Effects of Acute Ingestion of Sodium Citrate on Metabolism and 5-km Running Performance: A Field Study

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Abstract/Résumé

The purpose of the study was to (a) assess the effects of sodium citrate ingestion on metabolism and performance capacity in a 5-km competitive outdoor stadium run in trained male runners, and (b) elucidate the potential relationship between citrate-induced changes in plasma volume, body mass, and performance. Ten subjects (age 22.1 ± 2.5 yrs, body mass 74.1 ± 6.1 kg, height 180.1 ± 5.7 cm, VO₂max 60.8 ± 5.5 ml·kg⁻¹·min⁻¹) participated in the study. There was no effect of treatment on 5-km running time: 1100.0 ± 79.1 and 1082.7 ± 62.0s in citrate (CIT) and in placebo (PLC) trials, respectively, p = 0.09. Blood pH increased from 7.34 ± 0.07 to 7.49 ± 0.07 (p = 0.002) as a result of administering sodium citrate in the amount of 0.5 g·kg⁻¹ body mass in 1.5 litres of solution but remained stable while the equal volume of placebo drink was consumed: 7.40 ± 0.04 and 7.44 ± 0.09. The relative change in plasma volume after administering the drink was −1.99 ± 3.49% in the PLC and 9.75 ± 6.51% in the CIT trial (p = 0.001). Body mass did not differ before drinking; however, before the start the subjects were heavier in the CIT trial (74.2 ± 6.1 kg) vs. the PLC trial (73.4 ± 6.2 kg, p = 0.048). The shifts in plasma volume and body mass were not related to changes in performance. The results suggest that ingestion of sodium citrate induces an...
Le but principal de cette étude est d’analyser les effets de l’apport de citrate de sodium sur le métabolisme et sur la performance physique au cours d’une épreuve de course extérieure de 5 km chez des sujets masculins entraînés. De plus, cette étude se propose aussi d’évaluer la relation entre les variations de volume plasmatique, de masse corporelle, et de performance induites par le citrate. Dix sujets (âge: 22,1 ± 2,5 ans, masse corporelle: 74,1 ± 6,1 kg, taille: 180,1 ± 5,7 cm, VO₂ max: 60,8 ± 5,5 ml·kg⁻¹·min⁻¹) participent à cette étude. On ne dénote aucun effet du citrate de sodium sur le temps de performance: 1100,0 ± 79,1 et 1082,7 ± 62,0 s dans la condition de consommation d’une solution de citrate (CIT) et d’une solution-placebo (PLC), respectivement (p = 0,09). Le pH sanguin passe de 7,34 ± 0,07 à 7,49 ± 0,07 (p = 0,002) à la suite de la prise de 1,5 L de solution de citrate de sodium à raison de 0,5 g·kg⁻¹ de masse corporelle; dans la condition de placebo, le pH reste stable: 7,40 ± 0,04 et 7,44 ± 0,09. La variation relative du volume plasmatique après avoir pris la solution est de −1,99 ± 3,49% dans la condition PLC et de 9,75 ± 6,51% dans la condition CIT (p = 0,001). Avant la prise de la solution, la masse corporelle était la même, mais avant le début de l’effort, la masse était plus importante dans la condition CIT (74,2 ± 6,1 kg) que dans la condition PLC (73,4 ± 6,2 kg, p = 0,048). Les variations de volume plasmatique et de masse corporelle ne sont pas corrélées aux variations de performance. D’après les observations faites, le citrate de sodium favorise la rétention d’eau, augmente le volume plasmatique et le pH sanguin, mais n’améliore pas la performance de coureurs entraînés sur 5 km en terrain extérieur.

Introduction

Induced metabolic alkalosis by sodium bicarbonate or sodium citrate ingestion has been shown to increase blood lactate concentration during exercise when accompanied by an increase in the concentration of HCO₃⁻ (Hollidge-Horvat et al., 2000; McNaughton, 1992; Stephens et al., 2002). The increased blood lactate concentration commonly observed during and after exercise with induced metabolic alkalosis results from accelerated glycogenolysis and increased lactate accumulation in human skeletal muscle, as well as from increased lactate efflux from muscle cells (Hollidge-Horvat et al., 2000). Enhanced glycogenolytic ATP production apparently contributes to enhanced muscle function and improved performance observed in short-term intense exercise with induced alkalosis (Bird et al., 1995; Linossier et al., 1997; McNaughton et al., 1999a).

