SODIUM BICARBONATE AND HIGH-INTENSITY CYCLING CAPACITY: VARIABILITY IN RESPONSES

Original article
Running head: Sodium Bicarbonate Response Variability

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Abstract

Purpose: The aim of this study was to determine whether gastrointestinal (GI) distress affects the ergogenicity of sodium bicarbonate and whether the degree of alkalaemia or other metabolic responses are different between individuals who improve exercise capacity and those who do not. **Methods:** Twenty-one males completed two cycling capacity tests at 110% of maximum power output. Participants were supplemented with 0.3 $g \cdot kg^{-1}$BM of either placebo (maltodextrin) or sodium bicarbonate (SB). Blood pH, bicarbonate, base excess and lactate were determined at baseline, pre-exercise, immediately post-exercise and 5 minutes post-exercise. **Results:** SB supplementation did not significantly increase total work done (TWD) ($P = 0.16$, 46.8 ± 9.1 vs. 45.6 ± 8.4 kJ, $d = 0.14$), although magnitude based inferences suggested a 63% likelihood of a positive effect. When data were analysed without four participants who experienced GI discomfort, TWD ($P = 0.01$) was significantly improved with SB. Immediately post-exercise blood lactate was higher in SB for the individuals who improved but not for those who didn't. There were also differences in the pre to post-exercise change in blood pH, bicarbonate and base excess between individuals who improved and individuals who did not. C**onclusions:** SB improved high intensity cycling capacity, but only with the exclusion of participants experiencing GI discomfort. Differences in blood responses suggest that sodium bicarbonate may not be beneficial to all individuals. Magnitude based inferences suggested that the exercise effects are unlikely to be negative; therefore individuals should determine whether they respond well to sodium bicarbonate supplementation prior to competition.

Key words: Extracellular buffering, high-intensity exercise, gastrointestinal distress, blood responses, inter-individual variability

Introduction

The effects of sodium bicarbonate supplementation on exercise performance and capacity have been well researched (for review see [1]), and a recent meta-analysis showed that 0.3 g·kg^{-1}Body Mass (BM) sodium bicarbonate supplementation prior to a 60 s sprint improved performance by 1.7 ± 2.0% [2]. Despite this, the reported effects are equivocal, with several studies reporting no effect on exercise performance and capacity [3, 4, 5, 6, 7]. Inconsistencies in the performance outcomes of sodium bicarbonate supplementation studies can be partly attributed to differing dosing regimens [4], gastrointestinal (GI) discomfort experienced by some participants [8], exercise models insufficient to be limited by hydrogen cation (H^+) accumulation [5] and individual variation in the response to supplementation [9].

To determine the true effect of sodium bicarbonate supplementation on exercise, an appropriate exercise test to investigate the effects of increased buffering capacity should be of a sufficient intensity to result in a large accumulation of H^+, and therefore be limited by increasing muscle acidosis. Recently, Higgins et al. [10] showed that sodium bicarbonate improved exercise capacity at 100% peak mean minute power, but not 110 or 120%. However, using a high-intensity cycling capacity test performed to exhaustion at 110% of previously determined Powermax ($CCT_{110\%}$), previous studies have shown the $CCT_{110\%}$ to be positively influenced by a dietary intervention (β-alanine supplementation to increase muscle carnosine levels) known to increase intracellular pH buffering [11, 12]. This test was designed [11] to last between 120 and 240 s, and has been shown to be reliable with a coefficient of variation (CV) of 4.94% for total work done [13], which suggest that the $CCT_{110\%}$ is an appropriate model for examining the effects of dietary interventions designed to manipulate intramuscular changes in pH during exercise.

A potential moderator of the ergogenic effect of sodium bicarbonate supplementation on exercise capacity and performance is the gastrointestinal (GI) discomfort experienced by some participants. Price and Simons [9] suggested that the need to individualise supplementation with sodium bicarbonate was related to the individuals' susceptibility to GI discomfort, although GI discomfort was not correlated with performance decrements in their study. Van Montfoort et al. [14] measured the intensity of sickness and stomach ache prior to, and following high-intensity exercise, but reported little or no GI symptoms following supplementation with 0.3 g·kg^{-1}BM sodium bicarbonate. McNaughton [8] reported increased GI disturbance in all participants consuming doses above 0.3 g·kg^{-1}BM which may also explain the lack of a further increase in cycling capacity shown in these participants. The data of McNaughton [8] suggest 0.3 g·kg^{-1}BM to be the optimal dose to improve exercise performance or capacity with limited GI discomfort.

