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The relationship between lactic acid and work load: a measure for endurance capacity or an indicator of carbohydrate deficiency?

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Summary. The influence of low and high carbohydrate diets on the relationship between blood lactate concentration ([Lac]) and work load (WL) in incremental exercise tests (cycle ergometer) and endurance tests was evaluated in trained subjects. The relationship between relative work load (WL_{rel}) and [Lac] in arterialized blood was compared in untrained subjects (UT) and trained male athletes (TR) after 2 days without training while consuming a high carbohydrate diet (HCD). In both groups [Lac] of 2 mmol·l^{-1} was reached at about 60% [(mean ± SD) UT 57.7% ± 6%, TR 62.7% ± 3.8%] and 4 mmol·l^{-1} at about 75% (UT 75.2% ± 3.6%, TR 77.8 ± 2.2) of the maximal work load (WL_{max}). In eight cyclists the relationship between [Lac] and WL was not influenced by a 13-day training camp; however, heart rate was lower after the training camp. During their normal training programme, trained subjects had high relative work loads at their [Lac] thresholds, but after an HCD combined with an interruption of the training of 3 days, the relationship between [Lac] and WL_{rel} was the same as in UT. In six TR a low carbohydrate diet (LCD) combined with training led to high absolute (WL_{abs}) and WL_{rel} at [Lac] of 2 and 4 mmol·l^{-1}; an HCD combined with 3 days without training led to low WL_{abs} and WL_{rel} at the same [Lac] and to higher WL_{max}. In spite of the apparently lower endurance capacities TR were able to work significantly longer after HCD than after LCD (23 ± 10.5 min and 49 ± 16.2 min, respectively) at 65% of their WL_{max}. The variability of the relationship between [Lac] and WL following the dietary regimes leads to the conclusion that the "typical" [Lac] versus WL curve of endurance TR may result from a permanent glycogen deficiency.

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Key words: Diet — Endurance capacity — Glycogen — Lactate — Threshold

Introduction

It has been known for a long time that trained subjects (TR) can perform at higher fractions of their maximal work loads (WL_{max}) compared to untrained subjects (UT) in endurance exercise. This so-called higher endurance capacity in TR (Costill et al. 1973; Åstrand and Rodahl 1977) is thought to be the consequence of an increase in anaerobic metabolism only at higher percentages of WL_{max} compared to UT. Therefore, the endurance capacity is often "measured" by the relationship between blood lactate concentration ([Lac]) and work load (WL) (Stegmann et al. 1981; Davis 1985; Kindermann 1985; Heck et al. 1985; Wassermann 1986; Mader and Heck 1986). The prerequisites are that [Lac] should be lower at the same % WL_{max} and that the increase should begin at higher relative work loads (WL_{rel}) in TR compared to UT. Such a relationship has been found repeatedly (Hermansen and Stensvold 1972; Gollnick et al. 1986; Jacobs 1986).

On the other hand, it has been shown that the availability of substrates is an important determinant in the relationship between [Lac] and WL (Klausen and Sjøgaard 1980; Ivy et al. 1981; Heigenhauser et al. 1983; Yoshida 1984a; Neary et al. 1985). In particular, if the muscle glycogen is decreased by low carbohydrate diets (LCD) during training, [Lac] is lower in blood and muscle tissue at comparable absolute work loads (WL_{abs}) (Klausen and Sjøgaard 1980; Jacobs 1981), because muscle glycogen is the primary substrate for lactate production (Hagberg et al. 1982). Reduced

muscle glycogen content was shown by Costill and Miller (1980) during intensive daily training, even with a high carbohydrate diet (HCD). The aim of this study was to investigate whether the relationship between [Lac] and WL, especially WL_{rel}, can be characterized for different endurance capacities or if the relationship is an indicator of a carbohydrate deficit.

Subjects and methods

Subjects in varying states of physical fitness and different dietary and training conditions were compared to test the influence of carbohydrate availability on the relationship between [Lac] and WL.

Following informed consent, those subjects assigned to special diets were instructed in the use of a nutritive substance table to enable them to prepare diets consisting mainly of fat and protein (LCD) or with a high content of carbohydrate (HCD). The diets were not standardized.

In all experiments heart rate (HR) and [Lac] (Boehringer, Test Kit no. 124842, Mannheim) in arterialized blood were determined. Blood was taken either from a hyperaemized earlobe or a heated forearm vein. The individual anaerobic threshold (IAT) (Stegmann et al. 1981) was determined in every experiment.

