Effects of Lactate Consumption on Blood Bicarbonate Levels and Performance During High-Intensity Exercise

David M. Morris, Rebecca S. Shafer, Kimberly R. Fairbrother, and Mark W. Woodall

The authors sought to determine the effects of oral lactate consumption on blood bicarbonate (HCO$_3^-$) levels, pH levels, and performance during high-intensity exercise on a cycle ergometer. Subjects ($N=11$) were trained male and female cyclists. Time to exhaustion (TTE) and total work were measured during high-intensity exercise bouts 80 min after the consumption of 120 mg/kg body mass of lactate (L), an equal volume of placebo (PL), or no treatment (NT). Blood HCO$_3^-$ increased significantly after ingestion of lactate ($p<.05$) but was not affected in PL or NT ($p>.05$). No changes in pH were observed as a result of treatment. TTE and total work during the performance test increased significantly by 17% in L compared with PL and NT ($p=.02$). No significant differences in TTE and total work were seen between the PL and NT protocols ($p=.85$). The authors conclude that consuming 120 mg/kg body mass of lactate increases HCO$_3^-$ levels and increases exercise performance during high-intensity cycling ergometry to exhaustion.

Keywords: acidosis, buffering, pH, ergogenic

Substantial evidence suggests that metabolic acidosis is a contributing factor to fatigue during prolonged, high-intensity exercise (Adams, Fisher, & Meyer, 1991; Hultman, Del Canale, & Sjöholm, 1985; Raymer, Marsh, Kowalchuk, & Thompson, 2004; Spriet, Matsos, Peters, Heigenhauser, & Jones, 1985). Hydrogen ions produced in exercising muscle are transported to the bloodstream and buffered by blood bicarbonate (HCO$_3^-$) in an attempt to maintain normal pH in the muscle cell. It has been suggested that pH gradients between the muscle and blood influence the rate at which protons are transported out of the muscle and that alkalinizing the blood can improve buffering capacity and help maintain pH levels in the working muscle that are suitable for the maintenance of high-intensity exercise (Bishop, Edge, Davis, & Goodman, 2004). Indeed, numerous investigations have demonstrated that oral consumption of sodium bicarbonate (NaHCO$_3$) effectively increases blood HCO$_3^-$ and pH levels and improves tolerance of intense exercise (Matson & Tran, 1993). Furthermore, some of these investigations have revealed significant, positive correlations between the amount of NaHCO$_3$ consumed and both blood pH levels and exercise performance (Matson & Tran, 1993).

The metabolism of orally consumed lactate can also influence blood HCO$_3^-$ and pH levels. Metabolic fates of lactate include oxidation

\[ C_3H_5O_3^- + 3 O_2 + H^+ \rightarrow 3 CO_2 + 3 H_2O \]

and conversion into glucose

\[ 2 C_3H_2O_3^- + 2 H^+ \rightarrow C_6H_12O_6 \]

In either case, hydrogen ions are absorbed, which can lead to increases in pH or HCO$_3^-$ levels. Early studies of lactate consumption focused on its use as an energy substrate and its effects on endurance-exercise performance. Although ergonomic effects were not found, those investigations demonstrated that orally consumed lactate could raise blood HCO$_3^-$ and pH to levels similar to those found in studies of NaHCO$_3$ consumption (Bryner, Hornsby, Chetlin, Ullrich, & Yeater, 1998; Fahey et al., 1991; Swensen, Crater, Bassett, & Howley, 1994). The ability of orally consumed lactate to raise blood HCO$_3^-$ and pH levels has led to speculation that it may be effective in improving exercise performance during exhaustive, high-intensity exercise. Van Montfoort, Van Dieren, Hopkins, and Shearman (2004) studied the effects of lactate consumption on blood HCO$_3^-$ and pH and exercise performance during single, high-intensity treadmill runs to exhaustion. They found that blood pH and HCO$_3^-$ were higher after consumption of lactate than after consumption of a placebo, but preconsumption values for these variables were not reported, so the effects of their lactate-consumption strategy on blood pH and HCO$_3^-$ levels are not entirely clear. However, exercise performance did improve by 1.7% after the consumption of sodium lactate in comparison with placebo.