Matson and Tran [15] reported a relatively weak relationship (r = 0.42) between the dose of sodium bicarbonate and the resulting degree of blood alkalosis following supplementation using a meta-analysis of the literature. It was hypothesised that this was due to the large variability in individual pH and bicarbonate responses to supplementation, which suggests that the purported mechanism underlying a potential ergogenic effect of sodium bicarbonate supplementation might not have been present in all individuals. Most previous research has focused on the mean effect of sodium bicarbonate supplementation within the trial group, thereby disregarding individual variation, and this may have served to mask its true effect. Thus, inconsistencies in

previous findings could be explained by variability in individual responses to sodium bicarbonate supplementation, which, when analysed as group means with small sample numbers, do not represent its true effect. Therefore, it would be of interest to separate those who improved with sodium bicarbonate from those who did not, and investigate their blood responses to supplementation and exercise to determine any differences.

The present study was tightly controlled in an attempt to limit several contributing factors that may have contributed to equivocal results of sodium bicarbonate on exercise performance and capacity in the literature. This was achieved by employing the 'optimal' dose [8] using a split-dose strategy to minimise GI discomfort; employing a reliable [13] exercise test previously suggested to be limited by increasing muscle acidosis [11, 12]; and separating participants into those who improved their cycling capacity above the CV of the test and those who did not to determine any differences in blood responses. The primary aim of this study was to investigate the effect of sodium bicarbonate on cycling capacity and determine whether GI distress affects the efficacy. A secondary aim of this investigation was to determine whether the degree of blood alkalosis or other metabolic responses are different between individuals who improved exercise capacity and those who did not.

Materials and Methods

Participants

Twenty-one recreationally active males (mean ± SD; age 25 ± 5 y, body mass 80.7 ± 10.6 kg, height 1.79 ± 0.06 m, maximum cycling output [W_{max}] 316 ± 45 W) volunteered and gave their written informed consent to participate in this study. Participants were required not to have taken any supplement in the three months prior to taking part. The study was first approved by the institution's Ethics Review Committee.

Experimental Design

Participants were required to attend the laboratory on four separate occasions over a fourteen day period. All trials were performed at the same time of day to ensure results were not affected by circadian variation [16]. There were two preliminary trials, which comprised of an incremental cycle to exhaustion to determine W_{max}, followed by a habituation trial with the cycle capacity test to exhaustion at 110% of W_{max} ($CCT_{110\%}$). Participants then completed two repeated measures, counterbalanced and double-blind trials following the ingestion of 0.3 $g \cdot kg^{-1}$BM of either sodium bicarbonate (SB) or maltodextrin (P). All supplements were tested by HFL Sport Science prior to use to ensure no contamination with steroids or stimulants according to ISO 17025 accredited tests.

Design

Preliminary Testing

Each participant performed a graded cycle capacity test to exhaustion on a cycle ergometer (Lode Excalibur, Germany) to determine individual W_{max}. Exercise commenced at a self-selected power between 100 and 150 W, and was increased by 6 W every 15 s (ramp rate of 24 $W \cdot min^{-1}$) until participants reached volitional exhaustion. The maximum power output averaged over the final two stages was defined as an individual's W_{max}.

Every participant performed a habituation $CCT_{110\%}$ to minimise any learning effect during the main trials. A 5 min cycling warm up was performed at 100 W followed by a 2 min period of stretching. Since participants were not highly trained cyclists, each participant's $CCT_{110\%}$ was incremented over the first 30 s which corresponded to 80% W_{max} during the first 15 s, 95% W_{max} over the second 15 s followed by 110% W_{max} until volitional exhaustion [13]. Individual set up of the cycle ergometer (saddle and handlebar height and length) was determined prior to the initial W_{max} trial and was maintained for all subsequent $CCT_{110\%}$ trials. Participants pedalled at a self-selected pedal cadence (range 80-100 $rev \cdot min^{-1}$ across participants) and were required to maintain this cadence throughout the entire test. Verbal encouragement was given throughout. Volitional exhaustion was deemed to have occurred when participants dropped 20 $rev \cdot min^{-1}$ below their self-selected pedal cadence, at which point they were instructed to stop pedalling.

