The effects of sodium citrate ingestion on metabolism and physical performance capacity

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Summary

High-intensity exercise is associated with the accumulation of lactate and hydrogen ions (H+) within active muscle and blood. The rise in the concentration of H+ (fall in pH) induces fatigue. Ingestion of sodium bicarbonate or sodium citrate has been shown to increase blood pH and HCO₃⁻ concentration and to facilitate the efflux of intracellular lactate and H+ from contracting muscle cells. This, in turn, may delay the fall in intramuscular pH to the critical level, postpone the development of fatigue and improve performance during intense exercise. Unfortunately, ingestion of sodium bicarbonate induces gastrointestinal distress in many subjects. On the other hand, it is believed that sodium citrate administration has all the benefits of sodium bicarbonate without the associated negative side effects. However, analysis of the literature reveals that the possible ergogenic effect of sodium citrate has been assessed in sport-specific time-trials in very few studies and the results are inconsistent. Although in some cases ingestion of sodium citrate shortly before exercise has been shown to increase performance in running distances of 3000 and 5000 m as well as in 30-km cycling in male athletes, the results of other similar experiments have not confirmed these findings. In case an increased performance after sodium citrate ingestion has been detected, it has been associated with higher post-exercise blood lactate concentration in comparison with placebo treatment. In studies where the dose of sodium citrate of 400 mg · kg⁻¹ body mass or more have been administered, 74% of subjects have reported gastrointestinal distress, whereas lower doses seem to have no effect on performance in sport-specific time-trials. The results of some studies suggest that sodium citrate administered in 0.8 - 1.5 l of solution in a dose of 400 - 500 mg · kg⁻¹ induces an increase in water retention, plasma volume and body mass and restrains an increase in blood glucose concentration during intense exercise.

Keywords: running, cycling, buffer ingestion, ergogenic aid.

Introduction

In high intensity exercise anaerobic glycolysis significantly contributes to the energy supply in contracting muscles. Glycolysis produces lactic acid that dissociates into lactate ion (La⁻) and hydrogen ion (H+), which accumulate in muscle cell and blood. The rise in the concentration of H+ (fall in pH) in muscle and blood induces fatigue (Green, 1997). The latter is identifiable as a reduction in muscle force production (Green, 1997) and an increase in the perceived effort (Swank & Robertson, 1989). Substances that have potential to increase the buffering capacity of the body have also potential to increase physical performance capacity in high intensity exercise. The fact, that performance in high intensity exercise is improved by alkalotic treatment and impaired by dietary strategies that decrease blood pH, was observed many years ago (Dennig et al., 1931). The substances that have most frequently been used with aim of increasing the capacity of body to buffer H+ are sodium bicarbonate and sodium citrate (Requeña et al., 2005; Burke et al., 2006). These substances have similar biochemical and physiological effects, however, the use of sodium citrate is considered to be preferable to that of sodium bicarbonate because the former seems to induce less gastrointestinal upsets, cramps or diarrhoea (Kowalchuk et al., 1989; McNaughton, 1990; McNaughton & Cedaro, 1992; Parry-Billings & McLaren, 1986).

The present paper is intended to summarize the current body of data concerning the effects of sodium citrate ingestion on metabolic response to exercise and on physical performance capacity. In focus of the paper are the studies, in which the duration of exercise test has been at least 2 min, which have been carried out in field conditions or in laboratory settings as time trials, and in which appropriate doses of sodium citrate have been used.

1. Effects of sodium bicarbonate or sodium citrate ingestion on body pH regulation

Ingestion of Na-bicarbonate (NaHCO₃) induces an increase in the concentration of HCO₃⁻ [HCO₃⁻] in blood (Wilkes et al., 1983). Because the cell membrane is impermeable to HCO₃⁻ ions (Robin, 1961) the direct buffering effect of sodium bicarbonate occurs in the extracellular compartment only. However, buffering H+ ions in blood increases the intracellular/extracellular H+ gradient. Increased H+ gradient stimulates the La⁻/H+ cotransporter located in muscle cell membrane resulting in enhanced efflux of La⁻ and H+ from cell (Roth & Brooks, 1990).
In this way an increase in \([\text{HCO}_3^-]\) in blood has an indirect influence on intracellular \(pH\) regulation in muscle. Hence, during intense exercise after sodium bicarbonate ingestion elevated \([\text{HCO}_3^-]\) in blood may help to reduce the extent of the fall in \(pH\) in contracting muscle cells and, through that, improve performance.

