RESEARCH ARTICLE

Reliability of urine lactate as a novel biomarker of lactate production capacity in maximal swimming

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Abstract

Context: Postexercise urine lactate may be a novel biomarker of lactate production capacity during exercise.

Objective: To evaluate the reliability and utility of the urine lactate concentration after maximal swimming trials between different training protocols (6 × 50 m and 3 × 100 m) and training states (active and nonactive swimmers).

Materials and methods: Lactate and creatinine were determined by spectrophotometry in blood and urine.

Results: Blood and urine lactate concentrations were correlated in-between training protocols and in participants of different training states. The reliability of the urine lactate concentration was moderate for one of the training protocols and good or moderate for the two training states. Additionally, it was lower than that of the blood lactate concentration, and did not improve after normalizing to the urine creatinine concentration.

Discussion and conclusion: Although promising as a biomarker of lactate production capacity, urine lactate requires further research to improve its reliability.

Keywords

Swimming, anaerobic capacity, reliability, test–retest, intraclass correlation coefficient

History

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Introduction

Swimming is a competitive sport requiring a combination of physical capabilities (Smith et al., 2002). Lactate production training is of major importance for swimming performance, especially in short-distance events (Maglischo, 2003), such as those of 50 and 100 m, which rely mostly on anaerobic metabolism for ATP resynthesis (Colwin, 2003; Gastin, 2001; Maglischo, 2003; Mougios, 2006). Hence, a large part of swimming training is devoted to the improvement of lactate production capacity, which makes it highly desirable to evaluate performance during training phases in order to monitor the training process and ensure its success (Maglischo, 2003).

A valid and reliable biomarker of lactate production capacity and potentially anaerobic capacity is the peak blood lactate concentration after maximal exercise (Maglischo, 2003; Mougios, 2006; Viru & Viru, 2001). This parameter correlates with the muscle lactate concentration and anaerobic performance (Colwin, 2003; Maglischo, 2003; Mougios, 2006; Viru & Viru, 2001) and, thanks to its relative ease of measurement, it is used widely in training practice to evaluate and monitor adaptations of the lactate system of energy production.

The postexercise blood lactate concentration presents high repeatability in test–retest exercise protocols (Fatourou et al., 2011; Glaister et al., 2007; Mujika et al., 2006; Turner et al., 2008). In earlier studies, high lactate values were also found in urine after strenuous exercise (Johnson & Edwards, 1937; Judge & Eys, 1962; Liljestrand & Wilson, 1925; McKelvie et al., 1989), showing a positive relationship with the blood values (Johnson & Edwards, 1937; Miller & Miller, 1949). In addition, recent evidence suggests that the urine lactate concentration reflects the amount of lactate produced in the exercising muscles and may be a more sensitive biomarker of exercise metabolism than blood lactate (Pechlivanis et al., 2010; Saraslanidis et al., 2011; Tsalis et al., 2011). Nevertheless, to our knowledge, there are no studies regarding the reproducibility of the postexercise urine lactate concentration and its utility in assessing anaerobic capacity.

A number of swimming training sets are used by coaches, aiming at maximizing lactate production and, hence, swimming performance in short-distance events. The most suitable repeat distances in such sets are proposed to be 25 and 50 m; for example, a set of six maximal 50 m bouts on 5 min is considered one of the most appropriate for the development of lactate production capacity (Maglischo, 2003). Indeed, very high blood lactate values have been found after the end of such a set (Kabasakalis et al., 2014). The duration of each bout is suggested not to exceed 30 s because longer bouts...
might decelerate anaerobic metabolism due to early severe acidosis. Nevertheless, high blood lactate concentrations were also found after repeated maximal 100 m bouts lasting around 1 min (Collomp et al., 1992; Gao et al., 1988; Mero et al., 2004; Toubekis et al., 2008; Trappe et al., 1994). To our knowledge, there are no studies in which anaerobic swimming sets of different repeat distances have been compared in order to assess their suitability for the development of lactate production capacity in competitive swimmers. What is more, possible novel biomarkers, such as urine lactate, which could provide such information, have not been investigated.

