Effect of Beta-Alanine With and Without Sodium Bicarbonate on 2,000-m Rowing Performance

Ruth M. Hobson, Roger C. Harris, Dan Martin, Perry Smith, Ben Macklin, Bruno Gualano, and Craig Sale

Purpose: To examine the effect of beta-alanine only and beta-alanine with sodium bicarbonate supplementation on 2,000-m rowing performance. Methods: Twenty well-trained rowers (age 23 ± 4 y; height 1.85 ± 0.08 m; body mass 82.5 ± 8.9 kg) were assigned to either a placebo or beta-alanine (6.4 g·d⁻¹ for 4 weeks) group. A 2,000-m rowing time trial (TT) was performed before supplementation (Baseline) and after 28 and 30 days of supplementation. The post supplementation trials involved supplementation with either maltodextrin or sodium bicarbonate in a double-blind, crossover design, creating four study conditions (placebo with maltodextrin; placebo with sodium bicarbonate; beta-alanine with maltodextrin; beta-alanine with sodium bicarbonate). Blood lactate, pH, bicarbonate, and base excess were measured pre-TT, immediately post-TT and at TT+5 min. Performance data were analyzed using magnitude based inferences. Results: Beta-alanine supplementation was very likely to be beneficial to 2,000-m rowing performance (6.4 ± 8.1 s effect compared with placebo), with the effect of sodium bicarbonate having a likely benefit (3.2 ± 8.8 s). There was a small (1.1 ± 5.6 s) but possibly beneficial additional effect when combining chronic beta-alanine supplementation with acute sodium bicarbonate supplementation compared with chronic beta-alanine supplementation alone. Sodium bicarbonate ingestion led to increases in plasma pH, base excess, bicarbonate, and lactate concentrations. Conclusions: Both chronic beta-alanine and acute sodium bicarbonate supplementation alone had positive effects on 2,000-m rowing performance. The addition of acute sodium bicarbonate to chronic beta-alanine supplementation may further enhance rowing performance.

Keywords: beta-alanine, sodium bicarbonate, performance, rowing

Rowing races are performed over 2,000-m, requiring the individual to sustain high intensity activity over a 6–7 min period (for trained individuals). Stellingwerff et al. (2011) estimated that around 12% of energy comes from nonoxidative glycolysis during a 2,000-m rowing race meaning that there is a significant energy contribution from anaerobic glycolysis, resulting in the formation of two carboxylic acid groups from the oxidation of neutral hydroxyl groups on carbons 3 and 4 of each glucose or glycosyl unit metabolized. Anaerobic glycolysis is responsible for most of the hydrogen cation (H⁺) production (initially occurring in the Emden-Meyerhoff pathway with the ionization of the carboxyl group of 1,3-diphosphoglycerate) in skeletal muscle during high-intensity exercise, which can subsequently contribute to fatigue by impairing muscle function and force generation, resulting in a progressive deterioration in performance (Hultman et al., 1985). Therefore, reducing the negative impact of intracellular H⁺ accumulation is likely to attenuate reductions in performance in activities where reductions in pH are likely to be involved in the development of fatigue.

Carnosine (β-alanyl-L-histidine) is a cytoplasmic dipeptide and has an imidazole side chain with a pKa of 6.83. As a result, carnosine contributes to physicochemical buffering in the skeletal muscle over the exercise-induced pH transit range (pH 7.0 down to 6.0). In addition, carnosine is found in high concentrations within the skeletal muscle (Harris et al., 2006; Hill et al., 2007) and levels can be further increased with chronic beta-alanine supplementation (Harris et al., 2006; Hill et al., 2007). Synthesis of carnosine occurs in situ in muscle from the combination of beta-alanine with L-histidine.

Beta-alanine is the limiting factor to carnosine synthesis, and research has consistently shown that supplementation for 4 weeks or longer with beta-alanine increases muscle carnosine concentration (for reviews see Artioli et al., 2010; Derave et al., 2010; Sale et al., 2010). Several studies, though not all, have demonstrated that supplementation with beta-alanine improves exercise capacity and performance. A recent meta-analysis by Hobson et al. (2012) suggested that beta-alanine supplementation could improve the outcome of exercise tests by 2.85% (0.37–10.49%) overall. They also showed that...
there are significant improvements in exercise capacity tests ($p = .013$) but not for exercise performance tests ($p = .204$), although it was noted that there are fewer studies available using performance measures ($n = 12$) compared with capacity measures ($n = 27$). Thus, the efficacy of beta-alanine supplementation using appropriate exercise performance tests remains to be fully investigated.

