Effect of Acute Sodium Bicarbonate Intake on Sprint-Intermittent Performance and Blood Biochemical Responses in Well-Trained Sprinters

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Abstract
The present study was designed to determine the acute effect of sodium bicarbonate (NaHCO3) on the number of sprint repetitions during sprint high-intensity intermittent testing. In addition, blood biochemical (pH, HCO3-, and lactate) responses measured in three occasions were investigated. Thirteen male well-trained sprinters (24.65±3.44 yrs) performed two consecutive trials (7 days apart). Athletes were assigned randomly either to ingest a single dose of NaHCO3 (0.3 g/kg) 1 h prior to exercise or placebo using a double-blind crossover design. The intermittent sprint test consisted of 60 s treadmill sprints (90% of maximal work done) and 30-s recovery repeated intermittently until volitional exhaustion. Blood samples were collected from all athletes before exercise, after 1 h of dose intake, and after exercise in each trial. Paired sample t-testing showed that athletes complete significantly more sprint repetitions (p=0.036) during the intermittent sprint test with NaHCO3 (6.846±3.114) than with the placebo (5.538±3.872). Data also revealed no differences between trials in all blood responses at pre-exercise. After 1 h of dose consumption, however, blood pH and HCO3- were higher with NaHCO3 than with placebo (p<0.05), but no differences were noted in lactate between trials (p>0.05). After completion of the test, all blood responses were significantly higher with NaHCO3 than with placebo (p<0.05). In conclusion, intake of 0.3 g/kg of NaHCO3 1 h prior to treadmill sprint-intermittent performance increased sprint repetitions in well-trained sprinters, probably due to activated glycolysis caused by intracellular protons efflux into the blood.

Keywords: glycolytic enzymes, blood pH, buffering capacity, contractile force, fatigue

Introduction
High-intensity intermittent exercise results in a pronounced accumulation of glycolytic metabolites as a consequence of anaerobic energy delivery in the working muscles (da Silva et al., 2019; Danaher, Gerber, Wellard, & Stathis, 2014; Coso, Hamouti, Agudo-Jimenez, & Mora-Rodriguez, 2010; Sweeney, Wright, Brice, & Doberstein, 2010). As exercise progresses, the production of hydrogen cations (H+) increas-
es (Saunders et al., 2017) and the pH of the muscle declines (Hobson, Saunders, Ball, Harris, & Sale, 2012), which leads to acid-base imbalance. Increased acidity of the working muscles caused by H+ accumulation is a major cause of fatigue (Debold, Fitts, Sundberg, & Nosek, 2016; Fitts, 2016; Bishop, Edge, Davis, & Goodman, 2004) and can lead to performance impairments when exercise is performed at high intensities (de Salles Painelli et al., 2013; Tobias et al., 2013; Robergs, Hutchinson, Hendee, Madden, & Siegler, 2005). More specifically, muscle acidosis has been shown to impair energy transfer via the anaerobic system (da Silva et al., 2019), disrupt phosphoryl-creatine (PCr) resynthesis (Sahlin, Harris, & Hultman, 1975), and to inhibit the activity of key glycolytic enzymes, such as glycogen phosphorylase and phosphofructokinase (da Silva et al., 2019). Subsequently, the ability of the muscles to cope with high-energy demands decreases (Gladden, 2004).

Although a great portion of the contraction-induced H+ is rapidly transported out of the working myocytes to blood and buffered by bicarbonate (HCO3-) (de Salles Painelli et al., 2013; Requena, Zabala, Padial, & Feriche, 2005), blood acidity could also contribute to fatigue indirectly during high-intensity exercise (Price, Moss, & Prance, 2003; Hobson et al., 2012). In this context, nutritional supplements have been shown to attenuate acidosis and delay fatigue; sodium bicarbonate (NaHCO3) is one of them.

NaHCO3 is the most frequently alkalotic agent used by athletes who rely on glycolysis to delay fatigue (da Silva et al., 2019; Saunders et al. 2017) and reduce ratings of perceived exertion (RPE) (Carr, Slater, Gore, Dawson, & Burke, 2011). NaHCO3 can increase the extracellular buffering capacity by increasing blood HCO3- concentration (de Salles Painelli et al., 2013; Oliverra et al., 2017) in which it enhances H+ efflux from the working muscles to the blood (da Silva et al., 2019; de Salles Painelli et al., 2013) where they are neutralized (Bishop et al., 2004).