It is known that athletes lose part of their adaptations after interruption or cessation of training (Maglischo, 2003; Mougios, 2006). Such detraining compromises swimming performance (Mujika et al., 1995) and could induce small, nonsystematic changes in the activities of muscle glycolytic enzymes (Mujika & Padilla, 2001), which are directly related to the lactate system of energy production (Mougios, 2006). However, we are unaware of studies that have gone beyond 12 weeks of detraining or studies that have addressed the effect of detraining on the maximal lactate production capacity in former competitive athletes.

On the basis of the aforementioned shortcomings of the literature, we conducted two studies to investigate the reliability and utility of the postexercise urine lactate concentration as a biomarker of lactate production capacity by examining two practical issues in swimming training. Specifically, in Study 1, two swimming training sets, consisting of repeated 50 m or 100 m maximal bouts, were compared in order to investigate the suitability of each set for lactate production capacity training. In Study 2, we compared two different training states, active and nonactive former swimmers, in terms of maximal lactate production capacity.

**Methods**

**Study 1**

**Subjects**

Fourteen healthy adolescent, national-level competitive male swimmers (age, 15.3 ± 1.0 years; body mass, 70.2 ± 6.9 kg; height, 1.79 ± 0.05 m; BMI, 21.9 ± 2.1 kg·m⁻²; mean ± SD throughout) participated in the study. The participants trained at least six times a week and had a training experience of 4.6 ± 1.1 years. The swimmers and their parents were informed about the purpose, benefits, and risks of the study and then gave their written consent to participate. Procedures were in accordance with the Helsinki declaration and were approved by the Institutional Review Board.

**Exercise testing**

The study was conducted over two-week period in a test–retest fashion with all swimmers being at the same training phase. On the first week, the swimmers performed two freestyle swimming sets with maximum effort: six 50 m bouts on 5 min (that is, one every 5 min) and three 100 m bouts on 10 min. The sets were spaced three days apart and were executed in a random, counterbalanced order with passive recovery between swimming bouts. The same protocol was repeated the following week. Thus, each participant performed four trials (two of each kind), spaced 3–4 days apart. The swimmers started each bout by pushing off the wall inside the pool. Swimming times were recorded with the use of an electronic stopwatch (Stopwatch Selecta, W10710, Waterfly). Before each trial, the swimmers performed a light 10 min warm-up. The trials were performed in the afternoon (between 4 and 5 pm) in the fed state, in an indoor 50 m swimming pool at a water temperature of 26–27°C. On the week preceding the tests, the swimmers performed a 50 m and an 100 m freestyle maximal bout (on different days) in order to record each one’s current best performance and express performance at the subsequent tests as percentage of that (% seasonal best, SB).

**Blood and urine sampling**

Fourteen microliters of capillary blood were obtained from a fingertip at 2.5, 5, 7.5 and 10 min after the end of each set, hemolysed with 140 µL of 0.3 mol·L⁻¹ perchloric acid and stored at −80°C until analyzed to determine the peak postexercise lactate concentration. The highest lactate value among the postexercise samples of each individual was recorded as his peak postexercise value. The swimmers emptied their bladders 1.5 h before the onset of each test and then drank 0.5 L of water. They emptied their bladders again right before the test to ensure that any urine collected postexercise would have been produced from the onset of exercise onward. Then, they emptied their bladders into urine collection cups at 1 h after the end of each test. (Preliminary tests in our laboratory showed that waiting one hour after maximal exercise was necessary to maximize the urine lactate concentration). During this hour, the swimmers drank at least 0.5 L of water (or more, if desired). About 1 mL from each urine sample was stored at −80°C until analyzed for lactate and creatinine, while the rest was discarded.

