An Investigation Into the Effects of Sodium Citrate Ingestion on High-Intensity Exercise Performance

Ken van Someren, Kathy Fulcher, John McCarthy, Jonathan Moore, Gill Horgan, and Richard Langford

This study examined the effect of sodium citrate ingestion on high-intensity cycling performance in repeated 45-s bouts. Twelve subjects (9 male and 3 female) ingested either a sodium citrate solution (0.3 g · kg⁻¹ body mass [BM]) or a placebo 90 min prior to exercise. Postingestion blood HCO₃⁻ concentrations were significantly higher in the citrate trial (p < .01), but there was no difference in blood pH between trials. Peak power and total work significantly decreased over the five bouts (p < .05) and postexercise blood lactate concentrations significantly increased over the five bouts (p < 0.01), but there were no differences between trials. We conclude that sodium citrate ingestion (0.3 g · kg⁻¹ BM) is not an effective ergogenic aid for high-intensity, intermittent exercise as simulated in this protocol.

Key Words: alkalosis, blood HCO₃⁻, blood pH, cycling, ergogenic

High-intensity, short-duration exercise has been shown to produce lactic acid, which dissociates to form hydrogen ions (H⁺) (4, 6), which in turn decrease muscle pH (1, 10). Such a biochemical imbalance reduces the operational efficiency of the muscle’s contractile mechanism (3, 14) and inhibits the glycolytic enzymes phosphofructokinase and phosphorylase (11). This is commonly accepted to be the major cause of fatigue during short-duration, high-intensity exercise (4, 9, 15, 20).

Blood pH can be increased through the ingestion of acid-buffering agents (11). This increases the gradient between the intracellular muscle compartment and extracellular space (2), promoting an accelerated efflux of lactate and H⁺ from exercising tissues. Therefore, it has been proposed that elevated plasma bicarbonate levels would augment the body’s buffering capacity and thus improve anaerobic performance (1, 8, 10, 19). Consequently, the ingestion of sodium bicarbonate (known as bicarbonate loading) has received much attention from researchers over recent years. While improvements in anaerobic performance have been reported with bicarbonate loading, considerable gastrointestinal discomfort has also been observed (8, 22).

K. van Someren is with the Human Performance Lab, Department of Sport, Health and Exercise Science, St. Mary’s University College, Waldegrave Road, Strawberry Hill, Twickenham, Middlesex, TWI 4SX, UK. K. Fulcher, J. McCarthy, J. Moore, and G. Horgan are with the National Sports Medicine Institute, Medical College of St. Bartholomew’s Hospital, Charterhouse Square, London, EC1M 6BQ, UK. R. Langford is with the Department of Anaesthetics, St. Bartholomew’s Hospital, West Smithfield, London, EC1M 6BQ, UK.
Sodium citrate has been investigated as an alternative to sodium bicarbonate, since it can also induce alkalosis without the same extent of gastrointestinal disturbance (15). Current knowledge indicates that citrate is metabolized to bicarbonate, producing an increase in extracellular HCO$_3^-$ proportional to the quantity of citrate ingested (12, 15). A number of studies have demonstrated equivocal results. McNaughton (15) found that sodium citrate ingestion increased total work and peak power during a single bout of maximal 1-min cycle ergometry, with doses of 0.3, 0.4, and 0.5 g · kg$^{-1}$ body mass (BM). McNaughton and Cedaro (16) found sodium citrate ingestion (0.5 g · kg$^{-1}$ BM) to have no ergogenic effect for single bouts of 10 s and 30 s; however, they did observe improved exercise performance over 120-s and 240-s single-bout tests. In contrast, Cox and Jenkins (2) found that 0.5 g · kg$^{-1}$ BM produced no significant change in work output during cycle ergometry consisting of five 60-s bouts separated by a 5-min passive recovery. However, significant changes were observed in selected ventilatory and blood variables.

The purpose of this study was to determine the effects of sodium citrate ingestion (0.3 g · kg$^{-1}$ BM) on repeated 45-s bouts of supramaximal exercise. Blood chemistry and performance parameters were measured to assess potential ergogenic effects. The protocol of repeated supramaximal bouts was used to place high demands upon the glycolytic energy system and to simulate intermittent high-intensity exercise.

**Methods**

**Subjects**

Twelve healthy, active subjects (9 males and 3 females) volunteered for the study: age 28.1 ± 5.2 years (mean ± SD), height 177.3 ± 10.6 cm, body mass 71.1 ± 11.7 kg, peak oxygen uptake 3.68 ± 0.8 L · min$^{-1}$. All subjects were informed verbally and in writing of the nature and possible side effects associated with the project, and they all gave written informed consent. The study was approved by the District Research Ethics Committee.

