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High doses of sodium bicarbonate increase lactate levels and delay exhaustion in a cycling performance test

Luis H.B. Ferreira MSc [a,b,*], Andre C. Smolarek MSc [a,c], Philip D. Chilibeck PhD [b], Marcelo P. Barros PhD [d], Steven R. McAnulty PhD [e], Brad J. Schoenfeld PhD [f], Bruna Amorim Zandona MSc [a], Tácito P. Souza-Junior PhD [a,e]

[a] Research Group on Metabolism, Nutrition and Strength Training, Department of Physical Education, Federal University of Parana, Curitiba, Brazil
[b] University of Saskatchewan, College of Kinesiology, University of Saskatchewan, Saskatoon, Sakatchewan, Canada
[c] Centro-Oeste State University, Irati, Brazil
[d] Institute of Physical Activity and Sports Science (ICAFE), Cruzeiro do Sul University, São Paulo, Brazil
[e] Appalachian State University, Department of Health and Exercise Science, Appalachian State University, Boone, North Caroline, USA
[f] CUNY Lehman College, Bronx, New York, USA

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ABSTRACT

Objectives: It is well established that ingestion of sodium bicarbonate ($NaHCO_3$) causes metabolic alkalosis. However, there is no consensus in terms of optimal $NaHCO_3$ doses leading to enhanced performance. This study aimed to determine the effects of different $NaHCO_3$ doses on performance and lactate clearance in non-professional cyclists.

Methods: Twenty-one cyclists performed the following three double-blind trials: 1) ingestion of 0.3 g · kg^{-1} body weight (BW) of placebo; 2) ingestion of 0.1 g · kg^{-1} BW $NaHCO_3$ plus 0.2 g · kg^{-1} BW placebo (0.1 BC); and 3) ingestion of 0.3 g · kg^{-1} BW $NaHCO_3$ (0.3 BC). Performance was evaluated after warm-up on the bike followed by a performance test until exhaustion. Lactate levels were monitored in blood samples before and immediately after performance tests.

Results: Lactate levels in the blood were significantly higher after exercise in 0.3 BC and 0.1 BC (15.12 ± 0.92 versus 10.3 ± 1.22 and 13.24 ± 0.87 versus 10.3 ± 1.22 mmol/L; $P < 0.05$) compared with control. Significant improvements in performance were only identified in 0.3 BC group (76.42 ± 2.14; $P = 0.01$).

Conclusions: The present study found that 0.3 g · kg^{-1} BW $NaHCO_3$ is effective in improving performance and improving blood lactate levels in cyclists compared with control and 0.1 g · kg^{-1} BW $NaHCO_3$.

Introduction

During the last few years, more attention has been paid to understanding the relationship between high-intensity exercise (HIE) and buffering capacity [1]. As a result of HIE, an accumulation of specific glycolytic metabolites may be found in the blood circulation [2]. The lactate was misinterpreted through the years, where the exercise/fatigue relationship was associated with high lactate concentrations found in the blood after strenuous exercise [3]. Nevertheless, although lactate levels significantly increase during HIE, one of the main causes of fatigue may be a result of a high hydrogen (H^+) accumulation, a prominent product of glycogen degradation [1–4]. The acidity resulting from the H^+ accumulation is not considered the main cause of fatigue; however, this byproduct related to anaerobic metabolism presents some deleterious effects under the fatigue perspective, affecting whole glycolysis metabolism through phosphofructokinase (PFK) inhibition [5]. Therefore because of an increased competition of H^+ and calcium (Ca^{++}) concentration in the muscle cells, some impairments in performance may manifest during HIE [1].

Considering the importance of buffering capacity to expedite H^+ removal and retard the inhibition of glycolytic enzymes, the combination of nutritional strategies aiming to promote a better clearance of byproducts is an intriguing approach to improve the exercise performance [5–8]. According to American College of Sports Medicine, several substances are potentially efficacious in promoting improvements on buffering capacity, including sodium bicarbonate ($NaHCO_3$) and β-alanine supplementation [9].