Several investigations have shown that increased circulation-buffering capacity, achieved either by acute (single dose) or chronic (supplementation) NaHCO3 intake, improves performance and capacity at high intensities (Carr et al., 2011; Requena et al., 2005; Tobias et al., 2013; Lancha Júnior, de Salles Painelli, Saunders, & Artioli, 2015). This indicates that NaHCO3 has been reported to be beneficial in events with high-intensity exercise lasting from approximately 1 to 5 minutes (Carr et al., 2011; Saunders et al., 2017; Tobias et al., 2013), with utilized dose ranging from 0.1 to 0.5 g/kg body mass (McNaughton, Backx, Palmer, & Strange, 1999). The mechanism proposed to be reasonable for the effect of NaHCO3 involves increased activation of the glycolytic and the adenosine triphosphate (ATP)-PCr systems (Deb, Gough, Sparks, & McNaughton, 2018), although elevation in blood lactate has been demonstrated following NaHCO3 intake (Artioli et al., 2007).

Studies using repeated sprint bouts of high-intensity exercise have observed performance improvement (Price et al., 2003; Bishop et al., 2004; Tobias et al., 2013; Deb et al., 2018), but others failed to report beneficial effects (de Araujo Dias et al., 2015; de Salles Painelli et al., 2013; da Silva et al., 2019). Beside of discrepancies associated with the beneficial effect of NaHCO3, high volume and intensity exercise could induce acid-base disturbances (Carr et al., 2013). Additionally, although NaHCO3 has been studied for years, most investigations have been conducted using cycling ergometer tests. However, the effect of NaHCO3 intake on blood responses during repeated sprint-intermittent testing on a treadmill remains poorly investigated, and its effect on exercise capacity has yet to be demonstrated. Therefore, this study aimed to determine the number of sprint repetitions during treadmill high-intensity intermittent exercise protocol until volitional exhaustion. A secondary aim of this study was to investigate the concentrations of blood pH, HCO3-, and lactate in response to exercise. We hypothesized that extracellular buffering capacity by ingesting acute NaHCO3 might attenuate blood acidity and improve performance.

**Methods**

**Participants**

Thirteen male well-trained sprinting athletes (see Table 1 for participants demographic data) volunteered to participate in the present study, following being informed about the potential risks and benefits involved in participation. All athletes had been involved in a sprinting training at the Jordan Military Sports Federation for a minimum of five years. Other inclusion criteria for participation were the following: long male athletes; age ranged 20-30 years old; no previous injuries for at least four months, and not consuming NaHCO3 or any ergogenic aids seven days prior to participation. This study was approved in advance by the local scientific research committee of Yarmouk University (protocol. 11-2019 M.A). Each participant voluntarily provided written informed consent before participation.

**Table 1. Participants’ demographic data**

| Variables               | Mean ± SD       |
|-------------------------|-----------------|
| Age (years)             | 24.65 ± 3.44    |
| Height (cm)             | 181.55 ± 4.74   |
| Mass (kg)               | 79.34 ± 5.22    |
| BMI (kg/m²)             | 24.30 ± 2.46    |
| resting HR (bpm)        | 63.67 ± 3.92    |
| Training volume (min/week) | 420.33 ± 48.61 |
| Training experience (years) | 6.23 ± 1.98     |
| 100-m best time (s)     | 10.43 ± 0.60    |

**Experimental design**

Athletes performed two experimental trials in which they ingested a single dose of NaHCO3 (Premium sodium bicarbonate powder, VITADIRECT, USA) or maltodextrin (placebo). The trials were randomized and separated by one week to complete recovery, with both trials performed at the same time of the day (07:45 AM) to ensure that the findings were not affected by circadian rhythm. NaHCO3 and placebo were coded before data collection. The doses were administered in a crossover design, with the double-blind provision of NaHCO3 and placebo, as neither examiners nor athletes were aware of the experimental treatment. Each trial consisted of 1) intake...
of NaHCO₃ or placebo in the laboratory one hour prior to exercise, 2) a standardized 10-min warm-up (treadmill jogging with a speed of 7-8 km/h, joint mobilization, and stretching), and 3) repeated intermittent sprint test on a treadmill. Athletes were instructed to refrain from drinking water during the trial. The exercise protocol in both trials was performed in a cool environment (20-22°C) and 42-45% relative humidity.