The observation by Hollidge-Horvat et al. (2000) that induced metabolic alkalosis increases muscle glycogen use, lactate production and accumulation, calculated muscle H⁺ concentration, and the decline in muscle phosphocreatine content during 15 min of cycling at 75% maximal O₂ uptake (VO₂ max) suggests that this manipulation could be detrimental to physical working capacity during prolonged exercise. However, research data do not confirm any detrimental effect of alkalizers on endurance performance. For instance, ingestion of sodium bicarbonate or sodium citrate has been shown to have no effect on running time to exhaustion at various treadmill velocities (George and MacLaren, 1988; Hooker et al., 1987; Pottteiger et al., 1996b) or on performance during endurance cycling (Schabort et al., 2000; Stephens et al., 2002). Contrary to these data, some well
controlled studies have shown that ingestion (McNaughton et al., 1999b; Ööpik et al., 2003; Potteiger et al., 1996a; Shave et al., 2001) and intravenous infusion (Mitchell et al., 1990) of alkalizers before or during exercise may even improve prolonged exercise performance over approximately 10 to 60 min.

Thus the effect of administering alkalizers before or during endurance exercise on performance is equivocal. The exact mechanism as to why buffer ingestion improves endurance performance under some circumstances is also unclear. The results of the study of Mitchell et al. (1990) suggest that the ergogenic effect of sodium bicarbonate infused during endurance cycling may be attributed not only to the enhanced buffering capacity of the body but also to the increased plasma volume resulting from the administration of sodium-containing fluid. On the other hand, solution of sodium citrate in comparison with low-sodium drink has been shown to induce an increase in body mass 2 hours after administration (Ööpik et al., 2003).

This effect of sodium citrate ingestion could be expected to reduce the positive effect of alkalization and/or increase in plasma volume on endurance capacity, especially during running exercise. Indeed it is noteworthy that buffer ingestion has been consistently shown to have no effect on running time to exhaustion at different predetermined treadmill velocities (George and MacLaren, 1988; Hooker et al., 1987; Potteiger et al., 1996b). On the contrary, two groups of researchers (Ööpik et al., 2003; Shave et al., 2001) who allowed their subjects to choose their own running pace according to how they felt during exercise have reported significantly improved performance in 5- and 3-km time trials after sodium citrate consumption. It is evident that running to exhaustion at a constant velocity is not reflective of a competitive athletic event, whereas a well controlled time trial represents the behaviour of athletes during real competition considerably better. Thus the data of Ööpik et al. (2003) and Shave et al. (2001) suggest that sodium citrate ingestion may improve performance in long- and middle-distance running.

However, the generalizability of the results of these laboratory experiments to actual competitive situation remains unclear. Uncontrollable variables which exist outside laboratory conditions such as the weather, the influence of other athletes and spectators, etc., might cause variations in performance capacity as much as the possible effect of alkalizers. Moreover, the high potential for gastrointestinal distress (Ööpik et al., 2003; Schabort et al., 2000; Stephens et al., 2002) likely precludes the use of sodium bicarbonate and sodium citrate during competitions among athletes, and the potential relationships between the changes in plasma volume, body mass, and performance have not been systematically addressed. Therefore, in order to validate the previous laboratory research, the purpose of the present study was to assess the effects of sodium citrate administration shortly before exercise on metabolism and performance capacity in a 5-km competitive outdoor stadium run in trained male runners. The additional aim of the study was to elucidate the potential relationships between citrate-induced changes in plasma volume, body mass, and performance.

**Methods**

The two treatments, 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 12 days.
SUBJECTS

Ten trained young male 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 any with preexisting medical conditions that would contraindicate their involvement in the study. Their mean ± SD age, body mass, height, and maximal oxygen uptake (VO\textsubscript{2max}) at the beginning of the study were 22.1 ± 2.5 years, 74.1 ± 6.1 kg, 180.1 ± 5.7 cm, and 60.8 ± 5.5 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}, respectively. They had been involved in regular training for 11.9 ± 3.0 years.

PROCEDURE

All subjects were tested three times in a 3-hr postabsorptive state. The first occasion was to complete a maximal aerobic power test to determine VO\textsubscript{2max} in laboratory conditions, and the other two were to undertake a 5-km run in an outdoor stadium. The runners were instructed to abstain from vigorous exercise the day before 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 prior to 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 in order to ensure stable nutritional and training status of the subjects throughout the study period.