Although the results of Van Montfoort et al. (2004) are encouraging, their sparse reporting of key physiological data and relatively small performance improvement have left many questions about the metabolic and ergonomic effects of lactate consumption on...
high-intensity exercise performance. Furthermore, the dose used in their investigation (400 mg/kg body mass) required 90 min for complete consumption. Athletes often have numerous preparatory duties in the hours leading up to competitive events, and reducing the time commitment needed to consume an ergogenic aid could ease their preparation. The reproducibility of exercise tests to exhaustion has also been questioned. Variations in performance of 11% have been documented in a group of competitive runners who performed endurance runs to exhaustion at VO2max velocity on two separate occasions (Billat, Renoux, Pinoteau, Petit, & Koralsztein, 1994). Those results cast some doubt as to whether the use of repeated, high-intensity work bouts (Matson & Tran, 1993). Thus, we sought to study the effects of a relatively low oral dose of calcium lactate on blood HCO3–, pH, and exercise performance during repeated, high-intensity work bouts on a cycle ergometer.

**Methods**

The procedures of this investigation were reviewed and approved by the institutional review board of Appalachian State University. Eleven trained, competitive cyclists (9 men, 2 women) were recruited from the university’s cycling team and the local cycling community. All subjects had at least 1 year of competitive experience and were currently undergoing regular, high-intensity training bouts. All subjects demonstrated their willingness to participate by signing an informed consent before participating in the study. Mean (± SD) descriptive values for these subjects were 22 ± 2 years, 77.1 ± 6.7 kg, 175.5 ± 11.7 cm, and a VO2max of 60.5 ± 6.6 ml·kg⁻¹·min⁻¹.

During their initial visit, subjects performed a maximal exercise test to exhaustion to determine VO2max and maximal power output (MPO). After the maximal test, they performed a practice trial of an interval performance test (IPT) that they were to perform on subsequent visits as part of their experimental trials. All exercise testing was performed on a Lode Excalibur electronically braked bicycle ergometer (Lode, Groningen, Holland) placed in the hyperbolic (cadence-independent) mode with the handlebars and saddle adjusted to the dimensions of each subject’s bicycle.

After the initial visit, the subjects returned on three separate occasions to undergo their IPTs. On one of these occasions, they consumed only water before the test. On the other two visits they consumed either 120 mg of lactate per kilogram body mass or an equal volume of placebo (aspartame) with water before the test. This lactate dose was chosen based on results from a pilot study in which subjects consumed 20, 120, or 220 mg of lactate per kilogram body mass, in the form of calcium lactate, on three separate occasions. Blood HCO3– levels were measured every 20 min after the consumption of these three lactate dosages. In response to each dose, subjects’ blood HCO3– levels peaked at 80–100 min postingestion. Although sizeable increases in peak blood HCO3– levels were seen when the subjects consumed the 120-mg/kg body mass dose compared with the 20-mg/kg body mass dose, no further increases were observed when the dose was increased to 220 mg/kg body mass.

**Maximal Test and Determination of MPO**

The subjects performed a continuous, progressive exercise test to exhaustion beginning at a work rate of 3 W/kg body mass and increasing by 0.3 W/kg body mass every minute in a step-wise fashion until exhaustion. Ventilation volume and expired-gas concentrations were assessed continuously throughout the test and analyzed for oxygen consumption and carbon dioxide production using a Cosmed Quark b2 metabolic cart (Cosmed, Rome, Italy). Before commencement of the maximal test, the subjects performed a 10-min warm-up starting at 100 W below their work rate for the initial stage of the maximal test and progressing by 10 W/min. After the warm-up, subjects were allowed to rest for 5 min before beginning the maximal test. Criteria for a successful test included a plateau in oxygen consumption with an increase in work rate, a respiratory-exchange ratio greater than 1.15, and a maximal heart rate similar to the age-estimated maximal heart rate for the subject (220 – age). Pedaling cadence was monitored throughout the test. The subjects had to complete at least 30 s of the final stage for that work rate to be used as their MPO. Otherwise, the power output from the final completed stage was used for the MPO.