**Experimental procedure**

Each athlete visited the laboratory on four different occasions. Athletes' characteristics and vital factors were measured on the first visit. On the second visit, each athlete engaged in a warm-up to prepare themselves for running on a treadmill (Technogym Excite + RUN 1000-19” LED Touchscreen, Italy). They engaged in running three bouts with different speeds that ranged from low to moderate (7-13 km/h) for 10 min. On the next day (third visit), each athlete was familiarized with running on the treadmill for 15 min. After a 5 min rest, the athletes underwent a graded exercise test to determine VO₂max, in which exercise intensity is progressively increased while measuring ventilation and oxygen and carbon dioxide concentration of the inhaled and exhaled air. VO₂max is reached when oxygen consumption remains at a steady state despite an increase in workload (see Price et al., 2003). This regimen was done to measure the athletes’ greater speed (intensity) associated with VO₂max while performing in each trial for a 60 s sprint. Determination of VO₂max was to know the efficacy of cardiopulmonary status and to indicate a preparedness of athletes’ ability to perform the intermittent sprint test effectively. The results of athletes’ VO₂max and greater treadmill speed were 59.36±3.61 ml/kg/min; 17.05±1.71 km/h, respectively.

On the fourth visit, we repeated the regimen for each athlete to confirm the intensity (speed) of the exercise. Test-retest showed no difference in VO₂max (t=2.14, p=0.32) and maximal speed (t=0.65, p=0.73). Athletes then asked to perform the intermittent sprint test at a speed of 90% of their achieved maximal speed (range: 16.6-17.5 km/h) for 10 min. On the next day (third visit), each athlete was familiarized with running on the treadmill for 15 min. After a 5 min rest, the athletes underwent a graded exercise test to determine VO₂max, in which exercise intensity is progressively increased while measuring ventilation and oxygen and carbon dioxide concentration of the inhaled and exhaled air. VO₂max is reached when oxygen consumption remains at a steady state despite an increase in workload (see Price et al., 2003). This regimen was done to measure the athletes’ greater speed (intensity) associated with VO₂max while performing in each trial for a 60 s sprint. Determination of VO₂max was to know the efficacy of cardiopulmonary status and to indicate a preparedness of athletes’ ability to perform the intermittent sprint test effectively. The results of athletes’ VO₂max and greater treadmill speed were 59.36±3.61 ml/kg/min; 17.05±1.71 km/h, respectively.

All athletes were instructed to refrain from strenuous exercise in the 48 hours prior to each trial and also abstaining from drinking coffee for 12 hours. They were asked to avoid breakfast (eating) before beginning a trial to limit confounding nutritional effect on performance and to ensure NaHCO₃ absorption. Each athlete was asked to drink 500 ml of water 90 min prior to each trial to prevent possible dehydration.

**NaHCO₃ and placebo intake protocol**

Athletes were instructed to ingest 0.3 g/kg of NaHCO₃ orally 1 h prior to the experimental trial. NaHCO₃ was administered in 400 ml of chilled water (16°C) and mixed with 30 ml of strawberry flavour. The selected dose was used to avoid possible confounding factors that may impede performance. An intake acute dose of NaHCO₃ greater than 0.5 g/kg body mass can cause abdominal pain, flatulence, nausea, vomiting, and diarrhoea (Lancha Junior et al., 2015). In addition, all athletes were requested to ingest the supplement within 10 minutes to optimal absorption (Deb et al., 2018). In the placebo trial, athletes were asked to complete the same order with maltodextrin. The supplements were ingested using indistinguishable bottles so that the participants did not know which drink they had ingested.

**Sprint-intermittent test**

The intermittent sprint test consisted of treadmill repeated 60-s sprint bouts until volitional exhaustion (task failure). Rest periods were 30s between bouts. A speed of 16.6-17.5 km/h (the range of athletes’ maximal speed) was maintained in the treadmill throughout the bouts, in which the athletes were encouraged to complete as many as possible repetitions successfully. Task failure defined as the inability to maintain sprinting within 10 seconds of the preferred cadence.