The maximal oxygen uptake of the subjects was measured during a progressive exercise test performed on a treadmill (Runrace HC 1400, Technogym, Gambettola, Italy) as described by Ööpik et al. (2003). The test began with a 5-min warm-up. The speed was then increased from the initial rate of 8 km·h\textsuperscript{-1} after every 200 m by 0.5 km·h\textsuperscript{-1} until the runner could no longer maintain the pace. The protocol of the graded exercise used in this study is based on principles originally developed by Conconi et al. (1982). Expired gas was sampled and analysed continuously using an online system (True Max 2400, Parvo Medics, East Sandy, UT). The analyser was calibrated before each subject was tested.

The dose of sodium citrate used in the present study (0.5 g·kg\textsuperscript{-1} body mass) has been reported to be the most appropriate for inducing the greatest increase in HCO\textsubscript{3}\textsuperscript{-} concentration (McNaughton, 1990) and achieving an optimal alcalotic state 100–120 minutes after ingestion (Potteiger et al., 1996b). Our previous research (Ööpik et al., 2003) revealed that administering the needed amount of sodium citrate in 1 litre of solution during a short time interval (10 min) induced gastrointestinal distress and other negative side effects in most subjects. These symptoms may have been induced by high osmolality of the experimental drink (Linderman and Gosselink, 1994). Considering this, for the present study we prepared a less concentrated solution of sodium citrate and modified the procedure of administration. Thus, in the CIT trial the subjects ingested 1.5 litres of fluid containing sodium citrate (0.5 g·kg\textsuperscript{-1} body mass). The latter was dissolved in water and flavoured with very low energy flavouring. In the PLC trial, 1.5 litres of water was used and its taste was disguised with flavourings. The energy content of both drinks was less than 4.18 kJ·L\textsuperscript{-1}. 
The subjects started drinking 3 hours before exercise and administered the prescribed volume of fluid within 1 hour. For this purpose they were provided with 250-ml plastic cups and instructed to drink a cup of solution after equal time intervals (approx. 12 min) throughout the 1-hr administration period. The solutions were consumed in the same manner in both trials. The subjects were asked to report to the researchers any gastrointestinal distress experienced during the two trials. Body mass was measured just prior to administering the solution, immediately before starting the 5-km run, and after finishing it. The subjects were allowed to use the toilet between the first and second body mass measurement. They did not consume any food or other beverages after drinking the 1.5 litres of treatment solution throughout the testing procedure in each trial. They performed their customary prerace warm-up before undertaking the 5-km stadium run.

In order to create a real competitive situation during the test run, the subjects were pair-matched according to their expected performance capacity. During the race they were continuously encouraged to run as fast as they could, but they were not given any information on split times. Environmental conditions (ambient temperature 19 °C, humidity 50–60%, varied clouding) were similar on both outdoor testing days.

**BIOCHEMICAL ANALYSES**

Blood samples were drawn from an arm vein (v. intermedia cubiti). To facilitate the sampling procedure, a tourniquet was used for a few seconds prior to insertion of the needle. One sample was drawn before administering the solutions, the second before the standardised warm-up for each test run, and the third sample was obtained 5 min after the end of the 5-km run. Blood was collected into Vacutainer tubes (7.0 ml, no additives) and also into Vacutainer tubes containing EDTA (4.5 ml).

The whole blood sample without additives was then used for measurement of pH (pH meter Sentron 2001, Federal Way, WA), haemoglobin concentration (cyanmethaemoglobin method; Boehringer Mannheim GmbH, Mannheim, Germany; diagnostic kit No. 124729) and packed cell volume (by spun haematocrit). The values obtained were used to calculate changes in plasma volume (Dill and Costill, 1974). The blood samples containing EDTA were immediately cooled down by placing the vacutainer tubes into ice-cold water. The tubes were then centrifuged and the plasma was stored at –25 °C for later lactate and glucose analyses. Lactate and glucose concentrations were measured enzymatically in plasma samples using diagnostic kits purchased from Biocon, Vöhl-Marienhagen, Germany: No. 301 (lactate) and No. 458 (glucose). The intra-assey coefficient of variation for haemoglobin, packed cell volume, lactate, and glucose in our laboratory is 1.6% (n = 27), 0.68% (n = 22), 1.0% (n = 10), and 1.2% (n = 10), respectively.