Although intramuscular buffering is the first line of defense against $H^+$, and the only one when muscle blood flow is occluded (Sale et al., 2012), protons produced when muscles are working dynamically are transported out of the muscle cell allowing extracellular buffers to assist in acid-base regulation. As a result, circulating buffers, such as bicarbonate, also contribute to attenuating the accumulation of $H^+$ during dynamic exercise, preventing the development of metabolic acidosis. Supplementation with sodium bicarbonate has been shown to increase the circulating bicarbonate pool, increase blood pH and in some cases delay fatigue during high-intensity exercise (for a review, see McNaughton et al., 2008).

The majority of evidence suggests that both beta-alanine and sodium bicarbonate supplementation can improve exercise performance and capacity by attenuating the decline in pH in intracellular and extracellular compartments, although we cannot rule out the possibility that beta-alanine improves performance due to increased calcium sensitivity (Dutka et al., 2012). When beta-alanine supplementation was applied to a specific rowing test, Baguet et al. (2010) showed that those supplemented with beta-alanine completed a 2,000-m rowing ergometer time trial 2.7 ± 4.8 s faster than presupplementation, while those taking a placebo completed the same test 1.8 ± 6.8 s slower than presupplementation, although this failed to reach statistical significance ($p = .07$). Gains in performance were significantly correlated with the increase in muscle carnosine. Recently, Ducker et al. (2013) have also shown a trend for an ergogenic effect of beta-alanine supplementation on 2,000-m rowing performance, with those taking beta-alanine for 28 days going 2.9 ± 4.1 s faster and those taking a placebo going 1.2 ± 2.9 s slower ($p = .055$). In addition, McNaughton and Cedaro (1991) showed that participants covered a greater distance in a 6 min rowing ergometer test when supplemented with sodium bicarbonate compared with a placebo. Considering that the mechanisms by which beta-alanine and sodium bicarbonate prevent acidosis and improve performance are distinct, although related (McNaughton et al., 2008; Sale et al., 2010), one may speculate that the combination of these supplements would lead to further improvements in rowing performance.

The co-ingestion of beta-alanine and sodium bicarbonate has only recently been studied, with Sale et al. (2011) first showing that high-intensity cycling capacity lasting ~2–3 min was improved following 4 weeks beta-alanine supplementation. There was no significant additive effect when ingesting sodium bicarbonate, although further analysis using magnitude based inferences suggested a 70% chance that the further increase observed in capacity was a result of the addition of sodium bicarbonate. Subsequently, Bellinger et al. (2012) have shown that there was no additive effect of cosupplementation on 4-min cycling performance. Therefore, the effects of beta-alanine and sodium bicarbonate, administered in combination, on both cycling and rowing exercise performance remain unclear. As such, the current study investigated the effects of co-ingestion of beta-alanine and sodium bicarbonate on 2,000-m rowing ergometer performance. We hypothesized that there would be an additive ergogenic effect of cosupplementation on rowing performance.

Methods

Participants

Twenty trained, competitive, nonvegetarian, club-level rowers volunteered for the study and were randomly allocated, after stratification with respect to their 2,000-m ergometer rowing personal best times, into a beta-alanine and a placebo group (Table 1). Athletes were fully informed of any potential risks and discomforts associated with the study before completing a health screen and providing informed consent, after which their height and body mass were recorded. Athletes had not taken any supplements for 3 months and had not taken beta-alanine for at least 6 months before testing to allow for the long washout period of muscle carnosine (Stellingwerff et al., 2012). We did not class sports drinks or multivitamins as supplements. Athletes were asked to maintain their normal training regimen throughout the study period and to replicate their diet and activity patterns in the 36 hr before each trial; compliance with this was verbally confirmed before each trial. The study was approved by the Nottingham Trent University ethical advisory committee.