**Blood samples collection and analysis**

The blood samples were collected from each participant in both trials to measure blood pH, HCO₃⁻, and lactate on three occasions: pre-exercise, post-1 h of dose intake, and post-exercise. Venipuncture was used to obtain blood samples (4 ml). Blood pH and HCO₃⁻ were analysed using an ABL800 radiometer (Denmark). Blood lactate concentration was analysed using an Integral 400 device (Switzerland). The reference ranges of variables were as follows: 0.63-2.44 mmol/L for lactate, 22.0-29.0 mmol/L for HCO₃⁻. The normal blood pH is tightly regulated between 7.35 and 7.45.

**Statistical analysis**

The Shapiro-Wilk test was applied to check for normal distribution. All variables (blood pH, HCO₃⁻, and lactate) at the three time points were normally distributed (p<0.05). A repeated measures analysis of variance (ANOVA) with a Greenhouse-Geisser correction was used to determine possible differences in blood responses at the three time points within a trial. When a significant F rate was achieved, a post hoc test using the Bonferroni correction was used for pairwise comparison using adjusted means. A paired sample t-test was used to analyse the differences in the number of sprint repetitions between trials, and to analyse the differences in each measured point between trials. Frequentist inferences were assessed against the mean difference ± 95% confidence interval CI between trials in which that variances do not cross the zero-boundary interpreted as significant. All descriptive data are reported as mean ± SD. Significance was set at P<0.05 for all analyses. Statistical analysis was conducted using SPSS version 18.0 and Microsoft Excel.

**Results**

Data revealed that the number of sprint repetitions during the intermittent sprint test were significantly greater with NaHCO₃ (6.846±3.114) than with the placebo (5.538±3.872) (t=4.113, p=0.036). Table 2 illustrates the results of blood biochemical responses to the NaHCO₃ at three time points: 1) pre-exercise, 2) 1 h after dose intake, and 3) post-exercise. The analysed data showed statistical differences in all blood responses. Post hoc using Bonferroni with adjusted means revealed that both blood pH and HCO₃⁻ were significantly higher after 1 h of NaHCO₃ intake compared to pre- (F=4.201, p=0.027; F=3.817, p=0.030 for pH and HCO₃⁻, respectively) and post-exercise (F=3.522, p=0.034; F=2.961, p=0.041 for pH and HCO₃⁻, respectively), whereas blood lactate level was elevated after the finish of the test in comparison to the pre-exercise level (F=6.012, p=0.003) and 1 h post-dose (F = 8.976, p = 0.001), with no differences between pre-exercise and 1 h post-dose (F=0.351, p=0.468).

Table 3 illustrates the results of blood biochemical responses to the placebo at the same three time points. Data showed differences in all blood responses. Post hoc using Bonferroni with
Table 2. Results of blood biochemical responses to NaHCO3 at baseline (pre-exercise), 1 h after ingestion, and at after exercise in 13 well-trained sprinters. Data were analysed using one-way ANOVA

| Parameters  | Pre-exercise | 1-h post dose | Post-exercise |
|-------------|--------------|---------------|---------------|
| pH          | 7.42 ± 0.03  | 7.47 ± 0.02   | 7.37 ± 0.05   |
| HCO3 (mmol/L) | 25.81 ± 2.44 | 30.63 ± 4.01  | 17.28 ± 4.16  |
| Lactate (mmol/L) | 1.95 ± 1.88  | 2.21 ± 0.24   | 13.11 ± 3.09  |

Note. Significance level was set at p<0.05; Values expressed as mean ± SD; a Significantly different from pre-exercise; b Significantly different than 1 h post-dose.