The administration of $NaHCO_3$ as an alkalizing agent has been employed for decades as a means to enhance H^+ buffering capacity

* Corresponding author. Tel.: +554234222847.
E-mail address: lhboikoferreira@gmail.com (L.H.B. Ferreira).

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in blood during athletic performance [10]. The exercise-related acidosis and metabolic alkalosis (i.e., resulting from the ingestion of supplements) has been investigated thoroughly, particularly with respect to alkalotic agents such as $NaHCO_3$, sodium citrate, and the acidotic agent ammonium chloride [11]. During intense exercise, lactate accumulation in blood and muscles is coincident with pH decrease in both biological matrices [3]. The decrease of pH levels in the blood is related to the increased use of anaerobic glycolysis and consequent production of acidic compounds [3]. It has been proposed that supplementing exogenous $NaHCO_3$ provides an electrochemical gradient between the intra- and extracellular milieu, thus favoring the removal of H^+ protons during intense exercise [6]. It is widely believed that increased intramuscular acidity can drastically limit the capacity to perform high-intensity exercise [12], so the capacity to buffer the accumulation of H^+ protons is vital to sustain exercise performance.

High-intensity exercise requires muscle glycogen breakdown through anaerobic glycolysis to supply adenosine triphosphate (ATP) and maintain high force production. A few studies have already reported the effect of 0.3 $g \cdot kg^{-1}$ body weight (BW) $NaHCO_3$ as a particular dosage associated with exercise performance improvements in prolonged and high-intensity exercises [8,13,14]. Previous research has found that higher doses, such as 0.4 $g \cdot kg^{-1}$ BW $NaHCO_3$ [15] or 0.5 $g \cdot kg^{-1}$ BW $NaHCO_3$ [7], are effective in improving performance in prolonged high-intensity exercises. However, research has yet to determine if performance can be enhanced with lower dosages. Therefore, in an attempt to avoid the side effects related to ingestion of sodium bicarbonate, a lower dose than previously employed [7,8,12–14] may lead to performance gains while concurrently avoiding commonly encountered negative side effects.

Although some researchers have investigated the effects of $NaHCO_3$ on cycling [4,8,9,13], none have addressed the effect of different $NaHCO_3$ doses on performance or lactate levels in the blood. Therefore this study aimed to determine the ergogenic benefit of different doses of $NaHCO_3$ and the ratio of lactate levels in the blood during high-intensity cycling performance. Moreover, the present study endeavored to investigate the hypothesis that a higher dose of $NaHCO_3$ would be more effective than lower doses for improving performance and enhancing the glycolytic capacity, as analyzed through lactate levels.

Methods

Experimental approach

This study design was a double-blind, randomized, crossover study involving three different experimental trials performed across 3 consecutive weeks, whereby participants served as their own control. The tests included the following: 1) ingestion of 0.3 $g \cdot kg^{-1}$ BW of calcium carbonate ($CaCO_3$), used as placebo to provide a control analysis; 2) 0.1 $g \cdot kg^{-1}$ BW $NaHCO_3$ as a lower dose combined with 0.2 $g \cdot kg^{-1}$ BW $CaCO_3$, which was used in an attempt to equalize the volume and the weight of substances; and 3) ingestion of 0.3 $g \cdot kg^{-1}$ BW of $NaHCO_3$ as a higher dose, with a washout period of 7 d between each test session (Fig. 1).

Inclusion and exclusion criteria

All participants had to meet the following inclusion criteria: 1) male; 2) classified as physically active by International Physical Activity Questionnaire [16]; and 3) answered negatively to all items of the Physical Activity Readiness Questionnaire. The following criteria were causes for exclusion: 1) presence of joint, neurologic, cardiovascular, or respiratory issues that may impair performance; 2) use of medications that affect exercise responses; and 3) self-report of contraindication to high-intensity physical exercise, based on medical examinations performed within 12 months before the beginning of the evaluations. All participants signed an informed consent agreement.

