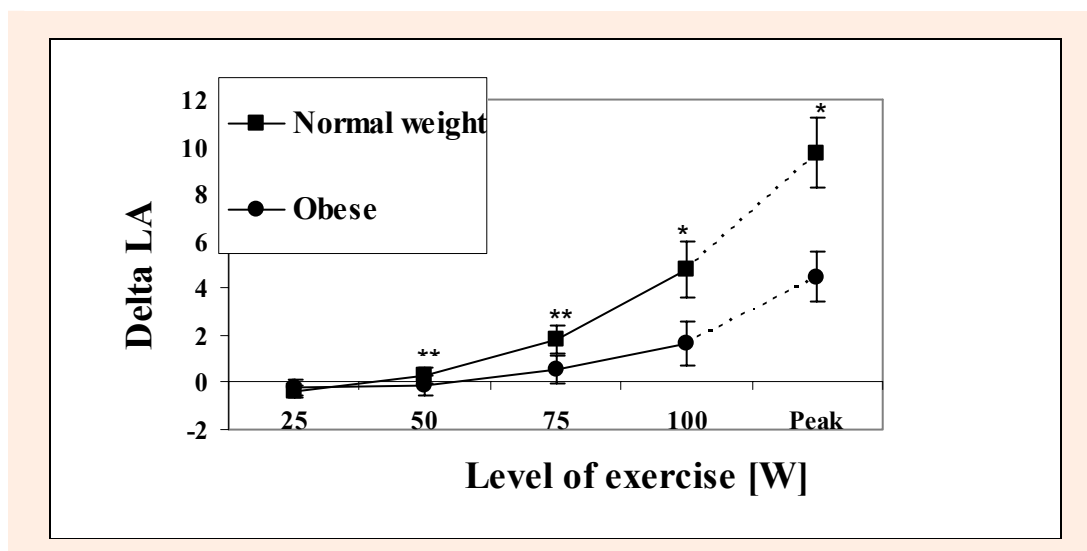


Figure 2. Difference between lactate concentration at each level during the test and resting value in obese and normal weight women. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

lactate thresholds were higher than ventilatory ones (Table 4). In the whole group and in obese but not in normal weight women a significant correlation between LT [$L \cdot min^{-1}$] and VT [$L \cdot min^{-1}$] was observed ($R = 0.398$, $p \leq 0.001$; $R = 0.366$, $p = 0.017$ and $R = 0.232$, $p = 0.34$, respectively).

The influence of weight reduction on thresholds

The obtained mean weight loss was 12.3 ± 4.2 % of the baseline weight (Table 1). After weight reduction only one woman had normal body weight. Eleven were overweight (BMI range $25.6-29.8 \text{ kg} \cdot m^{-2}$) and 7 were still obese (BMI range $30.1-39.7 \text{ kg} \cdot m^{-2}$).

After weight reduction blood lactate concentrations and RQ at rest did not change significantly. Also the increase of lactate concentration and RQ at exercise was similar as before weight reduction. The VO_{2peak} after weight reduction therapy did not change significantly ($1.66 \pm 0.28 \text{ L} \cdot min^{-1}$ vs $1.78 \pm 0.28 \text{ L} \cdot min^{-1}$).

The obtained weight reduction did not influence lactate threshold (1.12 ± 0.21 vs 1.13 ± 0.15) and ventilatory threshold ($0.89 \pm 0.17 \text{ L} \cdot min^{-1}$ vs $0.94 \pm 0.25 \text{ L} \cdot min^{-1}$) (Table 4). There was also earlier occurrence of ventilatory threshold after weight reduction therapy (Table 4). We did not observe any changes in oxygen consumption thresholds ($\% VO_{2peak}$) (Table 4).

Discussion

This study demonstrated that both ventilatory and lactate thresholds appear at higher oxygen consumption in obese than in normal weight women. Moreover the obtained 12% weight loss was insufficient to cause significant changes of the thresholds.

