

Use of blood lactate concentration as a marker of training status

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Abstract

The recent development of portable blood lactate analysers has made it relatively easy to test blood lactate concentration in the field. This paper discusses the validity and accuracy of measuring blood lactate concentration as a marker of training status or exercise intensity and examines the assumptions upon which the above practice is based.

The mechanisms responsible for blood lactate accumulation according to different theories are discussed, followed by a review of the literature regarding the measurement, tracking and interpretation of blood lactate concentration.

The use of blood lactate concentration to monitor either training status or intensity is complicated by the inaccuracy of portable lactate analysers, multiple confounding factors affecting blood lactate concentration such as carbohydrate depletion, mode of exercise, ambient temperature, muscle damage and overtraining and difficulty in interpreting the results. The relationship between changes in blood lactate concentration following training and subsequent performance in competition has not been convincingly established. Therefore it may be concluded that changes in blood lactate concentration should be interpreted with caution as the changes do not track training status or exercise intensity with sufficient precision to have a practical application.

Introduction

Blood lactate concentration during exercise is often used as a marker of exercise intensity and training status.^{6,37} This is due in part to the theoretical discourse on lactate and the 'lactate threshold' and the recent availability of less expensive and portable lactate analysers.

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The interpretation of blood lactate concentration is based on three assumptions: (i) blood lactate levels increase in a predictable and repeatable mode as exercise intensity increases; (ii) blood lactate concentration at a controlled submaximal exercise intensity decreases with increasing fitness; and (iii) training at 'threshold' intensity, which coincides with the 'lactate threshold', will result in the greatest improvement in fitness.^{17,20}

The validity of these assumptions has been questioned as the existence of the 'lactate threshold' has also been challenged.¹¹ Blood lactate concentrations are influenced by numerous factors, which affect the accuracy of repeated blood lactate sampling.¹⁰ Furthermore, the relationship between blood lactate concentration and changes in training status has also been challenged in subjects who are overtrained.²¹

The aim of this paper is to examine the assumptions upon which blood lactate concentrations are interpreted and to question whether measuring blood lactate concentration during exercise has any practical relevance in monitoring training status or exercise intensity. The approach will be firstly to discuss the principles of lactate metabolism and then to review the literature on the relationship between blood lactate concentration, exercise intensity and training status.

The principles of lactate metabolism

Lactate is a product of oxygen-independent metabolism of glycogen via the glycolytic pathway. For each molecule of glucose metabolised, two molecules of either lactate or pyruvate are formed. At high exercise intensity lactate accumulates in the blood. At present there are two prevailing theories explaining why this occurs.

The 'anaerobic threshold' theory

Traditionally it has been assumed that blood lactate and ventilation increase as a linear function of exercise intensity, until a turnpoint or 'lactate threshold' is reached. This observation was first made by Hill *et al.*¹⁸ in 1923. These studies formed the basis of the traditional theory of oxygen debt and lactate accumulation above the 'lactate threshold'. Above the 'threshold', blood lactate concentration and ventilation are assumed to increase rapidly as a result of the sudden onset of oxygen-independent glycolysis.³⁹

According to the 'anaerobic threshold' theory the change from oxygen-dependent to oxygen-independent metabolism is

presumed to be as a result of a limit in the supply of oxygen to working muscles at higher exercise intensities. Increases in cardiac output are insufficient to meet the demands for oxygen in working muscles and the production of lactate from oxygen-independent metabolism is presumed to then cause a metabolic acidosis.¹⁸

The protons responsible for the acidosis have traditionally been believed to arise from the dissociation of lactic acid into lactate and H⁺ ions¹⁹ and are buffered by the bicarbonate buffer system. Bicarbonate is then dissociated into H₂O and CO₂. A respiratory compensation for the 'lactic acidosis' leads to an exponential rise in ventilation (V_E) and expiration of this carbon dioxide. The ventilatory threshold is therefore thought to be linked directly to 'anaerobic metabolism' and the 'lactate threshold'. This theory suggests that above the 'lactate threshold' a shift away from oxygen-dependent metabolism would attenuate any further increases in VO₂. This results in a plateau of VO₂ at increasing exercise intensities above 'threshold' levels.²³