Table 3. Results of blood biochemical responses to placebo at baseline (pre-exercise), 1 h after ingestion, and at after exercise in 13 well-trained sprinters. Data were analysed using one-way ANOVA

| Parameters  | Pre-exercise | 1-h post dose | Post-exercise |
|-------------|--------------|---------------|---------------|
| pH          | 7.43 ± 0.02  | 7.43 ± 0.02   | 7.29 ± 0.04   |
| HCO3 (mmol/L) | 25.74 ± 2.74 | 25.59 ± 3.22  | 14.56 ± 4.38  |
| Lactate (mmol/L) | 2.07 ± 2.33  | 2.27 ± 0.44   | 10.46 ± 4.17  |

Note. Significance level was set at p<0.05; Values expressed as mean ± SD; a Significantly different than pre-exercise; b Significantly different than 1 h post-dose.

Table 4 illustrates the results of blood biochemical responses between NaHCO3 and placebo at the same three time points. There were no significant differences between trials in all blood responses at pre-exercise. After 1 h of dose consumption, blood pH and HCO3- were higher with NaHCO3 than with placebo, but no differences were noted in lactate between trials. After the finish of the test, however, all variables were significantly higher with NaHCO3 than with placebo.

Table 4. Results of blood biochemical responses to NaHCO3 and placebo at baseline (pre-exercise), 1 h after ingestion, and at after exercise in 13 well-trained sprinters. Data were analysed using paired sample t test

| Parameters  | Pre-exercise | 1-h post dose | Post-exercise |
|-------------|--------------|---------------|---------------|
| pH          | 7.42 ± 0.03  | 7.47 ± 0.02   | 7.37 ± 0.05   |
| HCO3 (mmol/L) | 25.81 ± 2.44 | 30.63 ± 4.01  | 17.28 ± 4.16  |
| Lactate (mmol/L) | 1.95 ± 1.88  | 2.21 ± 0.24   | 13.11 ± 3.09  |

Note. Significance level was set at p<0.05; Values expressed as mean ± SD; * Significantly different from placebo.

Discussion

The primary finding of the present study was that NaHCO3 ingestion was an effective strategy to complete significantly more sprint repetitions when compared to ingestion of the placebo. This result could be explained by the excessive blood HCO3- concentration due to NaHCO3 consumed before the beginning of the trial, in which it enhances intracellular H+ efflux. It has been documented that NaHCO3 administration can attenuate roughly 62% of the H+ diffused from working muscle flux. It has been documented that NaHCO3 administration can enhance muscle performance (4-30-s Wingate test, separated by 3 min) following chronic beta-alanine (BA) supplementation (6.4 g/day for 4 weeks) combined with 500 mg/kg of NaHCO3 ingested within seven days compared to a placebo in well-trained athletes. The total work done in that study was increased by 8% in the mode
of NaHCO₃ supplementation, which differed from our protocol. However, it has been suggested that chronic NaHCO₃ can elicit muscular capacity similarly with an acute intake (Artioli et al., 2007).

Our findings were contrary to further studies. Recently, da Silva et al. (2019) showed that a combination of BA supplementation (6.4 g/day for 28 days) and acute NaHCO₃ (0.3 g/kg) 60 min prior to cycling time-trial (60-s bouts at 110% of maximal power output, separated by 60-s rest) did not improve performance compared to each one alone and placebo in male cyclists. However, they suggested that NaHCO₃ increased the estimated glycolytic ATP-PCr systems. de Salles Painelli et al. (2013) reported that 0.3 g/kg of NaHCO₃ intake following BA supplementation (3.2-6.4 g/day for 4 weeks) had no ergogenic effect in 100- and 200-m swimming performance in swimmers. A limitation of that study was the lack of blood HCO₃⁻ measurement, so they failed to suggest an explanation for non-significant performance improvement. de Araujo Dias et al. (2015) observed that the ingestion of 0.3 g/kg of NaHCO₃ did not affect graded high-intensity cycling capacity test which initiated at 100 W and increased by 6 W every 15 s until volitional exhaustion compared to placebo. The explanation of their finding was attributed to a variability of monocarboxylate (MCT) transporter protein activity after NaHCO₃ intake and to H⁺ efflux ratio from myocytes into blood. However, participants recruited in that study were recreationally active individuals. In addition, conflicting findings have been observed when the duration of an exercise lasting less than 2 min (Requena et al., 2005). Danaher et al. (2014) showed no differences in repeated sprint ability (RSA) test (5 repeats of 6 s maximal effort cycling bouts, separated by 24 s rest) between NaHCO₃ (300 mg/kg), BA (4.8-6.4 g/day for 4 weeks) and placebo trials. However, time-to-exhaustion during cycling capacity test performed following RSA was increased 16% with a combination of NaHCO₃+BA. The lack of ergogenic effect of NaHCO₃ intake upon performance in previous studies might be associated with the type of exercise protocol, the duration of exercise, the small number of the sample, the intensity of exercise, and the environmental temperatures.