The issue of ventilatory threshold in obese has been previously investigated. Salvadori et al. examined the influence of obesity on anaerobic threshold measured by change of ventilatory parameters during physical effort

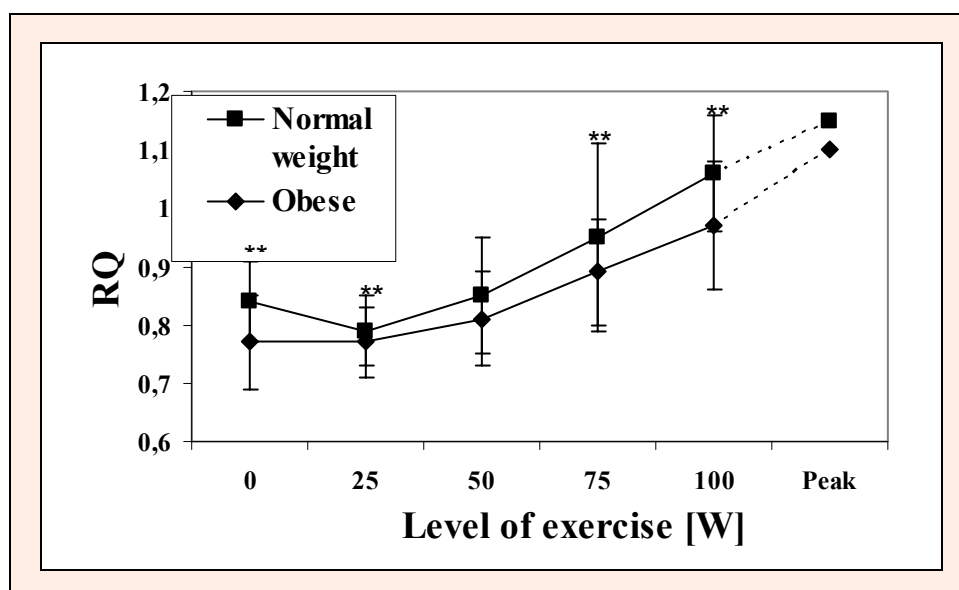


Figure 3. RQ at rest and during the test in obese women and normal weight controls. * $p < 0.05$; ** $p < 0.01$.

Table 2. VO_2 and VCO_2 at rest and during exercise in examined groups. Data are means (\pm SD).

	VCO_2 ($\text{L}\cdot\text{min}^{-1}$)			VO_2 ($\text{L}\cdot\text{min}^{-1}$)		
	Obese (n=42)	Normal weight (n=19)	P	Obese (n=42)	Normal weight (n=19)	P
At rest	.3 (.1)	.2 (.1)	<0.05	.3 (.1)	.3 (.1)	<0.01
25W	.6 (.1)	.5 (.1)	<0.05	.8 (.2)	.6 (.1)	<0.01
At VT	.7 (.2)	.6 (.2)	NS	.9 (.2)	.8 (.2)	<0.05
Peak	1.8 (.4)	1.6 (.4)	NS	1.7 (.3)	1.5 (.4)	NS

(Salvadori et al, 1991; 1992). Obese patients had a similar peak level, but the ventilatory threshold was significantly higher in normal body weight subjects than in obese ones. These results are opposite to our findings. Differences may result from a higher grade of obesity in patients examined by Salvadori et al. (mean BMI $40 \text{ kg}\cdot\text{m}^{-2}$ and $33 \text{ kg}\cdot\text{m}^{-2}$, respectively). It is worth to notice that the cited author did not determine thresholds in relation to absolute and fat free mass. We found that ventilatory threshold was significantly lower in obese than in normal weight women when presented in relation to body weight but similar in relation to fat free mass. In accordance with our results, Rowland (1991) showed that ventilatory threshold estimated in relation to body weight was significantly lower in obese patients.

Oxygen consumption at rest and during exercise was higher in obese than normal weight women due to increased energy expenditure for carrying of the self body and fitness. The lack of significant difference of oxygen consumption at the work load of 75, 100 and 125 W and at the peak of exercise may be explained by the limited number of the study group and by suggested by Salvadori et al. increased in peripheral O_2 extraction in obese (Salvadori et al., 2004; 2008).