The alternative theory

The presence of an anaerobic threshold has been challenged in the past.¹¹ Firstly, VO₂ has been shown to continue to increase as a function of workload, even at high intensities.³¹ This suggests a continued increase in oxygen-dependent metabolism despite higher exercise intensity. Secondly, it is possible to dissociate the lactate and ventilatory thresholds.¹⁴ Thirdly, it has been shown that muscle and blood lactate concentrations increase as a continuous curvilinear function of increasing exercise intensity, rather than as a threshold.¹¹ Furthermore, subjects with McArdles syndrome, a glycogen storage disease, have minimal increases in blood lactate concentration and an absent lactate threshold response to increasing exercise intensity despite a normal respiratory threshold response.²⁷

A plausible theory is that the accumulation of muscle and blood lactate is the result of the exponential increase in carbohydrate metabolism that occurs at higher workloads.¹¹ Metabolism of glucose via the oxygen-dependent and oxygen-independent pathways increase concurrently as a result of the increased flux through glycolytic pathways. There is also a continued increase in oxidation of pyruvate and lactate from glycolysis in the mitochondria of working muscles as well as a redistribution of lactate to non-working muscles and gluconeogenic tissues.²⁸

The progressive increase in blood lactate appearance (R_a) is matched at lower workloads by a progressive increase in blood lactate disappearance (R_d). This match is gradually lost at higher workloads, with R_a eventually exceeding R_d, resulting in an accumulation of blood lactate as a curvilinear function of increasing exercise intensity.^{11,12}

The accumulation of protons in the sarcoplasm from the breakdown of ATP to ADP shifts the lactate dehydrogenase and MCT4 equilibrium towards the production of blood lactate.¹¹ The protons generated by the hydrolysis of ATP are reconsumed during ATP synthesis by oxidative phosphoryla-

tion or creatine phosphate hydrolysis. During glycogenolysis this does not occur. These protons arise irrespective of whether the pyruvate or lactate formed from glycogenolysis are delivered to the mitochondria. The metabolic acidosis is therefore a function of the increase in carbohydrate metabolism. The metabolic acidosis is compensated for by an increase in ventilation that is indirectly related to the accumulation of blood lactate.¹⁵ This explains the dissociation between lactate and ventilatory thresholds observed in some studies.^{14,27}

Blood lactate testing and training

According to the traditional theory, the exercise intensity coinciding with the 'lactate threshold' is well defined' and is an accurate marker of training status. It has been suggested that training at and above the 'lactate threshold' will result in adaptations that reduce the concentration of blood lactate during subsequent submaximal and maximal exercise.⁴⁰ A common assumption is that training at the intensity coinciding with the 'lactate threshold' will cause an improvement of lactate clearance or a decrease in lactate production at submaximal workloads.⁴⁰ This theory has promoted the testing of blood lactate concentration in the field as a marker of training intensity and training status. This is based on the assumption that blood lactate concentrations are repeatable at controlled workloads and that the concentration of blood lactate decreases as training status improves. There are however multiple factors that may reduce the accuracy and interpretation of blood lactate concentration during monitoring of training status and intensity.

Factors affecting interpretation of steady state blood lactate concentration

1. Rate of change in exercise intensity. Blood lactate concentration is influenced not only by the exercise intensity but also by the duration of exercise prior to measurement and the rate of change in exercise intensity.²⁸ Submaximal exercise at a fixed intensity results in an initial increase in blood lactate concentration, followed by a reduction in blood lactate concentration after rates of carbohydrate oxidation in working and non-working muscles increase. A rapid increase in exercise intensity will therefore result in higher blood lactate concentrations than a situation where the increase to the same controlled workload is more gradual.²⁸

2. Carbohydrate depletion. Muscle glycogen content is associated with blood lactate concentration during exercise.³⁸ Carbohydrate loading before and carbohydrate depletion after exercise have been shown to increase and decrease lactate concentration respectively at any given subsequent exercise intensity.²¹ Prolonged exercise results in decreased blood lactate concentrations. This occurs because the reduction in muscle glycogen during prolonged exercise decreases glycogenolysis and therefore the rate of blood lactate accumulation.³⁸

3. Mode of exercise. Lactate production and oxidative lac-

tate clearance, and therefore the steady state blood lactate concentration, are affected by the mass of the recruited skeletal muscle during exercise, the pattern of intramuscular co-ordination and fibre type recruitment.⁵

Isolated upper-body exercise results in lower blood lactate concentration than exercise involving multiple limbs or the whole body.^{10,23,36,42} These differences are primarily due to the differences in the mass of the recruited muscles.

