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Use of Blood Lactate Measurements for Prediction of Exercise Performance and for Control of Training

Recommendations for Long-Distance Running

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Summary

Time over a distance, i.e. speed, is the reference for performance for all events whose rules are based on locomotion in different mechanical constraints. A certain power output has to be maintained during a distance or over time. The energy requirements and metabolic support for optimal performance are functions of the length of the race and the intensity at which it is completed. However, despite the complexity of the regulation of lactate metabolism, blood lactate measurements can be used by coaches for prediction of exercise performance.

The anaerobic threshold, commonly defined as the exercise intensity, speed or fraction of maximal oxygen uptake ($\dot{V}O_{2max}$) at a fixed blood lactate level or at a maximal lactate steady-state (MLSS), has been accepted as a measure of the endurance. The blood lactate threshold, expressed as a fraction of the velocity associated with $\dot{V}O_{2max}$, depends on the relationship between velocity and oxygen uptake ($\dot{V}O_2$). The measurement of the post-competition blood lactate in short

events (lasting 1 to 2 minutes) has been found to be related to the performance in events (400 to 800m in running). Blood lactate levels can be used to assist with determining training exercise intensity. However, to interpret the training effect on the blood lactate profile, the athlete's nutritional state and exercise protocol have also to be controlled. Moreover, improvement of fractional utilisation of $\dot{V}O_{2max}$ at the MLSS has to be considered among all discriminating factors of the performance, such as the velocity associated with $\dot{V}O_{2max}$.

The complexity of the regulation of lactate metabolism and the different influences on blood lactate level (e.g. glycogen storage) were recently presented in 2 excellent reviews.[1,2]

Despite controversy over the physiological basis for blood lactate accumulation (at its maximal or steady-state) and, more importantly, over the fact that blood lactate level does not reflect production in active skeletal muscles, most exercise laboratories regularly use this as a basis for assessing the metabolic profile of their sportsmen and women.[3] In a recent review, Bishop and Martino[4] focused on research addressing key considerations in the measuring and interpretation of blood lactate in exercise and post-exercise (recovery) lactate values. They emphasised that lactate values differ according to where the blood has been sampled from. The most common sampling sites are the ear lobe or finger tip which yield arterialised capillary blood.[4] Furthermore, Bishop and Martino[4] recalled that the environment and glycogen stores may influence the lactate curve. This must be considered when interpreting the effects of training and will be discussed in section 5.

Ten years after the review articles of Jacobs,[1] and Walsh and Banister,[2] this paper examines the use of blood lactate measurements for prediction of exercise performance from evaluation of physical capacities to assist with the preparation of training in different sports (short-, middle- and long-distance events). Is the lactate threshold (LT) at the same percentage of maximal oxygen uptake ($\dot{V}O_{2max}$) for all types of running or does it depend on the relationship between the energy cost and the velocity? Does this energy cost increase for swimming and rowing[5-7] or remain constant until velocity at $\dot{V}O_{2max}$ for running?[8]

1. Definition of the Blood Lactate Level

Time over a distance, i.e. speed, is the reference for performance for all events whose rules are based on locomotion within different mechanical constraints. A certain power output has to be maintained over distance or time done. Jones and Ersham[9] recall that the concept of an anaerobic threshold of blood lactate probably owes most to Margaria, whose work, extending over 30 years, developed the theory that exercise was accomplished by the use of aerobic processes up to $\dot{V}O_{2max}$, above which additional demands were met by anaerobic glycolysis. In fact, Margaria et al.[8] considered that the lactate accumulation occurred only above the minimal velocity which elicits $\dot{V}O_{2max}$. Before that, they considered that the athlete was evenly in the aerobic condition when considering the lactate accumulation at an intensity below $\dot{V}O_{2max}$.

Recently, it has been commonly accepted that the metabolic reaction to longer lasting dynamic exercise can be divided into 2 stages:[10]

(i) A load which can be sustained at steady-state for a long time. After about 2 to 5 minutes, a state of overall oxidative energy supply is established which is characterised by a balance of lactate production and elimination at a low level.[11] The characteristics of such endurance exercise is that lipid metabolism is a significant energy supply at slow and moderate speeds. Exercise is limited by the increase of the internal temperature associated with dehydration prevented by supplementation of water and substrate.

(ii) A load during which an additional net formation and accumulation of lactate is necessary to maintain power output. Such a load leads to exhaustion and fatigue through the disturbance of the

internal biochemical environment of the working muscles and whole body caused by a high or maximal acidosis. Depending on the necessary amount of additional lactate formation, the accumulation of lactate limits performance time from 30 seconds to about 15 minutes,[10] or probably less. This is because the time to exhaustion at the minimal velocity which elicits $\dot{V}O_{2max}$ averages 6 minutes 30 seconds[11] and is not correlated with the blood lactate accumulated during this all-out test at this velocity.[12]

Between these 2 metabolic states there is a transition stage called 'anaerobic threshold or lactate threshold', according to the different terminologies (table I). This means that there is a shift from a solely oxidative to an additional glycolytic energy supply. Lactic acid production would be due to the fact that activation of glycolysis is more rapid than activation of the oxidative phosphorylation. This results in a transient elevation of NADH in the cytoplasm and net lactic acid production.[23] This is indicated by a steep nonlinear increase of blood lactate in relation to power output and time.[10] The lactate accumulation with the increase of power output can be attributed to disparities in the rate of lactate production and removal, even if for these intensities under $\dot{V}O_{2max}$, lactate production is not related to an oxygen deficit but rather to the increase of the glycolysis flux. Skinner and McLellan[24] proposed an hypothetical model which clarified the different terminologies used for the anaerobic threshold (by the gas exchange, ventilatory and lactate approach).

Brooks et al.[25] underline that lactic acid is produced constantly, not just during hard exercise. In fact, lactic acid may be the most dynamic metabolite produced during exercise; lactate turnover exceeds that of any other metabolite yet studied. The constancy of blood lactic acid level during rest or exercise means that the entry into and removal of lactate from the blood are in balance. Increasing blood lactate levels indicate that entry exceeds re-

Table I. A classification of the different terminologies that exist in the literature to define specific changes in the exercise blood lactate response

Blood lactate value (mmol/L)	Definition and designation	Protocol	Reference
Baseline + 1	Onset of plasma lactate accumulation: the $\dot{V}O_2$ observed during the incremental exercise with a blood lactate concentration that is 1 mmol/L above the baseline blood lactate level	Discontinuous incremental test with 8 stages, of 10 min	13
2.2	Maximal steady-state: the oxygen, heart rate and/or treadmill velocity at which plasma lactate level was 2.2 mmol/L	2 discontinuous stages, of 10 or 15 min	14, 15
2.5	Lactate threshold: the exercise intensity that elicits a blood lactate level of 2.5 mmol/L after 10 min of exercise	Discontinuous incremental test with stages of 10 min	16
4	Anaerobic threshold: the $\dot{V}O_2$ or velocity associated with a blood lactate concentration of 4 mmol/L	Continuous incremental test with stages of 3 min	17, 18
4	Onset of blood lactate accumulation	Continuous incremental test with stages of 4 min	19
2-7	Individual anaerobic threshold: metabolic rate where the increase of blood lactate is maximal and equal to the rate of diffusion of lactate from the exercising muscle	Continuous incremental test with stages of 4 min with measurement of the lactate time course after the test	20
3.5-5	Lactate threshold: the starting point of an accelerated lactate accumulation around 4 mmol/L and expressed in $\%\dot{V}O_{2max}$	Continuous incremental test with stages of 3 min	21
2.2-6.8	Maximal steady-state of blood lactate level: the exercise intensity ($\dot{W}_{CL}$) which produces the maximal steady-state of blood lactate level	2 submaximal intensities (60-65% and 75-80% of $\dot{V}O_{2max}$) of 20 min carried out on the same day and separated by 40 min of complete rest	22

