Effects of sodium citrate ingestion before exercise on endurance performance in well trained college runners

V Oöpik, I Saaremets, L Medijainen, K Karelson, T Janson, S Timpmann

Objective: To test the hypothesis that sodium citrate administered two hours before exercise improves performance in a 5 km running time trial.

Methods: A total of 17 male well trained college runners (mean (SD) VO2MAX 61.3 (4.9) ml/kg/min) performed a 5 km treadmill run with and without sodium citrate ingestion in a random, double blind, crossover design. In the citrate trial, subjects consumed 1 litre of solution containing 0.5 g of sodium citrate/kg body mass two hours before the run. In the placebo trial, the same amount of flavoured mineral water was consumed.

Results: The time required to complete the run was faster in the citrate trial than the placebo trial (1153.2 (74.1) and 1183.8 (91.4) seconds respectively; p=0.01). Lower packed cell volume and haemoglobin levels were found in venous blood samples taken before and after the run in the citrate compared with the placebo trial. Lactate concentration in the blood sample taken after the run was higher in the citrate than the placebo trial (11.9 (3.0) vs 9.8 (2.8) mmol/l; p<0.001), and glucose concentration was lower (8.3 (1.9) vs 8.8 (1.7) mmol/l; p=0.02).

Conclusion: The ingestion of 0.5 g of sodium citrate/kg body mass shortly before a 5 km running time trial improves performance in well trained college runners.
METHODS

Study design

The two treatment conditions, sodium citrate (citric acid trisodium salt; CIT) and placebo (PLC), were administered in a counterbalanced, crossover, randomly assigned, double blind manner, with each trial separated by seven to eight days.

Subjects

A total of 17 male well trained college runners participated in the study, the protocol of which was approved by the ethics committee of the University of Tartu, Estonia. The subjects gave their written informed consent and were screened by questionnaire to exclude those with pre-existing medical conditions that would contraindicate their involvement in the study. Their mean (SD) age, body mass, height, and \(V_{\text{O}_2}\text{MAX}\) at the beginning of the study were 20.9 (1.9) years, 75.6 (5.4) kg, 182.9 (5.5) cm, and 61.3 (4.9) ml/kg/min respectively. They had been involved in regular training for 9.2 (3.6) years.

Procedure

All the subjects visited the laboratory three times. The first occasion was to complete a maximal aerobic power test to determine \(V_{\text{O}_2}\text{MAX}\), and the other two were to undertake a 5 km time trial on the treadmill. The subjects were instructed to abstain from vigorous exercise during the day preceding each test. They were also advised to follow their habitual eating pattern throughout the study period. For each test day and the day preceding the visit to the laboratory, the subjects kept detailed physical activity and food diaries. The information obtained from the diaries completed before the first visit to the laboratory was used to remind the athletes of the pattern of physical activity and eating to follow before each subsequent test day. These measures were undertaken to ensure a stable nutritional and training status throughout the study period of 11–12 days.

\(V_{\text{O}_2}\text{MAX}\) was measured during a progressive exercise test performed on a treadmill (Runrace HC 1400; Technogym, Gambettola, Italy). The test began with a five minute warm up. The speed was then increased from the initial rate of 8 km/h after every 200 m by 0.5 km/h until the athlete was unable to maintain the pace. The protocol of the graded exercise used in this study is based on principles originally developed by Conconi et al.\(^\text{29}\) Expired gas was sampled and analysed continuously using an online system (True Max 2400: Parvo Medics, East Sandy, Utah, USA). The analyser was calibrated before each subject was tested. Most of the subjects (11) reached their \(V_{\text{O}_2}\text{MAX}\) one to three stages before the last 200 m of the test. In the remaining six, \(V_{\text{O}_2}\text{MAX}\) was observed at the maximal running speed achieved during the test procedure. However, in all cases the respiratory exchange observed at the maximal running speed achieved during the study period of 11–12 days.

