Original Article

Sodium citrate ingestion increases glycolytic activity but does not enhance 2000 m rowing performance

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ABSTRACT

Martins AN, Artoli GG, Franchini E. Sodium citrate ingestion increases glycolytic activity but does not enhance 2000m rowing performance. *J. Hum. Sport Exerc.* Vol. 5, No. 3, pp. 411-417, 2010. Sodium citrate-induced alkalosis is an ergogenic strategy that has been proven to enhance physical performance in high-intensity exercises by increasing muscle buffer capacity and reducing the influence of H^+ on energy production and contractile processes. The objective of the present study was to evaluate whether acute sodium citrate ingestion may contribute to rowing performance in a 2000-m race simulation. Six well-trained competitive rowers took part in the study, but five of them have completed the whole experimental protocol. They were assessed twice for performance and lactate 2.5 h after the ingestion of a 750-mL natural mango juice containing sodium citrate (0.5 $g \cdot kg^{-1}$) or no substance added (placebo). The two experiments occurred 7-15 days apart. The study was conducted in a double-blind, placebo-controlled, cross-over fashion. Performance was assessed in a rower ergometer and blood lactate was determined in both conditions at rest and after exercise. Heart-rate and oxygen consumption were monitored throughout the tests. Data were analyzed using the Wilcoxon's signed rank test. Sodium citrate yielded a significantly higher lactate response to exercise than placebo ($p<0.05$), but no significant differences were found between treatments for performance, heart-rate and oxygen consumption. In conclusion, sodium citrate promoted a favorable metabolic environment for exercise performance but did not exert any influence on simulated rowing performance. **Key words:** SODIUM CITRATE, ALKALOSIS, ACID-BASE, FATIGUE, ROWING, PERFORMANCE.

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INTRODUCTION

Rowing is a popular sport in which practitioners may find a healthy way to exercise and improve their fitness levels as well as to compete recreationally or at high level, such as in Olympic Games. A standard competitive rowing race is of 2000 and typically lasts about 7 minutes. This may vary according to the type of racing boat, number of competitors in the same boat, number of oars used by each competitor or even according to the weight class (de Campos Mello, de Moraes Bertuzzi, Grangeiro, & Franchini, 2009).

Studies addressing the relative contribution of the energy transferring systems to the total energy produced during a ~7-min maximal exercise have shown that the approximate contribution of the aerobic metabolism is of ~80-85% whilst the anaerobic contribution accounts for ~15-20% of the total (Hagerman, 2000). In fact, Pripstein, Rhodes, McKenzie, and Coutts (1999) estimated that 88% of the energy contribution during the 2000 m on the ergometer came from the aerobic metabolism, leaving 12% for the anaerobic metabolism, while Russell, Le Rossignol, and Sparrow (1998) found values such as 84% of aerobic contribution and 16% of anaerobic contribution. On the other hand, studies conducted in older rowing ergometers (Hagerman, Connors, Gault, Hagerman, & Polinski, 1978; Mickelson & Hagerman, 1982) reported a smaller participation of the aerobic system, 70 and 72%, respectively. A recent study conducted on water revealed that the contribution was 87±2% aerobic, 7±2% alactic and 6±2% lactic (de Campos Mello et al., 2009).

Hence, anaerobic capacity may be responsible for 10-15% of competitive rowing performance. Furthermore, in the final 250 m athletes normally increase the intensity of their efforts, which is popularly known as the “final sprint”. By doing so, the anaerobic contribution increases substantially and this capacity to increase the speed and sustain it can indeed represent the difference between win and loss. This is especially evident in high-level competitions, in which the competitive level is barely identical and, therefore, any minimal increase in performance might be decisive to the final result.