Abbreviations: $\dot{V}O_2$ = oxygen uptake; $\dot{V}O_{2max}$ = maximal oxygen uptake.

moval; declining levels indicate the opposite. Radiotracer studies[25] have shown the turnover of lactic acid during exercise to be several times greater for a given blood lactate level during exercise than during rest. Similarly, for a given blood lactate level, blood lactate removal is several times greater in trained than in untrained individuals. Several factors appear to be responsible for the lactate inflection point during graded exercise: contraction itself stimulates glycogenolysis and lactate production. In addition, hormone-mediated accelerations in glycogenolysis and glycolysis, recruitment of fast-glycolytic muscle fibres, and a redistribution of blood flow from lactate-removing gluconeogenic tissues to lactate-producing glycolytic tissues cause blood lactate to rise during exercise protocols that call for continually increasing power output.[25]

2. Use of Blood Lactate Measurements According to Distance Events

2.1 Long-Distance Events

2.1.1 Blood Lactate Threshold and Aerobic Capability

Daniels,[26] stated that $\dot{V}O_{2max}$ (i.e. running economy, according to Conley and Krahenbuhl[27]) and $\%\dot{V}O_{2max}$ had to be regarded as important in determining success. Costill[28] reported that a very slight increase in lactic acid was found during 2 hours of running requiring between 55 and 67% of $\dot{V}O_{2max}$. Moreover, he noticed that highly trained distance runners were capable of utilising more than 90% of their $\dot{V}O_{2max}$. He concluded that training for competitive distance running appeared to permit a greater fraction of that capacity to be utilised without accumulation of blood lactate.[28] Coyle et al.[29] have shown that a competitive cyclist with a high LT 82% $\dot{V}O_{2max}$, had time to fatigue during exercise at 88% $\dot{V}O_{2max}$ more than 2-fold longer compared with a group whose LT was only 66% $\dot{V}O_{2max}$ (60.8 ± 3.1 *vs* 29.1 ± 5.0 minutes).

2.1.2 Lactate Threshold and Performance Over Long-Distance

Performance in long-distance running is related to several physiological variables, such as $\dot{V}O_{2max}$, the anaerobic threshold, running economy, anaerobic capacity, and the velocity associated with $\dot{V}O_{2max}$ (V_{amax}).[30-37] In fact, over 92% of the variance in performance was related to the $\%\dot{V}O_{2max}$ at LT and muscle capillary density.[38] The current concept is that LT determines (or is related to) the fraction of $\dot{V}O_{2max}$ that can be sustained by an individual in events lasting beyond 10 to 15 minutes, and that this value interacts with running economy to determine the actual running speed in competition.[16,29-39] The running speed at LT appears to be highly predictive of distance running performance at events including the 10 000m run and the marathon.[40] For trained and untrained women, LT and fixed blood lactate levels (2.0, 2.5 and 4.0 mmol/L) accurately predicted a 3000 or 3200m trial.[33,41]

Hagberg and Coyle[16] have shown that the racewalking velocity at the blood LT during incremental exercise performed on a treadmill was highly correlated to 20km racewalking pace (112 $\pm$ 6 minutes) and predicted performance times to within 0.6%. The 4 mmol/L $\dot{V}O_{2max}$ and power output performed on a cycle ergometer by 11 male swimmers, was correlated with a swimming performance, i.e. a speed (m/sec) over 1000m.[42]

Recently, Helgerud[43] evaluated performance-matched male and female marathon runners and reported that while men had a higher $\dot{V}O_{2max}$ and anaerobic threshold, women who had higher weekly training distance demonstrated a superior running economy (lower gross oxygen cost of running) and a higher exercise intensity (as $\%\dot{V}O_{2max}$) during the race. Therefore, the amount of training has to be considered when predicting performance[44] because the fractional utilisation of $\dot{V}O_{2max}$ during the race over long-distance depends on the kilometres covered every week, even if the anaerobic threshold remains unchanged. The ability to use lipid metabolism and the low mass fat induced by the number of kilometres are parameters which are more important for events which last for more than an hour, e.g. the marathon.[45] The $\dot{V}O_{2max}$ during a

race of 6.5 minutes in duration was related to the slow-twitch fibre content of the muscles and the anaerobic threshold and was inversely related to maximal blood lactate level.[46]

2.1.3 Lactate Threshold and Endurance Training

Increase of the LT (in absolute value of $\dot{V}O_{2max}$, also referred to as $\%\dot{V}O_{2max}$) has been found after endurance training.[47] Longitudinal changes in anaerobic threshold and distance running performances were assessed with a 4.5 month interval between pre-, mid- and post-tests in a relatively homogeneous sample of 21 male trained endurance runners. There was a higher relationship between the distance-running performances and anaerobic threshold-related attributes held up consistently over the 9-month training period. When the relationship between the absolute amount of change in the $\dot{V}O_{2max}$ anaerobic threshold and the absolute amount of change in distance running performance was evaluated, significant correlations were found in several different time periods. Moreover, Gaesser and Poole[48] showed that the LT increased during the first 2 to 3 weeks of training (6 days/week, 30 min/session at 70 to 80% of pretraining $\dot{V}O_{2max}$) in 6 untrained individuals. However, endurance performance (i.e. the total work output in a 90-minute maximal ergocycle test to exhaustion) responses to training have been shown as being largely genotype-dependant.[49,50]

Henriksson[51] reported lower respiratory quotient values and a lower release of lactate during submaximal exercise following endurance training. This was attributed to an increased muscle respiratory capacity estimated by muscle succinate dehydrogenase activity. Furthermore, it has been demonstrated that slow-twitch fibres have a higher respiratory capacity than fast-twitch fibres.[52] Moreover, Ivy et al.[53] have reported that the percentage of slow-twitch fibres was related to the absolute and relative LTs, and suggested that the ratio of slow-twitch to fast-twitch fibres may exert a genetic influence over LT and possibly control the range in which the relative threshold can shift, even if it did not prove a cause-and-effect relationship. Therefore the LT might be a high predictor of the aerobic ability before training.