4. Precision of monitoring blood lactate concentration.

The reduction in submaximal blood lactate concentration, secondary to improvements in training status, may be of smaller magnitude than the fluctuations in measurement due to lactate monitor reliability.³³ The reliability and accuracy of portable blood lactate analysers and the range of blood lactate concentration tested are illustrated (Table I).

At exercise intensity coinciding with the lactate threshold (maximal lactate steady state) an improvement in training status from pre-season to pre-competition is likely to result in a reduction in blood lactate concentration of approximately 0.7 mmol/l.³⁵ The changes in blood lactate concentration at threshold intensity may therefore be of a smaller magnitude than the level of error inherent in portable blood lactate analysers (up to 0.35 mmol/l at 5 mmol/l or 0.7 mmol/l at 10 mmol/l.¹³ This lack of precision confuses the interpretation of threshold blood lactate concentration measured in the field with a lactate analyser.

Changes in steady state blood lactate concentration of greater than 3 mmol/l only occur as exercise intensity increases above threshold values and closer to maximal intensity. To measure these changes would necessitate repeated bouts of exercise at high intensity, making the monitoring of training status an aversive test and therefore impractical.²¹

Although the reliability of portable lactate monitors has been measured in controlled laboratory settings,^{9,33} the practical reliability is questionable in field testing. Blood lactate values may differ depending on whether the blood is sampled from the finger capillary, earlobe capillary or forearm vein.¹⁰ Of the three available sampling sites, the ear capillary may perhaps be the preferred site as it is less affected by lactate released from nearby working muscles.¹⁰ Another potential problem with blood lactate testing in the field is the high concentration of lactate in sweat and the difficulty in cleaning the skin prick test sites.

5. Ambient temperature. A warm ambient temperature increases muscle glycogenolysis during exercise in subjects who are not heat acclimatised. This causes an increase in blood lactate concentration.³² Blood lactate concentration was over two-fold higher at fatigue during exercise at 40°C (36.7 mmol/l) compared with exercise at 3°C (16.3 mmol/l).³²

With acclimatisation, the difference in blood lactate concentrations at fatigue in hot conditions is attenuated.²⁴ Blood lactate concentrations during exercise should therefore be interpreted with caution in extreme environmental conditions when athletes are not acclimatised.

6. Overtraining syndromes. Repeated bouts of high-intensity exercise have been shown to reduce both submaximal and maximal blood lactate concentrations.²¹ These changes may be incorrectly interpreted as an improvement in training status. The reduced blood lactate concentrations seen in over-trained subjects may be as a result of carbohydrate depletion, resulting in reduced flux through glycolytic pathways at all intensities. This may be as a result of a decrease in catecholamines during overreaching and in overtraining syndromes. Catecholamines increase glycogenolysis via increased intracellular cyclic AMP. Both peripheral and central β -adrenergic receptors are downregulated during overtraining syndromes.²⁶ The cyclic AMP-mediated increases in glycolysis and glycogenolysis during exercise are therefore blunted during overtraining, even when muscle glycogen stores are normal. Reduced rates of glycolysis or glycogenolysis result in decreased blood lactate concentrations.²² Epinephrine and β -receptor interaction have also been shown to increase lactate uptake by contracting skeletal muscle during high blood lactate concentrations, thereby increasing rates of lactate disappearance.⁶

7. Muscle damage. Some studies have shown increased blood lactate concentrations during exercise with damaged, painful muscles. Gleeson *et al.*¹⁶ found that blood lactate concentration in athletes was increased during exercise following a bout of eccentric exercise eliciting delayed-onset muscle soreness. Peak blood lactate concentrations increased to 12.6 mmol/l from 10.9 mmol/l. This increase in blood lactate concentration is perhaps due to increased glycogenolysis³² as a result of increased type 2 muscle fibre recruitment¹⁶ or increased rate of efflux of lactate from the

TABLE I. Reliability and accuracy of portable blood lactate analysers and the range of blood lactate concentration tested