The body mass of the subjects measured immediately after the CIT or PLC drink did not differ in the two trials (table 1). During the two hour period between consumption of the drink and initiation of exercise, body mass of the subjects was reduced to a much greater extent in the PLC trial than in the CIT trial. As a result, the subjects in the CIT trial started the 5 km treadmill run on average 0.7 kg (\(p = 0.03\)) heavier than in the PLC trial.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Changes in body mass</th>
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<tbody>
<tr>
<td><strong>Body mass (kg)</strong></td>
<td><strong>Treatment</strong></td>
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<tr>
<td><strong>Placebo</strong></td>
<td>78.7 (5.9)</td>
</tr>
<tr>
<td><strong>Citrates</strong></td>
<td>78.9 (5.5)</td>
</tr>
</tbody>
</table>

Values represent mean (SD).

Significantly different (\(p < 0.05\)) from placebo treatment.
Sodium citrate and endurance performance

It took the subjects significantly (p = 0.01) less time to complete the 5 km run after drinking sodium citrate than after consuming placebo (1153.2 (74.1) vs. 1183.8 (91.4) seconds respectively). It is noteworthy that 13 of the 17 subjects achieved faster runs after sodium citrate ingestion, and only four were faster in the PLC trial. The average speed of running in the CIT trial exceeded that measured in the PLC trial during the 2nd and 4th kilometre of the distance (fig 1): 15.8 (1.2) vs. 15.4 (1.4) km/h (p = 0.01) and 16.2 (1.1) vs. 15.7 (1.2) km/h (p = 0.002). A trend towards a better performance in the CIT trial than the PLC trial was evident during the 3rd kilometre: 16.0 (1.2) vs. 15.7 (1.2) km/h (p = 0.06). In both trials the fastest average speed was achieved during the 5th kilometre, but there was no difference between the trials: 16.9 (1.3) km/h for the CIT trial and 16.7 (1.2) km/h for the PLC trial, p = 0.43. The maximum speed achieved in the two trials did not differ: 17.7 (1.4) km/h for the CIT trial and 17.7 (1.3) km/h for the PLC trial.

Heart rate during the run did not differ in the two trials, except that measured after three minutes (173.2 (12.0) beats/min in the CIT trial vs. 169.5 (13.5) beats/min in the PLC trial; p = 0.03). The maximum heart rate measured during exercise did not differ in the two trials: 194.8 (10.3) beats/min for the CIT trial and 193.7 (9.2) beats/min for the PLC trial.

The subjects’ perception of effort was the same in the two trials throughout the exercise period. Rating of perceived exertion during the run ranged from 14.9 (1.9) to 18.7 (0.8) for the CIT trial and from 15.4 (1.6) to 18.7 (1.3) for the PLC trial.

Packed cell volume and haemoglobin concentration increased significantly as a result of the run in both trials (table 2). In the CIT trial, significantly lower packed cell volume and haemoglobin levels were observed before and after exercise compared with the PLC treatment. The calculated relative decrease in plasma volume during exercise was similar in the two trials (table 2).

There were no differences in plasma lactate and glucose concentrations before the run between the two trials (table 3). A significant increase in the level of both metabolites was observed as a result of the 5 km run. However, the plasma concentration of lactate was significantly higher and that of glucose significantly lower after the run in the CIT trial than in the PLC trial (table 3). These significant between trial differences were evident also after correction of the measured concentrations of lactate and glucose for the individual changes in plasma volume (data not shown).

**DISCUSSION**

The primary finding of this investigation is that sodium citrate ingestion improved performance in a 5 km treadmill run in well trained college runners. The subjects covered the distance on average 30.6 seconds faster after receiving citrate than after receiving the placebo. A disadvantage of this study is that the coefficient of variation for 5 km treadmill runs in these subjects was not measured before the experimental trials. However, all the subjects had previous experience in treadmill running. Moreover, the treatments were administered in a counterbalanced manner—that is, nine subjects performed the CIT trial first while the other eight subjects started with the PLC trial. The effect of learning on the results should be negligible under these conditions, and the fact that 13 of the 17 athletes achieved a better result after sodium citrate ingestion probably reflects the true ergogenic effect of this substance.