Series I. After 2 days without training combined with HCD eight trained male cyclists (TR) (age: 19±3 years; weight: 74.5±6.8 kg; WL_{max}: 5.5 W·kg^{-1}±0.5) were tested with incremental work loads at a pedalling frequency of 95 rev·min^{-1}. Their own bicycles were used on a treadmill, using the method of Causin and Braumann (1987). The initial work load of 150 W (corresponding to about 36% of the WL_{max}) was increased by 16.6 W every minute until the subject was exhausted (100% relative performance). The HR and [Lac] were determined during the test and after 7 min of recovery (work load: 150 W). The relationship between [Lac] and WL_{rel} was compared to that of UT (UT; age: 29±6 years; weight: 75.8±7.3; WL_{max}: 4.0±0.6 W·kg^{-1}) who performed the same test on a cycle ergometer (Meditron Typ 799, Buchholz) with a pedalling frequency of 95 rev·min^{-1}. The initial load for the UT was about 33% of WL_{max}. To achieve similar conditions for the distribution of lactate in the whole body in TR and UT, it is necessary to produce tests of comparable duration and comparable increases in load relative to maximal performance. Therefore, the WL in UT was increased every minute with smaller WL steps so that the duration of the test was comparable to that of the TR cyclists (17.6±3.2 min UT, compared to 16.3±2.7 min TR). The WL during the recovery period was the same as the initial WL.

Series II. Eight cyclists from the Lower Saxony competition team [age: 19±3 years; weight: 73.3±5.2 kg; WL_{max} see Table 1 (II)] were tested by the method used in series I before and after a 13-day training camp to improve their endurance capacity. Before both tests the subjects interrupted their training for 2 days and ate an HCD.

Series III. Six TR (five highly trained cyclists and one highly trained long-distance runner; age: 24±5 years; weight: 73.7±8.3 kg) were tested twice at the end of their competition season, before and after a diet which is known to increase the state of muscle glycogen stores (Bergstroem et al. 1967). The first test was performed without any special preparation during their normal training programme. After this test, the subjects had an LCD combined with training (not standardized) for 3 days, followed by 3 days with an HCD without training. The second test was performed on the 7th day. Both tests took place in the early afternoon. A cycle ergometer (Meditron Typ 799, Buchholz) was used. The initial WL was 100 W. The WL was increased by 16.6 W every minute until the subject was exhausted. The pedalling frequency was 95 rev·min^{-1} as in series I.

Series IV. Six endurance TR (four cyclists, one long-distance runner and one triathlete; age 26±4 years: weight 73.7±8.6 kg; three of these subjects were in their recovery season) underwent the same dietary regime as in series III. The subjects were tested on the same days, and also on the last day of the LCD (day 3). Furthermore, these subjects performed an endurance test in the afternoon of the test days at about 65% of their maximal performance until exhaustion. The meals between the incremental test and the endurance test were the same and were almost free of carbohydrate on the 3 test days.

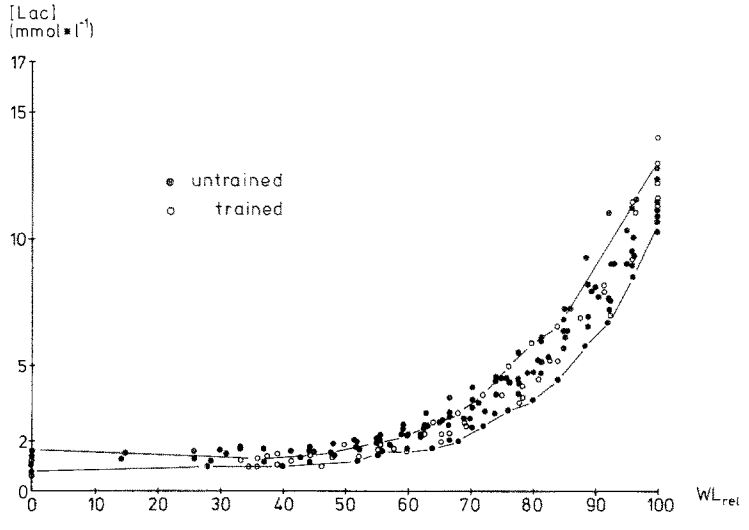

Fig. 1. Relationship between blood lactate concentration [*Lac*] in arterialized blood and the relative work load (WL_{rel}) of untrained subjects ($n=9$) and trained subjects ($n=8$) after a carbohydrate-rich diet combined with a 2-day training break (series I). The *left curve* relates to a cyclist (5.4 W·kg^{-1}), the *right curve* to an untrained subject (3.4 W·kg^{-1})