Our results showed that blood pH and HCO₃⁻ were significantly higher after the finish of the test in the NaHCO₃ trial than that of the placebo trial, which might indicate the effective buffering capacity of the extracellular median due to NaHCO₃ intake. Increased extracellular pH and raised HCO₃⁻ due to NaHCO₃ consume might raise the H⁺ and lactate efflux from working muscles (Requena et al., 2005) by increasing the activity of the lactate/-H⁺ cotransporter. This mechanism delays the drop in pH (Marx et al., 2002), delays the onset of fatigue (Hobson, et al., 2013), and leads to higher contractile force (McNaughton et al., 1999) by sustained muscle glycolytic ATP production (McKenzie, Couts, Stirling, Hoeben, & Kubara, 1986; Kemp & Foe, 1983). In this context, however, increased activation of glycolytic ATP-PCr systems induce high lactate levels (da Silva et al., 2019). Increased post-exercise lactate has been reported after NaHCO₃ intake (da Silva et al., 2019; Bishop et al., 2004), which might explain the elevated blood lactate after the finish of the test in the NaHCO₃ trial compared to the placebo in the present study. Additionally, the greater sprints repetitions as a result of NaHCO₃ consume was also contributed to elevation in blood lactate levels. In a study conducted by Deb et al. (2018), blood HCO₃⁻ was significantly higher in experimental trial after intermittent testing (16.0±2.0 mmol/L) compared to 13.0±3.0 mmol/L in a placebo trial, and blood pH was decreased from 7.47 (pre-test) to 7.31 (post-test) in NaHCO₃ trial compared to placebo (7.39; 7.20, respectively). In the same study, however, post-test blood lactate was significantly elevated in NaHCO₃ trial (17.9±5.9 mmol/L) compared to a placebo (13.9±4.3 mmol/L). da Silva et al. (2019) found that blood lactate (15.7 mmol/L), pH (7.30), and HCO₃⁻ (22.1 mmol/L) were significantly changed after exercise compared to placebo (12.0 mmol/L; 7.25; 19.5 mmol/L, respectively). Hobson et al. (2013) showed that chronic BA (6.4 g/day for 4 weeks) followed by acute NaHCO₃ (0.2 g/kg) were likely to be beneficial to 2,000-m rowing performance, with increased post-exercise blood pH, HCO₃⁻ and lactate compared to placebo group in rowers.

Conclusion

It can be concluded from the present data that acute (single dose) sodium bicarbonate can attenuate acidosis during high-intensity intermittent exercise and improve performance following intake 0.3 g/kg orally in well-trained sprinters, which indicates that sodium bicarbonate may act as a physicochemical buffer in the body and may represent, in part, an explanation for the ergogenic effect in sprint-intermittent exercise. Additionally, these data confirm that post-exercise blood lactate increases after the consumption of sodium bicarbonate.

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References

Artioli, G.G., Gualano, B., Coelho, D.F., Benatti, F.B., Gailey, A.W., & Lancha Jr, A.H. (2007). Does sodium-bicarbonate ingestion improve simulated judo performance? International Journal of Sport Nutrition and Exercise Metabolism, 17, 206-217. doi: 10.1123/ijsnem.17.2.206

Bishop, D., Edge, J., Davis, C., & Goodman, C. (2004). Induced metabolic alkalosis affects muscle metabolism and repeated sprint ability. Medicine & Science in Sports & Exercise, 36, 807-813. doi: 10.1249/01.MSS.0000126392.20025.17

Carr, A.J., Slater, G.J., Gore, C.J., Dawson, B., & Burke, L.M. (2011). Effect of sodium bicarbonate on [HCO₃⁻], pH, and gastrointestinal symptoms. International Journal of Nutrition and Exercise Metabolism, 21, 189-194. doi: 10.1123/ijsnem.21.3.189

