Chronic Oral Lactate Supplementation Does Not Affect Lactate Disappearance From Blood After Exercise

Fred Brouns, Mikael Fogelholm, Gerrit van Hall, Anton Wagenmakers, and Wim H.M. Saris

This study tested the hypothesis that a 3-week oral lactate supplementation affects postexercise blood lactate disappearance in untrained male subjects. Fifteen men were randomly assigned to either a lactate supplementation ($n = 8$) or a placebo ($n = 7$) treatment. During the treatment period they drank an oral lactate or a maltodextrin (placebo) supplement twice a day. The lactate drink contained 10 g of lactate as calcium, sodium, and potassium salts. Blood lactate concentrations were studied before, during, and immediately after three exercise tests, both pre- and posttreatment. Peak lactate values for placebo (PL) or lactate (L) treatment groups during different tests were as follows: Test 1 PL, $13.49 \pm 3.71$; L, $13.70 \pm 1.90$; Test 2 PL, $12.64 \pm 2.32$; L, $12.00 \pm 2.23$; Test 3 PL, $12.29 \pm 2.92$; L, $11.35 \pm 1.38$ and were reached 3 min postexercise. The decrease in blood lactate during the long (30- to 45-min) recovery periods amounted to $@ 10$ mmol/L. Blood lactate changes were highly reproducible. However, a 3-week oral lactate supplementation did not result in differences in lactate disappearance. This study does not support the hypothesis that regular oral lactate intake at rest enhances the removal of lactate during and following exercise, that is, not with the given lactate load and supplementation period.

Key Words: recovery, liver, enzyme induction, lactate clearance

Lactate, an intermediate in carbohydrate metabolism, is produced from pyruvate by cells of several tissues (e.g., muscle, liver, erythrocytes) during resting conditions (1). Physical activity such as running or cycling results in increased lactate production (1, 3). Muscle and blood lactate concentrations increase significantly when production rates exceed lactate clearance rates. This usually occurs during exercise at intensities above 50 to 60% of the maximal working capacity. The simultaneous decrease in muscle pH has been suggested to be associated with the development of local muscular fatigue (3, 5).

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High blood lactates are normally observed after maximal effort lasting 1 to 3 min such as middle-distance running and speed skating, but also after a number of repeated sprints in contact sports or between sprints in endurance events such as cycling competitions. Therefore it has been suggested that an increased removal of lactate may delay the development of fatigue in sports and thus lead to improved performance. Regular training resulting in repeated lactate production periods improves lactate clearance (2, 3) as well as recovery from performance.

Recently some food producers have claimed that regular lactate intake (e.g., by products containing mineral lactate) may improve lactate clearance. For example, Nycomed, the producers of the European sport drink XL-1, claim in their advertising and on their product packaging that the addition of magnesium lactate reduces lactic acid in muscle. To our knowledge there is no study supporting such claims. A possible rationale behind the hypothesis that oral lactate supplementation enhances lactate clearance may be that a regular oral lactate bolus results in a repeated lactate load to the liver, which may stimulate the liver to increase the absolute amount of lactate dehydrogenase, by enzyme induction. A comparable mechanism is observed after regular alcohol intake, which will increase the rate of removal from blood by induction of the enzyme alcohol dehydrogenase (7, 8). Additionally, an increased lactate load may induce the enzymes involved in gluconeogenesis (9).

In the present study we tested the hypothesis that a 3-week oral lactate supplementation affects blood lactate concentrations in untrained male subjects after different bouts of strenuous exercise, during both passive (sitting) and active recovery (at 15% and 45% of maximal work capacity). The rationale for supplying both active and passive recovery is that low intensity exercise during the recovery period following an intense bout of exercise has been shown to increase lactate clearance in comparison with passive rest (4), mainly by direct oxidation of lactate in muscle. We were interested in whether long-term lactate supplementation affects lactate clearance during any of these recoveries. A period of 3 weeks, two times a day, of lactate supplementation was thought to be long enough to exert an effect if any.

Methods

Subjects

Sixteen untrained male students volunteered to participate after they had been informed about the study and possible risks involved. One left the study because of health problems not related to this study. The subjects were randomized in a lactate supplement group ($n = 8$) and a placebo group ($n = 7$). Physical characteristics of both groups are presented in Table 1. Written informed consent was obtained from all subjects. The study was approved by the Ethics Committee of the University of Limburg.