**Procedures**

On the first visit to the laboratory, subjects were measured for height and body mass prior to testing. The first test performed by subjects was an incremental cycling test to determine peak oxygen uptake. Following a 5-min warm-up on a friction-loaded, mechanically braked cycle ergometer (Monark 818) at 75 W (75 revolutions · min$^{-1}$ with 1 kg), testing was commenced. Subjects were instructed to maintain a pedal rate of 75 rev · min$^{-1}$ throughout, and resistance was increased manually by 0.5 kg every 2 min. Respiratory data were collected on-line with a Jaeger EOS Sprint System, via a Salford valve attached to lightweight tubing. Expired gases were measured every 30 s via an infrared O$_2$ analyzer and a Zirconium CO$_2$ analyzer, and the volume of expired air was measured by a pneumotachograph. Heart rate and ECG data were monitored with a Hewlett Packard 43120A electrocardiograph defibrillator. The tests were terminated when the subject could no longer maintain a pedal rate of 70 rev · min$^{-1}$ and there was a plateau reflecting less than a 100 ml increase in $\text{VO}_2$ with a 35-W increase in work-rate. This $\text{VO}_2$ was accepted as a $\text{VO}_2$ peak for the purposes of subject description.
Following a minimum of 20 min of recovery, subjects performed a further 5-min warm-up at 70 W (70 rev \cdot min\(^{-1}\) with 1 kg) before being familiarized with the high-intensity exercise test protocol. This familiarization consisted of two supramaximal 45-s bouts on the cycle ergometer separated by 5 min of recovery (1 min passive recovery and 4 min active recovery, at 60 rev \cdot min\(^{-1}\) with 0.5 kg).

Subjects reported to the laboratory 120 min prior to each high-intensity exercise test. A whole-blood, fingertip capillary sample (60 \(\mu\)l) was taken to determine basal blood pH and HCO\(_3\)\(^-\) levels (Corning Blood Gas Analyser. 1312 Series, Instrumentation Laboratory). Subjects then ingested either 0.3 g \cdot kg\(^{-1}\) BM of sodium citrate (\(\text{C}_6\text{H}_5\text{Na}_2\text{O}_7 \cdot 2\text{H}_2\text{O}, \text{Merck, Germany}\)) or a placebo solution (0.3 g \cdot kg\(^{-1}\) BM calcium carbonate) in 500 ml sugar-free, black-current-flavored water over 30 min. These were administered in a random, double-blind, crossover procedure. Another whole-blood, fingertip capillary sample was taken for blood gas and blood lactate determination (Analox, GM7) 90 min after completion of consumption; the high-intensity exercise test was then performed. Subjects were tested at the same time of day to avoid any diurnal variations and were instructed to standardize both their diet and exercise for the 3 days preceding the high-intensity exercise tests. Weighed food intake was recorded prior to the first trial, and subjects were asked to repeat the same diet prior to the second trial. The 3-day weighed food records were analyzed for each trial using Compeat (Lifeline Nutritional Services).

Following a 10-min warm-up at 70 W (70 rev \cdot min\(^{-1}\) with 1 kg), subjects completed five all-out 45-s bouts. A modified basket-loaded, mechanically braked cycle ergometer (Monark 814E) was used for the 45-s tests, with computer software adapted from the Wingate Anaerobic Test (WAnT). Mean pedal rate, peak power, mean power, minimum power, total work, and the fatigue index were recorded for each sprint. Immediately prior to load application (4% BM), subjects cycled at 60 rev \cdot min\(^{-1}\) with 0.5 kg. Subjects were counted down to the start of each bout, whereupon the load was applied and subjects cycled maximally from the onset, without pacing. Standardized verbal encouragement was given throughout the tests. On completion of each bout, 1 min of passive recovery was followed by 4 min of active recovery (60 rev \cdot min\(^{-1}\) with 0.5 kg). A whole-blood, fingertip capillary sample was taken 3 min after each sprint for blood lactate determination (Analox, GM7).

**Statistical Analyses**

A series of repeated-measures ANOVAs were employed to compare peak power, total work, fatigue indices, postexercise blood lactate concentrations, blood pH, and HCO\(_3\)\(^-\) over time and between trials. A significance level of \(p < .05\) was established prior to investigation for all analyses. Where significance was found, post hoc Tukey tests, or paired-samples \(t\) tests with Bonferroni corrections, were employed.