Table 1. Results of the incremental exercise tests of series I and II. *I* Comparison between untrained subjects (UT) and trained subjects (TR) after 2 days on a carbohydrate-rich diet and without training. *II* Cyclists (CY) before (N) and after a training camp (TC). Numbers in parentheses indicate *n*. Values are means ± SD. Threshold values are given as a percentage of the maximal work load (WL_{max}) [IAT = individual anaerobic threshold (Stegmann et al. 1981)]. Blood lactate concentration ([Lac]) and heart rate (HR_{max}) values are for the end of the incremental test

		WL_{max} ($W \cdot kg^{-1}$)	2 $mmol \cdot l^{-1}$ (% WL_{max})	4 $mmol \cdot l^{-1}$ (% WL_{max})	IAT (% WL_{max})	[Lac] ($mmol \cdot l^{-1}$)	HR_{max} ($l \cdot min^{-1}$)
	UT (9)	4.0 ± 0.6	58 ± 6	75 ± 4	72 ± 6 (7)	11.2 ± 0.9	188 ± 11
I		$p < 0.001$	n.s.	n.s.	$p < 0.02$	$p < 0.05$	n.s.
	TR (8)	5.5 ± 0.5	63 ± 4	78 ± 2	81 ± 4 (6)	12.1 ± 1.0	191 ± 12
	CY N	5.3 ± 0.4	65 ± 5	80 ± 4 (7)	81 ± 4	11.3 ± 2.1	191 ± 7
II	(8)	n.s.	n.s.	n.s.	n.s.	n.s.	$p < 0.005$
	CY TC	5.4 ± 0.3	67 ± 4	82 ± 3	81 ± 4	11.2 ± 2.2	180 ± 6

In series I UT were tested against CY and in series II CY before the training camp (N) against after the training camp (TC)

Statistics. Values are presented as means ± standard deviation. Student's *t*-tests for paired and unpaired samples were used to determine significant differences between mean values.

Results

Series I

The relationship between [Lac] and WL_{rel} is shown in Fig. 1. No significant differences between TR after the 2 days of recovery and UT can be seen. The upper curve is that of a cyclist (TR) with a WL_{max} of 5.4 $W \cdot kg^{-1}$. The lower curve is that of a UT with a WL_{max} of 3.4 $W \cdot kg^{-1}$. There were no significant differences in either the WL_{rel} at [Lac] of 2 $mmol \cdot l^{-1}$ or 4 $mmol \cdot l^{-1}$. The maximal [Lac] at the end of exercise was significantly higher in TR than in UT. Maximal HR were not significantly different between both groups [Table 1 (I)].

Series II

The relationship between [Lac] and WL_{rel} of eight cyclists was unaffected by a 13-day training camp (1850 km) to improve endurance capacity (Fig. 2). However, the increase in HR was significantly smaller ($p < 0.05$) during the incremental test in all subjects after the training camp (Fig. 3).

Series III

The highly trained had high WL_{abs} (292 ± 48 W) and WL_{rel} (75%) at 2 $mmol \cdot l^{-1}$ [Lac] and high

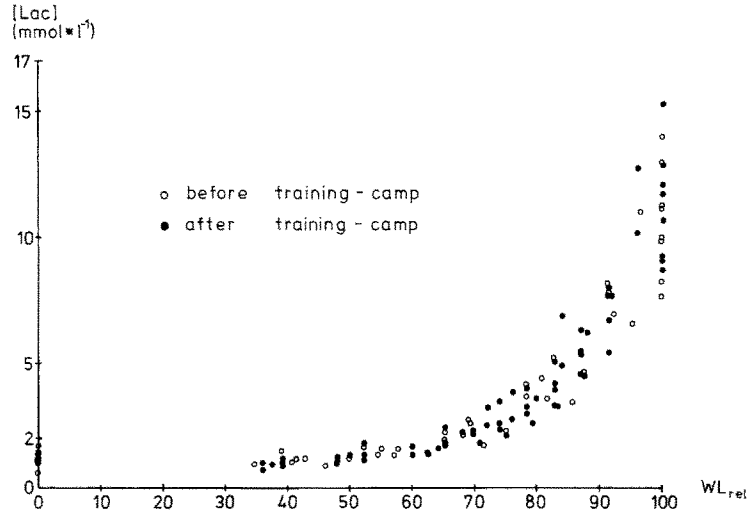

Fig. 2. Blood lactate concentration [*Lac*] — relative work load (WL_{rel}) relationship in cyclists (series II) before and after a training camp ($n = 8$)