Main Trials

Twenty-four hours prior to the main trials, participants were required to refrain from alcohol, caffeine and any strenuous exercise. Food intake was monitored during the twenty-four hours prior to the first main trial using a food diary and replicated prior to the second main trial. Following an overnight fast, participants arrived at the laboratory 4 h before the $CCT_{110\%}$. Baseline finger-prick blood samples were taken

before consuming a standardised breakfast of 3 slices of toast and jam at 09:00. Participants ingested 0.2 $g \cdot kg^{-1}$BM of sodium bicarbonate (SIS, UK) or matching placebo (maltodextrin; SIS, UK) alongside the breakfast. A final 0.1 $g \cdot kg^{-1}$BM was ingested 2 h after the standardised breakfast (11:00), 2 h prior to commencement of the $CCT_{110\%}$ (13:00). All supplements were administered in gelatine capsules. Participants were instructed to report any gastrointestinal or other symptoms experienced during the four hours prior to exercise. They were requested to note down the time, type (stomach cramps, bloating, headaches) and the severity (mild, moderate or severe) of symptoms.

Participants performed the $CCT_{110\%}$ as described above for the habituation trial, with TWD being recorded as the outcome measure. Arterialised finger-prick blood samples were taken at rest, immediately pre-, immediately post- and 5-min post-exercise. Blood samples were analysed for lactate (Lactate Pro, Arkray, Japan), pH, haemoglobin (Hb) and blood gases (Radiometer ABL 400, UK). Blood bicarbonate was calculated from PCO_2 and pH values according to the Henderson-Hasselbalch equation and base excess was calculated according to $((1 - 0.014[Hb]) \times ([HCO_3^-] - 24 + (1.43[Hb] + 7.7)(pH - 7.4)))$.

Statistical Analysis

All data were analysed using Statistica 9 (Statsoft, USA) and are presented as mean ± 1SD. Data were analysed for the trial effect of sodium bicarbonate supplementation in all participants (N = 21). The data were then analysed following the exclusion of participants experiencing GI discomfort (N = 17). In addition, the complete data set was split into two groups, categorising participants as those who improved (N = 9), in whom exercise capacity was improved above the CV of the $CCT_{110\%}$ (4.94%; Saunders et al. [13]), and those who did not (N = 12), in whom exercise capacity was not improved above the CV of the test. Paired samples t-tests were used to determine any differences in performance measures between supplementation trials. A two-way ANOVA (trial x time) with repeated measures was used to determine any difference in blood pH, lactate, bicarbonate and base excess levels. Mauchly's test of Sphericity was used to check the data for sphericity, and where it was violated, a Greenhouse-Geisser correction was applied. A post-hoc Bonferroni correction factor was used to test any differences indicated by the ANOVA. Effect sizes were calculated using Cohen's d [17]. Pearson's correlations were used to determine any association in exercise and blood variables. In addition, magnitude based inferences [18] were used to determine the practical significance of sodium bicarbonate on the $CCT_{110\%}$ using a spread sheet to establish the likelihood of a meaningful effect on exercise capacity. The smallest worthwhile improvement in TWD was 1.27 kJ which was equivalent to half the unbiased typical error associated with the measurement. Statistical significance was accepted at the $P \leq 0.05$ level.

Results

All Participants (N = 21)

Total work done was not significantly different between conditions (P = 0.16, d = 0.14) (Table 1). Magnitude based inferences showed that the effect of SB on TWD was possibly beneficial (63% positive, 36% trivial, 1% negative).

There was no difference in baseline pH, bicarbonate, base excess or lactate between trials (Table 2). Supplementation with SB, but not P, significantly increased pre-exercise pH, bicarbonate and base excess levels from baseline ($P \leq 0.001$). Blood pH, bicarbonate and base excess measured immediately post-exercise and 5 minutes post-exercise (Table 2) were significantly decreased from baseline in both P and SB ($P \leq 0.001$); with values being significantly higher in SB ($P \leq 0.001$). Blood lactate (Table 2) was significantly increased from baseline following exercise in both trials ($P \leq 0.001$), with significantly higher post-exercise concentrations shown following SB ($P \leq 0.001$).