**STATISTICAL ANALYSIS**

Conventional statistical analysis was used to calculate the mean and SD for each parameter investigated. The distribution pattern of the data was tested using a one-sample Kolmogorov-Smirnov test. A dependent t-test was used to determine differences between the trials in time taken to complete the 5-km run, whereas a repeated-measures analysis of variance was used to identify differences between
treatments for the remaining dependent variables. Significance was set at \( p < 0.05 \). Pearson product-moment correlation coefficients were computed to determine the relationships between variables.

**Results**

There was no significant effect of treatment on 5-km running time. It took 1082.7 ± 62.0 s in the PLC trial and 1100.0 ± 79.1 s (\( p = 0.09 \)) in the CIT trial to complete the 5-km competitive run. It is noteworthy that only 3 of the 10 subjects achieved a better result after sodium citrate ingestion, whereas 7 were faster in the PLC trial. The average speed of running (Figure 1) was similar in both trials and in the different stages of the distance. The only exception was the 4th km in the CIT trial, which was completed significantly faster than the 3rd km: 17.5 ± 2.3 vs. 15.3 ± 1.3 km·h\(^{-1}\) (\( p = 0.03 \)). This was mainly due to 4 runners who, after the relatively slow 3rd km, increased their pace. However, only 2 of them achieved marginally better finish time in the CIT trial compared with the PLC trial.

The runners’ body mass as measured prior to administering the PLC or CIT drink did not differ in the two trials (73.7 ± 6.2 kg and 73.8 ± 6.3 kg, respectively). However, before the start of the test run the runners were heavier in the CIT trial (74.2 ± 6.1 kg) vs. in the placebo treatment (73.4 ± 6.2 kg, \( p = 0.048 \)). This difference was also maintained after the race (72.6 ± 6.1 kg in the PLC and 73.5 ± 6.1 kg in the CIT trial, \( p = 0.005 \)).

In the CIT trial, significantly lower packed cell volume and haemoglobin concentration were observed before the exercise test compared to the values measured prior to administering the drink (Table 1). The calculated relative change in

**Figure 1.** Running speed of subjects at each kilometre of the 5-km outdoor stadium run (mean ± SD). *Significantly different (\( p < 0.05 \)) from preceding kilometre.
Table 1  Packed Cell Volume, Haemoglobin Concentration, and Relative Changes in Plasma Volume (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>Citrate</th>
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<tbody>
<tr>
<td>Packed cell volume (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>43.7 ± 1.1</td>
<td>44.9 ± 0.9 #</td>
</tr>
<tr>
<td>Before run</td>
<td>44.1 ± 1.5</td>
<td>42.5 ± 1.7 * #</td>
</tr>
<tr>
<td>After run</td>
<td>45.8 ± 1.7 * $</td>
<td>44.6 ± 1.0 $ #</td>
</tr>
<tr>
<td>Haemoglobin (g·100 ml⁻¹)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>14.3 ± 0.4</td>
<td>14.7 ± 0.7</td>
</tr>
<tr>
<td>Before run</td>
<td>14.5 ± 0.3</td>
<td>14.0 ± 0.8 *</td>
</tr>
<tr>
<td>After run</td>
<td>14.9 ± 0.5 * $</td>
<td>14.6 ± 0.6 $</td>
</tr>
<tr>
<td>Changes in plasma volume (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline → before run</td>
<td>−1.99 ± 3.43</td>
<td>9.75 ± 6.51 #</td>
</tr>
<tr>
<td>Before run → after run</td>
<td>−6.11 ± 4.53</td>
<td>−7.45 ± 5.97</td>
</tr>
</tbody>
</table>

Note: Significantly different (p < 0.05): * from baseline value; $ from before-run value; # from placebo treatment.

plasma volume during the period between administering the solution and starting the run was −1.99 ± 3.49% in the PLC and 9.75 ± 6.51% in the CIT trial (p = 0.001). Packed cell volume and haemoglobin concentration increased significantly as a result of the 5-km run in both trials (Table 1). The decrease in plasma volume during the race did not differ in the two trials (Table 1).

The relationship between the differences in body mass and performance times in the two trials was not statistically significant (r = −0.19; p > 0.05). Similarly, differences in plasma volume shifts were not related to performance times (r = −0.06; p > 0.05).

The pH value measured in blood increased from 7.34 ± 0.07 to 7.49 ± 0.07 (p = 0.002) as a result of sodium citrate ingestion, but remained stable when the placebo drink was consumed (Figure 2). The extent of the decrease in pH value during the 5-km race was the same in the PLC (−0.10 ± 0.06) and CIT trial (−0.10 ± 0.11).