2.2 Short-Distance Events

2.2.1 The Maximal Amount of Energy Released by the Anaerobic Process

Evaluation of the maximal amount of energy released by anaerobic processes (J/kg) is based on the following observations and hypotheses:

(i) In supramaximal exercise, the energy requirement per unit of time is greater than the maximal amount of energy released per unit of time by aerobic oxidations (i.e. the power output associated with $\dot{V}O_{2max}$). As a consequence, a fraction of the adenosine triphosphate (ATP) hydrolysed is resynthesised at the expense of creatine phosphate splitting and by anaerobic glycolysis at a rate proportional to energy output. It is assumed that the maximal energy output from lactic acid levels is determined by maximal lactic acid levels that can be accumulated in muscles before a critical value of intracellular pH is reached.[54] On this basis, it can be considered that the maximal value of anaerobic capacity is achieved at the end of supramaximal exercise performed until exhaustion.[54]

(ii) A constant fraction of the lactic acid accumulated in skeletal muscles diffuses in blood, causing an increase in blood lactate level towards a peak level reached 5 to 9 minutes after cessation of the exercise.[55,56] The maximum net increase of plasma lactate level, defined as peak plasma level minus resting plasma lactate level, is proportional to the net lactate accumulation in the whole body. It is assumed that the amount of lactate metabolised or leaving the muscles without increasing the maximum net increase of plasma lactate level before the peak blood lactate is reached, is negligible in comparison with the lactate appearing in blood during that period.[55]

(iii) The energy released by anaerobic process has been indirectly evaluated from the relationship between oxygen deficit and the maximum net increase of plasma lactate level, and was calculated as being equal to 260 J/kg.[57]

Camus and Thys[58] evaluated maximal anaerobic capacity, i.e. the maximal amount of energy released by the anaerobic process from maximal increase of plasma lactate, in a study of 8 men of different physical fitness submitted to supramaximal runs of various intensity performed until time to exhaustion (tlim). Using the relationship: maximum net increase of plasma lactate level/tlim = $f(E - \dot{V}O_{2max})$, and assuming that 1ml of oxygen = 20.9J (respiratory expiratory ratio = 0.96), these authors found maximal anaerobic capacity to range from 910 to 1330 J/kg. These values were only 8% lower than the oxygen deficit calculated by Medbo et al.[59]

In very short events, i.e. the 100 and 200m runs completed in 10.7 ± 0.2 and 22.6 ± 0.5 seconds, Hautier et al.[60] showed that blood lactate levels were high 3 minutes after the 100 and 200m run (8.5 ± 0.8 and 10.3 ± 0.8 mmol/L, respectively), but these were not correlated with performances. However, over 200m, blood lactate level was correlated with the velocity sustained over the last 165m. The difference between the blood lactate measured at the end of the 2 races was negatively correlated to the difference in velocities sustained.

2.2.2 The Blood Lactate Profile

Ryan et al.,[61] established the blood lactate versus swimming velocity profile in women swimmers during the collegiate season from September to March. These swimmers performed 3 × 500 yards (457m) with 30 seconds rest between bouts at intensities selected to elicit blood lactate in the range of 2 to 15 mmol/L. Their finding indicated that increases in the velocity eliciting a blood lactate of 4 mmol/L (identified in the profile of each swimmer) occurred early in the season in response to an increase in training metres to moderate levels. Increase in training distance to over 54 000 yards (49 377m) per week during the remainder of the training season did not alter the swimming velocity eliciting a blood lactate of 4 mmol/L. However, the question of the best method to assert the minimal individual load necessary to move the blood lactate profile remains. In fact, interpretation of blood lactate profile may be affected by varying muscle glycogen levels.[62]

Blood lactate levels of 20 to 25 mmol/L are often observed following competition in athletes in events requiring 1 to 2 minutes for completion.[2] Athletes in more prolonged competitive events may have blood lactate levels of 10 to 20 mmol/L following competition. Foster et al.[62] argue that a range of 20 to 30% of maximal blood lactate levels in the range of 12 to 20 mmol/L would be consistent with the 4 mmol/L reference standard. However, a previous study using a maximal lactate at steady-state (MLSS) showed that the MLSS value ranged from 2.2 to 6.8 mmol/L and might depend on the depletion glycogen state and the ratio of slow and fast twitch fibres.[22]

3. Influence of Models of Exercise on Blood Lactate Measurements: Incremental *vs* Constant Load Exercises

3.1 Incremental Model of Exercise

3.1.1 Blood Lactate Response to Incremental Exercise

LT can be evaluated by determining blood lactate level using graded load protocols or single steps of constant load of long duration (10 minutes and over) and nearly complete recovery between the steps (table I). Moreover, a given blood lactate level can be researched (2, 2.5 and 4 mmol/L) or an individual value (baseline value + 1 mmol/L, a value taking into account a MLSS) either by the incremental or discontinuous long-step protocols (table I).

Stegmann et al.[63] defined the individual anaerobic threshold (IAT) as the metabolic rate where the elimination of blood lactate during exercise is both maximal and equal to the rate of diffusion of lactate into the blood. The calculation of IAT involves the measurement of blood lactate during a stepwise increasing exercise test followed by a passive recovery period. The time during recovery when the blood lactate level equals the value measured at the end of highest exercise intensity is assumed to represent a time when the elimination of lactate is maximal and equal to the rate of diffusion.

A line drawn from this time during the recovery period that intercepts the exercise blood lactate curve will produce a time coordinate and, therefore, a power output that defines the IAT. McLellan et al.,[64] demonstrated that the IAT was not affected by an active recovery period.

3.1.2 Heart Rate and Blood Lactate Response to Incremental Exercise

In 1973, Keul[65] examined the relationship between heart rate and blood lactate during bicycle ergometer exercise with progressively increased work loads every 6 minutes. He showed that an increase in blood lactate level is first observed at a heart rate greater than 120 beats/min.[65]

Later, Conconi et al.[66] examined the relationship between running speed and heart rate in 210 runners. On a 400m track, the athletes ran continuously from an initial velocity of 12 to 14 km/h to submaximal velocities varying according to the athlete's capability. In all athletes examined, a deflection from expected linearity of the running speed-heart rate relationship was observed at submaximal running speed. They stated that this velocity where the deflection for the heart rate *vs* running speed and the velocity at the anaerobic threshold (breakpoint in the blood lactate response to exercise), was coincident in 10 runners. The same group of researchers reported a similar coincidence for swimmers, canoeists, cross-country skiers, cyclists, roller skaters, rowers, and ice skaters.[67,68] The so-called 'Conconi test' has been programmed in the software of the heart frequency meters. However, the protocols of determination of the velocity of the heart rate deflection and of anaerobic threshold were completely different. The former consisted of an increase of 0.5 km/h every 200m, heart rate being registered at the end of every fraction, and the latter was composed of 6 stages of 1200m (3 were run at a velocity under the velocity of the heart rate deflection and 3 above it) with 15 minutes of jogging between. Moreover, further studies were not able to find either the velocity of the heart rate deflection or the coincidence of it and the anaerobic threshold.[69-71]

Snyder et al.[72] proposed a more simple approach to estimating the MLSS (defined as the highest intensity in which blood lactate level increases <1.0 mmol/L between the tenth and thirtieth minute of exercise) by using the heart rate. Cross validation of the model for predicting MLSS was undertaken during 21 running exercises and 45 cycling exercises using a separate group. Using the MLSS upper 95% confidence interval model, 84% and 76% of those exercise periods, involving cyclists and runners respectively, were correctly predicted. The authors concluded that these simple heart rate models may predict MLSS with enough accuracy to be of use when direct blood lactate measurements are not available.[72]

3.1.3 Blood Lactate and a Category-Ratio Perceived Exertion Scale Response to Incremental Exercise

The category scale, commonly referred to as the Borg Scale,[73] has been widely used to study perception of exertion (rating of perceived exertion: RPE) during exercise in laboratory, occupational and clinical settings. This scale was developed by Borg so that the perceptual ratings increased linearly with power output and heart rate for exercise on a cycle ergometer.[74] Then, Borg et al.[73] developed a new psychophysical scale that perceptual ratings would increase as a positively accelerating function.[75] These authors concluded that the ratings from the category-ratio scale corresponded very well with the lactate accumulation during a maximal incremental test performed on cycle ergometer by 10 physically active men. The RPE value at the lactate fixed value of 2, 2.5 and 4 mmol/L were 11 ± 2, 14.5 ± 1.8 and 16.5 ± 2.3 mmol/L, respectively.[76] Seip et al.[76] consider that if blood lactate level is employed as an exercise intensity prescription, RPE-overall may provide a viable exercise prescription tool in pacing for running. Moreover, Steed et al.[77] showed that the relationship between RPE and LT remained constant during a constant 30-minute stage performed at the velocities corresponding to LT, 2.5 and 4.0 mmol/L blood lactate. This scale could therefore be used for training and to regulate the pace during a race,

especially for the events lasting 1 hour, such as the cycling world record.