Experimental Design

All rowers attended three testing sessions throughout the study; the athletes were already familiar with the test, having performed it numerous times as part of training and competition. A Baseline 2,000-m rowing ergometer time trial (TT) was completed before a 30 day double-blind supplementation period with either beta-alanine (BA; 6.4 g·d⁻¹; Carnosyn sustained-release tablets, NAI, USA) or a placebo (PLA; maltodextrin; NAI, USA). All athletes underwent two postsupplementation trials: one on Day 28 and one on Day 30.

Table 1 Participant Characteristics ($M \pm 1SD$)

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<tr>
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<th>BA ($n = 10$)</th>
<th>PLA ($n = 10$)</th>
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<tr>
<td>Age (y)</td>
<td>24 ± 3</td>
<td>23 ± 4</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.85 ± 0.07</td>
<td>1.85 ± 0.09</td>
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<tr>
<td>Weight (kg)</td>
<td>80.5 ± 8.8</td>
<td>84.5 ± 9.0</td>
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<tr>
<td>2,000-m best time (s)</td>
<td>407.5 ± 11.7</td>
<td>409.5 ± 19.5</td>
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Note. There were no significant differences between the groups.
The double-blinded supplementation regimen consisted of the consumption of two 800mg tablets, four times a day separated by 3–4 hr and athletes were asked to keep a supplementation log to record compliance (96% in the BA group and 94% in the PLA group). Supplementation with this dose of beta-alanine over 4 weeks has been shown to increase muscle carnosine concentrations by ~60% (Harris et al., 2006; Stellingwerff et al., 2012). Beta-alanine tablets were tested by the manufacturer before release for the study and conformed to the label claim for beta-alanine content. No participants reported experiencing symptoms of paraesthesia during supplementation with either beta-alanine or placebo.

In the Baseline trial, all athletes ingested maltodextrin, as a placebo, before the TT (MD; SIS, Blackburn, UK). For the trial on day 28 and day 30 they ingested either MD, as a placebo, or sodium bicarbonate (SB; SIS, Blackburn, UK) in a randomized, crossover design (Figure 1). In total, participants ingested 0.3 g·kg⁻¹ BM sodium bicarbonate in gelatin capsules, or the same number of capsules containing maltodextrin. Compliance with the consumption of these was checked verbally with participants before the TT (with 100% compliance being reported), and was also confirmed with the pre-TT blood sample. This dose is based upon supplementation strategies employed in other studies (Sale et al., 2011; McNaughton, 1991). The dose was divided so that participants ingested 0.2 g·kg⁻¹ BM four hours before the TT and 0.1 g·kg⁻¹BM two hours before the TT to minimize gastrointestinal discomfort; participants were asked to consume at least 500ml of fluids with each dose in an attempt to further reduce this. Of the participants, 19 reported no gastrointestinal discomfort after sodium bicarbonate ingestion, with only one participant in the BA group reporting severe symptoms (including stomach cramps and diarrhea). The data for this participant are included in the analysis. Supplements used in this study were batch tested by HFL Sports Science, Fordham, Suffolk, UK, before use to ensure no contamination with steroids or stimulants according to ISO 17025 accredited tests.

Participants in either group completed trials after 28 and 30 days of supplementation, with the result that four experimental conditions were used; placebo with maltodextrin (PLA.MD), placebo with sodium bicarbonate (BA.MD), b-alanine with maltodextrin (BA.MD), and b-alanine with sodium bicarbonate (BA.SB; Figure 1).

**Experimental Procedures**

The SB or MD supplements were delivered to the athletes in the 2 days before each TT. Participants were contacted on the day of testing to remind them to consume the supplement at the required times. Unfortunately, due to the availability of the athletes recruited to this study it was not possible to gain a presupplement blood sample before each TT.

Upon arrival at the testing venue, participants had a preexercise (Pre-TT) finger prick blood sample taken and then completed a warm-up of self-selected duration and intensity, which was then replicated in subsequent tests. Participants then manually set the drag-factor using the display screen on the rowing ergometer (Concept 2, Nottingham, UK), which was standardized for all subsequent tests. Participants used the same ergometer for each of their tests. Once ready, the TT started, with the participant blinded to all performance feedback other than the distance remaining. We did not allow the participants to see their stroke rate as this could be used as an aid to pacing the time trial. Time to complete the TT was recorded as the performance outcome measure. Immediately upon completion of the TT another finger prick blood sample was taken (Post-TT), and a final sample was taken 5 min after the completion of the test (TT+5).