Participants

At first, 26 cyclists were selected to compose the sample of the present study; however, after applying the eligibility criteria, only 21 cyclists (169 ± 12 cm, 20 ± 2 y, 85 ± 7 kg) were accepted for the study from our university student population. The cyclists were non-smokers and recreationally trained (e.g., usually practicing cycling activities four times a week for 1 h at each segment, reaching a mean of 11 ± 2 miles at each hour of cycling). Participants were informed of the risks associated with $NaHCO_3$ ingestion and the performance test. All participants provided written informed consent. The study was approved by the university's institutional ethics board.

Anthropometric components

In the first visit to the laboratory, we assessed participants' body weight and body fat (% BF) with a tetrapolar bioelectrical impedance analysis (BIA) using a Tanita-BIA (Model TFB-310, Tanita Corp., Tokyo, Japan). Height was measured with a stadiometer (Harpenden, Holtain Ltd., Crosswell, Wales) fixed in the wall following a standardized protocol [17].

Procedures

In the first visit to the laboratory, participants were familiarized with the HIE protocol that would be applied during the next three visits in an effort to avoid problems related to the lack of knowledge about the test. A food-intake record (R24) was collected and participants were advised to sustain a similar food intake for the next 4 wk of testing [18].

Study design

The study was conducted for 21 d, with the participants visiting the laboratory once a week, respecting a washout of 7 d between each session. Every day that the participants arrived in the laboratory, a food-intake record was collected followed by a baseline analyses for blood lactate, pH, and plasma HCO^3. Cyclists were given 10 min to ingest the $NaHCO_3/CaCO_3$ solutions. After ingestion, participants rested for 30 min before initiating a 5-min warm-up protocol on a cycle ergometer (Monark, Vansbro, Sweden) at 50 rpm against light resistance (1 kg), which was followed by the performance test [19].

The participants carried out the performance test immediately after the warm-up period by adding an extra load of 5% BW resistance and increasing cycling speed to 80 rpm. The cycling test was performed until exhaustion while time was recorded. After the cycling test, all participants performed an active recovery period, whereby resistance was reduced to 1 kg and pedaling frequency to 50 rpm for 15 min. Blood samples were collected at five different times: right after the arrival in the laboratory, 10 min before the cycling test (pretest), immediately after (posttest), and at 5 min and 15 min of active recovery period (+5 RP and +15 RP, respectively). The blood samples were analyzed for lactate 1 h after the blood collection with a YSI Xylem instrument (YSI, Yellow Springs, OH, USA). Both blood pH and HCO_3 were analyzed immediately using a Radiometer ABL800 FLEX blood gas analyzer (Radiometer America, Brea, CA, USA).

Statistical analysis

All data are presented as mean ± standard deviation. Differences between conditions are given as 95% confidence intervals (CIs). A Shapiro-Wilks test was used to verify normality of the data. Differences for performance scores (i.e., time to exhaustion) between conditions were assessed with repeated measures analysis of variance (ANOVA) with an a priori significance level set at $P < 0.05$. Blood lactate was assessed with a condition × time repeated measures ANOVA. The area under the blood lactate curve was determined across the exercise tests and compared between conditions with a repeated-measures ANOVA. To determine the effectiveness of the treatment group, the effects were quantified by calculating the effect size (ES), which is the difference between the placebo and the different doses of $NaHCO_3$, where the outcomes (means) was divided by the pooled pretest and posttest standard deviation of the three conditions. Classification of ES followed the guidelines proposed by Cohen [6]: 0 to 0.19 = trivial magnitude of effect; 0.20 to 0.49 = small magnitude of effect; 0.50 to 0.79 = moderate magnitude of effect; ≥0.80 = large magnitude of effect. Tukey's post hoc tests were used to determine differences between conditions when main effects or interactions were significant. All tests were analyzed using SPSS Software Version 20.0 (IBM Corp., Armonk, NY, USA). Significance was accepted at $P \leq 0.05$.