The use of buffering substances as ergogenic aids during endurance exercise has previously been studied. Sodium bicarbonate and sodium citrate have been shown to have no positive effect on running time to exhaustion.22–24 The subjects in those experiments were not allowed to alter their running pace, whereas in our investigation the runners chose their exercise intensity (speed of running) according to how they felt during the 5 km run. This is more representative of a competitive athletic event.

Three previous studies have shown an ergogenic effect of sodium citrate28 or sodium bicarbonate23, 25 during endurance exercise performed on a cycle ergometer. In two,26, 27 an intermittent intensity exercise model was used, as in our study. However, to the best of our knowledge, this investigation is the first to show improvement in endurance running performance. Thus the few data so far available suggest that the probability of achieving an improvement in endurance performance by ingestion of sodium citrate or sodium bicarbonate may be greater in cycling than running. In this respect, it is noteworthy that the changes in body mass during the two hour period after consumption of 1 litre of solution indicate significantly greater fluid retention with sodium citrate treatment compared with placebo (table 1). This could be expected because the sodium content of the sodium citrate drink was much higher than that of the placebo. Therefore, immediately before the run, the subjects in the CIT trial were heavier than those in the PLC trial (table 1). Consequently, owing to their approximately 1% greater body mass, they had extra work to perform during the run. This additional work load probably reduced the positive effect of sodium citrate ingestion on running capacity and at least partly explains why the improvement in performance of our subjects was relatively small compared with that observed in other studies in which endurance capacity was measured by cycle ergometry.25–27

The precise mechanism by which sodium citrate ingestion before the 5 km time trial improved performance in our well trained college runners remains obscure. Ingestion of sodium citrate has been shown to increase blood pH, HCO₃⁻ concentration, and base excess.28 The monocarboxylate transporter, which is thought to be responsible for lactate transport across the cell membrane,31 has been shown to be sensitive to pH gradient.31 Thus ingestion of sodium citrate, by increasing extracellular pH, may create a favourable pH gradient for efflux of intracellular lactate and H⁺. In the case of an intensively working skeletal muscle, this means a delay in the fall in intramuscular pH to the critical level at which glycolysis is inhibited. Against this background, it is not surprising that two26, 27 of the three groups who have found a positive effect of alkalisers on endurance performance capacity have concluded that the effect is probably due to increased efflux of intracellular lactate and H⁺ from contracting muscle cells.

Unfortunately we did not investigate the changes in blood pH, HCO₃⁻ concentration, and base excess. However, the

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*Significantly different (p < 0.05) from the placebo trial.
dose of sodium citrate used (0.5 g/kg body mass) has been reported to be the most appropriate for inducing the greatest increase in blood HCO₃⁻ concentration²⁵ and achieving an optimal alkaliolic state 100–120 minutes after ingestion.²⁵ Our subjects started the 5 km run 120 minutes after ingesting the citrate or placebo. The significantly higher lactate concentration measured in the plasma of our subjects after the run in the CIT trial compared with those in the PLC trial (table 3) is in accordance with the hypothesis that lactate efflux from muscle cells is facilitated during exercise after sodium citrate ingestion. This, in turn, may have increased the contribution of anaerobic glycolysis to energy production, enabling the muscles because of the more intense work that they had performed after sodium citrate ingestion.

Mitchell et al²⁵ found that, in exercise performed at 80% VO₂MAX, intravenous infusion of both sodium bicarbonate and sodium chloride improved endurance performance compared with control conditions (no infusion), although only sodium bicarbonate prevented the development of acidosis. In this situation, the ergogenic effect may be attributed to not only the enhanced buffering capacity of the body, but also to the increased plasma volume resulting from the infusion of sodium-containing fluids, which would result in better perfusion of the exercising skeletal muscle.²¹ The significantly lower packed cell volume and blood haemoglobin concentration observed before the run in the CIT trial compared with those in the PLC trial (table 3) may be explained by the increased uptake of blood sugar by the muscles because of the more intense work that they had to perform after sodium citrate ingestion.