3.2 Constant Output Model of Exercise and the Maximal Lactate at Steady-State

Olbrecht et al.[78] noticed a strong correlation between the swimming speed expressed as a percentage of the velocity at a blood lactate level of 4 mmol/L and the lactate level on a 30-minute swimming exercise that allowed assessment of the intensity during prolonged exercise using a percentage of the velocity at 4 mmol/L. However, the duration and the size of the intensity increments have been found to influence the value of the anaerobic threshold.[79] Thus, the intensity involving the MLSS has often been overestimated, especially in highly trained endurance athletes using the anaerobic threshold as a part of their training.[20,80-82] Mognoni et al.[80] have examined physiological responses during prolonged exercise at the power output corresponding to the blood lactate level of 4 mmol/L. Out of 34 moderately fit men, 20 were exhausted at a mean time of 38.2 minutes instead of 60 minutes scheduled and with a final blood lactate level equal to 5.3 ± 2.3 mmol/L. To achieve a real MLSS, it was necessary to have 4 or 5 prolonged exercise sessions of up to 30 minutes duration performed at exercise intensities between 50 and 90% of $\dot{V}O_{2max}$.[14,15,82,83]

Stegmann and Kindermann[20] have advocated an IAT determined from the changes in blood lactate both during and after an incremental exercise test. According to this model, the IAT has represented the maximal intensity where production and elimination of lactate are in equilibrium, so that higher exercise intensities lead to progressively increasing blood lactate.[20] Urhausen[83] has shown that IAT could be regarded as a reliable estimation of the range of MLSS although 100% of IAT did not necessarily represent exactly the MLSS in all individuals.

A recent study[22] has validated a protocol providing an immediate estimate of the exercise intensity corresponding to the MLSS using only 2 submaximal intensities lasting 20 minutes each and separated by only 40 minutes of rest in long-distance athletes (fig. 1).

The duration, intensity and recovery time of the 2 tests had no effect on the determination of the MLSS giving a steady-state checked during a 1-hour test. The blood lactate remained constant during exercise and ranged from 2.2 to 6.7 mmol/L (mean value equal to 3.9 ± 1 mmol/L) [fig. 2].

Only 2 tests of 20 minutes performed at 65 and 80% of the power at $\dot{V}O_{2max}$ and separated by 40 minutes of complete rest can be used to estimate MLSS. The MLSS is correlated and is close to the critical velocities[84] calculated from Monod and Scherrer's model.[85] The critical velocities can be an alternative way of calculating the LT, due to the easy way of calculation.[86]

Recently, Beneke and von Duvillard[87] reported that MLSS intensity as related to maximal workload was independent of the sports event (rowing, cycling, skating). However, the level of MLSS differed ($p < 0.001$) in rowing (3.1 ± 0.5 mmol/L), cycling (5.4 ± 1.0 mmol/L) and in speed skating (6.6 ± 0.9 mmol/L). Absolute MLSS work load was higher in rowing (316 ± 29.9W) and speed-skating (300.5 ± 43.8W) than in cycling (257.8 ± 34.6W). The level of MLSS might, therefore, be inversely related to the mass of dominantly working muscle. The authors concluded that the observed differences in MLSS and MLSS workload might correspond to the sport-specific mass of working muscle. They hypothesised that differences in the ratio between dominantly active muscle and assisting muscle, might affect the rates of glycolysis and muscular lactate oxidation. One reason for the latter may be lower or higher forces per stroke per given mass of muscle.

4. Age and Sex Effect on Blood Lactate Measurements

4.1 Women

Previous studies have delved into the sex differences in the relationships between long-distance performance and the anaerobic threshold.[38,88,89] No remarkable sex differences in LT $\%\dot{V}O_{2max}$

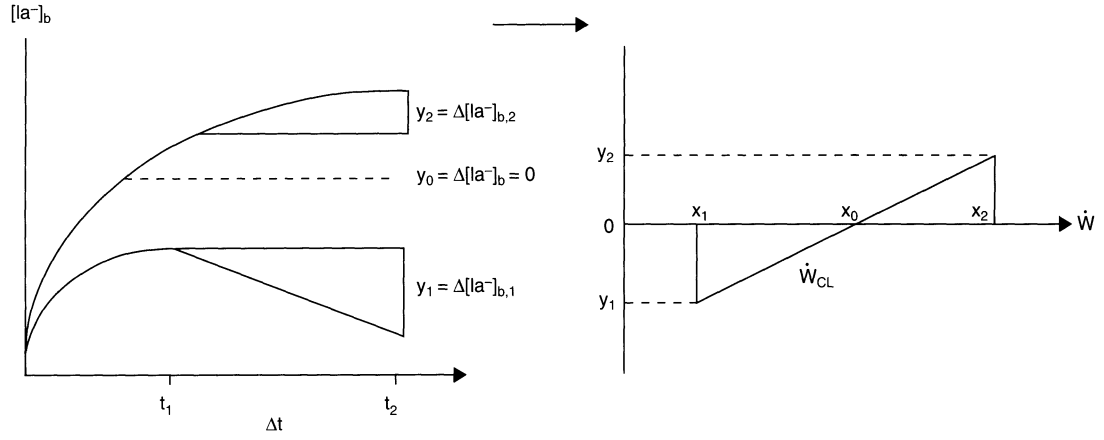

Fig. 1. Characteristic exercise intensity ($\dot{W}_{CL}$) determination from 2 exercises:
$x_0 = \dot{W}_{CL}$; $x_1 = \dot{W}_1$; $x_2 = \dot{W}_2$
$y_0 = \Delta[la^-]_b / \Delta t = 0$; $y_1 = \Delta[la^-]_{b,1}$ (decreasing values); $y_2 = \Delta[la^-]_{b,2}$ (increasing values)
it can be written:

$$\frac{y_2 - y_0}{y_0 - y_1} = \frac{x_2 - x_0}{x_0 - x_1}; \quad \dot{W}_{CL} = \frac{x_1 y_1 - x_2 y_2}{y_2 - y_1}$$

where $\Delta[la^-]_b$ is changes in blood lactate level, Δt is changes in time and x can be expressed in km/h, watts, or percentage maximal oxygen uptake (%$\dot{V}O_{2max}$) [as in this study].
For example, the first study participant had a maximal aerobic power (MAP) equal to 380W. The first and lowest stage ($\dot{W}_1 = x_1$) was 280W (73% MAP and 67% $\dot{V}O_{2max}$); the second and higher stage ($\dot{W}_2 = x_2$) was 320W (84% MAP and 82% $\dot{V}O_{2max}$); the blood lactate levels $\Delta[la^-]_b$ at the fifth and twentieth minute at 280W ($\dot{W}_1$) were 3.1 and 2.7 mmol/L respectively; $\Delta[la^-]_{b,1}$ (decreasing values, twentieth to fifth) = 2.7 – 3.1 = –0.4 = y_1; the $[la^-]_b$ at the fifth and twentieth minute at 320W ($\dot{W}_2$) were 3.7 and 5.4 mmol/L respectively; $\Delta[la^-]_{b,2}$ (increasing values, twentieth to fifth) = 5.4 – 3.7 = +1.4 = y_2. Similarly:

$$\dot{W}_{CL} = \frac{x_1 y_1 - x_2 y_2}{y_2 - y_1} = \frac{(280 \times 1.4) - (320 \times -0.4)}{1.4 - (-0.4)} = \frac{392 + 128}{1.8} = 288W$$