Results

Table 1 presents data on the concentration of lactate in blood (mmol/L) and cycling time until exhaustion (performance in seconds) of the recreational cyclists supplemented with 0.1 $g \cdot kg^{-1}$

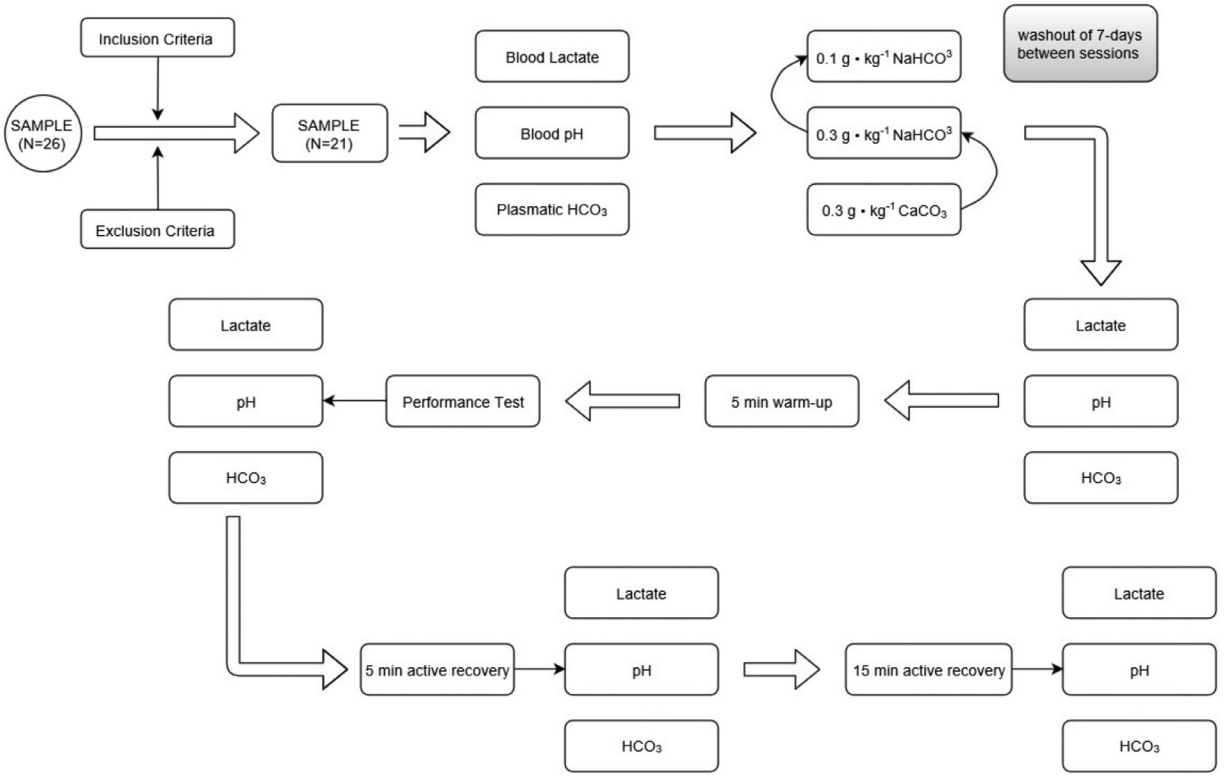

Fig. 1. Study protocol.

$NaHCO_3$ or 0.3 g · kg^{-1} $NaHCO_3$ before the test (pretest), immediately after the cycling test (posttest), and 5 and 15 min after the active recovery period (+5 RP and +15 RP, respectively). ($n = 21$; $P < 0.05$).

