Influence of fiber type composition and capillary density on onset of blood lactate accumulation.

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**Abstract:**
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Onset of blood lactate accumulation (OBLA) was determined in sixteen healthy and physically active men (23-33 yrs) during cycling using a continuous step-wise increased exercise intensity protocol. OBLA was defined as the exercise intensity corresponding to a lactate concentration of 4 mmol·l⁻¹ blood. Oxygen consumption, pulmonary ventilation, respiratory quotient (R), heart rate and lactate concentration were monitored during each exercise intensity. Muscle biopsies were obtained from m. vastus lateralis at rest for determination of fiber type composition and capillary density. Calculated values (mean±SD) for exercise intensity, oxygen consumption and ventilation at OBLA were 159±37W, 2.43±0.47 l·min⁻¹ and 49.8±10.5 l·min⁻¹ or 65±10 % of $\dot{V}_{O_2}\text{max}$. OBLA (? $\dot{V}_{O_2}\text{max}$) was found to correlate significantly ($r=0.75$, $p<0.001$) to the relative muscle area occupied by ST (Type I) fibers. Furthermore, 92% of the variance in OBLA could be explained by % ST area + capillary density. It is suggested that both inherent and adaptative qualities of the exercising muscle are of significance for the onset of blood lactate accumulation.
**Introduction**

The exercise intensity, corresponding to the onset of a net accumulation of lactate in blood, has been proposed to represent a metabolic shift from aerobic to prevalent anaerobic energy contribution (32). As originally described by Margaria et al. (22) a proportionally greater increase in pulmonary ventilation compared to oxygen consumption occurs at exercise intensities in the order of 50-60% of maximal oxygen uptake (VO$_{2\text{max}}$). To compensate for acidosis, caused by an enhanced lactate formation during heavy exercise ventilation increases out of proportion to oxygen uptake according to Wasserman et al. (37). Numerous studies have also reported lactate accumulation above resting values at exercise intensities equivalent to 50-60% VO$_{2\text{max}}$ (7, 17, 23). It has also been shown that onset of lactate accumulation occurs at higher absolute and relative exercise intensities in trained athletes than in moderately or non-trained individuals (6, 7). A recent study (13) emphasized the significance of muscle respiratory capacity and fiber type composition for onset of lactate accumulation during cycling exercise. It was found that individuals with a predominance of slow twitch (type I) fibers and a high muscle respiratory capacity in m. vastus lateralis had a higher lactate threshold than individuals with muscles rich in fast twitch (type II) fibers and a low capacity to oxidize pyruvate in vitro. In accordance, Sjödin and Jacobs (30) recently studied marathon runners and were able to demonstrate a relationship between fiber type composition and the running velocity at which lactate starts to accumulate in blood (VO$_{2\text{BLA}}$). Moreover, significant correlations between VO$_{2\text{BLA}}$ and both muscle
capillary density as well as the activity of key enzymes regulating glycolysis and the Kreb's cycle have been demonstrated (30, 31). The following experiments were intended to further study the influence of muscle fiber type composition and capillary density on onset of blood lactate accumulation during cycling exercise.

Methods

Sixteen male subjects volunteered for this study. They were all accustomed to heavy physical exercise and were engaged in different exercise programs (e.g. endurance running, strength-training, general conditioning) at the time of the study. Mean (range) values for age, height and weight were 27 (23-33) yr, 176 (165-185) cm and 78 (67-99) kg. Prior to giving their written consent, subjects were informed of the purpose and the risks associated with the experiments.

Maximal oxygen uptake ($\dot{V}O_{2max}$) was measured during cycling (60 rpm) on a Monark ergometer. Exercise intensity was increased by 30 W every second minute until exhaustion. Respiratory parameters and heart rate were monitored during the last 30 seconds at higher work loads and $\dot{V}O_{2max}$ was defined according to the "leveling off" criterion.