There was no difference in plasma lactate concentration between trials prior to administering the solutions as well as before the race (Figure 3). A significant increase in lactate level was observed as a result of the 5-km run in both trials. The measured concentration of lactate in plasma was significantly higher after the run in the CIT trial (13.6 ± 3.0 mmol·L⁻¹) than in the PLC trial (12.3 ± 1.5 mmol·L⁻¹, p = 0.04). However, this significant between-trial difference was not evident after correcting for the measured concentrations of lactate for the individual changes in plasma volume (11.5 ± 1.5 mmol·L⁻¹ in the PLC and 12.5 ± 2.6 mmol·L⁻¹ in the CIT trial, p = 0.16) (Figure 3).
Figure 2. Changes in blood pH (mean ± SD) under placebo and sodium citrate treatment. *Significantly different ($p < 0.05$) from baseline value.

Figure 3. Plasma lactate concentration (mean ± SD) under placebo and sodium citrate treatment. Significantly different ($p < 0.05$): * from baseline value; $ from before-run value.

Plasma glucose concentration did not differ prior to administering the PLC or CIT drink, but a significant difference was observed between the samples obtained before the test run (5.2 ± 0.3 mmol·L⁻¹ and 6.0 ± 0.4 mmol·L⁻¹, respectively, $p = 0.002$) (Figure 4). A significant increase in glucose level was observed as a result of the 5-km run in both trials; however, the extent of the increase was significantly higher in the PLC trial (4.54 ± 1.59 mmol·L⁻¹) vs. the CIT trial (2.32 ...
Effects of Sodium Citrate • 699

± 2.14 mmol·L⁻¹, p = 0.028) (Figure 4). The values of plasma glucose concentration measured after the PLC or CIT drink and after the run are presented as corrected for the individual changes in plasma volume.

Seven of the 10 athletes who participated in the study reported mild nausea and diarrhea following ingestion of the solution containing sodium citrate, whereas there were no such complaints in PLC trial.

Discussion

The primary finding of this study was that sodium citrate ingestion at a dose of 0.5 g·kg⁻¹ body mass shortly before exercise did not improve performance in a 5-km competitive outdoor stadium run in trained male runners. The ergogenic effect of sodium citrate on endurance running capacity observed in two recent time trial experiments (Ööpik et al., 2003; Shave et al., 2001) did not occur in competitive situation in the present study.

The positive effect of ingestion of alkalizers on endurance cycling performance has been attributed to increased efflux of lactate and H⁺ from contracting muscle cells (McNaughton et al., 1999b; Potteiger et al., 1996a). Indeed, the monocarboxylate transporter, which is thought to be responsible for lactate transport across the cell membrane (Roth, 1991), has been shown to be sensitive to pH gradient (Roth and Brooks, 1990). Increasing extracellular pH through the ingestion of alkalizers may facilitate the efflux of intracellular lactate and H⁺. In the case of an intensively contracting skeletal muscle, this means a delay in the decrease in intracellular pH to the critical level at which glycolysis is inhibited. Improved performance in an endurance running time trial, together with the significantly higher concentration of lactate measured in plasma after the run following citrate ingestion compared with the placebo treatment (Ööpik et al., 2003; Shave

Figure 4. Plasma glucose concentration (mean ± SD) under placebo and sodium citrate treatment. Significantly different (p < 0.05): * from baseline value; $ from before-run value; # from placebo treatment.
et al., 2001), is in accordance with this concept on the mechanism of action of alkalizers on physical working capacity.

Although the measured concentration of lactate in plasma was higher after the run in the CIT trial than in the PLC trial in the present study (see Results), this significant between-trial difference was not evident after correcting for the measured concentrations of lactate for the individual changes in plasma volume (Figure 3). Thus it seems there was no actual effect of sodium citrate ingestion on lactate efflux from working muscles in our subjects, and this could at least partly explain the lack of influence of this manipulation on performance.

Significant increase in blood pH (Figure 2) is a well-known effect of sodium citrate ingestion (Potteiger et al., 1996a, 1996b; Schabort et al., 2000). However, it is noteworthy that despite this change observed in the CIT trial, there were no between-trial differences in blood pH either before the start of the run or after finishing it. Thus there is no reason to expect that a more favourable pH gradient which would have stimulated the efflux of lactate and H+ from working muscles (Roth and Brooks, 1990) was achieved in the CIT trial compared with the PLC treatment in the present study. This in turn may explain why there was no between-trial difference either in lactate efflux from working muscles or in blood lactate accumulation during the race.