Results indicate a significant improvement in performance when a higher dose of sodium bicarbonate was used compared with a lower dose and placebo condition (76.42 ± 4.41 to 65.27 ± 8.31″ with $P = 0.001$, ES = 0.64; and 76.42 ± 4.41 to 68.03 ± 5.41 with $P = 0.001$, ES = 0.64). There was a condition × time interaction for blood lactate ($P < 0.01$; Table 1). Before cycling tests, no significant differences were identified in lactate. Posttest lactate results indicated that both 0.1 g · kg^{-1} $NaHCO_3$ ($P < 0.001$; 95% CI = 0.85–2.90 mmol/L) and 0.3 g · kg^{-1} $NaHCO_3$ ($P < 0.001$; 95% CI = 1.88–2.90 mmol/L) conditions were significantly higher than control. At 5-min active recovery, 0.3 g · kg^{-1} $NaHCO_3$ had significantly higher lactate than control ($P < 0.001$; 95% CI = 3.39–5.57 mmol/L). At 15 min active recovery, 0.3 g · kg^{-1} $NaHCO_3$ was also significantly higher than control ($P < 0.001$; 95% CI = 1.38–3.94 mmol/L). The $NaHCO_3$ experimental conditions were significantly

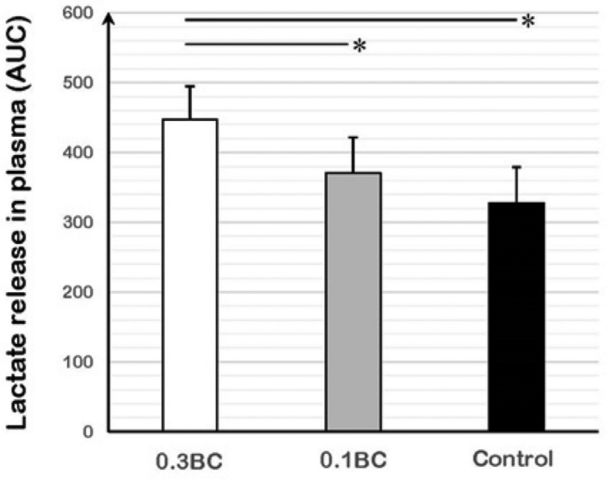

Fig. 2. Total lactate release in bloodstream (calculated as AUC; mmol · L^{-1} · s^{-1}) during a cycling test. AUC, area under the curve; BC, bicarbonate.

Table 1
Lactate concentrations in the blood (mmol/L) and performance test in seconds

	0.3 g · kg^{-1} $NaHCO_3$	0.1 g · kg^{-1} $NaHCO_3$	Control group	*P*
Pretest	1.75 ± 0.82	1.89 ± 0.23	1.96 ± 0.17	0.09
Posttest	15.12* ± 0.92	13.24* ± 0.87	10.33† ± 1.22	0.001
+5 RP	13.75* ± 1.19	11.92 ± 1.10	9.26† ± 0.94	0.001
+15 RP	7.57 ± 1.44	5.29 ± 1.20	4.90 ± 1.16	0.001
Performance (s)	76.42*,† ± 4.41″	65.27 ± 8.31″	68.03 ± 5.41″	0.001

RP, active recovery period.
All values are mean ± standard deviation.
Tukey's post hoc test on the significant condition × time interaction:
*Significantly different versus control ($P < 0.01$).
†Significantly different versus 0.1 g · kg^{-1} $NaHCO_3$ ($P < 0.01$).

different across all sampling periods ($P < 0.001$) except pretest, where no significant differences were identified.

Figure 2 depicts the total amount of lactate released in the blood of cyclists during the performance test (i.e., pre- to posttest). It is worth noting that these values are related to the exercising time until exhaustion, which differed across conditions. Nevertheless, both 0.1 g · kg^{-1} $NaHCO_3$ (17.3%) and 0.3 g · kg-1 $NaHCO_3$ (26.8%) conditions resulted in higher levels of total blood lactate during the cycling test compared with control ($P < 0.05$).

For performance (i.e., time to exhaustion), there was no difference between control and 0.1 g · kg^{-1} $NaHCO_3$ groups ($P = 0.53$), whereas time to exhaustion was significantly extended in the 0.3 g · kg^{-1} $NaHCO_3$ group compared with control ($P < 0.01$; 95% CI = 2.1–14.7 s; Fig. 1). The higher dose $NaHCO_3$ condition also had a greater time to exhaustion than the lower dose condition ($P < 0.001$; 95% CI = 4.86–17.43 s).

