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THE RELATIONSHIP BETWEEN IRON STATUS AND PERCEIVED EXERTION IN TRAINED AND UNTRAINED WOMEN

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The subjects for this study were 24 female, well-trained distance runners and 41 sedentary, female university students. On the basis of haematological measurements, the subjects were divided into four groups: a group with iron deficiency, non-anemic (normal haemoglobin and red cell count but serum ferritin less than 20 μ g^ol⁻¹) runners (IDR, $n = 11$), a group of runners with normal iron status (NR, $n = 13$), a group of sedentary women with normal iron status (NC, $n = 26$) and a group of iron deficient, non-anemic sedentary subjects (IDC, $n = 15$). Maximal oxygen uptake ($\rm\dot{VO}_{2}$ max) and lactate thresholds were determined during incremental bicycle ergometer exercise testing and on separate occasions the ratings of perceived exertion (RPE) were assessed at 25, 50, 75 and 90% $\rm\dot{VO}_2$ max using Borg's scale. There were no differences in the relationship between RPE and work load groups of runners or sedentary subjects with normal and deficient iron status. Iron deficiency did not affect VO,max and lactate thresholds. However, low body iron stores (serum ferritin level below 20 μ g \circ 1⁻¹) without overt anemia was associated with increased venous blood lactate concentration after maximal exercise load. This study demonstrated that physical work capacity and RPE were not affected by iron deficiency without anemia either in trained or untrained women.

Key words: iron deficiency, ferritin, ratings of perceived exertion, blood lactate, athletes

INTRODUCTION

Depressed serum ferritin levels with normal haemoglobin concentration during long term training have been reported in athletes involved in heavy exercise, especially women (1, 2, 3). There is general agreement that adults with iron deficiency are more quickly fatigued during brief, strenuous exercise and develop exaggerated tachycardia and lactic acidosis in proportion to the severity of their anemia (4, 5). Furthermore, blood lactate concentration is elevated during submaximal exercise and the lactate threshold (LT) and/or the onset of blood lactate accumulation (OBLA) may occur at a lower exercise intensity in iron deficient subjects than in normals (2). Iron deficiency without over anemia has been shown to affect muscle metabolism during exercise (6).

No specific studies have been carried out to explore the effect of iron deficiency in human subjects on the perceived exertion during physical work although there are data indicating an association between iron deficiency and

psychological functions (7, 8). The literature reveals that muscle lactate production may influence rating of preceived exertion (RPE) by increasing the intramuscular hydrogen ion concentration causing muscle fatigue (9).

In lactic acidosis is one of the main factors promoting development of fatigue, and blood lactate concentration is higher during submaximal exercise in iron deficiency, then subjects with low ferritin concentration should also demonstrate the changes in RPE. The aim of this work was to find out whether iron deficiency without anemia influences RPE and lactate production during exercise.

MATERIALS AND METHODS

Subjects

The subjects for the study were 32 well-trained female distance runners and 58 sedentary female university students. All runners had followed a regular training schedule (for approx. 4 years); their weekly running distance in the 2 months before the study was 50 ± 15 km, range 36—70 km. A questionnaire concerning training habits and medical history was completed. Subjects from the control group were healthy non-smokers, but none trained on a regular basis. Signed informed consent was obtained from all subjects and the project was approved by the University Committee for Human Research.

No dietary restrictions were imposed during the entire study, so it was not possible to make an analysis of the diet and report dietary iron intake but none of the subjects was treated by iron supplementation. Each subject had regular menstrual periods and some used oral contraceptives, but for no more than 2 yrs. Medical screening determined that the subjects were free of any inflammatory diseases.

Blood sampling and analysis

Haematological determinations were made on venous blood samples (10 ml) collected from an antecubital vein between 8.00 and 10.00 am on the 3rd to 5th day after the last day of menstrual flow in each woman. The runners had performed no acute exercise during 6 days before blood samples were collected. The determinations carried out on these blood samples included: hemoglobin concentration (Hb), packed cell volume (PCV), red blood cell count (RBC), serum iron (SI), and ferritin (Ferr) concentrations. Concentrations of Hb, PCV and RBC were determined by Coulter Counter (Remed, Poland), serum iron was measured using colorimetric kits (POCh, Poland) and serum ferritin was evaluated by a radioimmunoassay kit (Bio-Rad, Richmond, USA).