**IPT**

The IPT was similar to a protocol used by Costill, Verstappen, Kuipers, Janssen, and Fink (1984) in their investigation of NaHCO3 ingestion. Subjects performed four 1-min work intervals at 100% of their MPO, each followed by a 1-min recovery interval performed at 25% of MPO. After the final recovery interval, a fifth work interval at 100% of their MPO was performed to exhaustion. During the work intervals, subjects were required to maintain a pedaling cadence similar to that exhibited at their MPO during the maximal test. Exhaustion at the end of the fifth work interval was marked by volitional exhaustion of the subject or if cadence dropped below 50 rpm for more than 8 consecutive seconds. Subjects received verbal encouragement during each IPT from two members of the research team. Time to exhaustion (TTE) was measured on the final work interval, and total work performed during this interval was calculated by multiplying the TTE by the work rate. All IPT protocols were entered into the automatic program feature of the Lode ergometer. Thus, timing for the application and removal of workloads for each test was automatically controlled by the ergometer.
Study Protocol

Each subject underwent the IPT on four occasions. The initial IPT was performed on the subject's first visit, approximately 20 min after the termination of his or her maximal test. This application of the IPT was used as a familiarization trial, and no data from this trial were used in the data analyses. After this initial visit, the subjects returned to the laboratory on three occasions and performed the IPT after consuming 120 mg lactate per kilogram body mass with water (L), an equal volume of placebo with water (PL), or water with no treatment (NT). The water volume consumed was 30 ml for each gram of lactate consumed during the lactate trial and was standardized for all trials. Calcium lactate (Puracal, Purac America, Lincolnshire, IL) from a factory-sealed container was weighed to the nearest milligram, adjusted for calcium content, and provided to the subjects in gelatin capsules. An equal number of gelatin capsules, filled with aspartame, was used to provide the placebo. Both the lactate and placebo capsules were sprinkled with small amounts of aspartame to disguise them from any residual aspartame that may have been left on the outside of the capsules during the filling of the placebo. The experimental treatments were applied in a double-blind, randomized, crossover fashion. Subjects were 3 hr postprandial for each IPT, and each subject performed all of the tests at a similar time of day. At least 48 hr separated IPTs, and subjects were required to complete all three experimental IPTs within a 14-day period. The number of days between trials was kept as consistent as possible for each subject. The subjects were required to refrain from performing vigorous exercise on the day before each visit (no more than 30 min of total activity at a heart rate ≤65% of maximal heart rate) and were asked to maintain similar training schedules between trials and similar dietary regimens in the days leading up to each visit.

On arrival at the laboratory for each IPT, subjects were asked if they had followed the prescribed training and dietary guidelines. If the guidelines had been followed, a 22-g catheter was placed in a prominent antecubital vein, after which the subject rested quietly in a seated position for 10 min. A 1-ml blood sample was then drawn and discarded, followed immediately by a 1-ml sample that was analyzed for blood lactate, pH, and HCO$_3^-$ using an Abbott iStat1 analyzer (Abbott by a 1-ml sample that was analyzed for blood lactate, was then drawn and discarded, followed immediately in a seated position for 10 min. A 1-ml blood sample was obtained and analyzed as previously described, and RPI and RPSA were measured. The subject then mounted the cycle ergometer and began a standardized 10-min warm-up for the IPT. Work rate for the warm-up began at 50 W below the starting work rate for the maximal test, progressed by 10 W/min for 5 min, and then remained consistent for the remaining 5 min of the warm-up period. Immediately after the warm-up, the subject pedaled at a work rate of 25% of MPO for a period of 1 min before beginning the first work period of the IPT. Blood samples were obtained and analyzed and RPI and RPSA were measured before consumption of the experimental supplements (Pre-Con), before the beginning of the warm-up (Pre-WU), during the final 30 s of the warm-up (Pre-IPT), and immediately after reaching exhaustion (EXH).