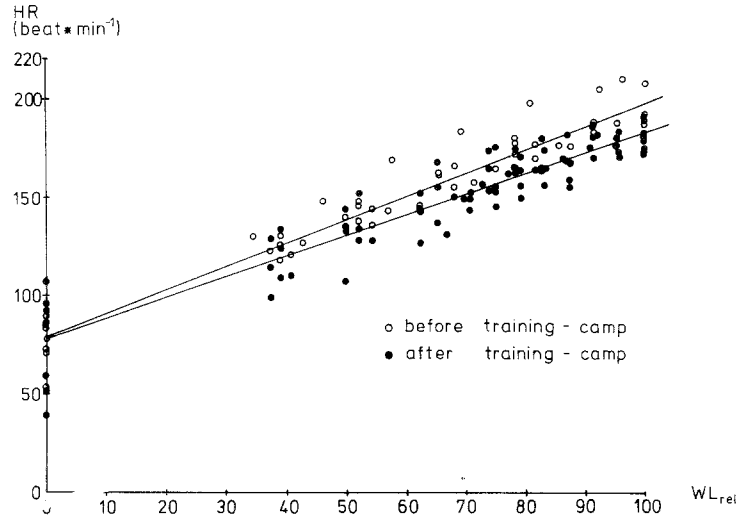

Fig. 3. Relationship between heart rate (*HR*) and relative work load (WL_{rel}) before and after a training camp (series II, $n=8$). Regression equations are $Y=79+1.18X$ before the camp and $Y=78+1.06X$ after the camp. The slopes are significantly different ($p<0.05$)

WL_{abs} (340±41) and WL_{rel} (87%) at 4 mmol·l^{-1} [Lac] when tested during their normal training programme. These values are suggestive of a high endurance capacity. After an HCD the WL_{abs} and WL_{rel} at 2 mmol·l^{-1} (244±31 W, and 59% respectively) and at 4 mmol·l^{-1} (319±24 W, and 77% respectively) were low. All subjects, however, reached higher WL_{max} and higher [Lac] at the end of exercise. The HR at the end of the tests was not significantly different between the tests [Table 2 (III)]. The IAT according to Stegmann et al. (1981) could not be determined in two subjects after an HCD: in one subject [Lac] decreased immediately after termination of the exercise, and in the other [Lac] in the 7th minute of recovery was still higher than at the end of exercise. The IAT of the remaining four subjects was 317±62 W (78%) on the normal day and 333±53 W (78%) after HCD.

Series IV

The relationships between [Lac] and WL_{abs} and WL_{rel} of one subject on the three diets are shown in Fig. 4a and b. With LCD the curves were shifted to the right, whereas an HCD resulted in a leftward shift from the normal curve. Group data are presented in Table 2 (IV). The WL_{abs} and WL_{rel} at fixed [Lac] were highest with an LCD, suggesting higher endurance capacities. However,

Table 2. Results of series III and IV. In both series $n=6$. Threshold values are given in absolute work loads (WL_{abs}) and as a percentage of WL_{max}. [Lac] and HR are for the end of the incremental test. (For definitions see Table 1)

		WL_{max} (W)	2 mmol·l^{-1}	4 mmol·l^{-1} (W_{abs} and % WL_{max})	IAT	[Lac] (mmol·l^{-1})	HR_{max} (l·min^{-1})
III	Normal	392±38	292±48 75%	340±41 87%	306±52 78%	8.1±2.0	184±14
		$p<0.005$	$p<0.005$	$p<0.02$		$p<0.005$	n.s.
	HCD	414±36	244±31 59%	319±24 77%	333±53 78% ($n=4$)	12.3±1.2	182±8
IV	Normal	392±23	256±44 65%	307±34 78%	289±23 74%	11.8±5.1	194±8
		n.s.	$p<0.02$	$p<0.02$	n.s.	$p<0.03$	$p<0.01$
	LCD	350±30	284±32 81%	324±34 91%	275±28 79%	6.6±2.9	182±12
		$p<0.002$	$p<0.001$	$p<0.004$	$p<0.02$	$p<0.001$	n.s.
	HCD	392±33	232±35 59%	292±30 75%	295±32 75%	13.6±3.9	187±7

The absolute values were used for Student's *t*-tests. In series IV normal and low carbohydrate diets (LCD) were tested against high carbohydrate diets (HCD)