Resting blood and urine samples were not collected, as their lactate concentrations are usually low, in the range of 1 to 2 mmol·L⁻¹ (Pechlivanis et al., 2010), and there was no point in examining the reliability of the resting urine lactate concentration.

**Dietary control**

The swimmers recorded their food intake (on dietary record forms, after having received detailed instructions) during the day before and the day of the first trial up to the time of arrival at the swimming pool. Then, they were asked to follow the same diet and record their food intake before the subsequent trials. The dietary records were analyzed for energy and macronutrients in Microsoft® Access through the use of a food database created in our laboratory on the basis of published data (Food Standards Agency, 2002).

**Study 2**

**Subjects**

Eighteen healthy male active and eighteen healthy male nonactive former swimmers participated in the study. The active group (age, 22.1 ± 2.6 years; body mass, 77.2 ± 7.9 kg; height, 1.80 ± 0.05 m; BMI, 23.9 ± 2.5 kg·m⁻²; mean ± SD throughout) were former competitive swimmers with a training experience of 9.1 ± 3.1 years, who had retired from...
characteristic training 3 ± 2 years before the study, but continued to train 3–5 times per week for about 75 min per day. The nonactive group (age, 23.9 ± 3.1 years; body mass, 83.9 ± 12.2 kg; height, 1.79 ± 0.05 m; BMI, 26.2 ± 3.2 kg·m⁻²) were also former competitive swimmers with a training experience of 7.2 ± 2.6 years, who had abandoned any exercise training program for at least one year before the study. The active and nonactive groups did not differ significantly in age, body mass, height or training experience. However, they differed in BMI (p = 0.026) and SB (12.9 ± 0.5 versus 13.9 ± 0.7 s, respectively, p < 0.001).

Both active and nonactive former swimmers had been skilled national-level swimmers, that is, finalists or medalists in national swimming championships of juniors, youth and/or open categories. The participants were informed about the purpose, benefits and risks of the study and gave written consent to participate. Procedures were in accordance with the Helsinki declaration and were approved by the Institutional Review Board.

Exercise testing

The participants performed eight 25 m maximal bouts of freestyle swimming on 2 min with passive recovery in between. The swimmers started each bout by pushing off the wall inside a 25 m indoor pool. The same trial was repeated one week later. Water temperature, warm-up, recording of swimming times and dietary control were the same as in Study 1. The trials were performed in the morning (10:30–11:30 am) in the fed state. One week before the first test, the swimmers performed a 25 m freestyle maximal bout in order to record each one’s current best performance and express performance at the subsequent tests as a percentage of that (% SB).

Blood and urine sampling

One capillary blood sample was obtained in the same manner as in Study 1, 5 min after the end of each test. Sampling time was dictated by a pilot study, which showed that blood lactate peaked at 5 min postexercise. The participants emptied their bladders right before the onset of the trials. Then, they emptied their bladders into urine collection cups at 1 h after the end of each trial. During the first half-hour postexercise, the swimmers drank 0.5 L of water. Urine samples were treated as in Study 1.

Biochemical analyses (for both studies)

After thawing, the hemolysates and urine samples from both studies were centrifuged at 1500 g for 5 min. Subsequently, the supernatant of each urine sample was diluted 100 times with water to bring the lactate and creatinine concentrations within measurable ranges. Blood and urine lactate, as well as urine creatinine, were then measured as described (Pechlivanis et al., 2010). All samples from one individual were analyzed on the same day. The intra-assay coefficient of variation was 4% for lactate and 1% for creatinine.