Increasing muscle buffer capacity has been a well documented strategy for enhancing performance in anaerobic-dependent exercises. A number of studies have shown the efficiency of sodium bicarbonate ingestion on high-intensity performance (Artioli, Gualano, Coelho, Benatti, Gailey, & Lancha, 2007; McNaughton, Siegler, & Midgley, 2008; Raymer, Marsh, Kowalchuk, & Thompson, 2004), but acute ingestion may cause some detrimental effects (e.g., gastrointestinal disturbances) and hampers exercise performance. Alternatively, sodium citrate has been proven to elicit similar positive effects on high-intensity performance being free of the side effects normally observed after sodium bicarbonate ingestion (McNaughton & Cedaro, 1992; Requena, Zabala, Padial, & Feriche, 2005). It is hypothesized that sodium bicarbonate and citrate ingestion increases the extracellular buffer capacity, augmenting the efflux of hydrogen ions (H^+) and lactate from muscle cells to the extracellular fluid, therefore resulting in a less acidotic environment in muscle cells (Artioli et al., 2007; Horswill, 1995). This would, in turn, allow the glycolytic pathway to be more active for a longer time during exercise, supplying more energy to the exercise and releasing more lactate to the blood. Such mechanism likely explains the increased blood and muscle lactate concentration after induced alkalosis as well as the ergogenic effects of induced alkalosis (Ball & Maughan, 1997; Bouissou, Defer, Guezennec, Estrade, & Serrurier, 1988).

Considering the significant contribution of the anaerobic metabolism to competitive rowing performance and the potential ergogenic effect of sodium citrate-induced alkalosis on exercises with these characteristics, the purpose of the present study was to investigate whether sodium citrate ingestion is or is not an effective strategy for improving performance in a simulated 2000-m rowing race.

MATERIAL AND METHODS

Participants

Six male competitive rowers took part in this study (age: 24±6 years; weight: 79.6±13.8 kg; height: 177.7±3.2). At the time of the data collection, they were regularly engaged in at least 6 rowing training sessions per week and actively competing in state and national level competitions. One voluntary, however, gave up from participation so a total of five subjects have completed the whole experimental protocol.

All subjects were informed about study's objectives and the procedures and risks involving their participation before signing the written informed consent. All procedures were approved by the Institutional Ethics Committee.

Study design

Each participant attended the laboratory on two different occasions (PRE and POST), 7-15 days apart. With the exception of the substance administered, the subjects were submitted to identical procedures on each occasion, which comprised: rowing performance assessment, blood collection for lactate determination and oxygen consumption (VO_2) measurement. One hundred and thirty minutes before the beginning of the experimental procedures, the subjects were required to ingest either sodium citrate (0.5 $g \cdot kg^{-1}$ of body weight diluted in 750 mL of natural mango juice) or placebo (750 mL of natural mango juice only). Both solutions were identical in appearance and flavor. The study was conducted in a double-blind, placebo-controlled and cross-over design. The order of the substances administered was chosen at random.

Procedures

Before the performance assessment, the subjects warmed-up at 100 W for 5 minutes in the same rowing ergometer used for the performance test. After that, they performed a 2000-m race simulation on a rowing ergometer (Concept II, Morrisville, USA). Power output was recorded at every 500 m and the time to complete the task was also recorded using a digital chronometer. Throughout the test, heart rate was measured through the use of an electronic frequencimeter (Polar Pace, Finland), VO_2 was determined with a portable system (VO_{2000}, Med Graphics) and blood samples were collected through by a small puncture in the earlobe. A drop of blood was then placed in a reagent strip and the blood lactate was determined with the Accusport device (Mannhein Boehringer).

Statistical Analysis

Data is presented as mean ± standard deviation. Due to the reduced sample size and to the non-homocedasticity of the data set, we have performed a non-parametric statistical analysis. Hence, the Wilcoxon's signed rank test was applied to each variable in order to compare the treatments (i.e., sodium citrate vs. placebo). The alpha level was previously set at 5%.

RESULTS

Sodium citrate ingestion resulted in no significant changes in rowing performance, as evidenced by both power output (Figure 1, panel A) and by the time-to-complete the 2000 m (panel B). As expected, the performance has apparently increased in the final 500 m, which represents the sprint. However, even during the sprint, sodium citrate ingestion elicited no performance improvements (Figure 1).