$\dot{W}_{CL}$ = 76% MAP and 79% $\dot{V}O_{2max}$, $\Delta[la^-]_{b,1}$, $\Delta[la^-]_{b,2}$ changes in blood lactate level in tests 1 and 2, respectively.

were found in long-distance runners.[38] That is in accordance with the fact that men and women have the same %$\dot{V}O_{2max}$ *vs* time relationship (calculated by the endurance index).[90] The $\dot{V}O_{2max}$ values are about 10 to 15% lower than the average value of 77 ml/kg/min and highest value of 84 to 85 ml/kg/min seen in comparable groups of men.[90] However, the mitochondrial adaptation in the skeletal muscles of highly trained men and women appear similar.[91]

Another study has looked at the sex differences between performance and running economy and showed a superior running economy among women.[92] As discussed in section 2.1.2, Helgerud[43] evaluated performance-matched male and female marathon runners and reported that while men had higher $\dot{V}O_{2max}$ and anaerobic threshold, women had higher weekly training distance and demonstrated superior running economy (lower gross oxygen cost of running) and a higher exercise intensity expressed as a percentage of $\dot{V}O_{2max}$ during the race.

In a recent study, Billat et al.[93] compared elite French male and female middle-distance runners. Each sex group was homogenous with respect to their International Amateur Athletic Federation (IAAF) performance scores, and there were no differences between the sexes. The results of this investigation indicated that when men and women are studied separately, the relationships between the bioenergetic parameters studied, and the 1500m track performances, i.e. the average velocity over 1500m, differ greatly. In fact, 4 parameters predicted 96% of the variance on 1500m track performance

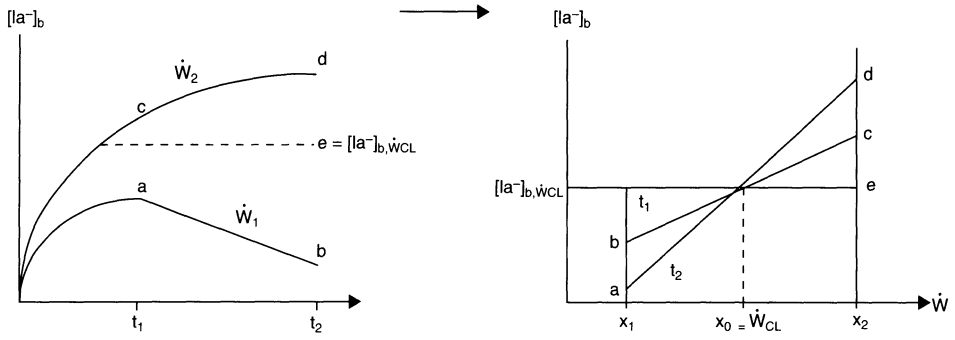

Fig. 2. The $[la^-]_{b,\dot{W}CL}$ determination by comparing 2 relationships: blood lactate level ($[la^-]_b$) and exercise intensity, at 2 times of observation, t_1 and t_2.
$x_0 = \dot{W}_{CL}$; $x_1 = \dot{W}_1$; $x_2 = \dot{W}_2$;
$a = [la^-]_{b1,t1}$; $b = [la^-]_{b1,t2}$; $c = [la^-]_{b2,t1}$; $d = [la^-]_{b2,t2}$; $e = [la^-]_{b,\dot{W}CL}$
The $[la^-]_{b,\dot{W}CL}$ (e) is where $[la^-]_b$ is identical at time t_1 and t_2 where exercise intensity is equal to characteristic exercise intensity ($\dot{W}_{CL}$). It can be written:

$$\frac{d-e}{e-b}=\frac{c-e}{e-a} \text{ and } [la^-]_{b,\dot{W}CL}=\frac{(da-bc)}{(d+a)-(c+b)}$$

For example, the first study participant had a maximal aerobic power (MAP) of 380W. The first and lowest stage ($\dot{W}_1 = x_1$) was 280W [73% MAP and 67% maximal oxygen uptake ($\dot{V}O_{2max}$)]; the second and higher stage ($\dot{W}_2 = x_2$) was 320W (84% MAP and 82% $\dot{V}O_{2max}$); the $[la^-]_b$ at the fifth and twentieth minute at 280W were 3.1 ($a = [la^-]_{b1,t1}$, and 2.7 mmol/L ($b = [la^-]_{b1,t2}$) respectively; the $[la^-]_b$ at the fifth and twentieth minute at 320W (W_2) were 3.7 ($c = [la^-]_{b2,t1}$) and 5.4 mmol/L ($d = [la^-]_{b2,t2}$) respectively;

$$[la^-]_{b,\dot{W}CL}=\frac{(da-bc)}{(d+a)-(c+b)}=\frac{(5.4\times3.1)-(2.7\times3.7)}{(5.4\times3.1)-(2.7\times3.7)}=\frac{16.74-9.99}{8.5-6.4}=\frac{6.75}{2.1}=3.20 \text{ mmol/L}$$

At $\dot{W}_{CL}$ (312W) the first participant had a $[la^-]_{b,\dot{W}CL}$ of 3.20 mmol/L.

in men: V_{amax}; velocity at the onset of blood lactate accumulation; tlim at 110% V_{amax}; and running economy, In contrast, for women there were no significant predictors. However, this study considered the velocity over 1500m, a distance in which aerobic and anaerobic contributions are similar.[94] However, for the same IAAF score, women spend 30 seconds more than men to achieve a 1500m event which involves more aerobic contribution for women than for men.

Hill and Smith,[95] showed that for the same all-out efforts of 30 seconds duration, women provided a relatively higher portion of aerobic energy than men (25 *vs* 20%). The performance difference between men and women was attributed to the $\dot{V}O_{2max}$ and V_{amax} differences. The fact that women have no significant aerobic predictor may seem to be surprising, since others who have found that $\dot{V}O_{2max}$ and V_{amax} were predictors of running performance in female long-distance runners.[31,33,36,37] This finding may be explained by the different training level of the different populations (not controlled in this study) and by the fact that some of the 1500m women runners are more 800 to 1500m runners (9 over 14) and the others were more 1500 to 3000/5000m specialists. There was no specific energetic profile for women to perform over 1500m. The velocity at the onset of blood lactate accumulation has also been proposed as a good predictor for 3000m track performance for 57 female distance-runners.[33] This was due to the large aerobic component of this race.[32]

Another study[96] demonstrated that anaerobic component (peak power and mean power using the Wingate test performed on a bicycle) was related only to 800m running performance. Once more, a 1500m race involves similar contribution of aerobic and anaerobic metabolism, but some runners

can perform this event with a different contribution of the various energy systems.