Statistical Analyses

Two-way ANOVAs with repeated measures were performed to detect differences in blood bicarbonate and pH resulting from time and treatment. Separate one-way ANOVAs were performed to detect differences between treatments for TTE and total work performed during the final interval of the IPT. Post hoc tests with Bonferroni’s correction procedure were used where appropriate. Pearson’s correlations were calculated between changes in blood HCO$_3^-$ levels and changes in TTE. Level of significance was set a priori at $p \leq .05$.

Results

Blood Measures

No significant differences were observed in the Pre-Con HCO$_3^-$ levels between the three experimental conditions. Seventy minutes after lactate consumption (Pre-WU), blood HCO$_3^-$ levels rose significantly from Pre-Con ($p = .04$). Comparatively, no significant differences were observed between the Pre-Con and Pre-WU HCO$_3^-$ levels in PL ($p = .79$) and NT ($p = .59$). Pre-WU HCO$_3^-$ levels were significantly higher in L than in PL ($p < .01$) and NT ($p = .03$). No significant differences were seen between NT and PL ($p = .86$). Pre-IPT HCO$_3^-$ levels were higher in L than in NT ($p = .03$) and PL ($p < .01$), and no differences were seen between NT and PL ($p = .64$). Significant decreases in HCO$_3^-$ were observed between EXH and all other time points for each treatment ($p < .01$). Treatment had no effect on blood HCO$_3^-$ levels at exhaustion. Blood HCO$_3^-$ data are presented in Table 1.

Blood pH for each treatment was significantly lower at EXH than at Pre-Con, Pre-WU, and Pre-IPT (all $p < .01$); no other time effects were detected. There were no effects of treatment on blood pH levels at any time point. Blood pH data are presented in Table 2.

There were no treatment effects on blood lactate levels at any of the measurement points. Blood lactate increased from 1.00 mmol/L at Pre-Con to 1.59 mmol/L at Pre-WU in L ($p = .07$), whereas differences between these time points were small and unremarkable in NT and PL. Blood lactate increased significantly at EXH.
compared with Pre-Con levels for each treatment. Blood lactate at exhaustion was higher in L than in NT and PL but did not reach the level of statistical significance ($p = .16, .18$ for L vs. NT and PL, respectively, observed power = .62). Blood lactate data are presented in Table 3.

**Exercise-Performance RPI and RPSA**

Supplementation with lactate resulted in significantly greater TTE than observed in NT and PL (both $p = .02$). No significant difference in TTE was observed between NT and PL ($p = .85$). Observed statistical power and effect size for these analyses were .91 and .43, respectively. Means, standard deviations, coefficients of variation, and 95% confidence intervals for TTE data are presented in Table 4. Total work performed during NT, PL, and L was 54 ± 12, 52 ± 19, and 62 ± 18 kJ, respectively. Levels of significance were identical in the analysis of work performed as observed in TTE. There was no order effect on IPT performance. TTEs were 149 ± 10, 145 ± 10, and 150 ± 13 s for the first, second, and third trials, respectively. RPI and RPSA were low and unremarkable throughout each trial, and when asked, no subject stated that illness or stomach ache affected his or her performance. RPI and RPSA data are presented in Table 5.
Discussion