Total work done was not correlated with pre-exercise pH (r = -0.05), bicarbonate (r = 0.03) or base excess (r = 0.01), nor with their changes from baseline to pre-exercise. However, TWD was significantly correlated with the changes in pH (r = -0.43, P = 0.004), bicarbonate (r = -0.41, P = 0.008) and base excess (r = -0.45, P = 0.003) from pre- to post-exercise, although there was no significant correlation with the change in lactate.

Participants Not Experiencing GI Discomfort (N = 17)

Any participant reporting symptoms of moderate to severe discomfort following SB ingestion was considered an individual with GI discomfort; four participants were categorised as such, with the most frequently reported symptoms being moderate to severe stomach cramps and diarrhoea. When data were analysed without those participants experiencing GI discomfort, TWD was significantly increased (P = 0.01, d = 0.25) in SB compared with P (Table 1). Magnitude based inferences showed that the effect of SB was probably beneficial (78% positive, 22% trivial, 0% negative).

Blood responses to supplementation and exercise were similar to the whole group blood responses (Table 2). In addition, the removal of participants who experienced GI discomfort from the analyses did not influence the significance of any of the correlations that were performed on the full data-set.

Improved (N = 9) and Non-Improved (N = 12)

There was a degree of individual variability in exercise capacity between P and SB for all participants, with the difference in TWD between trials ranging from -5.1 to +8.1 kJ (Figure 1). Twelve participants increased TWD following SB supplementation. Nine participants improved above the 4.94% test retest variability for TWD during the $CCT_{110\%}$ [13]. The remaining twelve individuals who did not improve (N = 9) or who did not improve above the CV of the $CCT_{110\%}$ (N = 3) were allocated to the non-improved group.

Exercise capacity was significantly different between trials for the improved group ($P \leq 0.001$) but not for non-improved (P = 0.12; Table 1). Magnitude based inferences showed that the effect of SB for the improved group was almost certainly beneficial

(100% positive, 0% trivial, 0% negative) and possibly trivial for the non-improved group (11% positive, 72% trivial, 16% negative).

Blood pH, bicarbonate and base excess levels were significantly increased in both groups, from baseline to pre-exercise in SB only (Table 3). In the group who improved, the reduction in pH, bicarbonate and base excess from pre- to post-exercise was greater in SB than in P ($P \leq 0.01$). In the non-improved group, there was no difference in the reduction in pH, bicarbonate or base excess from pre- to post-exercise between trials (all $P > 0.05$). Immediately-post exercise blood lactate concentrations were significantly higher in SB for the improved group ($P = 0.003$) but not for the non-improved group ($P = 0.35$).

Total work done was not correlated with any pre-exercise blood marker for the improved and non-improved groups, or with their changes from baseline to pre-exercise. Total work done was not significantly correlated with any blood changes from pre- to post-exercise in the individuals who improved, but was correlated to the change in pH, bicarbonate and base excess in the group who were not improved (all $P \leq 0.05$).

Discussion

The current study showed that TWD during the $CCT_{110\%}$ was unaffected by sodium bicarbonate supplementation in all participants, despite resulting in alkalaemia prior to exercise, although magnitude based inferences suggest a 63% likelihood that the difference between conditions was meaningful. The lack of an effect may have been due to GI distress experienced by several individuals, since a positive effect was shown following the removal of those experiencing GI discomfort. This could not explain the lack of an effect in all participants; there were some differences in blood responses to supplementation and exercise which may have contributed to the variability in results.

The results of the present study are in contrast to several studies using β-alanine supplementation [11, 12] that have shown significant increases in TWD using the same exercise test. However, any contrast in findings between β-alanine and sodium bicarbonate supplementation may be due to carnosine's more direct influence upon intramuscular pH. In addition, Sale et al. [12] showed a further 4.3% increase in TWD when participants supplemented with β-alanine co-ingested sodium bicarbonate, although this was non-significant, a 70% likelihood of a meaningful difference was shown. Similarly, the authors showed some variability in the exercise response to sodium bicarbonate which may have contributed to the lack of an effect.