Statistical analyses (for both studies)

Results are presented as mean±SD. Normality of data distribution was checked with the Shapiro–Wilks test. Characteristics of the two groups in Study 2 were compared by Student’s t-test. The reliability of all the variables that were measured in test and retest was assessed by calculating the intraclass correlation coefficient (ICC). The ICC is ideally 1 (full agreement), and reliability is considered significant when the ICC is significantly different from 0. In addition to significance, we adopted the following terminology to describe the meaningfulness of a significant reliability: low (ICC < 0.5), moderate (0.5 ≤ ICC < 0.65), good (0.65 ≤ ICC < 0.8), high (0.8 ≤ ICC < 0.9) and excellent (ICC ≥ 0.9). Two-way ANOVA, that is, trial (test, retest) × set (6 × 50 m, 3 × 100 m) for Study 1 and trial (test, retest) × group (active, nonactive) for Study 2, with repeated measures on trial and set, was used to compare variables related to the swimming tests. Significant interactions were followed up by simple main effect analysis. Pearson’s r or Spearman’s ρ correlation analysis, as appropriate, between swimming time, blood lactate, and urine lactate was also performed. The level of statistical significance was set at α = 0.05. Data were analyzed in SPSS 19 (SPSS, Chicago, IL).

Results

Study 1

Swimming speed at the 6 × 50 m and 3 × 100 m sets corresponded to 96.3 ± 1.9% and 97.4 ± 1.5% of the swimmers’ SB, respectively. Performance and biochemical data at test and retest, as well as their reliability, are presented in Table 1. Reliability of performance between test and retest was excellent for both sets (ICC > 0.9), providing a strong basis for examining the reliability of the biochemical parameters. In addition, all dietary parameters exhibited significant reliability that ranged from low to high (ICC 0.460–0.850, Table 2). The blood lactate concentration showed high repeatability in both sets (ICC 0.878 and 0.802 for 6 × 50 m and 3 × 100 m, respectively). However, the urine lactate concentration displayed significant (moderate) repeatability only after the 3 × 100 m set (ICC 0.558). To control for possible differences in urinary excretion rate between test and retest, we normalized the lactate concentration to the creatinine concentration (a widespread practice with urine metabolites in clinical chemistry). However, the repeatability of the urine lactate-to-creatinine ratio was lower than that of the urine lactate concentration and not significant in either set.

A significant correlation was found between the % SB of 100 m and the urine lactate concentration after the 3 × 100 m set (r = 0.400, p = 0.035), but not in the case of blood lactate concentration after the 3 × 100 m set (r = 0.158, p = 0.421, Figure 1). Additionally, significant correlations were found between the blood and urine lactate concentrations after both the 6 × 50 m and 3 × 100 m trials (r = 0.499, p = 0.007; and r = 0.380, p = 0.046, respectively, Figure 2).

No significant main effects of trial (p = 0.794) or set (p = 0.296) and no significant interaction of the two (p = 0.863) were found for the blood lactate concentration. This was also the case with the urine lactate concentration (p = 0.734, 0.347 and 0.871, respectively).
Study 2

Swimming speeds corresponded to 97.1 ± 3.2% of the active swimmers’ SB and 93.9 ± 3.8% of the nonactive swimmers’ SB (p < 0.001). Performance and biochemical data at test and retest, as well as their reliability, are presented in Table 3. Reliability of performance between test and retest was excellent for both groups (ICC > 0.9). The dietary parameters exhibited moderate to excellent repeatability (ICC 0.515–0.977, Table 4). The blood lactate concentration showed high repeatability in the active group and good repeatability in the nonactive group (ICC 0.828 and 0.713, respectively). Regarding the urine lactate concentration, repeatability was good for the active and moderate for the nonactive group (ICC 0.734 and 0.515, respectively).

Significant correlation was found between the blood and urine lactate concentrations in both the active and nonactive groups (r = 0.574, p < 0.001; and r = 0.400, p = 0.016, respectively, Figure 3).

Significant main effects of trial (p = 0.001) and group (p < 0.001), as well as a significant interaction between variables (p = 0.006), were found regarding 25 m performance. Simple main effect analysis pinpointed the reason for the interaction at the decrease in swim time between test and retest of the nonactive group (p < 0.001), suggesting a training effect of just one set in this group. A significant main effect of

Table 1. Performance and biochemical parameters (mean±SD), as well as intraclass correlation coefficient (ICC) and its 95% confidence interval (CI) between test and retest of the 6 × 50 m and 3 × 100 m swim trials (n = 14).