Athletes and non-trained subjects were identified as pre-latent anemic (iron deficiency status) by the criteria given by Hausman (10); i.e., subjects were considered to have normal iron status if Ferr > 20 μ g \circ 1⁻¹, SI > 90 μ g \circ dl⁻¹, and Hb > 12.0 g \circ dl⁻¹. Groups of iron deficiency athletes and iron deficiency controls consisted of subjects who had Ferr < 20 μ g \circ 1⁻¹, SI < 90 μ g \circ dl⁻¹ but

 $Hb > 12$ g \circ dl⁻¹. Twenty four athletes and 41 non-trained subjects which satisfied all of the three criteria for assignment were included for further studies. Finally, the subgroups were as follows: a group of control women with normal iron status (NC, $n = 26$), a group of iron deficient sedentary controls (IDC, $n = 15$), a group of runners with normal iron status (NR, $n = 13$) and a group of iron deficient runners (NDR, $n = 11$).

Exercise test

A continuous, progressive bicycle ergometer protocol was used to measure maximal oxygen uptake $(\dot{V}O_2)$ as described in our earlier paper (11). Basing on blood lactate concentration during the exercise test, the following indices were evaluated: 1) the lactate threshold (LT) determined graphically from the relationship between blood lactate and oxygen uptake $(\dot{V}O_2)$ and operationally defined as \overline{VO}_2 during the stage just prior to the rapid, sustained increase in blood lactate above the baseline level; 2) LT1 determined as $VO₂$ at which blood lactate had increased 1 mM above a resting level; 3) LT2 determined as the \overline{VO}_2 at which blood lactate concentration reached a value of 2 mM; 4) the onset of blood lactate accumulation (OBLA) determined as $\dot{V}O_2$ at which blood lactate concentration reached a value of 4 mM; 5) blood lactate concentration at the end of exercise test (LAmax) determined immediately after the subject stopped pedalling. Blood samples during exercise tests were obtained by an indwelling catheter in a forearm vein at rest prior to exercise and immediately following each test stage. Blood lactate was measured using an enzymatic method (Sigma, St. Louis, USA).

RPE exercise test

Within 3 days following the $VO₂max$ test the perceptual responses to various intensities of exercise were measured using the protocol described by us elsewhere (11) . The four power outputs (25, 50, 75 and 90% $VO₂max$) determined separately for each subject, were used for the RPE exercise test.

For the rating of perceived exertion Borg's scale was used (12). All subjects attended preliminary sessions to establish the low and high rating scale standards.

Statistics

The significance of iron status effect on lactate concentration and perceptual responses was determined by repeated analysis of variance followed by Sheffe contrasts for differences between the means. The values are presented as the means $(\pm SD)$. Analysis of variance was applied to point out the differences between the groups of athletes (NR vs IDR) and groups of controls (NC vs IDC) in lactate concentrations during exercise test.

RESULTS

The means of haematological and work capacity indices are summarized in Table 1. There were no significant differences in the haematological and iron status variables between iron deficient groups (i.e. IDC and IDR) and between groups with normal iron status (i.e. NC and NR). As expected, the trained women had higher $VO₂max$ than the untrained subjects. Mean differences in $VO₂max$ were 44 and 41% between NR and NC groups and between IDR and IDC, respectively. The $VO₂max$ between the groups of runners (NR and IDR) and between the control groups (NC and IDC) were almost the same. 418
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	NC	IDC	NR	IDR
Age [yr]	20.1(2.9)	21.3(2.0)	21.3(2.8)	21.9(3.2)
Height [cm]	164.2(3.6)	165.4(3.8)	167.2(3.9)	166.2(3.6)
Mass [kg]	59.4 (6.8)	61.1(5.3)	54.8(3.3)	56.4(6.8)
$\overline{\text{VO}}_{2\text{max}}$ [ml·kg ⁻¹ ·min ⁻¹]]	$32.3*(5.0)$	$34.7*(3.9)$	57.2(4.8)	58.1(5.5)
Hb $[g \cdot dl^{-1}]$	14.5(0.9)	12.8(1.2)	14.2(0.9)	12.7(1.0)
PCV	0.44(0.04)	0.41(0.03)	0.43(0.04)	0.41(0.05)
RBC $[10^9 \cdot 1^{-1}]$	4.7(1.2)	4.1(1.4)	4.7(0.8)	4.3(1.0)

Table 1. Means $(\pm SD)$ of physical and haematological characteristics of the subjects.