The results revealed that blood HCO$_3^-$ levels increased, and high-intensity exercise performance significantly improved, after the oral consumption of calcium lactate. Previous works have revealed increases in blood HCO$_3^-$ and pH levels after lactate consumption (Bryner et al., 1998; Fahey et al., 1991; Swensen et al., 1994); however, those investigations focused on lactate as an energy substrate and studied its effects on low-intensity endurance exercise. Van Montfoort et al. (2004) found that lactate ingestion increased TTE during single, high-intensity treadmill runs, but the improvement they observed was substantially smaller than what was observed in a study of the reproducibility of high-intensity treadmill runs to exhaustion (Billat et al., 1994). Furthermore, VanMontfoort et al. only reported post-ingestion blood pH and HCO$_3^-$ levels and did not report changes in these variables caused by lactate consumption. Thus, this is the first investigation to demonstrate substantial and significant metabolic and ergogenic effects of lactate ingestion in conjunction with high-intensity exercise.

There are a number of differences between the current study and that of VanMontfoort et al. (2004). Perhaps the most striking is the greater improvement in exercise performance in the current study despite the use of a lower dose of lactate. Subjects in the current investigation increased their performance by approximately 17% after consuming 120 mg of lactate per kilogram body mass, in the form of calcium lactate, compared with a 1.7% increase in performance after the consumption of 400 mg of sodium lactate per kilogram body mass in Van Montfoort et al.’s study. There may be a number of factors that contribute to this difference in performance.

The nature of the lactate consumption and the timing of its consumption in relation to the exercise test may have affected the results of these studies. Van Montfoort et al.’s subjects consumed sodium lactate over a 90-min period and began exercise 90 min after completing consumption. In the current study, subjects consumed their entire dose of lactate within 5 min and began their performance test 80 min after completing ingestion. In pilot work, we measured the temporal response of blood HCO$_3^-$ to lactate ingestion and noted a peak in blood HCO$_3^-$ 80–100 min after ingestion. These peak levels were followed by a gradual decline in blood HCO$_3^-$ in the remaining 20–40 min of the measurement period. Thus, Van Montfoort et al.’s subjects may have not been performing their exercise tests when blood buffering capacity was at its peak level. In regard to the lactate dose, we observed no greater blood HCO$_3^-$ response to 220-mg/kg body mass compared with the 120-mg/kg body mass dose, suggesting that there may be an upper limit to the dose-response relationship that is met by a dose of 120 mg/kg body mass.

There is also a concern about the different types of lactate used by VanMontfoort et al. (2004; sodium lactate) and the current study (calcium lactate). We are unaware of any differences in the way that these two forms of lactate are digested and metabolized. Calcium ion, with a double positive charge, binds two lactate molecules per ion, whereas sodium, with a single positive charge, only binds one. However, by weight, these two molecules have roughly the same percentage of lactate (~80%) because the mass of calcium is roughly twice that of sodium. In the current study, the mass of the calcium was factored out of the dosage so that each subject received 120 mg of lactate per kilogram body mass, and Van Montfoort provided 400 mg of sodium lactate per kilogram body mass, or about 320 mg of lactate per kilogram body mass. It should be recognized that the dissociation of lactate from calcium or sodium would leave cations that could affect strong ion difference and influence H$^+$ movement and buffering. However, because blood levels of sodium and calcium were not measured in this investigation, nor were they reported by Van Montfoort et al., we do not believe that it is appropriate to speculate on the possible effects these supplements may have had on strong ion difference and exercise performance.

Possibly the most substantial contributing factor to the differing performance responses of the current study and that of Van Montfoort et al. (2004) is the nature of the exercise performance test. Van Montfoort et al. used a single, high-intensity run to exhaustion as their performance measure, whereas our protocol had subjects perform four high-intensity intervals followed by a ride to exhaustion at 100% of their VO$_{2\text{max}}$ power output. Hermansen and Osnes (1972) demonstrated that blood lactate and pH exhibited greater changes in response