Price and Simons [9] reported no significant effect of sodium bicarbonate supplementation on high intensity running performance lasting around 75 s. The authors suggested that GI discomfort or individual differences in the blood responses to supplementation might explain the negative findings. Despite the split-dose strategy used in the present study, several participants reported symptoms of discomfort. As such, we analysed our data following the exclusion of the four participants who reported significant GI discomfort following sodium bicarbonate supplementation. None of the four participants reporting GI discomfort showed an increased exercise capacity following sodium bicarbonate supplementation, meaning that a significant improvement in high intensity exercise capacity was shown when group data were analysed following the exclusion of these participants. GI discomfort only partially explained the lack of an improvement in exercise capacity; however, twelve participants did not show any improvements in exercise capacity with sodium bicarbonate supplementation. This suggests that some other physiological differences between participants might also help to explain the individual capacity response.

Increases in blood bicarbonate concentration and subsequently blood alkalosis were shown in all participants prior to exercise following supplementation with sodium bicarbonate using a split-dose strategy. Pre-exercise blood bicarbonate concentrations compare favourably to those reported previously using different supplementation strategies but an identical dose [14, 19]. However, only nine participants showed an improved exercise capacity with sodium bicarbonate ingestion above the CV of the test (4.94% [13]). Blood data were also analysed according to the nine participants who showed an improved exercise capacity following sodium bicarbonate ingestion, and the twelve who did not. The change in blood bicarbonate, pH and base excess between baseline and pre-exercise following sodium bicarbonate ingestion were similar between individuals who improved and those who did not. This suggests that the underlying mechanism for an ergogenic effect of sodium bicarbonate supplementation was attained in all participants and thus was not an explanation for

the non-response. Further confirmation is provided by the fact that exercise capacity was not correlated to either the absolute concentration of, or the change in (from baseline to pre-exercise), any blood marker for all participants, suggesting that the degree of individual blood alkalosis prior to exercise did not influence the individual response in exercise capacity.

Whilst there were no differences between individuals who improved and who did not in the ability of sodium bicarbonate ingestion to promote blood alkalosis, the reduction in blood pH, bicarbonate and base excess from pre- to post-exercise was significantly greater in the sodium bicarbonate trial for the group who improved but not for those who did not. This might suggest that promoting blood alkalosis concentration through sodium bicarbonate supplementation does not necessarily increase blood bicarbonate buffering in all individuals during high-intensity exercise. As such, a potential difference exists in the ability of individuals to make full use of the induced blood alkalosis, which might explain the individual exercise capacity responses to sodium bicarbonate. Surprisingly however, TWD was not correlated to any change in blood measurements for the group of individuals who improved their exercise capacity, but was correlated to the change from pre- to post-exercise in blood pH, bicarbonate and base excess for the group who did not improve.

Ibanez et al. [20] reviewed the association between changes in peak blood lactate and exercise performance changes across 19 studies examining the potential ergogenic effects of alkalinising treatments. They suggested that a difference in blood lactate concentration of 2 $mmol \cdot L^{-1}$ between treatments was required to show a performance effect. In the present study, there was a difference between trials of +2.6 $mmol \cdot L^{-1}$ in peak blood lactate concentration immediately post-exercise in the group who improved, whereas there was an equivalent difference of only +0.7 $mmol \cdot L^{-1}$ in the group who did not improve their exercise capacity. As such, we provide some evidence to support the assertions of Ibanez et al. [20] since immediately post-exercise blood lactate concentrations were significantly higher in the sodium bicarbonate trial compared to the placebo trial for individuals who improved but not for those who did not. Furthermore, these results are consistent with a mechanism for the ergogenic effect of sodium bicarbonate being mediated by an increased efflux from muscle of lactate in association with H^+, along with improved intracellular pH regulation during exercise, and that where this does not occur there is no improvement. However, these findings are in contrast to those of Price and Simons [9], who showed that individuals whose performance worsened with sodium bicarbonate had a greater blood lactate response to exercise. This study investigated the variability in response to sodium bicarbonate supplementation during a single trial using a cycling capacity test previously shown to be limited by increasing acidosis. Further investigation should incorporate multiple sodium bicarbonate trials at the same intensity to determine whether exercise and blood responses to sodium bicarbonate supplementation are consistent within individuals.

Practical Applications

Sodium bicarbonate supplementation did not significantly improve exercise capacity, although exercise capacity was improved when the data from participants reporting GI discomfort were removed from the analyses. Furthermore, since magnitude based inferences suggest that sodium bicarbonate is unlikely to be negative, individuals

should engage in supplementation during training in order to determine whether they can tolerate the supplement, and if they attain any exercise benefit.