An alternative explanation is that, due to blunted catecholamine response to exercise, glucogenolysis and lactate production in muscles during the run was to some extent suppressed in the CIT trial compared with the PLC trial. Bouissou et al. (1988) demonstrated that in trained athletes, alkalosis induced by ingestion of sodium bicarbonate reduced epinephrine concentration in blood by 34% in comparison with control conditions in response to cycling exercise at 375 W until exhaustion. On the other hand, Febbraio et al. (1998) found intramuscular glycogen utilization, glycolysis, and carbohydrate oxidation to be augmented by elevated plasma epinephrine during a 40-min cycling trial at 71% VO2max in trained triathletes. In addition, both muscle and plasma lactate and plasma glucose concentrations were significantly elevated with increased epinephrine levels in plasma (Febbraio et al., 1998). Unfortunately, we did not measure the possible changes in blood hormones in the present study. However, a significantly smaller extent of an increase in blood glucose level during the race in the CIT trial compared with the PLC trial (Figure 4) may reflect a blunted catecholamine response.

In addition to alkalosis, an acute increase in plasma volume has been shown to blunt the exercise-induced rise in catecholamines and attenuate glycogen use during prolonged exercise (Grant et al., 1996; Green et al., 1989). In this respect it is noteworthy that the calculated relative change in plasma volume during the period between administering the solution and starting the race was different in the two trials, revealing a significantly greater increase in the CIT trial vs. the PLC trial (see Results).

Increased plasma volume in the CIT trial in the present study fits well with our previous observation about significantly lower packed cell volume and blood haemoglobin concentration 2 hours after sodium citrate ingestion compared with placebo administration (Ööpik et al., 2003). An increase in plasma volume after sodium citrate ingestion is an expected result. Most of the sodium absorbed from the intestinal tract remains in the extracellular fluids (Lindinger et al., 1999), and a
sodium load comparable to that used in the present study has been shown to result in an approximately 1-litre increase in plasma volume, which may persist for more than 3 hours after ingestion of sodium-containing solution (Lindinger et al., 2000).

Collectively the changes in plasma volume and body mass after consumption of 1.5 litres of solution (see Results) reveal significantly greater fluid retention with sodium citrate treatment compared with placebo. This could be expected because the sodium content of the citrate drink was much higher than that of the placebo. Therefore, immediately before the start of the run, our subjects were heavier in the CIT than in the PLC trial; moreover, this difference was maintained at the end of the race. Consequently, owing to their approximately 1% greater body mass, they had to work harder during the run in the CIT trial. This additional workload, together with gastrointestinal discomfort reported by 7 subjects in the CIT trial, could also help reduce any positive effect that sodium citrate ingestion may have had on endurance running performance. However, the weak relationship observed between the differences in body mass changes and performance times in the two trials (see Results) does not support this suggestion.

Although there are several plausible explanations for the lack of effect of sodium citrate ingestion on performance in the present study, it remains unclear why this dietary manipulation enhances physical working capacity during high-intensity endurance running exercise in well-controlled time trial experiments (Ööpik et al., 2003; Shave et al., 2001) but not in field conditions in a real competitive situation. Indeed, endurance working capacity has been found to be increased following citrate administration despite concomitant symptoms of gastrointestinal distress (Ööpik et al., 2003; Potteiger et al., 1996a; Shave et al., 2001) and increase in body mass (Ööpik et al., 2003). However, it is possible that the ingestion of sodium citrate in the present study was initiated too early before the start of the run. The solution was consumed within 60 min, beginning 180 min before exercise (see Methods). In the two time trial experiments mentioned above, the subjects began their fluid intake 120 min (Ööpik et al., 2003) or 90 min (Shave et al., 2001) before the start of the run and they consumed the prescribed amount of solution within 10 min or 30 min, respectively. The results of the present study also suggest that the positive effect of sodium citrate ingestion on endurance performance demonstrated in well-controlled time trial experiments may be negated by the influence of some uncontrollable variables present in a real competitive environment.

In conclusion, the results of the present study suggest that ingestion of sodium citrate in the amount of 0.5 g·kg⁻¹ body mass in 1.5 litres of solution induces an increase in water retention, plasma volume, and blood pH before exercise, but does not improve performance in a 5-km competitive run in field conditions in trained male runners. The shifts in plasma volume and body mass induced by sodium citrate administration are not related to changes in performance.

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Effects of Sodium Citrate • 703


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