Carr, B.M., Webstar, M.J., Boyed, J.C., Hudson, G.M., & Scheet, T.P. (2013). Sodium bicarbonate supplementation improves hypertrophy-type resistance exercise performance. European Journal of Applied Physiology, 113, 743-752. doi: 10.1007/s00421-012-2484-8

Coso, J.D., Hamouti, N., Agudo-Jimenez, R., & Mora-Rodriguez, R. (2010). Restoration of blood pH between repeated bouts of high-intensity exercise: effects of various active-recovery protocols. European Journal of Applied Physiology, 108, 523-532. doi: 10.1007/s00421-009-1248-6

da Silva, R.P., de Oliveira, L.F., Saunders, B., de Andrade Kratz, C., de Salles Painelli, V., da Eira Silva, V., Marinis, J.C.B., Franchini, E., Gualano, B., & Artioli, G.G. (2019). Effects of β-alanine and sodium bicarbonate supplementation on metabolism and repeated bouts of high-intensity exercise.
the estimated energy system contribution during high-intensity intermittent exercise. *Amino Acids*, 51, 83-96. doi: 10.1007/s00212-014-2895-9

Danaher, J., Gerber, T., Wellard, B.M., & Stathis, C.G. (2014). The effect of β-alanine and NaHCO₃ co-ingestion on buffering capacity and exercise performance with high-intensity exercise in healthy males. *European Journal of Applied Physiology*, 114, 1715-1724. doi: 10.1007/s00421-014-2895-9

de Araujo Dias, G.F., da Eira Silva, V., de Salles Painelli, V., Sale, C., Artioli, G.G., Gualano, B., & Saunders, B. (2015). (In) Consistencies in responses to sodium bicarbonate supplementation: a randomised, repeated measures, counterbalanced and double-blind study. *PLoS ONE*, 10(11), 1-13. doi: 10.1371/journal.pone.0143086

del Salles Painelli, V., Roschel, H., de Jesus, F., Sale, C., Harris, R.C., Solis, M.Y., Benatti, E.B., Gualano, B., Lancha Jr, A.H., & Artioli, G.G. (2013). The ergogenic effect of beta-alanine combined with sodium bicarbonate on high-intensity swimming performance. *Applied Physiology Nutrition and Metabolism*, 38, 525-532. doi: 10.1139/apnm-2012-0286

Deb, S.K., Gough, L.A., Sparks, S.A., & McNaughton, L.R. (2018). Sodium bicarbonate supplementation improves severe-intensity intermittent exercise under moderate acute hypoxia conditions. *European Journal of Applied Physiology*, 118, 607-615. doi: 10.1007/s00421-018-3801-7

Debold, E.P., Fitts, R.H., Sundberg, C.W., & Nosek, T.M. (2016). Muscle fatigue from the perspective of a single crossbridge. *Medicine & Science in Sports & Exercise*, 11, 2270-2280. doi: 10.1249/MSS.0000000000001047

Fitts, R.H. (2016). The role of acidosis in fatigue: a prospective. *Medicine & Science in Sports & Exercise*, 11, 2335-2338. doi: 10.1249/MSS.0000000000001043

Gladden, L.B. (2004). Lactate metabolism: a new paradigm for the third millennium. *The Journal of Physiology*, 558, 5-30. doi: 10.1113/jphysiol.2003.058701

Hobson, R.M., Harris, R.C., Martin, D., Smith, P., Macklin, B., Gualano, B., & Sale, C. (2013). Effect of β-alanine with and without sodium bicarbonate on 2.000-m rowing performance. *International Journal of Sport Nutrition and Exercise Metabolism*, 23, 480-487. doi: 10.1123/ijssnm.23.5.480

Hobson, R.M., Saunders, B., Ball, G., Harris, R.C., & Sale, C. (2012). Effects of β-alanine supplementation on exercise performance: a meta-analysis. *Amino Acids*, 43, 25-37. doi: 10.1007/s00726-011-1200-z

Kemp, R.J., & Foe, L.G. (1983). Allosteric regulatory properties of muscle phosphofructokinase. *Molecular and Cellular Biochemistry*, 57, 147-154. doi: 10.1007/BF00849191