**Results**

A repeated-measures ANOVA demonstrated that there was no difference in blood pH between trials, though there was an increase from pre- to postingestion in both trials \((F = 20.23, p = .004)\) (Table 1). A difference between trials was found for blood HCO\(_3\)\(^-\) \((F = 6.42, p = .04)\), and a follow-up paired-samples \(t\) test with Bonferroni correction showed postingestion HCO\(_3\)\(^-\) to be significantly higher \((p < .01)\) in the citrate trial than in the placebo trial. Blood HCO\(_3\)\(^-\) was also found to increase \((F = 53.12, p = .000)\) from pre- to postingestion in both trials (Table 1).
Figure 1 illustrates the peak power outputs during the five 45-s bouts for both trial conditions. No significant difference was found between trials; however, peak power significantly decreased over time in both conditions ($F = 4.37, p = .014$). Follow-up tests revealed that peak power decreased from Bouts 1 to 2, 2 to 3, and 3 to 4 ($p < .05$), but thereafter there were no differences over time.

There was no difference between trials for total work performed during the bouts (Figure 2). In both conditions, however, total work significantly decreased over time ($F = 6.36, P = 0.003$). Follow-up tests revealed that total work decreased from Bouts 1 to 2, 2 to 3 and 3 to 4 ($p < .05$), but thereafter there were no differences over time.

Table 1  Blood pH and Bicarbonate Concentrations for Pre- and Postingestion of Placebo and Sodium Citrate

<table>
<thead>
<tr>
<th></th>
<th>Placebo trial</th>
<th>Citrate trial</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preingestion</td>
<td>Postingestion</td>
</tr>
<tr>
<td></td>
<td>$M$  $SD$</td>
<td>$M$  $SD$</td>
</tr>
<tr>
<td>Capillary blood pH</td>
<td>7.40  0.02</td>
<td>7.44  0.02</td>
</tr>
<tr>
<td>Capillary blood HCO$_3^-$ (mmol-L$^{-1}$)</td>
<td>23.77  1.22  25.03  1.46</td>
<td>23.88  1.69  27.39  2.32*</td>
</tr>
</tbody>
</table>

*Significantly higher than postingestion placebo ($p < .01$).

![Figure 1 — Peak power outputs (mean ± SD) in high-intensity exercise tests. Open bars = placebo trial, black bars = citrate trial. No significant differences between trials were observed.](image-url)
Figure 2 — Total work output (mean ± SD) in high-intensity exercise tests. Open bars = placebo trial, black bars = citrate trial. No significant differences between trials were observed.

No differences were found in the fatigue indices between trials or over time. Neither were there differences in postexercise blood lactate concentrations between trials (Figure 3). However, postexercise blood lactate concentrations did increase over time \( (F = 21.10, p = .000) \). Follow-up tests revealed that there was a significant increase from Bout 1 to 2 \( (p < .05) \), but thereafter there were no differences over time.

Analysis of dietary records showed no significant difference between percentage carbohydrate, protein, and fat recorded for the 3 days preceding the two trials. Table 2 shows mean (±SD) values for macronutrients.

**Discussion**

An exercise bout of 45 s has been shown to stress the glycolytic energy system to a greater extent than does a 30-s bout (21). In theory, ingested sodium citrate is metabolized to bicarbonate, thereby increasing the extracellular buffering capacity and therefore potentially improving anaerobic performance. Consequently, it was anticipated that the buffering effect of sodium citrate ingestion would be ergogenic in a protocol of repeated bouts of this duration.

This study demonstrates that ingesting a sodium citrate solution \( (0.3 \text{ g} \cdot \text{kg}^{-1} \text{BM}) \) does not significantly improve any performance parameter during repeated 45-s supramaximal exercise. Previous studies have shown performance improvements in longer exercise bouts of 60 s (15), 120 s, and 240 s duration (16) following the ingestion of a sodium citrate solution. However, no such improvements have been demonstrated during 10 s and 30 s (16) or 60 s (2) duration. The current study suggests that 45-s exercise bouts are perhaps too short to benefit from sodium citrate ingestion.

Many researchers investigating the effects of sodium citrate ingestion have used a dose of \( 0.5 \text{ g} \cdot \text{kg}^{-1} \text{BM} \) (2, 16), though ergogenic effects have been demonstrated
Figure 3 — Postbout blood lactate concentration (mean ± SD) in high-intensity exercise tests. Open bars = placebo trial, black bars = citrate trial. No significant differences between trials were observed.

