Abstract

During the last two decades the concept of the MLSS (maximal lactate steady state) has been established. The MLSS detects the highest level of the BLC (blood lactate concentration) and the corresponding workload (MLSS workload) that can be maintained over time without continual BLC accumulation. In spite of a lack of experimental and/or theoretical foundation, it has been speculated that the level of the MLSS may decrease with increasing performance capacity. The potential inter-relationship between performance capacity and BLC response to prolonged constant workload will be analysed based on a recent study, which provided evidence that the MLSS is independent of performance whereas MLSS workload increases with performance capacity, and by a computer-aided simulation. The simulated model modifies and combines previous theories put forward to explain the response of BLC to exercise and incorporates a theory about limiting factors of oxygen transport to the muscle cell. Simulations consider the BLC response to selected prolonged constant workloads while paying special respect to changes in body structure and substrate utilization, which are generally accepted as limiting factors of performance capacity. This complex modulation of appearance and disappearance of lactate during constant prolonged exercise seems to support the experimental results, which indicated independence between MLSS and performance capacity.

Introduction

The BLC (blood lactate concentration) was established as a measure of exercise intensity early last century [1,2]. During the last two decades the concept of the MLSS (maximal lactate steady state) has been developed and validated as a method to describe the BLC response to prolonged constant workload exercises with the intention of detecting the highest level of BLC and workload (MLSS workload) that can be maintained over time without continual blood lactate accumulation [3–5]. The MLSS is considered to discriminate qualitatively between sustainable exercise intensities, in which continuous work is limited by stored energy (heavy exercise intensity), and exercise intensities, which have to be terminated because of a disturbance of cellular homoeostasis (severe exercise intensity) [6]. The level of the MLSS is speculated to decrease with increasing performance capacity [7–10]. Proof for such an inter-relationship is lacking.

The present paper describes two approaches to address this potential inter-relationship: firstly, an interpretation of experimental measures of BLC during prolonged constant workloads based on statistics and current knowledge [11], and secondly the application of a computer-aided simulation of BLC during prolonged constant workload [5], which incorporates selected potential modulators of the MLSS.

Experimental approach

The first study, designed to address a potential relationship between MLSS and performance capacity, investigated 33 male subjects (age, 23.7 ± 5.5 years; height, 181.2 ± 5.3 cm; body mass, 73.4 ± 6.4 kg) [11]. Peak workload reached at the end of an incremental exercise test (4.8 ± 0.6 W·kg⁻¹) served as a measure of maximum performance.
while the MLSS (4.9 ± 1.4 mM) and the corresponding workload (3.4 ± 0.6 W · kg⁻¹) were measured during three to six 30-min constant exercise tests on a cycle ergometer. In spite of the fact that the study fulfilled the inevitable prerequisite for such an analysis, i.e. a wide range of performance and BLC responses, the MLSS was not correlated with any measure of performance (Figure 1).

Although the MLSS appeared to be independent of performance capacity the latter study confirmed the generally accepted observation that production, and elimination of pyruvate, are modified by enhanced performance capacity which results in decreased levels of BLC at given workloads. This led to the conclusion that the complex combination of various effects on lactate appearance, and disappearance during exercise, may explain why subjects with higher maximum performance have higher MLSS workloads and why the MLSS is independent of performance.

This verbal explanation of experimental results concerning the MLSS considers selected aspects which possibly explain the independence between performance capacity and MLSS. However, the experimental results show a combined effect of factors which are considered to contribute to differences in performance capacity. The isolated effect of selected factors on the dynamic steady state of the BLC is almost impossible to anticipate under in vivo conditions based on such an experimental approach, statistics and verbal interpretation. Not only the magnitude but also the general effects of selected modulators on MLSS cannot be estimated without additional objective tools for data analysis. Appropriate computer models can potentially provide these tools.

**Computer-aided simulation**

Modelling of complex physiological responses to exercise serves as an alternative tool for data analysis and interpretation [12]. The model used here modifies and combines previous theories put forward to explain the response of the BLC to exercise [13–15] and incorporates a theory about limiting factors of oxygen transport to the muscle cell [16].

Basic assumptions are that performance is determined by mechanical power generated by working muscles (Mu). Depending on the mode of exercise the ratio between primarily engaged muscle (Mu₁) and assisting muscles (Mu₂) varies [17]. The lactate-formation rate is described as a function of V̇O₂ and/or exercise intensity (Int) of tissue with specific properties [5,14] equivalent to the activity of the respiratory chain [18] (for further details see [5]).

V̇O₂ is determined by cardiac output and the level of peripheral deoxygenation of the blood [19]. Cardiac output (the product of heart rate and stroke volume) and oxygen desaturation are functions of Int. Depending on Int the cardiac output is differentially distributed between non-muscular tissues and muscles. Desaturation of the blood perfusing different tissues is assumed to be linked to the specific loads and to constants describing the arterio-venous oxygen difference of tissues (for further details see [5]).

The overall rate of oxidative pyruvate utilization is a function of the V̇O₂ [20,21] but also of the activity of the pyruvate dehydrogenase. Pyruvate dehydrogenase activity depends on factors like ADP, Ca²⁺ etc, which relate to Int and therefore to the availability of pyruvate [14,15,22].

Net lactate appearance is the result of lactate production and utilization with respect to exercise intensity and oxygen availability, as shown by eqn (1). Due to different patterns of non-linearity for glycolytic rate and oxygen uptake there is a lack of pyruvate at low exercise intensities that has to be compensated by the utilization of fatty acids.