4.2 Children

In general, blood lactate level might be affected by the rate of: (i) lactate formation and accumulation in contracting muscles; (ii) efflux of lactate from muscle to blood; and (iii) uptake of lactate from blood by the heart, liver, active muscles and kidneys. Although no data are available for the rate of efflux and uptake of lactate in children, there are some suggestions regarding oxygen deficit[97,98] and muscle metabolic profiles.[99,100] It was demonstrated that the lactate level during submaximal work is related to the amount of oxygen deficit[101] and oxygen adjustment speed at the onset of work.[102]

Both blood lactate level and oxygen deficit, at the same relative work load, are smaller in boys than in adults. Exercise at the same relative submaximal intensity elicits lower blood lactate in children than in adults. Interpretation and identification of developmental and maturational patterns of response are limited, however, by the use of different testing conditions and reference points (e.g. LT, MLSS and fixed level reference points).[103] According to a recent article by Armstrong and Welsman[103] concerning the assessment and interpretation of aerobic fitness in children and adolescents, there is growing evidence to suggest that the 2.5 mmol/L reference blood lactate level should be used in preference to the 4.0 mmol/L level. This is due to the fact that the adult criterion occurs close to maximal exercise in many children and adolescents. However, it is difficult to elucidate the differences in blood lactate response between adults and children. There is a lack of conclusive evidence to support conventional explanations that differences are caused by insufficient testosterone, glycolytic enzymes, faster oxygen on-transients (i.e. the non–steady-state phases of oxygen kinetics during an exercise), a greater reliance on fat as a fuel, or high levels of physical activity.

Recently, Welsman et al.[104] found no significant correlation between testosterone and the percentage of peak oxygen consumption at blood lactate levels of 2.5 and 4.0 mmol/L. Macek and Vávra,[98] have observed a more rapid adjustment of oxygen in children at the onset of exercise than in adults, although this did occur during maximal work. Prepubertal children, notably those at the age of 10 years, reached a higher velocity at the LT than young untrained men. Furthermore, they attained similar velocities to those of trained young men. There was no significant difference between boys of all age groups (6 to 7, 8 to 9, 10 to 11, 12 to 13 and 14 to 15 years old), but a significant negative correlation between the velocity at the LT (i.e. velocity just below 2 mmol/L) and bone maturity score.[104] Heart rate and percentage maximal heart rate at the LT were dependant on age. These authors suggested that maturation is one of the factors influencing LT. However, this was the velocity at a blood lactate level of 2 mmol/L and the maximal blood lactate level decreased with age and was lower in children (4.7 ± 1.3 mmol/L at 6 to 7 years old and 11.1 ± 3.6 mmol/L at 14 to 15 years old). In this latter study, the children performed 4-minute runs at a fixed velocity with 2-minute rests between each exercise bout.

A recent study,[105] measured the running velocity corresponding to the individual MLSS in a group of 12-year-old boys and girls (6 boys and 7 girls) of the same school class whose pubertal maturation corresponded to the end of stage 2 and the beginning of stage 3.[25] The running velocity at the MLSS was expressed in $\%V_{amax}$ and was calculated using only the 2 submaximal stages of 15 minutes at 60 and 75% of V_{amax}, each separated by a 40-minute interval of rest. This test was previously validated and planned for adults with 2 stages of 20 minutes as shown in figures 1 and 2.[22] V_{amax} was significantly higher in the boys than in the girls (12.6 *vs* 11.2 km/h) and the MLSS velocity was equal to 68% of V_{amax} for both girls and boys (8.5 ± 0.7 and 7.7 ± 0.7 km/h, respectively). These values were lower than those reported by Mocellin et al.[106] using rectangular protocols lasting 16 minutes, they showed that 12-year-old boys demonstrated a steady-state in blood lactate level at 89% of V_{amax} and 88% of $\dot{V}O_{2max}$. However, these

authors considered that the MLSS was assumed if the difference between the last level measured after 3 and 5 minutes of running exceeded 1 mmol/L (and not strictly equal to 0 as calculated in figures 1 and 2) during a constant speed, and the increase was discontinuous, or if a continuous increase in blood lactate exceeding 0.5 mmol/L was observed during the run.

Macek et al.[107] have shown that 10 prepubertal boys aged 12 years could easily run 1 hour at 60% of $\dot{V}O_{2max}$, with blood lactate level decreasing from 1.53 to 1.09 mmol/L between the tenth and the sixtieth minute. Whatever, the maximal lactate definition chosen, determination of the maximal blood lactate velocity is of particular interest when beginning an endurance training programme without risk of overloading for children.[105] It would appear that children can train at least at the same relative load of V_{amax} than adults.

The rise in performance which accompanies growth could be explained by the improvement in running efficiency as shown by Daniels and Oldridge[108] (52 ml/kg/min at 202 m/min *vs* 45.5, 22 months later), or by Davies.[109] However, as for all criteria, few data are available on the influence of training at the LT, on the performance in children, as done for adults.[47] In a recent paper, Unnithan et al.[110] reported that overall major variables associated with increase in distance running performance for adults were: running economy; $\dot{V}O_{2max}$; and velocity at the LT. Peak $\dot{V}O_2$ appeared to be the most important factor associated with success at 3000m (12:51 ± 1:04 min:seconds) in pre-pubertal distance runners (average age of 11.7 ± 1.1 years, 60.5 ± 3.1 ml/kg/min of peak $\dot{V}O_2$).

4.3 Older Athletes

The popularity of competitive involvement among older athletes has increased to such an extent that regular world championships are held for this age group (over 40 years old).[111] Maffuli et al.[111] ascertained the fact that tests originally developed to monitor various indices of sustained aerobic power in younger adults are equally applicable to older trained individuals. They tested 20 male athletes (average age 58.7 ± 3.2 years; range 52 to 66 years), training 55 ± 14 km/week and racing from 1500m to marathon distance. The performance of master athletes, when expressed relative to the onset of blood lactate accumulation, is quite similar to that of adult endurance athletes. For similar performance, they have the same onset of blood lactate accumulation as younger individuals. As is the case with younger competitors, master athletes probably do not run 10 000m and marathon races at speeds that require $\dot{V}O_{2max}$. In older individuals there is some evidence that absolute $\dot{V}O_{2max}$ at which the LT occurs remains constant or only declines minimally with aging. This means that the LT as a percentage of $\dot{V}O_{2max}$, may increase somewhat with aging.[40,112] If the LT changes in this way, then such alterations would serve to maintain performance in the face of declines in $\dot{V}O_{2max}$. Such a mechanism could explain the continued world class performances of some individuals in open competition into their late thirties and early forties.[39] This possibility is consistent with observations that suggest that training preserves the mitochondrial adaptations in skeletal muscles that are thought to play a key role in regulating lactate production in contracting skeletal muscles.[112]

5. Specificity of Sports and Blood Lactate Use for Training

5.1 Running

Förenbach et al.[113] showed that the range of running speed at a lactate level between 2 to 3 mmol/L could be considered the 100% intensity level for training and competition for a marathon. However, improvements in performance occur in trained runners when intensity of training is increased[114] and it is known that optimum improvements in cardiorespiratory fitness occur when training is at an intensity corresponding to 90 to 100% of $\dot{V}O_{2max}$.[115] Other studies suggest that a blood lactate level of 4 mmol/L, corresponding to 80 to 90% of $\dot{V}O_{2max}$ in male runners, represents the optimum intensity to aerobic fitness[2,115,116] and corresponds to 10 to 16km race pace.[47] How-

ever, both authors were correct when considering the range of running distance events.