Study Design

The study consisted of three pre- and three posttreatment exercise tests. The last pre- and first posttest were separated by 28 days. During the last 3 weeks of this period the subjects ingested an oral lactate or a maltodextrin (placebo) supplement
Table 1  Physical Characteristics of the Subjects (means ± SD)

<table>
<thead>
<tr>
<th>Treatment group</th>
<th>Age (yr)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Body mass index (kg · m⁻²)</th>
<th>$W_{\text{max}}$ (watts)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo (n = 7)</td>
<td>23</td>
<td>184</td>
<td>78</td>
<td>22.8</td>
<td>304</td>
</tr>
<tr>
<td>±2</td>
<td>±8</td>
<td>±19</td>
<td>±4.3</td>
<td>±11</td>
<td>±33</td>
</tr>
<tr>
<td>Lactate (n = 8)</td>
<td>24</td>
<td>184</td>
<td>71</td>
<td>21.0</td>
<td>294</td>
</tr>
<tr>
<td>±2</td>
<td>±6</td>
<td>±4</td>
<td>±1.1</td>
<td>±33</td>
<td></td>
</tr>
</tbody>
</table>

twice a day. The first bolus was ingested before breakfast and the second bolus just before bedtime. This was done to induce rapid lactate uptake by the gastrointestinal tract, resulting in a high lactate load to the liver.

The lactate drink contained 10 grams of lactate in the following composition: 8.2 g calcium lactate powder, 6.25 ml sodium lactate solution (60% lactate), 2.05 ml potassium lactate solution (60% lactate), diluted to 100 ml with water. The mineral quantity ingested as a result of the lactate salts amounted to 1.5 g calcium, 1 g sodium, and 0.5 g potassium. These samples were prepared by PURAC b.v., The Netherlands. The placebo samples contained 10 g of maltodextrin, Passelli MD6 (AVEBE, The Netherlands) in 100 ml water.

**Exercise Tests**

**Test 1.** This was an incremental maximal workload test ($W_{\text{max}}$) to exhaustion, followed by passive recovery (6). The exercise test began with a 5-min warm-up period at 50 watts. The load was then increased by 50 watts every 2-1/2 min until the subject's heart rate reached 160 bpm; then the increments were reduced to 25 watts every 2-1/2 min until exhaustion. Heparinized blood samples were taken from a forearm venous catheter before the test, at the end of each workload increment, and then after 1, 3, 5, 7, 9, 12, 20, 30 and 45 min of recovery. The subjects were seated during the entire recovery period (Figure 1). The maximal work capacity observed in this test was set at 100% $W_{\text{max}}$ according to Keizer (6) and used to determine the workloads in the other tests.

**Test 2.** This involved supramaximal exercise with active recovery at 15% $W_{\text{max}}$. After a warm-up period at 50 watts, the subjects cycled four repetitions of 1 min at 120% $W_{\text{max}}$. Because of the ergometer's electronic braking system, which results in a 20-sec delay before reaching a certain power output (acceleration time), the effective working time on 120% $W_{\text{max}}$ was about 40 sec. There was a 1-min rest between the supramaximal work bouts. During the recovery period the subjects cycled at 15% of their $W_{\text{max}}$ (Figure 2). Blood samples were taken before the test, at the end of each workload increment, and then after 1, 3, 5, 7, 9, 12, 20, 30, and 45 min of recovery.

**Test 3.** This involved supramaximal exercise with active recovery at 45% $W_{\text{max}}$. The test protocol was identical to Test 2 except that the workload during recovery was 45% $W_{\text{max}}$. The 30-min recovery interval was followed by 3 min
Figures 1, 2, and 3 — Arrows indicate blood sampling. Black areas represent exercise protocol, with exercise intensity as given on the y axes.
at a workload of 100% $W_{\text{max}}$ and a recovery period at 45% $W_{\text{max}}$ of 15 min. Blood samples were taken at rest, after each intense exercise bout, and during the recovery periods at 1, 3, 5, 7, 12, 15, and 30 min (Figure 3).

**Analytical Methods**

**Lactate Analysis**

Blood samples were kept on ice and were centrifuged within 1 hr at 3,500 rpm. Plasma was immediately frozen and kept at $-20^\circ\text{C}$ for not more than 2 weeks. Plasma lactate concentration was determined spectrophotometrically by the lactate dehydrogenase method using a COBAS Bio analyzer (Roche Diagnostica, Switzerland). The intra- and interrun imprecision were 2.0 and 4.1% at mean plasma lactate concentrations of 7.8 and 7.5 mmol · L$^{-1}$, respectively.

**Statistical Analysis**

We were testing the hypothesis that oral lactate supplementation may influence postexercise lactate clearance from the blood by means of enzyme induction in the liver and possibly in muscle tissue. For the statistical analysis, the mean maximal lactate values occurring at 3 min postexercise for all three tests were chosen as a starting point because this reflects the baseline for decreases in lactate concentration caused by clearance. In total, the following 7 sampling points were included in the comparison against this baseline: 2, 4, 6, 9, 12, 17, and 27 min after the reference 0 point.