<table>
<thead>
<tr>
<th>Variables</th>
<th>6 × 50 m</th>
<th>3 × 100 m</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test</td>
<td>Retest</td>
</tr>
<tr>
<td>Average time of each bout (s)</td>
<td>29.0 ± 1.1</td>
<td>28.8 ± 1.1</td>
</tr>
<tr>
<td>Peak blood lactate (mmol·L⁻¹)</td>
<td>18.1 ± 2.5</td>
<td>18.5 ± 2.7</td>
</tr>
<tr>
<td>Urine lactate (mmol·L⁻¹)</td>
<td>93.5 ± 53.5</td>
<td>82.4 ± 37.0</td>
</tr>
<tr>
<td>Urine creatinine (mmol·L⁻¹)</td>
<td>4.4 ± 2.0</td>
<td>4.4 ± 1.6</td>
</tr>
<tr>
<td>Urine lactate/creatinine</td>
<td>23.7 ± 18.4</td>
<td>19.8 ± 9.0</td>
</tr>
</tbody>
</table>

Table 2. Dietary parameters (mean±SD), as well as intraclass correlation coefficient (ICC) and its 95% confidence interval (CI) between test and retest of the 6 × 50 m and 3 × 100 m swim trials (n = 14).

<table>
<thead>
<tr>
<th>Variables</th>
<th>6 × 50 m</th>
<th>3 × 100 m</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test</td>
<td>Retest</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy intake (kcal)</td>
<td>5014 ± 1392</td>
<td>4928 ± 1636</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>604 ± 206</td>
<td>648 ± 289</td>
</tr>
<tr>
<td>Carbohydrate (% energy)</td>
<td>94 ± 17</td>
<td>94 ± 21</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>210 ± 64</td>
<td>204 ± 71</td>
</tr>
<tr>
<td>Fat (% energy)</td>
<td>76 ± 16</td>
<td>75 ± 18</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>176 ± 48</td>
<td>190 ± 55</td>
</tr>
<tr>
<td>Protein (% energy)</td>
<td>29 ± 4</td>
<td>31 ± 5</td>
</tr>
</tbody>
</table>

All dietary parameters correspond to total energy or nutrient intake during one and a half day before each trial.

Figure 1. Correlation of the blood lactate concentration (A) and urine lactate concentration (B) after the 3 × 100 m test with swimming speed as percentage of seasonal best at 100 m (n = 14). Regression lines and 95% confidence intervals are shown.
trial \((p = 0.006)\) was found in the blood lactate concentration, being the retest values lower than the test ones in both groups. No significant outcome was produced by the analysis of variance (ANOVA) of the urine lactate data.

**Discussion**

The present work examined if urine lactate could serve as a novel biomarker of lactate production capacity during exercise by addressing two research questions of not only theoretical but also practical interest in competitive sport. First, which intermittent training protocol was best suited for maximizing lactate production capacity and, secondly, if there was a detraining effect on maximal lactate production capacity. Ideally, these questions should have been answered through muscle lactate determination. However, it is not practical to obtain muscle biopsy. Thus, we resorted to blood lactate, a widely accepted index of muscle metabolism, and introduced urine lactate, for which there is evidence that it reflects the amount of lactate produced in the exercising muscles and could be a more sensitive index than blood lactate (Pechlivanis et al., 2010; Tsalis et al., 2011).