Sodium citrate (\(\text{Na}_2\text{C}_6\text{H}_5\text{O}_7\cdot\text{H}_2\text{O}\)) dissociates into sodium (\(\text{Na}^+\)) and citrate (\(\text{Cit}^-\)) ions in body fluids. The \(\text{Cit}^-\) is expelled from the plasma, and, as a result of that, electrical equilibrium is disturbed. Electrical neutrality is restored by increasing \([\text{HCO}_3^-]\) and decreasing \([\text{H}^+]\) in plasma. The precise biochemical mechanism behind \(\text{HCO}_3^-\) production from sodium citrate is unknown (Potteiger et al., 1996a). However, due to the rise in \([\text{HCO}_3^-]\) in blood the effect of sodium citrate ingestion on body \(pH\) regulation is essentially the same as that achieved by sodium bicarbonate administration.

2. Effects of sodium citrate ingestion on performance: significance of dose and type of exercise

McNaughton (1990) studied the effect of different doses of sodium citrate (from 100 to 500 mg · kg\(^{-1}\) body mass) on performance in cycle ergometer exercise. Significant improvement in 1 min sprint performance was observed after subjects ingested 300 - 500 mg · kg\(^{-1}\) of citrate, with the greatest amount of work being performed in the trial where 500 mg · kg\(^{-1}\) dose was administered.

In another study McNaughton and Cedaro (1992) assessed the effect of ingestion of sodium citrate in a dose of 500 mg · kg\(^{-1}\) body mass on performance in cycle ergometer exercise of 10, 30, 120 and 240 s duration. Their main finding was that both total work performed and peak power achieved were significantly increased as a result of sodium citrate ingestion in maximal exercise of 120 and 240 s of duration, but not in efforts lasting only 10 or 30 s.

Ibanez et al. (1995) have suggested that a minimum production of lactic acid and \(\text{H}^+\) may be needed during exercise for any significant effect of pre-exercise ingestion of alkalinizers to be observed on the contribution of anaerobic glycolysis to overall energy generation in intensively working muscle. They noted that in studies in which no effect was found after alkalosis, peak blood lactate under the placebo condition was lower (6 mmol · l\(^{-1}\)) than in the studies in which a significant metabolic effect was observed (9 - 18 mmol · l\(^{-1}\)) (Ibanez et al., 1995).

3. Effects of sodium citrate on metabolic response to exercise and on physical performance capacity in running and cycling

The summary of the studies of the effects of sodium citrate ingestion in which the duration of test exercise employed has been at least 2 min and which have been carried out in field conditions or in laboratory settings as time trials is presented in Table 1.

Tiryaki and Atterbom (1995) studied the effects

<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects</th>
<th>Dose (mg · kg(^{-1}))</th>
<th>Mode of study</th>
<th>Mode of exercise</th>
<th>Distance</th>
<th>Performance</th>
<th>Blood lactate</th>
<th>Blood glucose</th>
<th>GI distress (number of cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tiryaki and Atterbom, 1995</td>
<td>15 female</td>
<td>300</td>
<td>field</td>
<td>running</td>
<td>600 m</td>
<td>NS</td>
<td>NS</td>
<td>Not measured</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>Ööpik et al., 2007 *</td>
<td>17 female</td>
<td>400</td>
<td>field</td>
<td>running</td>
<td>1500 m</td>
<td>NS</td>
<td>NS</td>
<td>▼</td>
<td>15 (88.2%)</td>
</tr>
<tr>
<td>Shave et al., 2001</td>
<td>7 male, 2 female</td>
<td>500</td>
<td>field</td>
<td>running</td>
<td>3000 m</td>
<td>†</td>
<td>†</td>
<td>Not measured</td>
<td>8 (88.9%)</td>
</tr>
<tr>
<td>Ööpik et al., 2003</td>
<td>17 male</td>
<td>500</td>
<td>laboratory</td>
<td>running</td>
<td>5000 m</td>
<td>†</td>
<td>†</td>
<td>▼</td>
<td>17 (100%)</td>
</tr>
<tr>
<td>Ööpik et al., 2004</td>
<td>10 male</td>
<td>500</td>
<td>field</td>
<td>running</td>
<td>5000 m</td>
<td>NS</td>
<td>NS</td>
<td>▼</td>
<td>7 (70%)</td>
</tr>
<tr>
<td>Potteiger et al., 1996</td>
<td>8 male</td>
<td>500</td>
<td>laboratory</td>
<td>cycling</td>
<td>30 km</td>
<td>†</td>
<td>†</td>
<td>Not measured</td>
<td>3 (37.5%)</td>
</tr>
<tr>
<td>Schabert et al., 2000</td>
<td>8 male</td>
<td>200</td>
<td>laboratory</td>
<td>cycling</td>
<td>40 km</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>Not measured</td>
</tr>
<tr>
<td></td>
<td></td>
<td>400</td>
<td></td>
<td></td>
<td></td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td></td>
</tr>
</tbody>
</table>