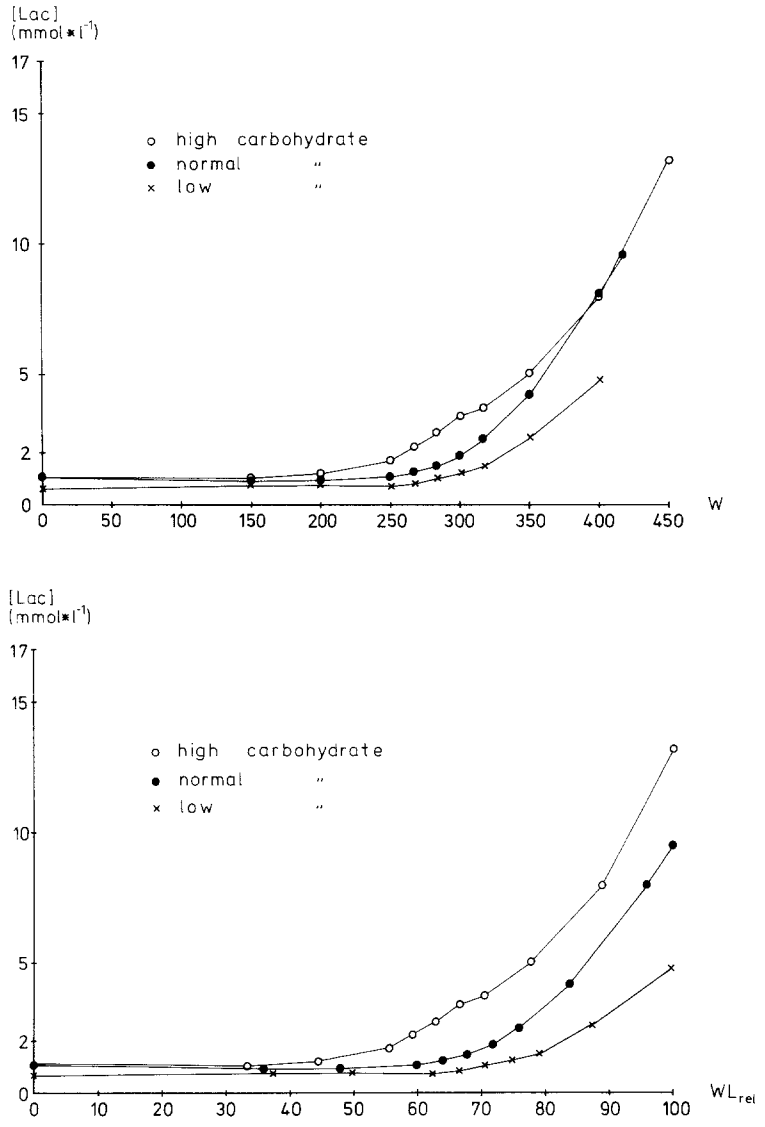

Fig. 4. a, b Blood lactate concentration [*Lac*] — work load curve. **a** Absolute WL (WL_{abs}), **b** relative WL (WL_{rel}) after different dietary and training regimes in one subject

the WL_{max} were higher and the performance times in the endurance tests longer (26 ± 12 min) after an HCD compared to an LCD (Table 3). It should be noted that no carbohydrate intake was allowed between the test in the morning and the endurance test. Mean [Lac] during and at the end of the endurance tests were higher after HCD (Table 3, Fig. 5).

Table 3. Results of the endurance tests with endurance-trained athletes ($n = 6$). The mean WL was 266 ± 12 W corresponding to 67% ± 3% of WL_{max}. (For definitions see Table 1)

	Performance time (min)	$[Lac]_{max}$ ($mmol \cdot l^{-1}$)	$[Lac]_{end}$ ($mmol \cdot l^{-1}$)	HR_{end} ($l \cdot min^{-1}$)
Normal	35 ± 12	4.4 ± 2.2	3.7 ± 2.0	182 ± 16
	$p < 0.01$	n.s.	$p < 0.05$	n.s.
LCD	23 ± 11	3.2 ± 1.6 ($n = 5$)	2.3 ± 1.3 ($n = 5$)	163 ± 22
	$p < 0.002$	$p < 0.02$	$p < 0.01$	$p < 0.05$
HCD	49 ± 16	5.6 ± 2.9	5.4 ± 2.9	178 ± 10

p indicates significantly different from HCD

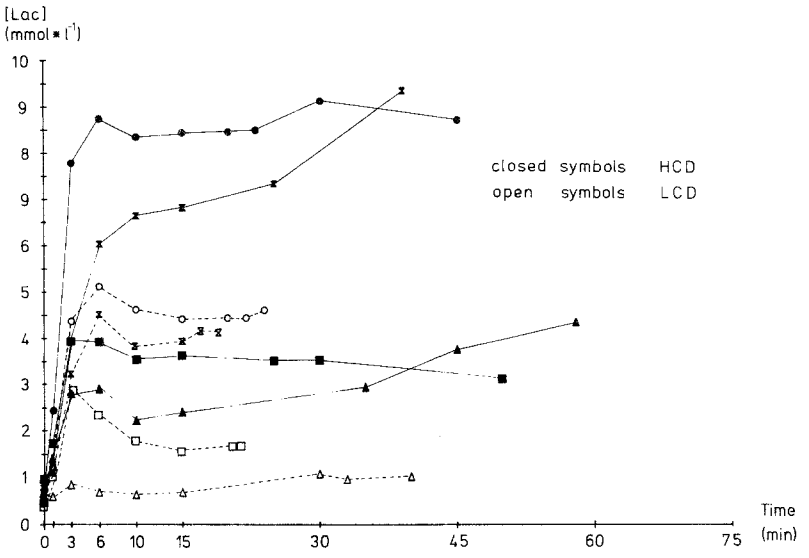

Fig. 5. Blood lactate concentration [*Lac*] in arterialized blood during endurance tests at 65% of maximal work loads after high and low carbohydrate diets (*HCD* and *LCD* respectively). The various symbols indicate only four subjects for the sake of clarity