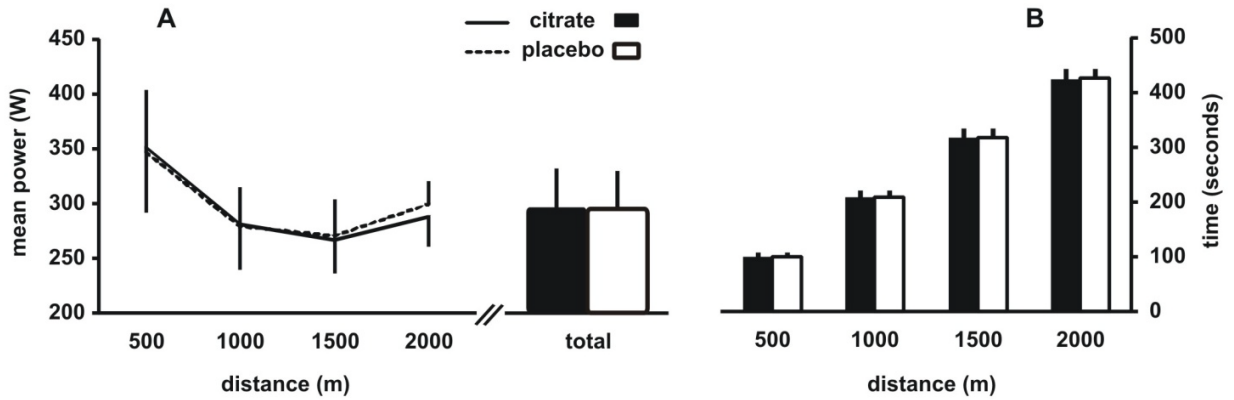

***Figure 1**. Rowing performance evaluated by power output (panel A) and time-to-completion (panel B) after ingestion of sodium citrate and placebo. No significant differences between treatments were observed.*

Heart-rate (Figure 2, panel A) and oxygen consumption (Figure 2, panel B) were not significantly different between treatments. Resting blood lactate was similar between treatments but exercise has promoted a higher lactate response when sodium citrate was ingested in comparison to placebo (Figure 3).

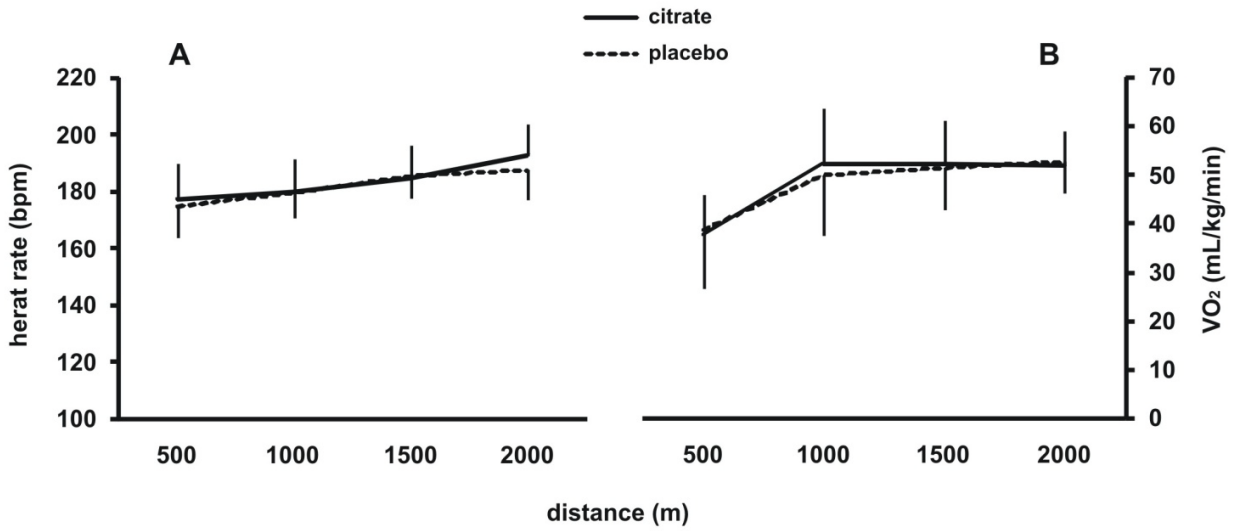

***Figure 2**. Heart-rate (panel A) and oxygen consumption (panel B) responses to exercise after sodium citrate and placebo. No significant differences were observed.*