**Table 3.** Performance and biochemical parameters (mean± SD), as well as intraclass correlation coefficient (ICC) and its 95% confidence interval (CI) between test and retest for active \((n = 18)\) and nonactive swimmers \((n = 18)\).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Active</th>
<th></th>
<th>Nonactive</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test</td>
<td>Retest</td>
<td>ICC</td>
<td>95% CI</td>
</tr>
<tr>
<td>Average 25 m time (s)(^a)</td>
<td>13.3 ± 0.6</td>
<td>13.3 ± 0.6</td>
<td>0.981(^b)</td>
<td>0.952–0.993</td>
</tr>
<tr>
<td>Blood lactate (mmol·L(^{-1}))</td>
<td>17.1 ± 2.7</td>
<td>16.5 ± 3.2</td>
<td>0.828(^b)</td>
<td>0.606–0.932</td>
</tr>
<tr>
<td>Urine lactate (mmol·L(^{-1}))</td>
<td>110.6 ± 44.4</td>
<td>110.9 ± 53.9</td>
<td>0.734(^b)</td>
<td>0.427–0.891</td>
</tr>
</tbody>
</table>

\(^a\)Significant main effects of trial (test, retest) and group (active, nonactive), as well as a significant interaction \((p<0.01)\).

\(^b\)Significantly different from 0 \((p<0.05)\).

\(^c\)Significant main effect of trial \((p = 0.006)\).

**Table 4.** Dietary parameters (mean± SD), as well as intraclass correlation coefficient (ICC) and its 95% confidence interval (CI) between test and retest for active \((n = 18)\) and nonactive swimmers \((n = 18)\).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Active</th>
<th></th>
<th>Nonactive</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Test</td>
<td>Retest</td>
<td>ICC</td>
<td>95% CI</td>
</tr>
<tr>
<td>Energy intake (kcal)</td>
<td>2893 ± 724</td>
<td>2836 ± 701</td>
<td>0.977(^a)</td>
<td>0.941–0.991</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>367 ± 104</td>
<td>362 ± 104</td>
<td>0.943(^a)</td>
<td>0.858–0.978</td>
</tr>
<tr>
<td>Carbohydrate (% energy)</td>
<td>51 ± 5</td>
<td>51 ± 6</td>
<td>0.790(^a)</td>
<td>0.531–0.915</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>107 ± 40</td>
<td>104 ± 38</td>
<td>0.886(^a)</td>
<td>0.726–0.955</td>
</tr>
<tr>
<td>Fat (% energy)</td>
<td>33 ± 8</td>
<td>33 ± 8</td>
<td>0.835(^a)</td>
<td>0.619–0.934</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>113 ± 39</td>
<td>113 ± 37</td>
<td>0.954(^a)</td>
<td>0.883–0.982</td>
</tr>
<tr>
<td>Protein (% energy)</td>
<td>16 ± 4</td>
<td>16 ± 4</td>
<td>0.893(^a)</td>
<td>0.743–0.958</td>
</tr>
</tbody>
</table>

All dietary parameters correspond to total energy or nutrient intake during one and a half day before each trial.

\(^a\)Significantly different from 0 \((p<0.05)\).
Specifically, in two intermittent running tests differing in rest interval duration, a significant effect on postexercise urine lactate (but not on blood lactate) was found, while urine lactate showed a greater increase postexercise than blood lactate (on average 45-fold versus 6-fold). These findings suggest that urine lactate could be a more sensitive biomarker of lactate production capacity than lactate itself. However, a novel biomarker must be reliable in order to be usable. Since no data on the reliability of the urine lactate concentration were available, we performed all exercise tests in duplicate, using the well-accepted reliability of the blood lactate concentration as benchmark.

The significant correlation between the blood and urine lactate concentrations and the near identity of the ANOVA outcomes (i.e. no significant findings with either the blood or urine lactate concentrations) in both studies of the present work enhance the possibility of urine lactate being a valid biomarker of anaerobic metabolism in muscle during exercise. In addition, the significant correlation between an index of performance (% SB) and the urine, but not blood, lactate concentration in the 3×100 m set suggests, again, that urine lactate might be a more sensitive exercising biomarker than blood lactate. To this, one should add the advantage of the noninvasive nature of urine sampling, while, of course, acknowledging practicality issues such as having to wait for one hour to sample and the lack of portable devices for urine lactate analysis at present. Additionally, further research will be needed to examine whether urine is a suitable biofluid for frequent postexercise sampling and lactate measurement as a way of evaluating lactate clearance and, through this, aerobic capacity, a possibility offered by blood lactate.