On the other hand, total lactate in the blood in the posttest was significantly higher in the groups treated with 0.3 g · kg^{-1} $NaHCO_3$ and 0.1 g · kg^{-1} $NaHCO_3$ compared with the control group. This condition was 31.8% higher than control (Fig. 2). No significant difference was identfied when comparing 0.3 g · kg^{-1} $NaHCO_3$ and the 0.1 g · kg^{-1} $NaHCO_3$ conditions.

Figure 3 depicts the ratio of difference between blood pH pre- and postsupplementation and the differences during the performance test and active recovery period. During presupplementation, no significant differences were identified between groups. However, after the supplementation, in the pretest period, a significant difference was identified in the group that ingested a higher dose of $NaHCO_3$ compared with the control group (7.6 ± 0.26 to 7.1 ± 0.21 with $P = 0.001$). In the posttest analysis, the group that ingested a higher dose of $NaHCO_3$ presented significantly higher values of blood pH compared with the group with a lower dose and control (7.4 ± 0.3 to 6.9 ± 0.21 with $P = 0.01$ and 7.7 ± 0.31 to 6.7 ± 0.14 with $P = 0.001$, respectively). The same behavior was identified after 5 min of active recovery, whereas the higher dose significantly improved the blood pH compared with the other two conditions (7.5 ± 0.17 to 7.1 ± 0.11 with $P = 0.04$ and 7.5 ± 0.17 to 7.0 ± 0.09 with $P = 0.01$). No other significant difference was identified during conditions ($P > 0.05$).

The blood HCO_3 analysis can be visualized in Figure 4, where the values are reported for the presupplementation, pretest, posttest, 5 min of active recovery, and 15 min of active recovery in both conditions. No statistical differences were identified in the presupplementation period ($P > 0.05$). In the pretest, after supplementation, a significant improvement was identified with a higher dose of $NaHCO_3$ compared with a lower dose and the control group (33.4 ± 1.32 to 28.2 ± 1.29 with $P = 0.001$ and 33.4 ± 1.32 to 27.9 ± 1.48 with $P = 0.001$, respectively). The blood HCO_3 presented a similar pattern in the posttest condition (29.7 ± 2.88 to 22.4 ± 1.89 with $P = 0.01$ and 29.7 ± 2.88 to 18.6 ± 1.77 with $P = 0.001$). With 5 min of active recovery, blood HCO_3 was significantly higher in the 0.3 bicarbonate compared with control (22.7 ± 1.79 to 16.88 ± 1.32 with $P = 0.001$), followed by similar improvements after 15 min of active recovery, whereas a higher dose promoted a significant improvement compared with control (26.9 ± 1.2 to 21.3 ± 1.77 with $P = 0.03$). No other statistical difference was identified in testing conditions ($P > 0.05$).

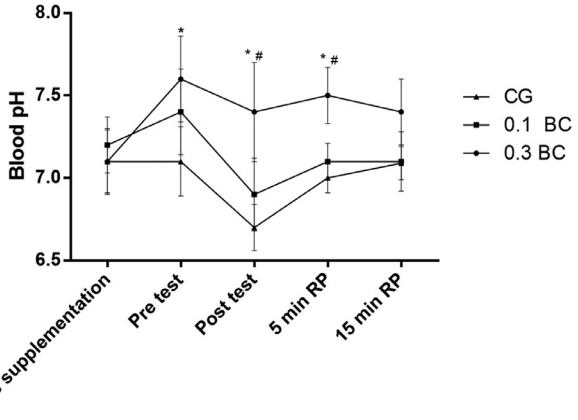

Fig. 3. Blood pH ratio during the whole intervention conditions. *Significant difference between 0.3 BC and CG (with $P < 0.05$); #Significant difference between 0.3 BC and 0.1 BC (with $P < 0.05$); 0.1 BC = 0.1 g · kg^{-1} $NaHCO_3$; 0.3 BC = 0.3 g · kg^{-1} $NaHCO_3$. BC, bicarbonate; CG, control group; RP, active recovery period.