The following protocol was applied to determine the onset of blood lactate accumulation (OBLA) during cycling (21, 30). After a 3-4 min warm-up period at 60-90 W, initial work load was set individually. This load ranged from 90 to 150 W and equaled 45 (33-58) % of $\dot{V}O_{2max}$. Continuous exercise was performed at
a pedaling frequency of 60 rpm and with a 30 W increment every fourth minute. Subjects cycled at least 4 loads until voluntary exhaustion or near exhaustion. Respiratory parameters and heart rate were monitored during the final 30 seconds of each work load. Venous blood samples were simultaneously collected through an indwelling catheter for subsequent analyzes of lactate concentration (28). The relationship of lactate concentration, oxygen consumption and pulmonary ventilation to exercise intensity was plotted for each subject. OBLA was defined as the exercise intensity which corresponded to a lactate concentration of 4 mmol·l⁻¹ blood. OBLA was found to vary less than 2% in three subjects, who repeated the experiments on two different occasions.

Muscle biopsies (4) were obtained from the vastus lateralis muscle of the left leg at rest. Tissue samples were frozen, treated and analyzed for muscle fiber type composition (% slow twitch (ST) fibers, % ST area), fast twitch (FT), ST and mean fiber area as described by Tesch (34) as well as capillary density (1).

Results

Maximal oxygen uptake and fiber type composition in the present study ranged 39-62 ml·kg⁻¹·min⁻¹ and 34-82% ST area, respectively. Information on some physiological and histochiemical parameters is listed in Table 1. Blood lactate concentration at rest averaged (±SD) 1.4 (±0.3) mmol·l⁻¹. Mean (±SD) values for oxygen consumption, pulmonary ventilation, respiratory quotient (R), heart rate and blood lactate concentration at
different exercise intensities are described in Table 2. Calculated values, obtained from individual plottings, for exercise intensity, oxygen consumption and ventilation at OBLA were 159 (±37) W, 2.43 (±0.47) l·min\(^{-1}\) and 49.8 (±10.5) l·min\(^{-1}\). These values corresponded to an exercise intensity equivalent to 65 (±10) % of \(\dot{V}_{O2max}\). Exercise intensities calculated to correspond to blood lactate concentrations of 3 and 5 mmol·l\(^{-1}\), respectively were found to correlate highly to OBLA (4 mmol·l\(^{-1}\)). The correlation coefficients were 0.94 (3 vs. 5 mmol·l\(^{-1}\)) 0.95 (3 vs 4 mmol·l\(^{-1}\)) and 0.99 (4 vs. 5 mmol·l\(^{-1}\)). A positive relationship (\(r=0.75, p<0.001\)) was demonstrated between the relative exercise intensity (% of \(\dot{V}_{O2max}\)) equivalent to OBLA and % ST area (Fig. 1) i.e. the larger the volume of ST fibers the closer to \(\dot{V}_{O2max}\) the subjects could exercise. Positive relationships (\(r=0.71-0.72, p<0.01\)) were also present between OBLA and the corresponding \(O_2\) consumption, expressed either absolutely or relative to body weight. No correlation was demonstrated between \(O_2\) consumption at OBLA and % ST area. No significant correlations were obtained between capillary density nor \(\dot{V}_{O2max}\) and OBLA. However, multiple regression analyses revealed that 92% of the variance in OBLA could be explained by % ST area + capillary density (cap·mm\(^{-2}\)) (R=0.96). A slightly, but not significantly higher correlation (R=0.98) was obtained if \(\dot{V}_{O2max}\) was added as a third independent variable. OBLA did not parallel changes in \(\dot{V}_E/\dot{V}_{O2}\), and in most cases a "break away" in \(\dot{V}_E/\dot{V}_{O2}\) response was not even observed.

Discussion

In numerous previous studies onset of lactate accumulation in blood during exercise with progressively increases in intensity
has been examined to describe metabolic changes referred to as aerobic and anaerobic thresholds as suggested by e.g. Skinner and McLellan (32). To assess the "breaking point", which represents a pronounced, increased glycolytic energy contribution, Mader et al. (21) have presented data supporting a "lactate threshold" corresponding to 4 mmol·l⁻¹ blood. Likewise, a leveling-off in lactate release from the exercising muscle occurs when the intramuscular concentration of lactate has reached approximately 4-5 mmol·kg⁻¹ wet weight (16, 18). This is consistent with the findings that the maximal exercise intensity which can be maintained by athletes in endurance events in the order of 30-60 minutes, seems to be performed with similar blood lactate levels (5, 20, 35). In the present study the experimental design introduced by Mader and co-workers was applied to define onset of blood lactate accumulation. To assure adequate metabolic adjustments due to the progressive increments in exercise intensity during testing, each load was performed for four minutes (30). The rationale for using a concentration of 4 mmol·l⁻¹ to define onset of lactate accumulation may be questioned. The fact that the exercise intensity corresponding to this level was highly correlated to exercise intensities equivalent to both 3 and 5 mmol·l⁻¹ blood suggests that alternative concentrations (at least in the range 3-5 mmol·l⁻¹) may reflect the same metabolic events.