Finger prick blood samples were taken for the assessment of blood lactate (Lactate Pro, Arkray, Japan) pH, hemoglobin (Hb) and blood gases (Radiometer ABL 400, UK). Subsequently, bicarbonate was calculated from PCO₂ and pH values according to the Henderson-Hasselbalch equation, and base excess was calculated according to \((1 − 0.014[Hb]) × ([HCO₃⁻] – 24 + (1.43[Hb] + 7.7) × (pH –7.4)).\)

**Statistical Analyses**

All data were checked for normality of distribution using the Shapiro-Wilk test. Unpaired *t* tests were used to compare the groups at baseline. TT performance data were analyzed using a contemporary magnitude-based

![Figure 1](image_url) — Study design. All participants undertook a baseline trial, after which they were assigned to either the PLA or BA group for 28 days of chronic supplementation. They then completed two more trials in a randomized order, involving acute supplementation with either SB or MD. *Denotes counterbalanced order of trials.
inferences approach (Hopkins, 2007) to detect small effects of practical importance in an applied setting. This establishes the likelihood (in percentage terms) of each experimental manipulation having a positive/trivial/negative effect. A Cohen’s unit of 0.2 was employed as the smallest meaningful change in performance. Where the chance of benefit or harm were both >5%, the true effect was deemed unclear. Qualitative descriptors were assigned to the quantitative percentile scores as follows: 25–75% possible; 75–95% likely; 95–99% very likely; >99% almost certain (Batterham & Hopkins, 2005; Hopkins, 2002). We have chosen a priori not to present P values for the performance data, as these do not allow the reader to interpret the magnitude of any effect (Cummings & Koepsell, 2010; Hackshaw & Kirkwood, 2011; Sterne & Smith, 2001). The effect of placebo was taken as the difference between Baseline and PLA.MD. The effect of sodium bicarbonate only was taken as the difference between PLA.MD and PLA.SB, since this would be the more likely timeframe (i.e., 2 days between trials, rather than the 4 weeks between Baseline and PLA.SB) used to assess the acute effects of sodium bicarbonate on 2,000-m rowing performance. The effect of beta-alanine was taken as the difference between Baseline and BA.MD against the difference between Baseline and PLA.MD, with the additional effect of acute sodium bicarbonate ingestion to chronic beta-alanine supplementation being taken as the difference between BA.MD and BA.SB.

Haematological data, which are secondary data but necessary to confirm a potential mechanism of action of the supplements, were analyzed using a conventional three factor ANOVA (Group x Trial x Time; SPSS v19, Chicago, USA) and these data are reported as mean ± 1SD unless otherwise stated. The alpha level of significance was set at p < .05.

**Results**

**TT Performance**

The mean performance data (± 1 SD) are shown in Table 2. Baseline TT performance was similar in the two groups (PLA 411.9 ± 17.5s; BA 412.1 ± 13.2s; mean ± 1 SD). The effect of beta-alanine on 2,000-m rowing performance was very likely to have been positive (96% chance of a positive effect) compared with the effect of placebo (a difference of 6.4 ± 8.1 s; 1.5 ± 1.9% between the response to beta-alanine and the response to placebo; Figure 2). Sodium bicarbonate alone was also of likely benefit (87% chance of a positive effect; 3.2 ± 8.8 s; 0.8 ± 0.9%; Figure 2), with the addition of acute sodium bicarbonate supplementation to chronic beta-alanine

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<th>Baseline</th>
<th>MD</th>
<th>SB</th>
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<tr>
<td>PLA</td>
<td>411.9 ± 17.5</td>
<td>416.4 ± 20.4</td>
<td>413.2 ± 18.7</td>
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<tr>
<td>BA</td>
<td>412.1 ± 13.2</td>
<td>410.3 ± 9.4</td>
<td>409.2 ± 10.7</td>
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*Figure 2* — Mean ± 1SD percentage change scores in TT performance from Baseline for the MD and SB trials.
supplementation having a small (1.1 ± 5.5 s; 0.3 ± 0.48%; Figure 2) additional effect which was possibly beneficial (64% chance of an additional positive effect). However, the relatively large standard deviations shown for these differences suggest something of an individual response to supplementation. Individual performance changes within each group were as follows: two subjects improved and eight were worse with placebo, six improved and four were worse with beta-alanine, seven improved and two were worse with sodium bicarbonate and seven improved and three were worse with combined beta-alanine and sodium bicarbonate.