Test unit	Gold standard	Reliability	Limits of agreement (mmol/l) (2 SD)	Range tested (mmol/l)	Ref. no.
Accusport	Microstat LM3	SEM = 0.5 mmol/l, R = 0.99	-2.2 - 1.9	1 - 14	9
Accusport	Microstat LM3	$r = 0.99$	0.5 - 3.0	1 - 13	33
Accusport	Kodak Ektachem	$r = 0.99$	-2.34 - 2.34	1 - 24	13
Lactate Pro	ABL 700	$r = 0.99$	-1.83 - 2.09	1 - 18	34
Lactate Pro	YSI 2300	$r = 0.99$	-0.98 - 2.02	1 - 18	34
Lactate Pro	Accusport	$r = 0.99$	-2.14 - 2.02	1 - 18	34

R = intraclass correlation coefficient; r = correlation coefficient; SEM = standard error of the mean; SD = standard deviation.

muscle fibres secondary to increased membrane permeability.¹⁶ Therefore the use of blood lactate concentration as a marker of training status and intensity in athletes suffering from delayed-onset muscle soreness may be inappropriate.

Interpretation of blood lactate concentrations

Blood lactate concentration increases as a curvilinear function of increasing workloads¹¹. As a result of this relationship the change in blood lactate concentration from basal concentration to 80% of VO_{2max} is approximately 3.5 mmol/l. At higher workloads the blood lactate concentration increases rapidly to a maximum concentration which can exceed 14 mmol/l.

A reduction in blood lactate concentration as a result of prior exercise bouts may be due to overtraining rather than to improvements in training status. This may be interpreted incorrectly and a diagnosis of overtraining may be missed. A consequence of this interpretation might be that the athlete does not reduce the training load, exacerbating the symptoms of overtraining. The reduction of submaximal blood lactate concentration due to overtraining is also associated with a lower maximal blood lactate concentration. However, tests to elicit maximal blood lactate concentrations are exhaustive, aversive and impractical from the perspective of monitoring training. This limits the use of maximal blood lactate measurement as a diagnostic adjunct.²¹

Tracking blood lactate concentration and exercise performance

The exercise intensity coinciding with the onset of blood lactate accumulation (OBLA)^{2,6,7} has been used as a predictor of exercise performance and has correlated well with ranking performance in cross-country skiers.²⁵ Lactate threshold values have also been used to predict performance accurately in runners.²⁰

The competition performance of elite-level marathon runners has however been found to be independent of their OBLA values.⁸ Wiswell *et al.*⁴¹ have also shown a concurrent decline in lactate threshold values and running performance with ageing in both male and female athletes.

Myburgh *et al.*³⁰ have found no relationship between plasma lactate concentrations for self-selected maximal efforts of 1 hours' duration.³⁰ Other studies have also shown a poor relationship between changes in submaximal blood lactate concentrations and changes in performance during competition.^{3,4,29,35}

Although 4 mmol/l is most often considered to be the onset of blood lactate accumulation, there is actually great variation among individual subjects (range 2 - 8 mmol/l).⁶ The criteria defining the lactate threshold are also not well defined and the abovementioned studies used differing criteria.

In conclusion, studies show conflicting findings with regard

to lactate as a predictor of exercise performance. Further research as well as more accurate definition of the lactate threshold are necessary to clarify this issue.

Conclusions and practical guidelines

The use of blood lactate concentration as a marker of training status and training intensity is confounded by complications in accuracy and difficulty in interpreting the results.

The rate of change in exercise intensity, the mode of exercise, carbohydrate intake, sampling procedures, and ambient temperature are all confounding factors that reduce the repeatability of blood lactate sampling. The reduction in blood lactate concentration during overtraining syndromes also make changes in blood lactate concentration with training difficult to interpret.

A change in training status from early season to pre-competition results in a change in submaximal blood lactate concentration of about 0.7 mmol/l, or less, coinciding with the intensity at lactate threshold.^{17,35} Portable lactate analysers have been shown to differ from laboratory testing apparatus by 1 mmol/l - 2 mmol/l. The reliability of portable lactate analysers, although good, may still result in inaccuracies of up to 7%. The accuracy of portable lactate analysers may therefore confound testing over periods of short duration, as the changes in submaximal blood lactate concentrations with changes in training status may be of a similar or smaller magnitude than the error inherent in portable lactate analysers. The changes in submaximal blood lactate concentration induced by training and the correlation of these changes with competition performance has also not been convincingly established. Therefore it may be concluded that changes in blood lactate concentration should be interpreted with caution.

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