Table 2  Macronutrient Percentages in Recorded Diets Prior to Trials

<table>
<thead>
<tr>
<th></th>
<th>Placebo trial</th>
<th>Citrate trial</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( M )</td>
<td>( SD )</td>
</tr>
<tr>
<td>Carbohydrate (%)</td>
<td>54.6</td>
<td>6.2</td>
</tr>
<tr>
<td>Protein (%)</td>
<td>13.9</td>
<td>5.6</td>
</tr>
<tr>
<td>Fat (%)</td>
<td>30.2</td>
<td>6.7</td>
</tr>
</tbody>
</table>

at lower doses (15). In the current study, 0.3 g · kg\(^{-1}\) BM of sodium citrate significantly elevated blood bicarbonate levels above those seen in the placebo trial, supporting the findings of a number of previous studies (12, 15). In addition, Parry-Billings and MacLaren (19) showed 0.3 g · kg\(^{-1}\) BM sodium citrate to significantly increase blood buffering capacity but produce no significant increases in mean power over three 30-s Wingate tests. However, in contrast to previous studies (12, 15, 19), we failed to observe a difference in postingestion blood pH between trials with a dose of 0.3 g · kg\(^{-1}\) BM, despite elevated blood bicarbonate concentrations. This may explain why no ergogenic effects were observed in our study. In addition, the sarcolemma is impermeable to HCO\(_3\)\(^-\) and therefore sodium citrate ingestion will have no effect upon resting muscle pH. It is also likely that extracellular changes in bicarbonate will have little effect on muscle pH during short-duration exercise (19), thereby precluding any ergogenic effects.

Higher blood lactate concentrations generally reflect an increased anaerobic energy yield, which is facilitated by an increased buffering capacity. Although we
found that ingestion of the sodium citrate solution significantly increased blood HCO₃⁻, postexercise blood lactate concentrations did not increase in the citrate trial. This supports our finding that the observed increase in blood buffering capacity failed to increase exercise performance. Sodium citrate has been associated with nonsignificant increases in postbout blood lactate concentrations following repeated 30-s Wingate tests (19) but a significant increase following a single 60-s bout (15). Although oxidative energy yield becomes more significant as test duration increases, the maximal accumulated oxygen deficit, which has been proposed as a valid measure of anaerobic capacity, reaches a peak value during a 2-min exhaustive exercise bout (17). This suggests that the glycolytic system is maximally stressed during exercise in the region of 2 min, and therefore an increased buffering capacity would be most beneficial over this duration rather than over 45 s. This is supported by previous studies showing beneficial effects of increased buffering capacity during exercise bouts of 120 s and longer (16). In addition, it is likely that the 5-min rest between each bout in our protocol provided for complete phosphocreatine resynthesis and myoglobin saturation (5), thus also reducing the glycolytic energy demands of the exercise.

In contrast to much of the previous research, this study used a subject group of both males and females. This explains the slightly lower mean values for physical characteristics and performance parameters in relation to previous studies that used only male subjects (15, 16). Although our subjects had a variety of sports backgrounds, they were not well-trained sprint and power athletes.

An advantage of using sodium citrate rather than sodium bicarbonate is the reported lesser extent of gastrointestinal disturbance (15, 19). However, 5 subjects in this study reported feeling nauseous before and during the high-intensity exercise test. Of these subjects, 1 suffered severe gastrointestinal upset after the test. Two subjects suffered similar upset during the placebo trial. Such effects appear minimal compared with those noted by Cox and Jenkins (2), who found that with a dose of 0.5 g • kg⁻¹ BM sodium citrate, 7 of the 8 subjects felt nauseous, 5 of whom vomited.

In summary, a dose of 0.3 g • kg⁻¹ BM sodium citrate significantly increases blood buffering capacity but fails to produce ergogenic effects over this exercise protocol. This is perhaps due to an insufficient stress imposed upon the glycolytic energy system during repeated 45-s bouts of exercise, preventing performance improvements. Further research is needed to determine whether higher doses of sodium citrate can improve performance during repeated 45-s bouts. In addition, investigation with a highly anaerobic trained subject group is recommended, where ergogenic effects might be evident.

References


**Acknowledgments**

This study was supported by a grant from the Gatorade Sport Science Institute. We would like to thank Henryk A. Lakomy for writing the computer software for our protocol and Amanda Daley for her help in the statistical analyses.

*Manuscript received: March 3, 1997*  
*Accepted for publication: April 14, 1998*