The main finding of the present study was the relationship established between OBLA on one hand and fiber type distribution and capillary frequency. Recently, Ivy et al. (13) demonstrated a very close relationship between in vitro pyruvate oxidation
capacity of muscle homogenates and either absolute (oxygen consumption) or relative (to maximal oxygen consumption) lactate threshold. Positive correlations were also observed between lactate threshold and percentage of ST fibers and $\dot{V}O_2^{\text{max}}$.

Sjödin and Jacobs (30) and Sjödin et al. (31) have presented information along the same line. Thus, in marathoners, the mean marathon velocity was directly related to the treadmill running velocity required to elicit the onset of lactate accumulation. Furthermore, the velocity corresponding to OBLA ($V_{OBLA}$) was positively related to % ST area, capillary density and citrate synthetase (CS)/phosphofructokinase or CS/lactate dehydrogenase activity ratio. This suggests that key steps in the metabolic pathways, associated with the regulation of glycolysis and muscle respiration are of significance for the exercise intensity, where lactate starts to accumulate in blood.

The efficiency in the rate of lactate release from muscle is probably reflected by the density of the vascular bed surrounding the activated muscle fibers. One can therefore speculate that the effects of a well developed capillary network during submaximal exercise, where lactate formation occurs would be less likely concerned with the oxygen delivery capacity but probably more related to facilitating efflux of lactate from muscle. In short term, intense, local muscular performance, where oxygen transport to the exercising limb is not essential for actual performance (19, 34), "lactate release" following exercise was found to correlate to capillary density. In contrast during very heavy exercise activating large muscle groups and with a pronounced oxygen demand, the rate of acceleration of $O_2$
uptake at onset of exercise seemed to be dependent on capillary density (Tesch, unpublished observations).

Ample information concerning the differences in the metabolic profile of the two main fiber types in human (1, 8, 29, 34, 36) provides support for a delayed onset of blood lactate accumulation in individuals with muscles rich in ST fibers. As concluded by Ivy and co-workers the "cause-effect" relation in this context is ambiguous. Even though individual fiber type composition seems to be rather constant, modifications may take place as a result of physical training (15). The findings obtained by Pedersen (24) can be interpreted to reflect individual variation in training background. He observed a correlation between lactate threshold and percentage of type IIA fibers. These fibers have been found to increase in proportion to total percentage of type II fibers due to moderately intense aerobic training (10). In contrast to fiber type composition, enzyme activities representing glycogenolysis, Kreb's cycle and the electrontransport chain as well as capillary density are very sensitive to changes in the environmental stress (26). Interestingly, Sjödin et al. (31), who recorded training volume for their subjects during a two month period, were also able to relate differences in OBLA to individual variations in training distance covered prior to their investigation. In running experiments interindividual differences in mechanical efficiency have also to be considered. One can speculate that alterations in mechanical efficiency, independent of changes in the cardiovascular system or the ATP regenerating system of the muscle, may occur as a result of training and thus influencing OBLA (Komi, pers. comm.). In
cycling exercise, on the contrary, mechanical efficiency is relatively constant when comparing individuals (2). Moreover, the efficiency of muscular work during cycling below 80% of \( \dot{V}_{O_2} \text{max} \) at 60 rpm is not influenced by differences in muscle fiber composition (33). Hence, variations in OBLA can solely be explained by differences in the metabolic response to exercise which is determined by factors such as oxygen delivery capacity (25, 27), muscle metabolic profile (26), diet (14), hormonal regulation (9), physical activity level (11, 12, 25) etc.