Conclusions

Sodium bicarbonate supplementation did not significantly improve exercise capacity during a cycling test likely to be limited by increasing muscle acidosis, although magnitude based inferences suggest a 63% likelihood of a significant positive effect. Furthermore, exercise capacity was improved when the data from participants reporting GI discomfort were removed from the analyses, although GI discomfort could not explain a lack of an effect in all participants. Variability in exercise capacity and some blood responses between trials suggests that sodium bicarbonate supplementation may be beneficial to some, but not all individuals.

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Figures

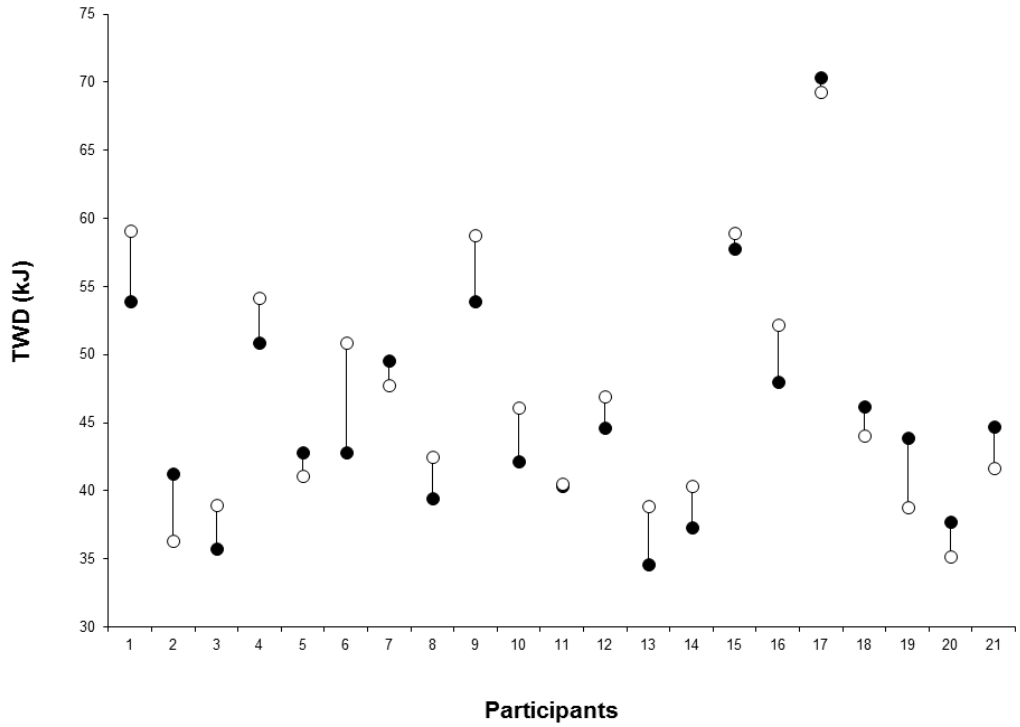

Figure 1. Individual TWD (kJ) in the $CCT_{110\%}$ in both P (black) and SB (white). Participants 18 – 21 are the participants who experienced gastrointestinal symptoms.

TABLE 1. TWD for all participants (N = 21), excluding those who experienced gastrointestinal discomfort (N = 17) and for participants who improved exercise capacity (Improved) and participants who did not improve exercise capacity (Non-Improved). *P ≤ 0.01 from placebo trial.

	TWD (kJ)
N = 21	
Placebo	45.6 ± 8.4
$NaHCO_3^-$	46.8 ± 9.1
N = 17	
Placebo	46.2 ± 9.2
$NaHCO_3^-$	48.4 ± 9.3*
Improved (N = 9)	
Placebo	43.1 ± 7.3
$NaHCO_3^-$	47.5 ± 8.1*
Non-Improved (N = 12)	
Placebo	47.5 ± 9.0
$NaHCO_3^-$	46.2 ± 10.1

TABLE 2. pH, bicarbonate, base excess and lactate for all participants (N = 21) and excluding those who experienced gastrointestinal discomfort (N = 17). Data are mean ± SD. (*$P \leq 0.01$ from baseline; ^ $P \leq 0.01$ from placebo trial at the same time point).