* Unpublished work. NS: no difference between sodium citrate and placebo treatment. †: significant improvement in performance after sodium citrate ingestion compared with placebo treatment, or significantly higher post-exercise blood lactate concentration in sodium citrate trial compared with placebo trial. ▼: significantly lower post-exercise blood glucose concentration in sodium citrate trial compared with placebo trial. GI distress: the number of subjects who reported the symptoms of gastrointestinal distress and their proportion (percentage) in the whole study group is indicated.
of sodium bicarbonate and sodium citrate ingestion on 600-m running performance in female runners. The substrates were administered randomly in a crossover and double-blind fashion for each subject. The doses were 300 mg · kg⁻¹ in case of both alkalinizers, the substrates as well as placebo (sugarless drink) were ingested 2.5 hours prior to the start of the race. Although pH and [HCO₃⁻] in blood were significantly higher in sodium bicarbonate and sodium citrate trials in comparison with placebo trial before and after the run, no between-trials differences were observed in performance times. Moreover, the concentration of lactate in blood was similar in different trials immediately before and after the race. Thus, alkali ingestion resulted in significant shifts in the blood acid-base balance, but apparently did not facilitate the efflux of lactate from working muscles and failed to affect the 600-m running performance in young female runners.

The purpose of our study (Ööpik et al., 2007, unpublished work) was to assess the effects of sodium citrate ingestion on metabolic response to exercise and performance in a 1500-m competitive run in trained female middle-distance runners in field condition. Sodium citrate (400 mg · kg⁻¹) and placebo (1.0% sodium chloride) were administered in a counterbalanced, crossover, randomly assigned double-blind manner two hours prior to the race. The time taken to cover the 1500-m distance did not differ in the citrate and placebo trial. In citrate trial compared to placebo trial greater relative increase in plasma volume after administering the experimental solution, increased body mass immediately before and after the race and restrained increase in blood glucose concentration during the race were observed. The results suggest that sodium citrate induces an increase in water retention before exercise and may modify carbohydrate metabolism in high intensity running, but does not improve performance in 1500-m competitive run in female middle-distance runners.

Elite triathletes and modern pentathletes (7 men and 2 women) completed two 3000-m runs after administering 500 mg · kg⁻¹ of sodium citrate or placebo (NaCl, 100 mg · kg⁻¹) (Shave et al., 2001). The substrates were dissolved in 1 l of sugar-free, fruit-flavoured fluid and administered in a double-blind and randomly assigned manner 1 hour before the start of the time-trial. Performance time was significantly faster (by 10.7 s; 1.7%) in the sodium citrate trial compared with the placebo trial. Better performance was associated with significantly higher blood lactate concentration in blood samples taken after the run in citrate trial compared with placebo trial. The authors concluded that the ergogenic effect of sodium citrate might be explained by preservation of intracellular environment, facilitated by an enhanced efflux of lactate and H⁺.

Well-trained male college runners performed a 5-km treadmill run with and without sodium citrate ingestion in a random, double-blind, crossover design (Ööpik et al., 2003). In the citrate trial the subjects consumed 1 l of solution containing 500 mg · kg⁻¹ of sodium citrate two hours before the run. In placebo trial flavoured mineral water was administered in the same manner. Much greater fluid retention during the period between administering the solution and starting the run was observed with sodium citrate treatment compared with placebo. As a result, the subjects in the citrate trial started the run on average 0.7 kg heavier than in placebo trial. Despite that, the time required to complete the run was significantly faster (by 30.6 s; 2.6%) in citrate trial than in the placebo trial. Significantly higher lactate concentration was measured in the plasma of the subjects after the run in citrate trial compared with the placebo trial. This suggests that improvement in performance in citrate trial was associated with facilitated lactate and H⁺ efflux from contracting muscles during the run. Contrary to lactate plasma glucose concentration was much lower in samples taken after the run in citrate trial compared with placebo. Taken together the results of this study show that ingestion of sodium citrate shortly before start may improve performance in 5-km run in well-trained young runners.