Blood Analyses

Blood pH \((p = .458)\), bicarbonate \((p = .391)\), bases excess \((p = .393)\) and lactate \((p = .227)\) were not significantly different between PLA and BA groups at any time point in all trials (Table 3). In PLA.MD and BA.MD, blood pH, bicarbonate, base excess and lactate were not significantly different from Baseline trial values (Table 3). In the PLA. SB and BA.SB trials, there were significant differences \((p \leq .01)\) in all hematological parameters compared with the Baseline trials as well as compared with PLA.MD and BA.MD.

On all trials, bicarbonate and base excess were significantly reduced from pre-TT to post-TT \((p \leq .001)\), and were further reduced by TT+5 \((p \leq .004)\; \text{Table 2}\). There was a significant Time × Trial interaction for both bicarbonate \((p = .012)\) and base excess \((p = .028)\), showing that for PLA.SB and BA.SB, values were higher than at the same time point in PLA.MD or BA.MD. Lactate concentrations increased \((p < .001)\) and pH values decreased \((p < .001)\) as a result of the exercise, but there was no difference for either lactate or pH from post-TT to TT+5 (Table 3). Blood lactate concentrations were also significantly higher (Time × Trial interaction; \(p = .041)\) on PLA.SB and BA.SB than on PLA.MD and BA.MD.

Discussion

This is the first study to examine the combined effects of chronic beta-alanine supplementation over 4 weeks with the acute supplementation of sodium bicarbonate on 2,000-m rowing performance. We hypothesized that beta-alanine and sodium bicarbonate supplementation in isolation would result in faster 2,000-m rowing times compared with placebo but that the effect of combined supplementation would be greater than the effect of each supplement taken individually. It is very likely that the effect of beta-alanine on 2,000-m rowing performance was beneficial and there was a small but possibly beneficial effect when acute sodium bicarbonate ingestion

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<th>PLA</th>
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<tr>
<td>pH</td>
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<tr>
<td>Baseline</td>
<td>7.417 ± 0.017</td>
</tr>
<tr>
<td>MD</td>
<td>7.420 ± 0.022</td>
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<tr>
<td>SB</td>
<td>7.451 ± 0.031</td>
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<tr>
<td>Lactate (mmol·L⁻¹)</td>
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<tr>
<td>Baseline</td>
<td>1.7 ± 3.0</td>
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<tr>
<td>MD</td>
<td>1.6 ± 0.4</td>
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<tr>
<td>SB</td>
<td>1.8 ± 0.9</td>
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<tr>
<td>Bicarbonate (mmol·L⁻¹)</td>
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<tr>
<td>Baseline</td>
<td>24.47 ± 1.59</td>
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<td>MD</td>
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<td>SB</td>
<td>30.42 ± 2.51</td>
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<td>Base excess (mmol·L⁻¹)</td>
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<td>Baseline</td>
<td>0.84 ± 1.21</td>
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<tr>
<td>MD</td>
<td>0.83 ± 1.38</td>
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<tr>
<td>SB</td>
<td>6.20 ± 2.08</td>
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*Denotes a significant effect of exercise within a trial.

Denotes a significant effect of time post-TT to TT+5.

Denotes a significant difference to the equivalent MD trial.
was added to chronic beta-alanine supplementation. This suggests a slightly greater benefit of cosupplementation over beta-alanine supplementation alone.