	Baseline	Pre-exercise	Post-exercise	Post-ex +5 min
N = 21				
pH				
Placebo	7.407±0.021	7.402±0.024	7.236±0.044*	7.229±0.056*
$NaHCO_3^-$	7.401±0.015	7.461±0.020*^	7.292±0.054*^	7.283±0.054*^
Bicarbonate (mmol·L^{-1})				
Placebo	24.79±1.14	24.96±0.99	14.43±1.89*	12.82±2.10*
$NaHCO_3^-$	24.66±1.44	30.40±1.01*^	18.39±2.52*^	15.26±2.78*^
Base excess (mmol·L^{-1})				
Placebo	0.78±0.98	0.82±0.78	-10.48±2.06*	-12.69±2.80*
$NaHCO_3^-$	0.54±1.28	6.49±1.03*^	-6.89±3.11*^	-9.60±3.38*^
Lactate (mmol·L^{-1})				
Placebo	1.2±0.4	1.2±0.5	12.6±2.4*	12.4±2.0*
$NaHCO_3^-$	1.1±0.4	1.2±0.3	14.4±3.4*^	14.5±2.9*^
N = 17				
pH				
Placebo	7.407±0.023	7.398±0.024	7.226±0.039*	7.215±0.048*
$NaHCO_3^-$	7.400±0.017	7.459±0.020*^	7.276±0.036*^	7.268±0.041*^
Bicarbonate (mmol·L^{-1})				
Placebo	24.79±1.24	24.87±1.07	15.16±1.78*	12.32±1.84*
$NaHCO_3^-$	24.51±1.41	30.33±1.08*^	17.52±1.68*^	14.41±2.07*^
Base excess (mmol·L^{-1})				
Placebo	0.70±1.08	0.66±0.75	-10.90±1.77*	-13.40±2.40*
$NaHCO_3^-$	0.39±1.27	6.39±1.05*^	-7.94±1.98*^	-10.61±2.53*^
Lactate (mmol·L^{-1})				
Placebo	1.2±0.3	1.3±0.5	13.0±2.4*	12.9±1.4*
$NaHCO_3^-$	1.2±0.4	1.2±0.3	15.5±2.6*^	15.5±1.8*^

TABLE 3. Changes in pH, bicarbonate, base excess and lactate from baseline to pre-exercise and pre-exercise to post-exercise for participants who improved exercise capacity (Improved) and participants who did not improve exercise capacity (Non-Improved) in SB. (*P ≤ 0.001 from placebo trial; ^P ≤ 0.01 from placebo trial).

	Δ Baseline to Pre-Ex	**Δ Pre-Ex to Post-Ex**
pH		
Improved		
Placebo	- 0.014 ± 0.036	- 0.158 ± 0.029
$NaHCO_3^-$	+ 0.060 ± 0.020*	- 0.184 ± 0.031^
Non-Improved		
Placebo	+ 0.002 ± 0.020	- 0.173 ± 0.047
$NaHCO_3^-$	+ 0.060 ± 0.015*	- 0.158 ± 0.056
Bicarbonate (mmol·L^{-1})		
Improved		
Placebo	+ 0.41 ± 0.83	- 9.19 ± 1.42
$NaHCO_3^-$	+ 5.94 ± 0.90*	- 12.79 ± 1.84*
Non-Improved		
Placebo	+ 0.00 ± 0.35	- 9.78 ± 2.10
$NaHCO_3^-$	+ 5.58 ± 1.53*	- 11.43 ± 2.46
Base Excess (mmol·L^{-1})		
Improved		
Placebo	+ 0.01 ± 0.70	-10.89 ± 1.45
$NaHCO_3^-$	+ 6.10 ± 0.72*	- 14.36 ± 1.97*
Non-Improved		
Placebo	+ 0.08 ± 0.51	-11.60 ± 2.38
$NaHCO_3^-$	+ 5.84 ± 1.35*	- 12.65 ± 3.03
Lactate (mmol·L^{-1})		
Improved		
Placebo	+ 0.1 ± 0.5	+ 11.0 ± 2.4
$NaHCO_3^-$	+ 0.1 ± 0.4	+ 14.0 ± 3.5*
Non-Improved		
Placebo	+ 0.0 ± 0.5	+ 11.8 ± 2.7
$NaHCO_3^-$	+ 0.1 ± 0.4	+ 12.6 ± 3.5