Means that are marked by $*$ are significantly (p < 0.05) different for NR vs NC and IDR vs IDC. .

Mean RPEs at various percentages of $VO₂$ max in the four groups of women are shown in Fig. 1. At work loads above 75% $VO₂$ max mean RPEs of the trained subjects, both from NR and IDR groups were significantly lower than those of the untrained groups (i.e., NC and IDC) by an average of 2.2 points for NR vs NC and 1.3 points for IDR vs IDC. There were no differences in the relation of RPE to work load between groups of runners (i.e., NR vs IDR) and groups of sedentary subjects (i.e, NC vs IDC).

The perceptual responses to exercise at the LT and at the OBLA in the four group of women are compared in Table 2. The LT expressed as $\sqrt[6]{\text{VO}_2}$ max was significantly higher in the athletes, both from NR and IDR groups, than in the untrained women, both from NC and IDC groups. Mean RPEs at LT differed as follow: by 0.9 point for NC vs IDC and by 0.6 point for NR vs IDR. At OBLA mean RPEs differed as follow: by 1.1 points for NC vs IDC ($p < 0.05$) and by 0.7 point for NR vs IDR.

The maximal blood lactate concentrations and lactate thresholds are summarized in Table 3. The lactate threshold occured at 40.2%, 36.8%, 61.1% and 57.5% VO_2 max for NC, IDC, NR and IDR group, respectively. There was no effect of iron status on LT, LT1, LT2 and OBLA when NC and NR groups were compared with IDC and IDR groups, respectively. Low iron

Fig. 1. Means (\pm SD) of RPE at various percentages of $\rm \dot{VO}_2$ max. $\rm \dot{VO}_2$ max $-$ [ml·kg⁻¹·min⁻¹]; RPE – points; the percentages of \overline{VO}_2 max are marked by: x – 25%, \triangle – 50%, \circ – 75%, \bullet - 90%; LT - lactate threshold; OBLA - onset of blood lactate accumulation; \pm SD for the ordinate and the abscissa are given; means that are connected by an $*$ are significantly ($p < 0.05$) different for NR vs NC and for IDR vs IDC.

Table 2. Means (\pm SD) of metabolic [as the % $\rm \dot{VO}_{2max}$] and perceptual responses [as the RPE scale points] to exercise at the LT and the OBLA.

	NC		IDC		NR		IDR	
	LT	OBLA	LT	OBLA	LT	OBLA	LT	OBLA
$VO2$ max RPE	40.2 (6.7) 3.2 (0.5)	77.2 (5.1) $7.3*$ (1.1)	36.8 (7.1) 2.3 (0.4)	75.8 (5.4) 6.2 (1.0)	61.0 (5.2) 3.6 (0.9)	81.9 (4.7) 5.7 (0.9) $\overline{}$	57.5 (6.0) 3.0 (0.5)	80.5 (4.1) 6.4 (1.2)

 $*p<0.05$ for NC vs NR

status was associated with a significant increase $(p<0.05)$ in lactate concentration only in untrained subjects (NC and IDC) which was estimated immediately after the subject stopped pedalling.

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	NC	IDC	NR	IDR
LT	13.0	12.8	35.0	33.4
LT1	(2.7) 20.5	(3.3) 22.0	(3.8) 38.1	(4.2) 36.9
	(3.5)	(4.1)	(4.2)	(3.9)
LT2	20.9	21.8	38.7	37.2
	(4.3)	(4.0)	(4.4)	(5.3)
OBLA	26.8	24.1	46.9	46.8
LAmax	(4.8) $10.8*$	(4.5) 12.3	(5.1) 10.0	(5.3) 11.2

Table 3. Means $(\pm SD)$ of the venous blood lactate parameters expressed \overline{VO}_2 [ml·kg⁻¹·min⁻¹] derived during exercise test to exhaustion. LT, LT1, LT2 and OBLA are 420

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VO₂[ml·kg⁻¹·min⁻¹] derived during exercise to

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NC expressed in ml·kg⁻¹·min⁻¹; LAmax in mmol·l⁻¹.