There are now three studies investigating the effects of beta-alanine supplementation on 2,000-m rowing performance. Although the results of Baguet et al. (2010) and Ducker et al. (2013) are statistically nonsignificant, they both show a strong trend for a positive effect of beta-alanine, with 4.5-s and 4.1-s improvements over the placebo groups respectively, while our data show a 6.4-s improvement in the beta-alanine group over the placebo group. It is possible that differences in the overall conclusions of these studies are due to differences in methods of analysis, rather than the size of the effect. For instance, in Ducker et al. (2013), the authors report \( p = .055 \) in a comparison of rowing times between the beta-alanine and placebo groups using a split-plot ANOVA, whereas the difference is \( p = .034 \) when the two means are compared using Student’s \( t \) test for unpaired means. Taken together, these results suggest a worthwhile effect of beta-alanine supplementation on 2,000-m rowing performance and, as such, competitive rowers might wish to consider the addition of beta-alanine to their training and competition nutritional strategies.

There was also a likely effect of sodium bicarbonate supplementation alone on 2,000-m rowing performance, which is supported by the biochemical data confirming that sodium bicarbonate ingestion increased pH, base excess, bicarbonate and lactate concentrations on BA.SB and PLA.SB compared with BA.MD and PLA.MD. Our results are in line with the results of McNaughton and Cedaro (1991) who showed a significant effect of sodium bicarbonate on a 6 min rowing ergometer test (with 10 days between tests), but not with those of Carr et al. (2012) who showed no benefit on 2,000-m rowing (separated by 2 days). However, even despite this, it should be noted that the effect of sodium bicarbonate supplementation alone was less than the effect of beta-alanine supplementation alone. Therefore, where well tolerated, our data suggest that rowers might wish to consider additional acute supplementation with sodium bicarbonate, on top of supplementation with beta-alanine.

To our knowledge, there have been two other studies that have examined the effect of combining beta-alanine and sodium bicarbonate on exercise capacity and performance. Sale et al. (2011) has conducted the only study to-date on exercise capacity, reporting a significant effect of beta-alanine on high-intensity cycling capacity and a 70% likelihood of a worthwhile effect from cosupplementation with sodium bicarbonate. Similarly, Bellinger et al. (2012) examined the effects of beta-alanine plus sodium bicarbonate on exercise performance tests. Bellinger et al. (2012) showed a significant effect of sodium bicarbonate on a 4 min cycling time trial but only a small and nonsignificant effect of beta-alanine and beta-alanine plus sodium bicarbonate. Thus, the results of the current study and those of Bellinger et al. (2012) are not in agreement with regards to an additive effect of supplementation with beta-alanine plus sodium bicarbonate. In addition, Bellinger et al. (2012) reported a greater effect on 4 min cycling performance from supplementation with sodium bicarbonate, whereas the greater effect on 2,000-m rowing performance came from beta-alanine and the additive effect of cosupplementation in the present investigation. The results of Bellinger et al. (2012) showed that the effect of increased indirect buffering (via sodium bicarbonate) may be more effective than an increase in intracellular buffering (via beta-alanine). With the beta-alanine dose (65 mg·kg−1·BM·d−1) employed by Bellinger et al. (2012), a worthwhile increase in muscle carnosine would be expected along with a subsequent increase in intracellular buffering. Without confirmation of the muscle carnosine content it is possible that not all participants responded to supplementation, or that some increased only slightly their muscle carnosine level (see Baguet et al., 2009), and the same could be suggested of the current study without muscle biopsies for confirmation. However, current evidence from studies measuring carnosine changes by muscle biopsy or \(^1\)H-MRS, suggests that the former is unlikely. It is possible though that there were a high proportion of low responders from the seven participants taking part in the Bellinger et al. (2012) study.

The trained participants in the current study completed the 2,000-m row in 6 min and 50 s on average, which would have required a significant contribution from anaerobic glycolysis (Stellingwerff et al., 2011), with the oxidation of the two neutral hydroxyl groups on each glycosyl (or glucosyl) unit metabolized to two carboxylic acid groups, ending with the accumulation of lactate anions and \( \text{H}^+ \) under these conditions. Accumulation of these \( \text{H}^+ \); as high intensity exercise continues, contributes to the disruption of metabolism (Harris et al., 1976; Spriet et al., 1989; Sählin 1992; Sählin & Harris, 2011) and muscle function (Donaldson & Hermansen, 1978; Fabiato & Fabiato, 1978), eventually leading to fatigue. Support for an effect of reduced intracellular \( \text{pH} \) on fatigue at the whole body level is provided by Hultman et al. (1985), who used ammonium chloride ingestion (0.3 g·kg\(^{-1}\)·BM) to generate acidosis before percutaneous electrical stimulation of the skeletal muscle. Force output was reduced in the ammonium chloride condition, which coincided with a lower intracellular \( \text{pH} \) at the end of 75 s of electrical stimulation.