Lancha Junior, A.H., de Salles Painelli, V., Saunders, B., & Artioli, G.G. (2015). Nutritional strategies to modulate intracellular and extracellular buffering capacity during high-intensity exercise. *Sports Medicine*, 45(Suppl 1), S71-S81. doi: 10.1007/s40279-015-0397-5

Marx, J.O., Gordon, S.E., Vos, N.K., Nindl, B.C., Gomez, A.L., Volek, J.S., Pedro, J., Ratamess, N., Newton, R.U., French, D.N., Rubin, M.R., Hakkinen, K., & Kraemer, W.J. (2002). Effect of alkalosis on plasma epinephrine responses to high intensity cycle exercise in humans. *European Journal of Applied Physiology*, 87, 72-77. doi: 10.1007/s00421-002-0591-7

McKenzie, D.C., Couts, K.D., Stirling, D.R., Hoeben, H.H., & Kubara, G. (1986). Maximal work production following two levels of artificially induced metabolic alkalosis. *Journal of Sports Sciences*, 4, 35-38. doi: 10.1080/02640418608732096

McNaughton, L., Backx, K., Palmer, G., & Strange, N. (1999). Effects of chronic bicarbonate ingestion on the performance of high-intensity work. *European Journal of Applied Physiology*, 80, 333-336. doi: 10.1007/s004210050600

Medbo, J.I., & Tabata, I. (1993). Anaerobic energy release in working muscle during 30 s to 3 min of exhausting bicycling. *Journal of Applied Physiology*, 75(4), 1654-1660. doi: 10.1152/jappl.1993.75.4.1654

Oliveria, L.F., de Salles Painelli, V., Nemezio, K., Goncalves, I.S., Yamaguchi, G., Saunders, B., Gualano, B., & Artioli, G.G. (2017). Chronic lactate supplementation does not improve blood buffering capacity and repeated high-intensity exercise. *Scandinavian Journal of Medicine and Science in Sports*, 27, 1231-1239. doi: 10.1111/smss.12792

Price, M., Moss, P., & Prance, S. (2003). Effects of sodium bicarbonate ingestion on prolonged intermittent exercise. *Medicine & Science in Sports & Exercise*, 35(8), 1303-1308. doi: 10.1249/01.MSS.0000079067.46555.3C

Requena, B., Zabala, M., Padial, P., & Feriche, B. (2005). Sodium bicarbonate and sodium citrate: ergogenic aids?. *The Journal of Strength and Conditioning Research*, 19(1), 213-224.

Robergs, R., Hutchkinson, K., Hendee, S., Madden, S., & Siegler, J. (2005). Influence of pre-exercise acidosis and alkalosis on the kinetics of acid-base recovery following intense exercise. *International Journal of Sport Nutrition and Exercise Metabolism*, 15, 59-74. doi: 10.1123/ijssnm.15.1.59

Sahlin, K., Harris, R.C., & Hultman, E. (1975). Creatine kinase equilibrium and lactate content compared with muscle pH in tissue samples obtained after isometric exercise. *Biochemical Journal*, 152, 173-180. doi: 10.1042/bj1520173

Saunders, B., Elliott-Sale, K., Artioli, G.G., Swinton, P.A., Dolan, E., Roschel, H., Sale, C., & Gualano, B. (2017). β-Alanine supplementation to improve exercise capacity and performance: a systemic review and meta-analysis. *British Journal of Sports Medicine*, 51, 658-669. doi: 10.1136/bjsports-2016-096396

Sweeney, K.M., Wright, G.A., Brice, A.G., & Doberstein, S.T. (2010). The effect of β-alanine supplementation on power performance during repeated sprint activity. *Journal of Strength and Conditioning Research*, 24, 79-87. doi: 10.1519/JSC.0b013e3181c63bd5

Tobias, G., Benatti, F.B., de Salles Painelli, V., Roschel, H., Gualano, B., Sale, C., Harris, R.C., Lancha Jr, A.H., & Artioli, G.G. (2013). Additive effects of beta-alanine and sodium bicarbonate on upper-body intermittent performance. *Amino Acids*, 45, 309-317. doi: 87.236.233.3