The effects of group (lactate or placebo, before or after treatment) and time (minutes after maximal lactate concentration) on plasma lactate were tested by analysis of covariance for repeated measurements, using BMDP statistical software (2V program). A significant group-by-trial or group-by-trial-by-time interaction would indicate a possible effect of treatment. In case of a significant interaction, an orthogonal polynomial test (BMDP 2V program) was used to evaluate when and how the slopes (change in blood lactate as a function of time) differed between the groups.

**Results**

Plasma lactate concentration at rest was between 1 and 2 mmol · L$^{-1}$ in all tests and increased during exercise with increasing workload. In Test 1 (see Figure 4) the highest lactate values were observed 3 min after the moment of exhaustion, due to the release of lactate from muscle. Lactate concentration decreased in the recovery period but did not reach preexercise values within 45 min of passive recovery. No significant effects of lactate supplementation were found ($p > 0.05$). There were no differences in total work output between lactate and placebo treatment pretreatment ($W_{\text{max}} \pm SE$, watt), respectively, 304 ± 4 and 294 ± 13, or posttreatment, 296 ± 10 and 291 ± 14.

In Test 2 the peak lactate values 3 min after the fourth supramaximal repetition at 120% of $W_{\text{max}}$ were only slightly lower (Figure 5) than after the maximal workload achieved in Test 1. Plasma lactate decreased steadily during active (15% $W_{\text{max}}$) recovery but did not reach resting levels at 30 min postexercise.
Figure 4 — Incremental maximal workload test ($W_{\text{max}}$) followed by passive recovery (Test 1). Pre- and posttreatment values for placebo ($n = 7$), respectively, □, Δ, and for the lactate group ($n = 8$), ■, and ▲.

Compared to all other lactate curves, the placebo group had lower postexercise lactate levels before the treatment. This was statistically verified by significant group-by-trial ($p = 0.03$) and group-by-trial-by-time ($p = 0.0005$) interactions. According to the orthogonal polynomial analysis, the slopes for changes in blood lactate concentration differed between groups before ($p = 0.004$) but not after ($p > 0.05$) the treatment.

In Test 3 the maximal lactate concentrations similar to Test 2 were measured 3 min after the fourth supramaximal repetition (Figure 6). Also the blood lactate decrease was similar in Tests 2 and 3. Lactate concentrations after the 3-min maximal work were somewhat lower than after the supramaximal repetitions. No significant treatment effects were found ($p > 0.05$).

**Discussion**

Oral lactate supplementation did not lead to changes in resting blood lactate as a result of lactate absorption. However, this absence of an increase in forearm venous blood lactate does not necessarily imply that the lactate load given did not reach liver and muscle. The lactate may all be absorbed and appear in the vena portae. No increase in venous forearm lactate will be seen, however, when the liver extracts all the lactate from the blood or when liver and skeletal muscle together extract all the lactate. Blood sampling at multiple sites (e.g., arterial sampling) or isotopic lactate tracers are required to investigate the absorption and metabolic availability of the lactate in greater detail.

In none of the experiments did the 3-week daily lactate supplementation result in a difference in blood lactate concentration during and following exercise.
Figure 5 — Supramaximal exercise with active recovery at 15% $W_{\text{max}}$ (Test 2). Pre- and posttreatment lactate concentrations for placebo ($n = 7$), respectively, $\square$, $\triangle$, and for the lactate group ($n = 8$), $\blacksquare$, and $\triangle$.  

Figure 6 — Supramaximal exercise with active recovery at 45% $W_{\text{max}}$ (Test 3). Pre- and posttreatment lactate concentrations for placebo ($n = 7$), respectively, $\square$, $\triangle$, and for the lactate group ($n = 8$), $\blacksquare$, and $\triangle$. 
Evidently lactate treatment had no effect on postexercise blood lactate disappearance in Tests 1 and 3. For reasons unknown, the placebo group had lower postexercise lactate levels in Test 2 before the treatment, but no changes were observed posttreatment in the lactate group either.

One may speculate on possible reasons for the absence of the hypothesized effect: First, it is possible that the lactate is metabolized by the intestinal cells, thus not leading to a significant lactate supply to the liver. Second, it is possible that the given dose of lactate per se is too small to lead to enzyme induction. To our knowledge there is no information on this quantitative aspect. It is not ethical to perform liver biopsies in healthy individuals, and direct measurements of lactate dehydrogenase enzyme in liver are therefore not possible.

In conclusion, the present data do not support the hypothesis that oral lactate consumption enhances lactate removal from blood during and after exercise. Because dairy products and sport drinks containing mineral lactates are well below the level of lactate supplemented in this study, promotional claims by food producers to this effect must be discounted.

References


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