<table>
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<tr>
<th>Trial</th>
<th>Time</th>
<th>Pre-Con</th>
<th>Pre-WU</th>
<th>Pre-IPT</th>
<th>EXH</th>
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<td>$&lt;1 \pm 1/0 \pm 0$</td>
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<td>$&lt;1 \pm 1/0 \pm 0$</td>
<td>$&lt;1 \pm 1/0 \pm 0$</td>
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<tr>
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<td>$&lt;1 \pm 1/1 \pm 2$</td>
<td>$&lt;1 \pm 1/1 \pm 2$</td>
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</tr>
<tr>
<td>Lactate</td>
<td>$&lt;1 \pm 1/0 \pm 0$</td>
<td>$&lt;1 \pm 1/1 \pm 1$</td>
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<td>$&lt;1 \pm 1/1 \pm 1$</td>
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Note. Pre-Con = before consumption of the experimental supplements; Pre-WU = before the beginning of the warm-up; Pre-IPT = during the final 30 s of the warm-up; EXH = immediately after exhaustion.
to interval work than with single exercise bouts. This has led some investigators to suspect that alkalinizing agents may be more ergogenic for interval events than for single bouts of exercise. In fact, meta-analyses have suggested that when high-intensity exercise to exhaustion is preceded by several high-intensity interval efforts, a greater ergogenic effect from NaHCO₃ is seen than in protocols that use single, high-intensity exercise bouts (Matson & Tran 1993).

In the current study, lactate consumption increased TTE by 18% over PL and 17% over NT, which is consistent with, albeit somewhat less than, most studies using NaHCO₃ and similar exercise protocols. Costill et al. (1984) had cyclists perform four 1-min efforts followed by a fifth effort to exhaustion at 125% of VO₂max power output and reported a 42% increase in TTE after the consumption of 200 mg NaHCO₃ per kilogram body mass. Wijnen, Verstappen, and Kuipers (1984) used an identical exercise protocol to that of Costill et al., finding a 4% improvement in performance after ingestion of 180 mg NaHCO₃ per kilogram body mass and a 22% improvement after consumption of 360 mg/kg body mass. McKenzie, Coutts, Stirling, Hoeben, and Kuzara (1986) also measured the performance response in repetitive bouts of high-intensity cycling after the consumption of 150- and 300-mg NaHCO₃ per kilogram body mass doses and saw improvements in TTE of 49% and 42%, respectively.

A notable difference between the previous studies of NaHCO₃ and the current study is the effects of the supplement on blood pH levels. Each of the three previous studies of NaHCO₃ found significant increases in blood pH after NaHCO₃ consumption, whereas we observed no changes in blood pH as a result of lactate consumption. It is suspected that NaHCO₃ consumption achieves its ergogenic effect by alkalinizing the vascular space and promoting H⁺ efflux from the working muscle during high-intensity work. These actions have been shown to maintain higher pH levels in the working muscle during high-intensity exercise (Raymer et al., 2004), which may result in increased glycolytic rate (Bishop et al., 2004; Hullahide-Horvat, Parolin, Wong, Jones, & Heigenhauser, 2000; Stephens, McKenna, Canny, Snow, & McConell, 2002). We did observe higher levels of blood HCO₃⁻ in the preexercise period as a result of lactate consumption, which would increase the buffering capacity of the blood. However, the effect of increased blood HCO₃⁻, without a concomitant increase in blood pH, on intramuscular pH during exercise is currently unknown.

We are troubled by the lack of change in blood pH while seeing simultaneous increases in blood HCO₃⁻ and TTE. Previous investigations using NaHCO₃ (Matson & Tran, 1993) typically observed changes in both HCO₃⁻ and pH. Relative to these studies, we observed a modest increase in blood HCO₃⁻, and we suggest that this small change may not have been sufficient to exert a substantial mass action effect on the carbonic anhydrase reaction at rest. However, during exercise, the increased blood HCO₃⁻ could have absorbed enough H⁺ to facilitate H⁺ efflux from the working muscle, thus maintaining a more favorable environment for muscle contraction. Furthermore, even though they did not reach statistical significance, we did observe higher blood lactate levels at exhaustion in L than in NT and PL, which would support the contention of greater H⁺ efflux from the working muscle during the L trial.