Discussion

The relationship between [Lac] and WL of highly trained subjects was investigated under different dietary and training regimes, which were known to alter the muscular glycogen store (Bergstroem et al. 1967; Heigenhauser et al. 1983), and compared with that of UT. Unquestionably, the relationship between [Lac] and WL_{abs} is dependent on the physical fitness of an athlete. However, [Lac] has to be lower at the same percentage of WL_{max} in endurance trained athletes if a conclusion is to be drawn about the endurance capacity. After a recovery period of 2 days without any physical activity combined with an HCD the relationship between [Lac] and WL_{rel} was almost identical for TR and UT when the durations of the tests were in the same range. The influence of test duration on [Lac] and thus on thresholds was shown by Hughes et al. (1982), Yoshida (1984b), and Heck et al. (1985).

Reproducibility of thresholds determined by fixed concentrations, breaking points, and the increase above the resting value

The relationship between [Lac] and WL is dependent on the carbohydrate content of the food and on the intensity of training. All tested methods to determine aerobic and anaerobic thresholds show distinct differences under differing conditions. If fixed concentrations (see Tables), breaking points (Beaver et al. 1985) (Fig. 6), or an increase above the resting concentration (Aunola

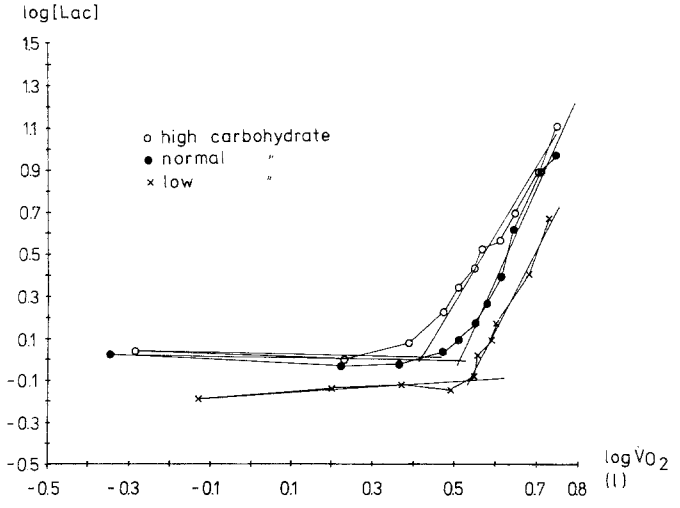

Fig. 6. Reproducibility of thresholds determined according to Beaver et al. (1985) under different dietary and training conditions. *Lines* are drawn by eye. [*Lac*], blood lactate concentration; $\dot{V}_{O_2}$, oxygen consumption

and Rusko 1984) (Fig. 4a) are used as criteria, intensive training on the days before the test results in high WL at these thresholds (series III). The same is valid for LCD.

The AT determined by ventilatory parameters do not show the same variability (Ivy et al. 1981; Heigenhauser et al. 1983; Neary et al. 1985). These AT are shifted to lower power outputs with glycogen depletion (Hughes et al. 1982), indicating that there is no causal relationship between [Lac] in arterial blood and ventilation. A simple cause and effect relationship does not even exist during exercise between muscle venous (Heigenhauser et al. 1983) or arterial acidosis (Hagberg et al. 1982; Busse and Maassen 1987) and ventilation. In contrast, ventilation is correlated with plasma $[K^+]$ (Tibes 1977; Busse and Maassen 1987), which in turn does not seem to be dependent on acidosis during exercise (Busse and Maassen 1989). Therefore, AT determined by ventilatory parameters do not simply reflect the increase of anaerobic metabolism.

Under the HCD regimes the WL_{rel} at the fixed [Lac] are very low; usually this would lead to the conclusion that the endurance capacity would be low. In fact, the performance times at about 65% of WL_{max} were significantly longer. In consequence, low WL at the thresholds were connected with high endurance times. The fact that the [Lac]-WL relationship was not altered after the training camp is also contrary to the opinion that it is a measure of endurance capacity. We were not able to test the endurance performance in series II, but, according to the coach, the cyclists were in much better condition after the training camp. The lower HR during the incremental tests may be used as an indicator of this positive effect of the camp.