Coen et al.[117] pointed out the possibility of using the IAT as a basis for advice on training. They assumed that the appropriate intensity of an extensive endurance training programme was between 85 and 92% IAT because the lactate level increases to above 92% of the IAT speed when endurance runs are performed on flat terrain. Furthermore, they considered the fact that the intensity of the speed session programmes, with the lactate level exceeding 4 mmol/L, could also be determined on the basis of the speed of the IAT. The regression line between the threshold speed and lactate level of a 5 × 1000m programme allowed accurate predictions to be made concerning the corresponding lactate level for a given intensity. Under good climatic conditions, the lactate level increased at 116 and 120% IAT to approximately 6 and 9 mmol/L respectively. Coen et al.[117] emphasised the importance of taking external factors into consideration in order to provide the correct advice for training (e.g. terrain gradation and climatic conditions). However, in reality it seems that training pacing is far from these intensities.

Robinson et al.[118] attempted to quantify training by the use of objective, longitudinal training data. They showed that the mean intensity of steady-state running for the participants in this study is considerably lower than the optimal training intensities suggested by some authors quoted above. However, the differences in relative training speed between participants were highly significant depending on the training distance (middle-distance *vs* long-distance). Robinson et al.[118] showed that these differences were partly explained by the negative correlation between relative training speed and intended event distance, which demonstrates that individuals training for long-distance events do so generally at a lower intensity than those who are training for middle-distance events. They demonstrated that the training intensity is being chosen by the athletes or coaches as appropriate for the intended event rather than being limited by the volume of training, the correlation between relative training speed and the mean weekly training distance was not significant. Tables II and III show an example of training 5 months before the first important competition for a middle-distance runner having a $\dot{V}O_{2max}$ equal to 73 ml/kg/min associated with a V_{amax} of 21.5 km/h. His MLSS (according to the method of Billat et al.[22]) was equal to 80% of V_{amax}. After these 4 weeks, V_{amax} reached 22 km/h and maximal blood lactate velocity remained at 80% of V_{amax} (increasing in absolute value). Further studies are needed to appreciate the influence of particular training on both V_{amax} and maximal blood lactate velocity.

Table II. Personal best times and velocities for a 26-year-old male middle-distance runner[a] over 9 years

Distance (m)	Personal best [h:min:sec] (year)	Velocity	
		km/h	% V_{amax}
800	0: 1: 55.83 (1988)	24.9	115[b]
1 000	0: 2: 28.92 (1990)	24.2	112[b]
1 500	0: 3: 57.34 (1990)	22.8	105[b]
3 000	0: 8: 28.59 (1995)	21.2	98[b]
5 000	0: 14: 40 (1994)	20.5	95[b]
10 000	0: 31: 34 (1995)	19.0	88[b]
21 100	1: 10: 38 (1993)	17.9	85[c]

a This runner has already run for 8 weeks with 70km at 60-70% of the velocity associated with $\dot{V}O_{2max}$ [V_{amax}]. V_{amax} = 21 km/h; maximal lactate steady-state velocity ($\dot{W}_{CL}$) = 17 km/h (81% of velocity associated with $\dot{V}O_{2max}$); 5 training sessions per week. The purpose of this first training plan over 4 weeks is to improve V_{amax} (with the second training session) and to improve maximal lactate steady-state velocity (with the fourth training session).

b V_{amax} = 21.5 km/h.

c V_{amax} = 21 km/h.

Abbreviation: V_{amax} = the velocity associated with maximal oxygen uptake; $\dot{V}O_{2max}$ = maximal oxygen uptake.

Table III. Training plan performed at maximal lactate steady-state velocity in a 26-year-old male middle-distance runner[a] 1995/96 (trainer: V. Billat)

Weeks	Training session no.				
	1 (70-75% V_{amax})	2 (90-110% V_{amax})	3 (70-75% V_{amax})	4 (velocity at the maximal lactate steady-state = 80-85% V_{amax})	5 [hills = (slope 4-8%) at 100% of $\dot{V}O_{2max}$]
15-21 Jan 1996	1h at 14 km/h	Warmup: 20 min at 12km/h + 15 × 200m in 31 sec (23.2 km/h = 110% V_{amax}) Recuperation = 1 min at 12 km/h for approximately 200m + 15 min slow run at 60% V_{amax}	Slow run (14.5-15 km/h) for 1h 30 min	2 × 12 min at 17 km/h for 3400m Rest between = 5 min run at 12 km/h	Hills 8 × 1 min 30 sec at 6% Heart rate = 190 beats/min at the top of hills Rest between = 1-2 min Heart rate = 140 beats/min
22-28 Jan 1996	1h at 14 km/h	10 × 400m in 1 min 9 sec (20.9 km/h = 99% V_{amax}) Rest between = 1 min at 12 km/h (200m)	Slow run (14.5-15 km/h) for 1h 30 min	2 × 15 min at 17 km/h for 4250m Rest between = 5 min run at 12 km/h	Hills 10 × 1 min at 6%
29 Jan-4 Feb 1996	1h at 14 km/h	5 × 800m in 2 min 20 sec (20.6 km/h = 98% V_{amax}) Rest between = 1 min at 12 km/h (200m) Active recovery of 15 min	Slow run (14.5-15 km/h) for 1h 30 min	1 × 15 + 1 × 20 min at 17 km/h = 4250m + 5650m Rest between = 5 min run at 12 km/h	Hills 8 × 1 min at 6%
5-11 Feb 1996	1h 30 min at 14 km/h	5 × 1000m in 2 min 55 sec (20.5 km/h = 97.6% V_{amax}) Rest between = 2 min 30 sec at 12 km/h (400m)	Slow run (14 km/h) for 45 min	2 × 20 min at 17 km/h = 2 × 5650m Rest between = 7 min run at 12 km/h	Hills 8 × 2 min at 4%

a Summer objectives: June 1996: <3 min 58 sec over 1500m (personal best 3 min 57 sec in 1990). September 1996: less than 14 min 45 sec over 5000m (personal best 14 min 40 sec in 1994).

Abbreviations: V_{amax} = the velocity associated with $\dot{V}O_{2max}$; $\dot{V}O_{2max}$ = maximal oxygen uptake.

Recently, Casaburi et al.[119] showed that there were no significant differences between a group which was trained at the LT and a group which was trained above the LT. These groups were followed during 5 cycle ergometer training sessions per week for 5 weeks with the same total training work at or above the LT. They concluded that in healthy individuals, exercise which does not elevate blood lactate alters power output responses as effectively as exercise which elevates lactate, provided that total training work is the same.