Heart rate and rating of perceived exertion were similar in the two trials. These findings are in accordance with other results.²² ²⁵ ²⁶ ²⁸

Alkalisers, especially sodium bicarbonate,²⁵ have been reported to induce gastrointestinal distress after ingestion. The side effects of sodium citrate observed in this study included nausea and thirst in 12 subjects combined with headache in two of them. Moreover, all 17 subjects reported an urge to defecate or diarrhoea after citrate ingestion. However, they remarked that these disturbances were comparatively mild and transient, occurring within the first hour. Schabort et al²⁷ reported that gastrointestinal discomfort and stomach cramps were experienced by five of their eight subjects during a 40 km cycling time trial after ingestion of 0.6 g of sodium citrate/kg body mass, with only two subjects complaining of symptoms after ingestion of the 0.4 g/kg body mass dose. Similarly, Potteiger et al²⁸ mentioned that some gastrointestinal distress was associated with consumption of 0.5 g of sodium citrate/kg body mass in three out of their eight subjects. In contrast, no gastrointestinal discomfort was reported in two other studies,²⁹ ³⁰ although 0.5 g/kg body mass doses of sodium citrate were consumed by the subjects participating. Thus it is evident that the ingestion of sodium citrate at a dose of 0.4–0.6 g/kg body mass has the potential to cause gastrointestinal distress. Because the response seems to vary between individuals, the efficacy of sodium citrate should be individually tested by the athletes before using it as an ergogenic aid in competitions. Moreover, before sodium citrate ingestion can be recommended for improvement of endurance performance in an actual competitive situation, appropriate field studies should be undertaken to prove the efficacy of this manipulation.

In conclusion, the results of the study indicate that the ingestion of 0.5 g of sodium citrate/kg body mass improves

<p>| Table 2 | Packed cell volume, haemoglobin concentration, and relative change in plasma volume |
|------------------------------------------|---------------------------------------------|---------------------------------------------|-------------------------------|</p>
<table>
<thead>
<tr>
<th>Treatment</th>
<th>Packed cell volume (%) Before run After run</th>
<th>Haemoglobin (g/100 ml) Before run After run</th>
<th>Change in plasma volume (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>44.5 (2.6) 45.8 (2.7)*</td>
<td>15.3 (1.0) 15.6 (0.9)*</td>
<td>–4.5 (3.1)</td>
</tr>
<tr>
<td>Citrate</td>
<td>42.5 (1.9)† 43.7 (2.2)†</td>
<td>14.7 (1.2)† 15.1 (1.1)†</td>
<td>–4.9 (1.8)</td>
</tr>
</tbody>
</table>

Values represent mean (SD).
*Significantly different (p < 0.05) from before run.
†Significantly different (p < 0.05) from placebo treatment.

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Changes in the concentrations of lactate and glucose in plasma</th>
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</thead>
<tbody>
<tr>
<td>Metabolite</td>
<td>Treatment</td>
</tr>
<tr>
<td>----------</td>
<td>-----------</td>
</tr>
<tr>
<td>Lactate (mmol/l)</td>
<td>Placebo</td>
</tr>
<tr>
<td>Citrate</td>
<td>2.2 (1.1)</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>Placebo</td>
</tr>
<tr>
<td>Citrate</td>
<td>4.9 (0.7) †</td>
</tr>
</tbody>
</table>

Values represent mean (SD).
*Significantly different (p < 0.05) from before run.
†Significantly different (p < 0.05) from placebo treatment.
Take home message

- Ingestion of sodium citrate shortly before endurance running may improve performance.
- Ingestion of sodium citrate at a dose of 0.4–0.6 g/kg body mass has the potential to cause gastrointestinal distress.
- The efficacy of sodium citrate should be individually tested before its use as an ergogenic aid in competitions.

5 km running time trial performance in well trained college runners. The precise mechanism of action remains to be elucidated.

ACKNOWLEDGEMENTS

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REFERENCES