Sale et al. (2012) have recently shown a 13.2% improvement in isometric knee extensor endurance at 45% of maximal voluntary isometric contraction (MVIC) with beta-alanine supplementation. The increase in intramuscular pressure at 45% MVIC results in occlusion of muscle blood flow, allowing only minimal loss of lactate anions and \( \text{H}^+ \) from the contracting muscle. The estimated increase in \( \text{H}^+ \) production (estimated from the known rate of lactate plus pyruvate production at this intensity (Ahlborg et al., 1972) was closely matched with the theoretical increase in \( \text{H}^+ \) buffering capacity from the estimated increase in muscle carnosine which occurs with this supplementation protocol. Given these findings, we suggest that the most plausible explanation for the very likely improvement to 2,000-m rowing
performance shown following 4 weeks of beta-alanine supplementation at 6.4 g·d⁻¹ in the current study is via an increase in intracellular buffering capacity as the result of an elevation in muscle carnosine. However, we cannot rule out the possibility that the improved performance was due to increased calcium sensitivity in the skeletal muscle fibers that could augment force production and the total work done during exercise (Dutka et al., 2012).

It should be noted that this study is not without limitation. No muscle biopsies were taken, primarily due to the nature of the participants and the testing venue. As such, we, like the other studies examining cosupplementation with beta-alanine and sodium bicarbonate previously (Sale et al., 2011; Bellinger et al., 2012), cannot confirm an increase in muscle carnosine and we cannot determine the direct mechanism for an effect of beta-alanine in muscle. However, given the results of other muscle-based studies—for a review, see Harris et al. (2012)—we are confident that an increase would have occurred in the current study. Similarly, given that this increase is highly likely, it is also the case that intracellular buffering capacity will have been improved, given that carnosine has a side chain pKa of 6.83 and is present in the skeletal muscle in millimolar concentrations. Despite these assertions, it would be of benefit for future studies of the effects of beta-alanine plus sodium bicarbonate supplementation to report the increase in muscle carnosine content and perhaps even the change in muscle pH, which we could not do in the current study. Furthermore, these results were obtained after just 4 weeks of beta-alanine supplementation, which does not correspond to the maximum increase in muscle carnosine (Hill et al., 2007). It is possible that still greater increases in muscle carnosine content as a result of a longer supplementation period, and thus a greater buffering capacity, would further increase the impact of beta-alanine supplementation on exercise performance. Finally, one participant had severe gastrointestinal distress as a result of the sodium bicarbonate supplementation. Although we analyzed the data with n = 19, there was little difference in the mean overall values to those presented and therefore we have included these data, despite the potential removal of the blinding for this individual.

In conclusion, the results of the current study show a very likely positive effect of beta-alanine supplementation alone, a likely positive effect of sodium bicarbonate supplementation alone, and a possibly positive effect with the addition of acute sodium bicarbonate supplementation to chronic beta-alanine supplementation on 2,000-m rowing performance. In view of these results, rowers might consider the use of beta-alanine or sodium bicarbonate and, if well tolerated by the individual, they might consider supplementation with both chronic beta-alanine and acute sodium bicarbonate. It should also be noted, however, that the responses to supplementation were individual.

Declaration of Funding

No funding was received for this study.

Conflict of Interest

We declare that we received beta-alanine and maltodextrin supplies from NAI to undertake this study, though no additional funding was provided. Roger Harris is an independent paid consultant of NAI, is named as an inventor on patents held by NAI.

Acknowledgments

The authors would like to thank National Alternatives International, San Marcos, California for providing the beta-alanine (Carnosyn SR) and placebo supplements. The authors would also like to thank Professor Will Hopkins for his expert advice on magnitude-based inferences, and the athletes of Nottingham Rowing Club, Leicester University Boat Club, and Nottingham Trent University Boat Club for participating.

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