In another study conducted by the same group (Ööpik et al., 2004) trained young male runners were studied in field conditions. In general features the study design followed that employed in the experiment described above (Ööpik et al., 2003), the dose of sodium citrate was also the same, i.e. 500 mg · kg⁻¹. However, in order to reduce the likelihood of gastrointestinal distress less concentrated solution of sodium citrate was prepared and the procedure of administration was modified. Thus, in the citrate trial the subjects ingested 1.5 l of fluid containing sodium citrate and in placebo trial the same amount of flavoured water was ingested. The athletes started drinking 3 hours before the start of the run and administered the prescribed volume of fluid within 1 hour. In order to create a real competitive situation during the test run, which took place on the outdoor stadium, the subjects were pair-matched according to their expected performance capacity. Ingestion of
sodium citrate induced an increase in water retention, plasma volume, and blood pH before exercise. However, no between-trial differences were observed in performance times. These findings show that the generalizability of the results of well-controlled laboratory experiments to actual competitive situations is questionable. Uncontrollable variables which exist outside laboratory conditions such as the weather, the influence of other athletes and spectators, etc., may cause variations in performance capacity as much as the possible effect of alkalinizers.

Trained male competitive cyclists participated in the study conducted by Potteiger et al. (1996b). The subjects performed two 30-km cycling time trials. The two treatment conditions (sodium citrate and placebo) were administered in a counterbalanced, crossover, randomly assigned double blind manner. Both sodium citrate and placebo (wheat flour) were ingested in a dose of 500 mg · kg⁻¹ with 1 l of water 1.5 hours before exercise. Performance time was significantly faster (by 102.7 s; 2.9%) in the sodium citrate trial compared to the placebo trial. Better performance in citrate trial was associated with higher blood pH and lactate concentration during exercise. The authors concluded that the ergogenic benefit of sodium citrate was most likely due to the establishment and maintenance of favourable metabolic conditions, which facilitated lactate and H⁺ ion efflux out of the working muscles.

Schabort et al. (2000) studied trained male cyclists and triathletes with aim to determine whether sodium citrate enhances endurance cycling performance and, if so, what dosage is needed for that. The experimental conditions involved the ingestion of three dosages of sodium citrate (200, 400 and 600 mg · kg⁻¹) and placebo (calcium carbonate, 100 mg · kg⁻¹) 1 hour prior to exercise. The substances were dissolved in 400 ml of water. The subjects performed four 40-km time-trials on their own bicycles mounted on an ergometer. In order to mimic the cycle road races, the time-trials included four 500-m, four 1-km and two 2-km sprints. The highest citrate dose induced the highest blood pH and [HCO₃⁻] before as well as during exercise. However, no significant differences were observed in blood lactate concentrations between the four treatments. Likewise, no between-trial differences were measured in power output, sprint performance or time taken to complete the 40-km simulated race. The authors concluded that additional factors, rather than the accumulation of H⁺ ions in the muscle, are the cause of fatigue and exhaustion during long-lasting cycle exercise.

4. Negative side effects of sodium citrate ingestion

Although ingestion of sodium bicarbonate has potential to enhance performance in high intensity exercise, its use in competitive sports is limited because it induces severe gastrointestinal distress in many subjects (Requena et al., 2005; Lindeman & Gosselink, 1994). Sodium citrate compared with sodium bicarbonate has often been considered as less dangerous substance (Kowalchuk et al., 1989; McNaughton, 1990; McNaughton & Cedaro, 1992; Parry-Billings & McLaren, 1986). However, the analysis of the papers referred to above shows, that in case the dose of sodium citrate ingested has been 400 mg · kg⁻¹ or more, 25-100% of individuals in different studies have experienced gastrointestinal problems (Table 1). Pooling the number of subjects studied and that of those who reported about gastrointestinal symptoms reveal, that the incidence of gastrointestinal distress following sodium citrate ingestion is as high as 74%. Some authors suggest that the failure of sodium citrate to produce improvements in performance could be due to gastrointestinal symptoms experienced by many subjects (Shabort et al., 2000), whereas others have observed increased performance despite moderate (Potteiger et al., 1996) to very high (Shave et al., 2001; Ööpik et al., 2003) incidence of complaints like nausea, diarrhoea, feelings of bloatedness and increased flatulence. Lower dosages of sodium citrate are apparently less dangerous in this regard, however, they also seem to be ineffective in respect of performance (Table 1). Anyway, the potential of sodium citrate to induce gastrointestinal distress should not be underestimated and its use in real competitive situation should be preceded by individualized trials in training conditions.