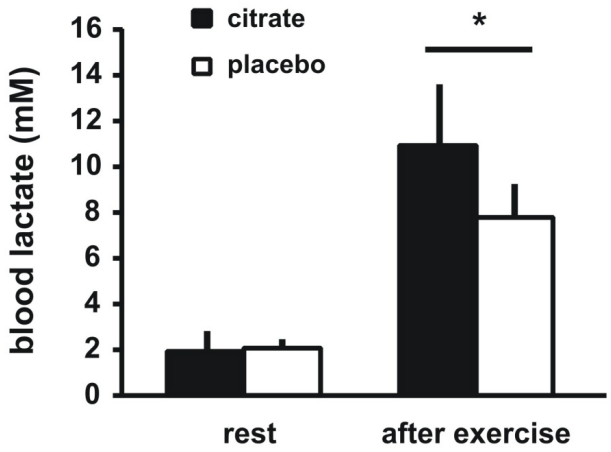

***Figure 3**. Blood lactate response at rest and after exercise following the ingestion of sodium citrate and placebo (*p<0.05).*

DISCUSSION

The main finding of the present study is that acute sodium citrate ingestion, despite resulting in a higher lactate response to exercise, did not improve simulated rowing performance. This result is of great practical application as it demonstrates that an ergogenic strategy which has been extensively used in high-intensity exercises is not effective in this specific task. To the best of our knowledge, this is the first report to evaluate sodium citrate in 2000 m rowing performance.

Literature has been consistently shown the efficiency of the same supplementation protocol used in this study (i.e., 500 mg·kg^{-1} of body weight ingested 150 minutes prior to the exercise) for increasing exercise performance (McNaughton, 1990; Potteiger et al., 1996). Moreover, McNaughton (1990) tested different dosage strategies for sodium citrate (i.e., from 0.1 to 0.5 g·kg^{-1} of body weight) and found that 0.5 g·kg^{-1} is the mostly effective dose. Thus, the supplementation protocol is unlike to explain the lack of positive results in our study. On the other hand, literature has unequivocally demonstrated that induced alkalosis is most ergogenic in exercises whose glycolytic activity and acidosis are markedly high (Artioli et al., 2007; Bishop, Edge, Davis, & Goodman, 2004; McNaughton, 1992). Even though 2000 m rowing is a high-intensity exercise, the anaerobic glycolysis contribution is about 7% (de Campos Mello et al., 2009), which may be not enough for evidencing any performance improvement promoted by citrate ingestion. We hypothesized that rowing performance could be enhanced due to increased in exercise intensity during the final 500 m. However, even considering performance at this stage, no effects of sodium citrate were observed. In fact, most of studies using single bouts of exercise lasting more than 3 minutes have not found any ergogenic effect of alkalosis (McNaughton, 1992; Stephens, McKenna, Canny, Snow, & McConell, 2002). Thus, the relative low glycolytic contribution is the most reasonable explanation for our results. This has happened despite the more favorable metabolic environment resulted by citrate ingestion, as evidenced by the higher lactate concentration.

In the present study, heart rate remained unchanged under the two treatments. Although a lower blood acidosis is expected to occur after sodium citrate ingestion (Requena et al., 2005) and, therefore, a lower heart rate response to exercise would be also expected, data by Bracken, Linnane, and Brooks (2005) demonstrated that blood catecholamine response to exercise is not influenced by sodium citrate ingestion, which provides a good reason for the lack of effects of alkalosis on heart rate. Additionally, studies with

sodium bicarbonate ingestion have repeatedly shown no effects of metabolic induced alkalosis on this parameter (Siegler & Hirscher, 2010), therefore supporting our findings. Similarly, VO_2 was also kept unchanged under the experimental conditions. This is not surprising as a number of previous studies on metabolic induced alkalosis did not show significant alterations of oxygen consumption in maximal exercises (Cox & Jenkins, 1994; Kozak-Collins, Burke, & Schoene, 1994) although it can affect VO_2 kinetics (Berger et al., 2006).

CONCLUSION

In short, acute sodium citrate ingestion elicited a higher lactate response to exercise, suggesting greater lactate efflux from the muscle as well as greater glycolytic activity and, consequently, an intramuscular environment more favorable to performance. In spite of that, 2000 m rowing performance was not influenced by treatment, even during the final sprint. No other physiological alterations were observed after citrate ingestion. In conclusion, sodium citrate is not an ergogenic aid in rowing competitions.

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