Depending on the ratio between glycolytic and oxidative rate (eqn 1) a steady state of the BLC will be established after an initial increase of BLC, or the BLC will increase proportionally to the cube root of time, or the BLC attains an increase that is proportional to time as a linear function of the difference between lactate production and elimination.

The metabolic power can be calculated by summing the rate of glycolysis, oxygen uptake contributing to pyruvate and fat utilization times their corresponding caloric equivalents, respectively. Mechanical power is the product of metabolic power and the mechanical efficiency for the specific task (for further details see [5]).

$$\frac{dLa}{dt} = \frac{dLa}{dt} gly - \frac{dLa}{dt} ox = rM \left( \frac{M_{u_1}}{1 + K^*} \left( 1 - \frac{\text{Load}_{\text{gly}}}{\text{Load}_{\text{max}}} \right)^3 \right) + M_{u_2} \left( 1 + K^* \left( \frac{\text{Load}_{\text{gly}}}{\text{Load}_{\text{max}}} \right)^3 + \frac{dLa}{dt} gly \left( \frac{\text{Load}_{\text{gly}}}{\text{Load}_{\text{max}}} \right)^3 \right)$$

Net lactate production results from the glycolytic rate and the pyruvate-consumption rate. Net lactate production increases with exercise intensity, fat oxidation compensates for the
lack of pyruvate. In eqn 1, \( dLa/dt \) is the net glycolytic rate, \((dLa/dt)\text{Gly}\) is the glycolytic rate, \((dLa/dt)\text{Ox}\) is the rate of pyruvate oxidation, \( \text{M}_{\text{1}} \) is the mass of primarily engaged muscle, \( \text{M}_{\text{2}} \) is the mass of assisting muscle, \( rM \) is the muscle mass related to body mass, \( \text{Load}_{\text{1}} \) is the load related to maximum load per unit of primarily engaged muscle, \( \text{Load}_{\text{2}} \) is the load related to maximum load per unit of assisting muscle, \( \text{Load}_{\text{nMu}} \) is the load related to maximum metabolic load per unit of non-muscular organs, \( \text{Glymax} \) is the maximum glycolytic rate, \( K' \) is the constant of half-maximal activation of cellular performance capacity, \( K'' \) is the constant of half-maximal activation of glycolysis, \( \text{Kel} \) is the constant of half-maximal velocity of pyruvate dehydrogenase, \( La \) is the lactate concentration in distribution space, \( \text{O}_2\text{Equ} \) is the lactate oxygen equivalent and \( \dot{\text{V}}\text{O}_2 \) is the rate of oxygen uptake.

**Application**

Using the proposed model, constant load tests in cycling were simulated with differences in workload of approximately 15 W (Figure 2), which is comparable with practically applied testing conditions. The simulations modulate training-related changes in body mass (75–70 kg), relative muscle mass (36–44%), cardiac output (19.8–26.3 l·min\(^{-1}\)) and pyruvate utilization expressed as the half-maximal velocity constant for the pyruvate dehydrogenase activity \([4.5–5.0 \text{ (mmol·l}^{-1})^2]\). The first simulation gives typical results for an untrained male subject aged 20–30 years (Figure 3; MLSS I) and is comparable with the experimental results described above. An increase in relative muscle mass combined with a loss of total body mass results in a significant increase in the BLC response (Figure 3; MLSS Ia). The simulated behaviour of the BLC shows an increase over time that is beyond the experimentally used definition of the MLSS. However, the simulation still identifies a steady-state condition (Figure 3; MLSS Ia) where the lactate-appearance rate is lower than the rate of pyruvate utilization. The higher level of the MLSS did not cause a significant increase in the corresponding workload. Relevant improvement of the MLSS workload demands a corresponding increase in the cardiac output (Figure 3; MLSS Ib). The increase in cardiac output provides an increase in the \( \dot{\text{V}}\text{O}_2 \) at an MLSS workload of almost the same magnitude. The level of the MLSS (Figure 3; MLSS Ib) is decreased. An increase in the metabolic rate of fat utilization by 1.6% elevates the BLC level of the MLSS to a value (MLSS II) that is almost identical to the level of MLSS I (Figure 3).

The simulations of MLSS I, MLSS Ib and MLSS II meet the criteria of the experimentally used definition of MLSS and reflect the experimental results. The simulations indicate that selected changes which are generally accepted as factors of improved endurance capacity may decrease but can also increase the level of the BLC at a given workload. The combined effect decreases the BLC and the reliance on glycogen at a given workload and increases the MLSS workload but does not change the level of the MLSS.

**Conclusion**

Experimental results provided evidence that the MLSS is independent of performance whereas MLSS workload increases with performance capacity. The application of a computer-aided simulation of BLC during prolonged constant workload incorporated selected potential modulators of the performance capacity. The complex modulation of appearance and disappearance of lactate during constant prolonged exercise enabled the analysis of both the isolated and the combined effects of such parameter modulations on the...
BLC response to defined conditions of constant prolonged exercise and MLSS. The simulations seem to support the experimental results, which indicated the independence between MLSS and performance capacity. They seem to give evidence that the independence between the level of MLSS and endurance capacity results from the combination of an increased aerobic rate combined with fine-tuning of the rate of fat utilization. Therefore, the complementary use of an experimental approach and computer-aided simulations confirms objectively that modulation of selected factors which have been discussed in conjunction with increased endurance performance modulate the power output at given exercise intensities but do not necessarily alter the level of MLSS.

I gratefully thank R.M. Leithäuser and C. Angus for many stimulating discussions and their helpful comments on earlier drafts of this paper.

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Received 6 June 2003