Conclusion

The available data on the effect of ingestion of sodium citrate on performance in sport-specific exercise tasks of 2 min or longer duration is contradictory. Although in some cases ingestion of sodium citrate shortly before exercise has been shown to increase performance in running distances of 3000 and 5000 m as well as in 30-km cycling in male athletes, the results of other similar experiments have not confirmed these findings. In case an increased performance after sodium citrate ingestion has been detected, it has been associated with higher post-exercise blood lactate concentration in comparison with placebo treatment. Female subjects have been studied in two
occasions only and the results suggest that ingestion of sodium citrate shortly before exercise does not improve performance in middle-distance running in female athletes. In studies where the dose of sodium citrate of 400 mg · kg⁻¹ body mass or more have been administered, 74% of subjects have reported gastrointestinal distress, whereas lower doses seem to have no effect on performance in sport-specific time-trials. The results of some studies suggest that sodium citrate administered in 0.8 - 1.5 l of solution in a dose of 400-500 mg · kg⁻¹ induces an increase in water retention, plasma volume and body mass and restrains an increase in blood glucose concentration during intense exercise. Further studies are needed in order to enhance the current comprehension of the potential advantages and disadvantages of using sodium citrate in sports practice.

REFERENCES

SODOS CITRATO VARTOJIMO ĮTAKA MEDŽIAGŲ APYKAITAI IR FIZINIAM PAJĖGUMUI

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SANTRAUKA

Didelio intensyvumo fizinė veikla susijusi su laktato ir vandenilio jonų (H⁺) kaupimu dirbančiuose raumenyse ir kraujoje. H⁺ didėjimas (ir atitinkamai pH mažėjimas) sukelia nuovargi. Pastebėta, kad sodos bikarbonato arba sodos citrato vartojimas (tirpalo pavidalu, geriant) didina kraujo pH ir bei HCO₃⁻ koncentraciją kraujoje bei palengvina tarpšlėtiniu laktato ir H⁺ ištekėjimą iš susitraukiančių raumenų lašelių. Tai savo ruožtu gali sulėtinti tarpšlėtinio pH mažėjimą iki kritinio lygio, atitolinti nuovargio
Santrauka

Tyrimo tikslas – nustatyti šildymo ir šaldymo poveikį raumens nuovargiui ir atsigavimui, priklausomai nuo lyties. Tiriamačių kontingentą sudarę 19–23 metų moterys (n=10) (ūgis 166,4 ± 5,6 cm; kūno masė 56,2 ± 6,1 kg) ir 18–23 metų vyrai (n=10) (ūgis 177,8 ± 5,8 cm; kūno masė 78,2 ± 6,1 kg). Tiriamieji buvo testuoti izokinetiniu dinamometru. Registruotas maksimaliosios jėgos momentas (MJM). Buvo atliekami dideliu (450°/s) greičiu, sumažino netiesioginá raumenų pažeidimų simptomá – kreatinkinazës aktyvumá (tiek vyrø, tiek moterø) yra didesnë už lenkiamøjø. Tiek šildymas, tiek šaldymas prieš krûvá, kuris tačiau vyrø raumenų izokinetinio susitraukimo jėgos reikšmës buvo didesnës negu moterø. Blauzdos tiesiamojø raumenø MV, kad temperatūros pokytis nepriklausomai nuo lyties nepadidino blauzdos tiesiamøjø ir lenkiamøjø raumenø MJM, praëjus 24 h po krûvá, reikšmingai (p < 0,05) skiriasi tarp ÁTR ir šaldytø bei tarp ÁTR ir šildytø raumenø. Iš gautø rezultatø ir šaldytø raumenø bei šaldytø ir šildytø raumenø jėgos pokyčiø skirtumo. Vyrø ir moterø CK aktyvumas kraujo serume, pastebëtas joks teigiamas poveikis sporto rezultatams. Kai kurių tyrimo rezultatai rodo, kad sodos citrata, kai jo rekomenduojama išgerti 0,8–1,5 l (tirpalo), skaičiuojant 400–500 mg vienam žmogui. Kainų kiekio ir kuno masės sulaikymà bei apribojus glikozës koncentracijos kraujo serume padaugėjo didējimas intensyvios fizinës veiklos metu.

Raktažodžiai: šildymas, šaldymas, sportas, sportininkø medžiagų važiavimas, metų temperatūros pokyčiai, veiklos sutrikimai, kūno vietojû, kūno temperatûroje, sporto rezultatai, jėgos momentas.