In conclusion, the combined effect of fiber type composition and capillary density predicts with high accuracy the fraction of \( \dot{V}_{O_2} \text{max} \), which can be utilized during submaximal cycling exercise, without pronounced blood lactate accumulation. It is suggested that both inherent and environmental factors will contribute to this relationship.
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<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
<th>Range</th>
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<tbody>
<tr>
<td>( \dot{V}_{O_2}) max 1·min(^{-1} )</td>
<td>3.78 ± 0.49</td>
<td>3.00-4.50</td>
</tr>
<tr>
<td>( \dot{V}_{O_2}) max ml·kg(^{-1})·min(^{-1} )</td>
<td>48.7 ± 7.7</td>
<td>38.6-61.5</td>
</tr>
<tr>
<td>% ST</td>
<td>54 ± 12</td>
<td>41-81</td>
</tr>
<tr>
<td>% ST area</td>
<td>49 ± 15</td>
<td>34-82</td>
</tr>
<tr>
<td>Mean fiber area, ( 100 \cdot \text{m}^2 )</td>
<td>66 ± 17</td>
<td>39-107</td>
</tr>
<tr>
<td>( F_F/ST ) area</td>
<td>1.22 ± 0.21</td>
<td>0.84-1.52</td>
</tr>
<tr>
<td>Capillary density, ( \text{cap} \cdot \text{mm}^2 )</td>
<td>409 ± 126</td>
<td>240-652</td>
</tr>
<tr>
<td>( \text{cap} \cdot \text{fib}^{-1} )</td>
<td>1.70 ± 0.33</td>
<td>1.12-2.25</td>
</tr>
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TABLE 2. Mean (±SD) values for respiratory parameters, heart rate and blood lactate concentration

<table>
<thead>
<tr>
<th>Work load, W (W)</th>
<th>90</th>
<th>120</th>
<th>150</th>
<th>180</th>
<th>210</th>
<th>240</th>
<th>270</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n)</td>
<td>(9)</td>
<td>(13)</td>
<td>(16)</td>
<td>(16)</td>
<td>(13)</td>
<td>(6)</td>
<td>(2)</td>
</tr>
<tr>
<td>Oxygen consumption, l·min^-1</td>
<td>1.50 ± 0.12</td>
<td>1.87 ± 0.14</td>
<td>2.29 ± 0.16</td>
<td>2.79 ± 0.18</td>
<td>3.16 ± 0.19</td>
<td>3.54 ± 0.17</td>
<td>-</td>
</tr>
<tr>
<td>Ventilation, l·min^-1</td>
<td>38.4 ± 14.1</td>
<td>41.5 ± 9.6</td>
<td>50.3 ± 10.7</td>
<td>71.0 ± 21.9</td>
<td>82.3 ± 26.1</td>
<td>93.3 ± 27.1</td>
<td>127.0 ± -</td>
</tr>
<tr>
<td>Respiratory quotient (R)</td>
<td>0.96 ± 0.12</td>
<td>0.97 ± 0.07</td>
<td>0.97 ± 0.07</td>
<td>1.02 ± 0.09</td>
<td>1.03 ± 0.09</td>
<td>1.04 ± 0.06</td>
<td>1.06 ± -</td>
</tr>
<tr>
<td>Heart rate, beats·min^-1</td>
<td>125 ± 23</td>
<td>132 ± 24</td>
<td>143 ± 23</td>
<td>161 ± 23</td>
<td>169 ± 18</td>
<td>172 ± 10</td>
<td>177 ± -</td>
</tr>
<tr>
<td>Lactate, mmol·l^-1</td>
<td>2.0 ± 0.6</td>
<td>3.0 ± 0.8</td>
<td>3.9 ± 1.3</td>
<td>5.7 ± 2.1</td>
<td>7.0 ± 2.6</td>
<td>8.2 ± 3.1</td>
<td>-</td>
</tr>
</tbody>
</table>
y = 0.52x + 39.11
r = 0.75
p < 0.001
FIGURE LEGEND

Fig. 1. The relationship between muscle fiber type composition (% ST area) of m. vastus lateralis and onset of blood lactate accumulation, expressed as percentage of $\dot{V}_{O_2}$max.