Despite the aforementioned potential advantages of the urine over the blood lactate determination, the reliability of the former was lower than that of the latter. This may be due to the fact that any lactate leaving the exercising muscles encounters additional sources of biological variability (such as absorption by other organs and glomerular filtration) before reaching the urine one hour later, as compared to blood lactate that is measured a few minutes after exercise. Seeing this difference in Study 1 and assuming that hydration might play a role, we established a stricter hydration control in Study 2, that is, a fixed amount of water (equal or less, compared to Study 1) in a shorter period of time. This resulted in improved reliability of the urine lactate concentration, although it remained lower than the corresponding in blood. Anastasio et al. (2001) reported that, in healthy men, glomerular filtration rate was influenced by the hydration level, being higher with low compared to high hydration. Therefore, it is possible that increased glomerular filtration rate, due to decreased hydration, results in more lactate being excreted in the urine and less being absorbed by other organs, thus diminishing the variability of its concentration in urine.

Regarding the two swimming sets, we found that the 6×50 m on 5 min and 3×100 m on 10 min did not differ in terms of blood or urine lactate concentration, which suggests that both sets are equally suitable for the improvement of lactate production capacity for healthy adolescent national-level competitive male swimmers. Although we recently reported on the blood lactate concentration after a 6×50 m swimming set (showing values around 18 mmol·L⁻¹, Kabasakalis et al., 2014, as in the present study), this is the first time where two anaerobic sets are compared. Our findings indicate that sets constructed with 100 m repeated bouts can improve the maximal lactate production capacity as efficiently as sets constructed with shorter distances. Nevertheless, the rest interval between bouts should be proportional to the distance covered in each bout to ensure adequate recovery and, hence, equivalence of the sets for the development of maximal lactate production capacity.

Regarding the detraining effect on lactate production capacity, we found that active and nonactive swimmers did not differ in either their blood or urine lactate values, despite a clear detraining effect on performance. It seems that retaining a part of swimming training for a limited period had less influence on lactate production capacity than the long periods of past competitive swimming training. Thus, lactate production capacity is probably a rather residual training adaptation in competitive swimmers. The different BMI of the
two groups could, in theory, have affected the results, although lactate kinetics was not found to be influenced by different weight or BMI in other athletes (Karniničić et al., 2013). Our findings are also in agreement with those of studies that have reported well maintained adaptations in the activities of muscle glycolytic enzymes (summarized in Mujika & Padilla, 2001), although none of those studies exceeded 12 weeks of detraining. Thus, our data, based on comparison between active swimmers training 3–5 times per week and nonactive swimmers, suggest that maximal lactate production capacity is maintained after at least one year of swimming training cessation.

In practice, our findings suggest that swimming coaches may use sets of either 50 m or 100 m bouts, adding up to the same total distance, with proportional recovery between bouts, in order to develop the lactate production capacity of their swimmers. They should probably not be concerned about their swimmers’ losing part of their established maximal lactate production capacity during rest periods or taper and may thus spend time on other important training parameters instead.

Conclusion

Although the reliability of the postexercise blood lactate concentration ranged from good to high, that of the urine lactate concentration ranged from non-significant to good in different trials and participants. Nevertheless, the blood and urine lactate concentrations were correlated, while urine lactate was the marker that correlated with performance in the case of the longer distance used in the present study. Thus, the utility of the urine lactate concentration as a biomarker of lactate production capacity merits further investigation. Future research with a variety of exercise modalities, types of participants, hydration regimens etc. should aid in exploring the validity and reliability of this parameter to the benefit of accurate planning of training and assessment of training outcomes.

Declaration of interest

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References