* p<0.05 for NC vs IDC

DISCUSSION

 (2.1) (1.7) (1.7) (2.0) (1.6)

A pattern of changes in blood lactate concentration during exercise has been shown to be an important determinant of work capacity (14) and to play an important role in the development of fatigue (9). Willis et al. (15) reported that the marked deficit of endurance in iron-deficient sedentary rats was associated with 15-fold higher concentration of blood lactate compared with their iron-sufficient counterparts. It is hypothesized that the iron-deficient rodents exhibit marked impairments in aerobic work capacity by reduction in the iron-containing mitochondrial oxidases of skeletal muscle which limits cellular oxidative ATP production (16, 17). Some studies have indicated α -glycerophosphate changes in muscle cells as the cause of impaired muscle function and lactic acidosis in iron deficiency (18, 19). It has been reported that submaximal exercise resulted in significantly greater blood lactic acid concentration, glucose turnover and metabolic clearance rates (e.g., lactate conversion to glucose via the Cori cycle) in the iron-deficient rats (17, 20).

There are only few studies concerning the effects of decrease in iron pool without overt anemia on exercise capacity in human subjects. Recently, mildly iron-deficient women athletes have been investigated to determine whether iron deficiency without anemia limits physical work capacity. Newhouse et al. (21) studied 40 prelatent iron deficient female endurance runners and noted that the presence of serum ferritin below 20 μ g ^o 1⁻¹ did not significantly reduce physical work capacity. The results of the experiment conducted on 100 female college athletes by Risser et al. (22)

indicated that iron deficiency and its treatment had no significant impact on mood state, but slightly affected subjective assessment of performance. Nilson et al. (23.) studied the effect of iron supplementation upon endurance capacity, VO,max and lactate production in iron-deficient non-anemic athletes. The data showed improvement in $VO₂$ max or time to exhaustion although serum iron levels returned to normal in the previously iron-deficient subjects. Moreover, it has been shown by Celsing et al. (24) that a 4-week period of severely depleted tissue iron stores (serum ferritin about 7 μ g \circ 1⁻¹) did not affect the maximal activities of various human skeletal muscle enzymes.

On the other hand the recent human studies (4, 14, 25) demonstrated a progressive lactic acidosis directly proportional to the severity of the iron deficiency and inversely related to maximal aerobic capacity in human volunteers performing maximal exercise tests. Demello et al. (13) postulate that the lactate threshold (LT) appears to be a crucialpoint for perception of effort during exercise.

In the present study we demonstrated that iron deficient non-anemic subjects, both trained and untrained, did not have significantly higher blood lactate concentration during the exercise test compared to subjects that normal iron status. However, we found that at higher exercise intensity, above 80% VO,max, low iron and ferritin concentration in blood was associated with an increase of venous blood lactate concentration in untrained iron deficient subjects. Our findings are inconsistent with the observations in untrained women. Lukaski et al. (26) studied metabolic responses during a standardized, progressive maximal work capacity test of 11 non-anemic, iron deficient women. They found that reduction of body iron stores without overt anemia affects exercise metabolism by reducing total aerobic energy production, increasing glycolytic metabolism and increasing post exercise lactate concentration.

In conclusion, the results of this study show that the parameters of physical fitness (LT, LT1, LT2 and OBLA) and Borg's scale are not associated with iron status. Low ferritin concentration, about 20 μ g^o 1⁻¹ without any haematological abnormalities, does not affect physical fitness and ratings of perceived exertion in trained or untrained women.

REFERENCES

^{1.} Deuster PA, Kyle SB, Moser PB, Vigersky RA, Singh A, Schomaker EB. Nutritional survey of highly trained women runners. Am J Clin Nutr 1986; 44: 954–962.

^{2.} Fredericson LA, Puhl J, Runyanh WS. Effects of training on indices of iron status of young female cross-country runners. Med Sci Sports Exerc 1983; 15: 271—276.