Reproducibility of IAT

The IAT (Stegmann et al. 1981) is also dependent on the muscular glycogen availability. But in contrast to the thresholds at fixed [Lac] the WL_{abs} increased slightly with HCD. Under these conditions the IAT was similar to the WL at 4 $mmol \cdot l^{-1}$ (Table 2), and [Lac] also increased to about 4 $mmol \cdot l^{-1}$. The lower [Lac] at the IAT of highly trained TR compared to UT (Stegmann et al. 1981) seems also to be the consequence of a reduced glycogen availability.

Reproducible IAT values require that the "overshoot" and subsequent decrease of [Lac] after exercise changes in proportion to [Lac] at the end of exercise (Stegmann et al. 1981). The time-course of [Lac] during recovery was not dependent on [Lac] at the end of exercise (Fig. 7), therefore the cause of increased IAT after HCD appears to be the higher [Lac] at the end of the exercise.

Thresholds, [Lac], and performance times during endurance tests

The [Lac] in arterialized blood during an endurance test is also dependent on the availability of carbohydrates. After HCD, [Lac] was higher at the same WL, and the performance time also increased despite higher [Lac]. Similar increases in the performance times after HCD were shown by Saltin (1973) and Bergstroem et al. (1967). Performance times with higher WL_{rel} are also increased (unpublished results). In summary, the endurance capacities for exercise of up to 1-h duration are enhanced after HCD.

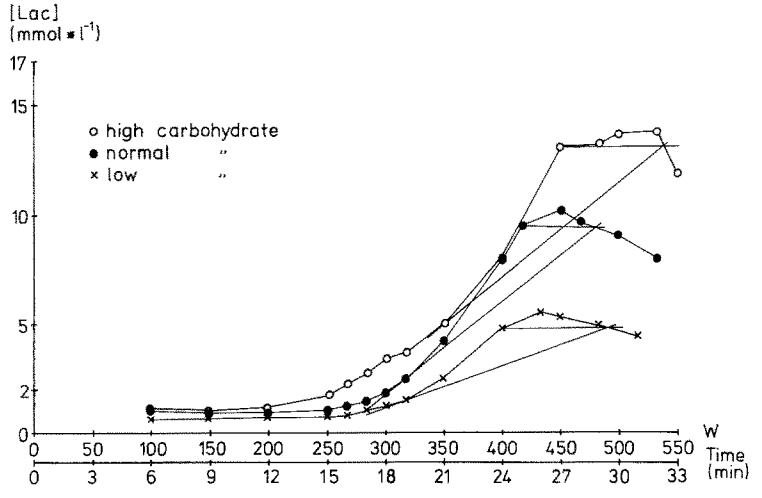

Fig. 7. Determination of the individual anaerobic thresholds (*IAT*) according to Stegmann et al. (1981) under the different conditions. [*Lac*], blood lactate concentration

WL_{max} and maximal [Lac]

An increase in performance time with increasing [Lac] after HCD, was also associated with higher WL_{max} in the incremental tests. In series III, [Lac] was about 4 $mmol \cdot l^{-1}$ higher on the 2nd test day (HCD) and the WL_{max} was increased by about 20 W. In series IV, [Lac] was increased two-fold, WL_{max} rose by about 40 W and $\dot{V}_{O_{2max}}$ by about 300 ml (4.45 l ± 0.55 l to 4.74 l ± 0.53 l, $p < 0.05$, measured with the closed system Magna Test 710; Meditron, Buchholz) at comparable HR. Apparently high [Lac] do not compensate for an oxygen deficit, but are accompanied by an increase in $\dot{V}_{O_2}$. Therefore, a high [Lac] seems to be necessary to attain the highest $\dot{V}_{O_{2max}}$. This agrees with the hypothesis of Connett et al. (1985), who suggested that muscle lactate is of minor importance concerning direct energy supply, but of great importance as a store of substrate and as a buffer in connection with the redox state of the cytosol. This improved redox potential would improve the mitochondrial aerobic ATP production and therefore $\dot{V}_{O_{2max}}$ and WL_{max} would be significantly higher when [Lac] is high.