As mention previously both ventilatory threshold and VO_2 peak did not change after the complex weight reduction therapy, which was not followed by normalisation of body weight. Thus all but one study subjects remained overweight or obese. The similar results was observed by others (Hakala et al., 1996, Larsson and Mattsson, 2003). These authors did not observe the improvement in physical efficiency after VLCD without regular physical activity. Shinkai et al. (1994) despite the usage of the moderate physical activity (50-60% VO_2max , 3-4 times a week for 45-60 min) also did not observe an increase of oxygen uptake at peak exercise. The similar results were presented by Utter et al. (1998) who examined the influence of diet alone and together with increased physical activity (5 days weekly 60-80% VO_2max). The weight reduction therapy lasted 12 weeks. 9.7% increase of VO_2max was observed only in the group with increased physical activity. Also, Ashutosh (1997) observed 11.6% increase of VO_2 at the peak of exercise after weight reduction therapy with regular physical activity (3 times per week 45-60 min). These results might

indicate that the recommended activity for our subjects during weight reduction program was insufficient to affect their capacity measured by ventilatory threshold and oxygen uptake. Each participant has obtained recommendation for increase of daily activity and implementation of self-organize exercise three times a week for at least 30 minutes. Only some of them declared to attend to a structured club. In such circumstances it is difficult to expect the physical activity at the level of 50-70% VO_2max . The low level of physical activity during weight reduction therapy in our study is in line with the significant loss of lean body mass. A characteristic feature of comprehensive weight loss therapy is to restrain loss of lean body mass.

We also observed the higher lactate threshold in obese than in normal weight women. In obese and normal weight women lactate threshold appeared at higher oxygen consumption than ventilatory threshold. The time issue at which lactate and ventilatory threshold occur needs to be discussed, taking also into account the limitations of lactate threshold estimation (determination of blood lactate concentrations in 3 minutes intervals).

Numerous thresholds determination methods were described (Bosquet et al., 2002; Ekkekakis et al., 2008; Meyer et al., 2005; Ohuchi et al., 1996). Available lactate models almost exclusively fit into one of two categories: 1) the first increase in blood lactate concentrations above resting values during incremental exercise (aerobic lactate threshold, LT1) and 2) the maximal lactate steady state representing the exercise intensity above which a continuous increase in blood lactate is unavoidable (anaerobic threshold, LT2) (Meyer et al., 2005). According to Wasserman's theory, the ventilatory threshold is an answer to increased ventilation driven by an elevated VCO_2 which results from the buffering of protons (Wasserman et al., 1973). However, further studies proved that the appearance of ventilatory threshold is independent of lactate concentration (Cecca et al., 1986; Gaesser and Poole, 1986; Hagberg et al., 1982; Hughes et al., 1982) but related to the other factors such as: serum potassium concentration (Lindinger and Sjogaard., 1991). It was suggested that during exercise, overweight subjects predominantly utilise free fatty acids (FFA) due to the decreased tissue glucose consumption, as the effect of insulin resistance and increased intramuscular fat storage

Table 3. VE and VE/VCO_2 at rest and during the exercise in examined groups. Data are means (\pm SD).

	VE ($\text{L}\cdot\text{min}^{-1}$)			VE/VCO_2 ($\text{L}\cdot\text{min}^{-1}$)		
	Obese (n=42)	Normal weight (n=19)	P	Obese (n=42)	Normal weight (n=19)	P
At rest	8.5 (2.0)	7.3 (1.3)	<0.05	32.2 (3.9)	32.1 (4.4)	NS
25W	17.3 (3.3)	14.7 (2.1)	<0.05	30.0 (4.1)	29.8 (3.2)	NS
At VT	19.9 (4.2)	18.1 (5.0)	NS	29.2 (3.5)	28.4 (3.6)	NS
Peak	53.2 (11.4)	49.4 (10.0)	NS	30.4 (4.0)	30.7 (4.1)	NS

Table 4. Thresholds in the examined groups. Data are means (±SD).

Parameters	Normal weight (n=19)	Obese (n=42)	significance obese vs control	Obese (n=19)		significance before and after
				Before	After	
LT [L·min ⁻¹]	.94 (.15) #	1.11 (.21)*	0.01	1.12 (.21) †	1.13 (.15) ‡	NS
LT/BM [mL·min·kg ⁻¹]	16.7 (3.2)	12.1 (2.4)	0.001	12.2 (2.3)	14.0 (1.7)	0.01
LT/FFM [mL·min·kg ⁻¹]	22.4 (4.1)	21.2 (4.1)	NS	21.3 (3.9)	22.4 (2.8)	NS
VT [L·min ⁻¹]	.79 (.23)	.88 (.18)	0.05	.89 (.17)	.94 (.25)	NS
VT/BM [mL·min·kg ⁻¹]	13.8 (.2)	9.6 (2.0)	0.001	9.7 (2.2)	11.5 (2.4)	0.01
VT/FFM [mL·min·kg ⁻¹]	18.4 (4.7)	16.9 (3.4)	NS	17.0 (3.6)	18.4 (4.5)	NS
LT/VO ₂ peak [%]	67.3 (12.4)	65.9 (11.2)	NS	68.4 (13.1)	64.7 (9.9)	NS
VT/VO ₂ peak [%]	53.8 (12.1)	53.7 (9.8)	NS	54.9 (11.5)	52.5 (8.4)	NS