- 3. Parr RB, Bachman LA, Moss RA. Iron deficiency in female athletes. Physican Sports Med 1984; 12: 81—86.
- Gardner GW, Edgerton VR, Senewiratne B, Bernard RJ, Ohira Y. Physical work capacity on metabolic stress in subjects with iron deficiency anemia. Am J Clin Nutr 1977; 30: 910—917.
- Lukaski HC, Hall CB, Siders WA. Altered metabolic response of iron deficient women during gradet maximal exercise. Eur J Appl Physiol 1991; 63: 140-145.
- 6. Blum SM, Sherman AR, Boileau RA. The effects of fitness-type exercise on iron status in adult women. Am J Clin Nutr 1986; 43: 456—461.
- 7. Beutler E, Harsh BE, Gurney CW. Iron therapy in chronically fatigued nonacademic women: A double blind study. Ann Intern Med 1960; 52: 378—381.
- Tuckner DM, Dandstead HH, Penland JG, Dawson SL, Milne DB. Iron status and brain function: serum ferritin level associated with asymmetries of cortical electrophysiology and cognitive performance. Am J Clin Nutr 1984; 39: $105-110$.
- 9. Robertson RJ, Falkel JF, Drash AD. Effect of blood pH on peripheral and central signals of perceived exertion. Med Sci Sports Exerc 1986; 18: 114—122.
- 10. Hausman K. Eisenvertungsstorungen: Chronische Anamie, sideroblastische Anamie und residulle Endothelsiderose. In Probleme der Erythrozytopoese, Granulozytopoese und des Malignen Melanoms, GW Lohr, R Engelhardt, W Mobius (eds). Springer-Verlag, 1978, pp. $145 - 163$.
- 11. Spodaryk K, Szmatlan U, Berger L. The relationship of plasma ammonia and lactate concentrations to perceived exertion in trained and untrained women. Eur J Appl Physiol 1990; 61: 309—312.
- 12. Borg GAV. Psychophysical bases of perceived exertion. Med Sci Sports Exerc 1982; 14: $377 - 381.$
- 13. Demello JJ, Cureton KJ, Boineau RE, Singh M. Ratings of perceived exertion at the lactate threshold in trained and untrained men and women. Med Sci Sports Exerc 1987; 19: 354— 362.
- 14. Schoene RB, Escourrou P, Robertson HT. Iron repletion decreases maximal exercise lactate concentration in female athletes with minimal iron deficiency anaemia. J Lab Clin Med 1983; 102: 306—312.
- 15. Willis WT, Dallman PR, Brooks GA. Physiological and biochemical correlates of increased work in trained iron-deficient rats. J Appl Physiol 1987; 65: 256-263.
- 16. Davies KJA, Magpie J, Brooks GA, Dallman PR, Packer L. Muscle mitochondrial bioenergetics, oxygen supply and work capacity during dietary iron deficiency and repletion. Am J Physiol 1982; 242: E418—E427.
- 17. Henderson SA, Dallman PR, Brooks GA. Glucose turnover and oxidation are increased in the iron-deficient anaemic rat. Am J Physiol 1986; 250: E414—E421.
- 18. Finch CA, Miller LR, Inamdar AR, Person R, Seiler K, MacKler B. Iron deficiency in the rat. Physiological and biochemical studies of muscle dysfunction. J Clin Ivest 1976; 58: 447 —453.
- 19. Finch CA, Gollnick PD, Hlastala MP, Miller LR, Dillman E, Macller B. Lactid acidosis as a result of iron deficiency. J Clin Invest 1979; 64: 129—137.
- 20. Greeg SG, Kern M, Brooks GA. Acute anemia results in an increased glucose dependence during sustained exercise. J Appl Physiol 1989; 66: 1874–1880.
- 21, Newhouse IJ, Clement DB, Tauton JE, McKenzie DC. The effects of prelatent/latent iron deficiency on physical work capacity. *Med Sci Sports Exerc* 1989; 21:
263–268.
- 22. Risser WL, Lee EJ, Poindexter HB, et al. Iron deficiency in female athletes: its prevalence and impact on performance. Med Sci Sports Exerc 1988; 20: 116—121.
- 23. Nilson K, Schoene RB, Robertson HD, Escourron P, Smith NJ. The effect of iron repletion on exercise-induced lactate production in minimally iron-deficient subjects. Med Sci Sports Exerc 1981; 13: 92—96.
- 24. Celsing F, Blomstrand E, Werner P, Piloted P, Ekblom B. Effect of iron deficiency in endurance and muscle enzyme activity in man. Med Sci Sports Exerc 1986; 18: 156—161.
- 25. Rowland TW. Iron deficiency in the young athlete. Med Clin North Am 1990; 37: 1153— 1163.

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