The combination of 300 and 100m run times, percentage of body fat, running economy and $\dot{V}O_{2max}$ as independent variables, accounted for the greatest amount of the total variance ($R^2 = 0.89$) of the performance over 800m.[120] They concluded that these data offered additional support for the notion that much of the intramuscular ATP was produced and utilised during an 800m run (132 ± 7.3 seconds). The later study showed that these shorter distances can be used as performance prediction. However, Hirvonen et al.[121] has shown that after a maximal 200m run, the speed of running decreased, although the creatine phosphate (–47%) was not depleted and lactate level (6.3 ± 0.7 mmol/L) was not at maximum level. Complete fatigue occurred over 400m when creatine phosphate stores were depleted (–89%) and blood lactate attained an individual maximum (10.3 ± 0.7 mmol/L), peak lactate value being 14.9 ± 0.3 mmol/L. These values are far below those reported by Kinderman and Keul[122] i.e. 24.9 mmol/L at the end of a 400m run in 45.5 seconds.

Lacour et al.[123] reported blood lactate values of 20.1, 21.9 and 20.8 mmol/L for men at the end of 400, 800 and 1500m, respectively. It seems that, in relation to the duration of effort, there was a continual increase of the maximal lactate level from 100 to 400m.[122] In fact, Karlsson and Saltin[124] had demonstrated that the breakdown of the phos-

phogens was already maximal after 2 minutes of work at 120 and 100% of the maximal aerobic power (i.e. the power which elicits $\dot{V}O_{2max}$). The accumulation of lactate in the muscle and the blood increased continuously until exhaustion. They concluded that low ATP and creatine phosphate stores was not the reason for muscular fatigue, muscular lactate level (acidosis) might be the limiting factor for power outputs of 120 and 100%.

5.2 Swimming

In swimming, the measurements have to be performed in the water. Keskinen et al.[125] showed that the blood lactate value of 4 mmol/L in 2 (submaximal and maximal) sets of 400m, 3 mmol/L in a number of sets of 300m (progressive speed), 2 mmol/L in a number of sets of 100m (progressive speed) were found to correspond to the same velocity. They concluded that to measure the whole intensity area, a combination of tests should be applied by performing an incremental set with several steady-state loadings (aerobic) and one or two 100m swims (anaerobic) in one test session.

Treffene et al.[126] investigated the rate of lactic acid accumulation during swimming at a range of relative intensities below and above the minimal velocity which elicits the maximal heart rate and $\dot{V}O_{2max}$, i.e. V_{amax} as defined above in other studies and obtained at the end of an exhaustive 365.8m swim (in accordance to Costill et al.,[127] close to the Lavoie et al.'s protocol which used a 400m[128] swim). They found that the speed at which a 4 mmol/L blood lactate level occurred, was equal to $95.6 \pm 2.6\%$ of V_{amax} (i.e. 0.05 m/sec below) and 88% of $\dot{V}O_{2max}$. They showed that the lactate accumulation gradient for each 2% range of relative intensity was from 86 to 110% of V_{amax}. The mean lactate accumulation gradient (mmol/L/min) at all constant intensities below 100% of V_{amax} was less than 0.55 mmol/L/min. Marked changes in the rate of venous lactate accumulation occurred immediately when V_{amax} was exceeded; the rate being proportional to the intensity above V_{amax}. At 100.8% of V_{amax}, the gradient was 2.08 ± 0.7 mmol/L/min and at 108.8% of V_{amax}, the gradient was 9.9 ± 1.4 mmol/L/min. The findings of high lactate accumulation rates (proportional to intensity) at speeds above V_{amax} agreed with the findings of di Prampero et al.[55] whose data indicate that the lactate would continue to accumulate at this rate had the Treffene et al.[126] study investigated longer sprint performance.

In contrast to running, blood lactate accumulates at a velocity which is very close to V_{amax} and $\dot{V}O_{2max}$ in swimming. This is the result of the exponential relationship between the velocity and the oxygen uptake ($\dot{V}O_2$) in swimming. However, Poole et al.[129] showed a slow component of $\dot{V}O_2$ kinetics during exercises above the LT, where an additional slow phase of $\dot{V}O_2$ is superimposed upon the underlying kinetics. Recently, Zoladz et al.[130] confirmed the nonlinearity of the $\dot{V}O_2$-power output relationship on a cycle ergometer. However, in swimming there is a net nonlinearity of the relationship between $\dot{V}O_2$ and speed due to drag (for review on the energetics of competitive swimming see Toussaint and Hollander[131]). Toussaint and Hollander[131] demonstrated that the 10% increase in propelling efficiency resulted in both a reduction in time over short- and long-distance, which was superior to the gains found when increasing the maximal aerobic or anaerobic power by 10%.

Miserocchi et al.[132] demonstrated that the biomechanical constraint due to the aquatic environment could explain the smaller value of the slope of the relationship between velocity and time found in swimmers compared with track and field runners.[90] It means that swimmers lose less speed as the time of the event increases. This reflects the difficulty for swimmers to develop high maximum velocities over shorter distances due to the much higher drag in water compared with air.[133]

5.3 Rowing

With a race duration of approximately 6 to 7 minutes for the 2000m course, rowing may be regarded as a sport model for one key exercise physiological variable: $\dot{V}O_{2max}$.[134] The anaerobic threshold reported in elite rowers is around 85% of $\dot{V}O_{2max}$. Kramer et al.[135] showed that $\dot{V}O_{2max}$ was

the variable which produced the highest correlations with rowing performance among field variables (e.g. 90 second ergometer distance) and laboratory variables (e.g. concentric peak torque or maximum heart rate). However, LT was not examined. In a group of 12 rowers who trained for the World Championships, the anaerobic threshold (the power output at 4 mmol/L) was significantly improved in the preparatory phase (March), and declined in the post-competitive phase (November).[136,137] Training which elicits a blood lactate above 4 mmol/L, sprint training and athletic training complete the training schedule, which may reach 1000 hours or 5000 to 7000km per year.[138] The increased anaerobic threshold was accompanied by a significant increase in physical fitness. A strong correlation was found between the mean power output in the exercise simulating the 2000m race and power output at the anaerobic threshold in both preparatory and competitive periods. This supports the view that high aerobic fitness is one of the principal factors determining the competition outcome in rowing; anaerobic threshold being equal to 76% of the mean power recorded in the 2000m test.

6. Conclusions

Blood lactate measurements are an approximate way to appreciate the equilibrium between the rate of lactate production and elimination determined by the relative kinetics of glycolysis, lactate dehydrogenase and mitochondrial respiration. However, this complexity in the regulation of lactate metabolism does not prevent general practitioners or coaches from measuring or using the blood lactate for prediction of exercise performance.

The anaerobic threshold, commonly defined as the exercise intensity, speed, or fraction of $\dot{V}O_{2max}$ at a fixed blood lactate level or a MLSS, has been accepted as a measure of endurance ability. The blood LT expressed as a fraction of $\dot{V}O_{2max}$ is the same in elite long-distance athletes and does not depend on the specificity of the sport. On the contrary, the blood LT expressed as a fraction of the velocity associated with $\dot{V}O_{2max}$ depends on the relationship between velocity and $\dot{V}O_2$, for example swimmers who have a blood LT very close to the velocity associated with $\dot{V}O_{2max}$.

The measurement of the postcompetition blood lactate in short events has found to be related to the performance in events lasting from 1 to 2 minutes (400 to 800m in running). Blood lactate measurements can help when selecting the correct exercise intensity for training. However, to interpret the blood lactate profile modification after training, the athlete's nutritional state and exercise protocol have to be controlled. Moreover, improvement of fractional utilisation of $\dot{V}O_{2max}$ at the MLSS, for example, has to be considered as an assessment tool like all the factors of performance such as the velocity associated with $\dot{V}O_{2max}$.

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