* p < 0.001 LT vs VT in obese women, # p < 0.01 LT vs VT in normal weight women, † p < 0.001 LT vs VT in obese women before therapy, ‡ p < 0.01 LT vs VT in obese women after therapy. LT: lactate threshold; VT: ventilatory threshold; BM: body mass; FFM: fat free mass.

(Perseghin et al., 1999, Salvadori et al., 2004; Weiss R et al., 2003). This hypothesis is supported by the lower levels of lactate during exercise. In consequence, in obese higher lactate threshold and lower RQ values at rest and during the exercise is observed. Our results are in line with previous studies (Kelman et al., 1975; Maughan et al., 1978; Salvadori et al., 2008; Yoshida, 1986).

Diet is another factor that may influence lactate threshold. This issue was previously investigated in normal weight subjects producing conflicting results. Ivy et al. (1981) and Hughes et al. (1982) have shown that lactate threshold was increased in elevated FFA conditions or depletion of glycogen in muscles. On the other hand, Powers et al. (1983) have demonstrated, that increased serum concentration of FFA after coffee ingestion did not influence blood lactate concentration. Also, Yoshida (1986) did not observe changes in lactate and ventilatory thresholds on high and low carbohydrate diet. The lack of expected changes in ventilatory threshold was explained by the immediate buffering of lactate in the blood by the bicarbonate system.

It may be supposed that higher serum FFA concentration in obese facilitates fat metabolism during exercise. Indeed, Goodpaster et al. (2002) showed that during moderate exercise, obese sedentary men had increased rates of fatty-acids oxidation from nonplasma sources and reduced rate of carbohydrate oxidation, particularly muscle glycogen, in comparison to lean sedentary men. This observation could support our finding - the low increase of lactate concentration in blood during exercise and higher lactate threshold in obese than in normal-weight women. The higher lactate thresholds in obese women were also previously shown by Ardevol et al. (1998).

The limitation of our study is rare (in 3 minutes intervals) determination of blood lactate concentration decreasing the accuracy of threshold estimation. In consequence we may suppose that lactate thresholds values are somehow overestimated. However this error is similar for both obese and normal weight women. Thus further studies are necessary to estimate lactate threshold with higher precision in obese.

It is worth to stress that the majority of performed studies concerning the influence of weight reduction on ventilatory parameters, and on ventilatory threshold (Colak and Ozcelik, 2004; Ozcelik et al., 2006; Salvadori et al., 1992; 1993; 1999; Sartorio et al., 2003) do not provide information about lactate concentration and lactate

threshold during physical exercise before and after weight reduction therapy. The published studies concern mostly healthy individuals, athletes, or people suffering from diabetes t.2 and coronary artery diseases (Belli et al., 2007; Omija et al., 2004). According to the best of our knowledge the relationship between threshold values measured by ventilatory parameters and lactate concentration was not studied in obese.

Conclusion

The results concluded that; 1. The higher lactate threshold noted in obese women may be related to the increased fatty acid usage in metabolism. 2. Both in obese and normal weight women lactate threshold appears at higher oxygen consumption than ventilatory threshold. 3. The obtained (12%) weight reduction, without weight normalisation was insufficient to cause significant changes of lactate and ventilatory thresholds in obese women.

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Key points

- Results showed that adolescent young female gymnasts have an altered serum inflammatory markers and endothelial activation, compared to their less physically active peers.
- Physical activities improved immune system.
- Differences in these biochemical data kept significant after adjustment for body weight and height.

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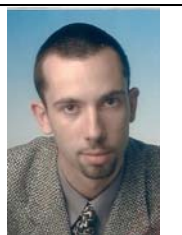
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