Despite the evidence suggesting that increased blood pH results in a more favorable muscular environment for high-intensity exercise, the mechanisms of action for the ergogenic effects of NaHCO₃ or lactate consumption remain nebulous. In their meta-analyses of NaHCO₃ consumption, Matson and Tran (1993) found a positive effect on performance, especially when measuring exercise TTE. However, the relationships between the magnitude of the performance improvements and the levels of blood alkalosis and HCO₃⁻ were practically nonexistent (r = .21 for alkalosis, r = .10 for HCO₃⁻). We also observed improvements in performance after consumption of lactate; however, like previous studies of NaHCO₃, correlations between increases in blood HCO₃⁻ caused by lactate consumption and improvements in performance in L compared with NT and PL were low (r = -.10 NT vs. L, r = -.36 PL vs. L).

Exercise performance data also suggest that there is great inter- and intrasubject variation in ability to respond to alkalinizing agents. Wijnen et al. (1984) had 5 subjects perform 18 trials of their exercise test, 6 each under the placebo, 180-mg/kg body mass, and 360-mg/kg body mass doses, and found significant improvements from NaHCO₃ use in 2 of the 5 subjects. Extreme variation in the remaining 3 subjects’ performances within each treatment prevented statistical significance, and some subjects saw their average performance decrease after NaHCO₃ use compared with placebo. We also observed variations in subjects’ responses to lactate ingestion. Compared with NT, lactate consumption resulted in an average 17% increase in TTE, with individual changes ranging from a 42% improvement to a 7% reduction in performance. Despite this wide range, the improvements of 7 of the 11 subjects were in the 10–20% range, and the remaining 2 subjects improved by approximately 3%. The only subject without improved TTE after the consumption of lactate was also the only one who did not exhibit increases in blood HCO₃⁻ in response to lactate consumption.

The TTE performance test has been recently criticized for having poor reliability (Currell & Jeukendrup, 2008). In reality, there are few studies that have assessed the test–retest reproducibility of TTE performance tests, and many of those focused on long, ≥1.0-hr protocols. Factors such as boredom, monotony, and discomfort from sitting on a bicycle saddle are far more likely to occur in long exercise protocols than in shorter (<5 min) ones. Thus, in comparison with shorter tests, performance in long TTE tests is more likely to be influenced by factors other than physiological capacity, which could contribute to higher levels of inconsistency in performance. In fact, Billat et al. (1994) reported an average 11% variation in test–retest performance of runs to exhaustion at 100%
of VO2max velocity, which is well below our observed improvement in performance of approximately 17% after lactate supplementation. It also should be noted that although the current results did exhibit typically high coefficients of variation for TTE tests, 10 of the 11 subjects did improve after lactate consumption compared with both NT and PL treatments. Furthermore, we did attempt to address the issue of intersubject variance by converting the performance test times to percentage of improvement. Analyses of these transformed data revealed similar p values to the raw data, and we chose to report the raw times because we believed that they would provide a better description of the exercise challenge and assist in generalizing the results to real-world competitive applications. Nevertheless, future investigations may wish to focus on performance tests that use time-trial formats, as they appear to be a more reliable measure of performance than are TTE tests (Currell & Jeukendrup, 2008).

In conclusion, the current investigation demonstrated increased blood HCO3− levels and improved exercise performance during repetitive, high-intensity cycling ergometry after the ingestion of calcium lactate. Blood pH was unchanged by lactate consumption. Mechanisms for the improvement of exercise performance remain unclear, because correlations between increases in blood HCO3− and improvements in exercise performance were low. Future work should investigate the effects of lactate ingestion on muscle metabolism and pH and differences in the effects of sodium lactate and calcium lactate consumption. In addition, optimal lactate dosage to elicit shifts in blood bicarbonate and pH should be explored.

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References