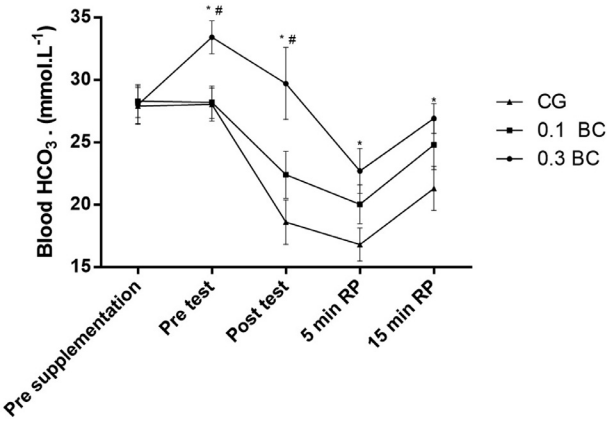

Fig. 4. Blood HCO_3 behavior with different doses of $NaHCO_3$ and placebo. *Significant difference between 0.3 BC and CG (with $P < 0.05$); #Significant difference between 0.3 BC and 0.1 BC (with $P < 0.05$); 0.1 BC = 0.1 g · kg^{-1} $NaHCO_3$; 0.3 BC = 0.3 g · kg^{-1} $NaHCO_3$. BC, bicarbonate; CG, control group; RP, active recovery period.

Discussion

To our knowledge this is the first study to examine two different doses of $NaHCO_3$ within the same study and analyze its time-dependent effects on the performance and lactate concentration in cyclists. This study found that 0.3 g · kg^{-1} BW $NaHCO_3$ increased cycling performance (based on time until exhaustion) and increased lactate levels during the pre- and postexercise period, whereas a lower dose of 0.1 g · kg^{-1} BW only resulted in significantly higher lactate levels immediately posttest compared with placebo condition.

Early metabolic concepts (from the 1930s) proposed that mammalian skeletal muscles were not able to synthesize glycogen from lactate [20]. In the 1970s, researchers established that our body could actually convert lactate into glucose and produce ATP for contractile activity in working muscles. From a biochemistry perspective, the cellular production of lactate during the exercise has several benefits for force production based on a retrograde glucose-generating system involving liver metabolism (the Cori cycle). Several studies have already reported the deleterious effects of H^+ accumulation on energy-supplying processes [2,3,21], which reinforces the hypothesis that H^+ buffering capacity could be vital to exercise performance. Cytosolic lactate dehydrogenase (LDH) catalyzes the pyruvic acid reduction to lactic acid with the concomitant regeneration of cytosolic NAD^+ for further oxidative processes (e.g. the glyceraldehyde 3-phosphate dehydrogenase reaction). This, in turn, sustains the cytosolic redox potential (NAD^+/NADH), supports continued substrate flux through phase two of glycolysis, and allows continued ATP regeneration from glycolysis [3].

Regardless, during intense exercise the high anaerobic energy turnover with resultant intramuscular H^+ accumulation (or inorganic phosphate; Pi) can impose unfavorable pH conditions to the contracting muscle leading to early fatigue. Following the perspectives proposed by the biochemical metabolism, the group that ingested higher doses of $NaHCO_3$ presented higher pH values compared with lower doses or the placebo condition. These results are similar to those reported in the literature [9,10,22,23], although these increases are usually related to improvements in performance parameters during high-intensity exercises [9]. One of the main causes for improvement is related to the increased glycolytic capacity prompted by the higher alkalosis and therefore higher removal rate of H^+. In addition, the following other mechanisms have been implicated in causing (early) fatigue: 1) the depolarization of the resting membrane potential as a result of disturbances in muscle sodium (Na^+), potassium (K^+), and chloride (Cl^-) homeostasis [21]; 2) pH-dependent inhibition of the myofibrillar adenosine triphosphatase and other glycolytic anaerobic enzymes [24]; and 3) the unstable cross-bond formation at the troponin C (TnC) site of troponin by Ca^{2+}-mediated inhibition [24,25].