Causes for the increase of [Lac]

Independent of the role of lactate, the causes of [Lac] increase remain open. A deficit in muscle oxygen supply does not seem to be the primary factor, especially at moderate work loads (Connett et al. 1983, 1984). It is unlikely that higher [Lac] at submaximal WL resulted from a lesser oxygen supply because lactic acid is itself a vasodilator, thereby increasing oxygen delivery, and increased [Lac] also enhances oxygen dissociation from haemoglobin (Braumann et al. 1982). According to Donovan and Brooks (1983), the elimination of lactate from the blood is the main factor determining [Lac]. From the present experiments it cannot be judged whether high production or low elimination of lactate was the main cause of elevated [Lac] during exercise. However, the nearly identical relationships between [Lac] and WL_{rel} of TR and UT indicate that at the same WL_{rel} the production and the elimination rates of lactate are not different between these two groups under these conditions. Therefore, it is unlikely that the lactate elimination rate in TR is an important determinant of endurance capacity.

The effect of carbohydrate availability on [Lac] is well known. Muscle glycogen depletion reduces [Lac], whereas the availability of carbohydrate increases [Lac] during exercise (Bergstroem et al. 1967; Klausen and Sjøgaard 1980; Jacobs 1981; Heigenhauser et al. 1983). The LCD combined with training causes a decrease in muscular glycogen content (Klausen and Sjøgaard 1980; Jacobs 1981; Heigenhauser et al. 1983), which is the primary source for the circulating lactate (Hagberg et al. 1982). Costill and Miller (1980) showed that the glycogen stores of the muscle decrease during 2 h of intensive daily training even with HCD. The time taken for a complete replenishment of the stores is 2–3 days (Hultmann 1967; Piehl et al. 1974), if the subjects are physically inactive. However, exercise of the lowest intensity seems to result in a breakdown of glycogen and impedes the synthesis of glycogen (Bonen et al. 1985). Therefore, it can be supposed that highly trained athletes, who usually exercise more than 2 h/day, have partially depleted glycogen stores. By means of the HCD, combined with 2 days without physical activity, the stores may be filled. As a consequence the relationship between [Lac] and WL_{rel} is shifted to the left and becomes almost the same as in UT. Therefore, increased lactate production is probably the cause for the higher [Lac]. Assuming filled muscle glycogen stores, the increased glycogenolytic flux with exercise leads to an increase in intramuscular glucose-6-phosphate concentration (Klausen and Sjøgaard 1980) and to an increased lactate production. At the same level of exercise, an artificially increased glycogenolysis was shown to enhance lactate production by Stainsby (1986). The assumption that the increase of [Lac] is mainly due to a higher substrate supply is further supported by Helyar et al. (1988), who found significant changes in [Lac], but no changes in the maximal activity of the key enzymes of either glycogenolysis, glycolysis, citric acid cycle, or β-oxidation, after a 5-day training programme. The fact that the relationship between [Lac] and WL_{rel} of TR is identical with that of UT after HCD would indicate that the glycogen turnover is similar in TR and UT at the same WL_{rel} (the same percentage of $\dot{V}_{O_{2max}}$) if the stores are sufficiently filled, as reported by Saltin (1973).

In addition, the availability of free fatty acids might play a role in [Lac] during exercise. If the plasma concentration of free fatty acid [FFA] is high the glycogen breakdown may be reduced and therefore [Lac] would be low (Ivy et al. 1981; Rennie et al. 1976). On the other hand, an influence of plasma [FFA] on [Lac] could not be demonstrated by Quirion et al. (1988) and Yoshida (1984a). The reason for this discrepancy might be the higher

[FFA] or the additional heparin infusion in the study of Ivy et al. (1981).

With HCD the [FFA] is lower and with LCD [FFA] higher than under normal conditions (Yoshida 1984a). Under the HCD conditions low plasma [FFA] are associated with longer performance times for high work loads of about 2 min duration (unpublished results), for moderate WL up to 1 h duration, and to high WL_{max} during incremental tests. The latter is in contrast to the findings of Ivy et al. (1981), who showed an unchanged WL_{max}, but in their glucose infusion experiments the muscular glycogen stores were probably unaltered; and the high insulin levels following their infusions may have reduced the maximal work capacity. The question arises whether filled muscle glycogen stores and in addition an activated fat metabolism is beneficial for enhanced endurance capacity.

Conclusions

1. It is not possible to draw any conclusions from the relationship between WL and [Lac] about the endurance capacity of an athlete.
2. Subjects after complete recovery from exercise have a nearly constant relationship between percentage of WL_{max} and [Lac], regardless of the aerobic capacity (similar to the HR and WL relationship).
3. No prognosis about [Lac] during an endurance test can be deduced from an incremental WL test. Furthermore, [Lac] during these endurance tests seems to be dependent on the availability of muscle glycogen.
4. In athletes after complete recovery from exercise low threshold values are associated with longer performance times at 65% WL_{max}.
5. The high thresholds related to the WL_{max} of highly trained TR are possibly due to a continuous carbohydrate deficiency. The same is true for the low [Lac] at IAT in highly trained TR.

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