The hypothesis that H^+ ions are effectively buffered in muscle cells is discarded here because the sarcolemma is impermeable to HCO_3^- ions [26], presenting different effects when compared with β-alanine, which presents an intracellular effect [1]. However, according to Brooks [27], the lactate efflux from muscle cells is enhanced by the increase of HCO_3^- levels, which may be related to a monocarboxylate transporter activation (MCT). Similar to previous results [7,11], the HCO_3 indicated a major effect from higher doses of $NaHCO_3$. Monocarboxylate transporter activation is responsible for H^+ and lactate transport from the sarcolemma to the extracellular blood. The significant increases of HCO_3^- enhance the lactate and H^+ transporters, resulting in a greater efflux of these coproducts to the blood circulation, where they can be buffered or transported to another inactive muscle fibers [28]. The capacity to remove these high concentrations of H^+ on active muscles allows continuous contractile capacity, beyond the continuation of glycolysis process, which may in fact delay the onset of muscle fatigue during intense exercises [29]. It is worth investigating whether the positive effects of $NaHCO_3$ (improving performance associated with higher lactate levels in the blood) comes from the putative activation of the LDH reaction in liver, which maintains the Cori cycle, or the eventual preservation of muscle glycogen as a source of glucose 6-phosphate to fuel glycolysis. It has been reported that energy sources such as pyruvate could alter the stoichiometry among glycolytic flow, H^+ release, and lactate/H^+ consumption [3].

After the significant improvements in pH levels when higher doses of $NaHCO_3$ were administrated, lactate experienced a significant impact from the supplementation protocol. Similar results were found in Judo fighters by Artioli [6], with higher lactate levels identified after the ingestion of $NaHCO_3$. However, it is important to mention that the study by Artioli [6] focused on athletes, who most likely exhibited higher percentages of type II fibers and lower LDH activities in muscles compared with recreational and sedentary participants [30]. Conversely, our volunteers here were merely recreationally active cyclists. Furthermore, $NaHCO_3$ supplementation in Artioli's study was mixed with low-carbohydrate solutions, which may have induced additional effects from a lactate standpoint. The digestion of the carbohydrate solution could increase HCL levels, thereby degrading the concentration of $NaHCO_3$. Moreover, 0.3 g · kg^{-1} $NaHCO_3$ supplementation in our study delayed exhaustion in performance tests [8]. In another study [14] it was reported that alkalosis caused by the ingestion of sodium bicarbonate was significantly lower when 0.1 g · kg^{-1} of $NaHCO_3$ was used. However, there were no significant differences between 0.2 g · kg^{-1} and 0.3 g · kg^{-1} of $NaHCO_3$, so the main cause of the inefficiency of lower doses could have been related to the lower potential to improve metabolic alkalosis by increasing the pH levels in the blood.

Our study had some notable limitations. For one, we did not standardize participants' diets, which may have confounded findings. Moreover, the sample was composed solely of recreationally active cyclists, making it impossible to generalize findings to other populations. Nonetheless, other investigators should consider our model when treating athletes with $NaHCO_3$ for a better comparison about the impacts on performances. We assume the biomechanical, psychological, and physiological differences between our model and realistic road-cycling races may vary substantially during real competition. Moreover, our conclusions could be extended to high-intensity exercises and different sports. Therefore the efficiency of $NaHCO_3$ supplementation must take into account the duration and the intensity of the exercise, whereas the applicability of supplementation on short high-intensity exercises is a question that remains unanswered.

Conclusions

The present study found that 0.3 g · kg^{-1} BW $NaHCO_3$ was effective in improving performance, presenting higher lactate levels in the blood of cyclists associated with significant improvements on pH levels, whereas a lower dose (0.1 g · kg^{-1} BW $NaHCO_3$) did not provide similar beneficial effects. Future studies should consider a comparison with a dose higher than 0.3 g · kg^{-1} BW $NaHCO_3$ without reaching the 0.5 g · kg^{-1} BW $NaHCO_3$ to find a dose that provides fewer side effects while simultaneously enhancing effects on performance.

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