**Title:** The effects of sodium bicarbonate ingestion on cycling performance and acid base balance recovery in acute normobaric hypoxia.

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**Abstract**

This study investigated the effects of two separate doses of sodium bicarbonate (NaHCO$_3$) on 4 km time trial (TT) cycling performance and post-exercise acid base balance recovery in hypoxia. 14 club-level cyclists completed four cycling TT’s, followed by a 40 min passive recovery in normobaric hypoxic conditions (FiO$_2$ = 14.5%) following one of either: two doses of NaHCO$_3$ (0.2 g kg$^{-1}$ BM; SBC2, or 0.3 g kg$^{-1}$ BM; SBC3), a taste-matched placebo (0.07 g kg$^{-1}$ BM sodium chloride; PLA), or a control trial in a double-blind, randomized, repeated-measures and crossover design study. Compared to PLA, TT performance was improved following SBC2 ($p = 0.04$, $g = 0.16$, very likely beneficial), but was improved to a greater extent following SBC3 ($p = 0.01$, $g = 0.24$, very likely beneficial). Furthermore, a likely benefit of ingesting SBC3 over SBC2 was observed ($p = 0.13$, $g = 0.10$), although there was a large inter-individual variation. Both SBC treatments achieved full recovery within 40 min, which was not observed in either PLA or CON following the TT. In conclusion, NaHCO$_3$ improves 4 km TT performance and acid base balance recovery in acute moderate hypoxic conditions, however the optimal dose warrants an individual approach.

**Keywords:** Buffering, personalised nutrition, individual pursuit, alkalosis
Introduction

Exercise and training programmes with a hypoxic stimulus have been of interest to both exercise physiologists and athletes, as they may augment exercise performance upon a return to sea level (Holliss et al., 2013, Sinex and Chapman, 2015). One common issue with incorporating a hypoxic stimulus however, is the ability of the athlete to sustain overall training intensity and volume, as exercise performance represents a curvilinear decline with increasing elevations (Deb et al., 2018a). In cycling time trial (TT) performance, Amann et al. (2006) displayed a 5.4% reduction in 5 km TT completion time in acute moderate hypoxic conditions (FiO$_2$ 15%; 2700 m). Consequently, this reduction in volume and intensity in consecutive single sessions in hypoxia can potentially limit the efficacy of hypoxic training schedules, such as intermittent hypoxic training (IHT) (Sinex and Chapman, 2015). Athletes and coaches may therefore consider interventions that mitigate the decline in performance observed in single sessions of exercise at acute hypoxia, in an attempt to sustain overall training volume and intensity.

Decreases in performance shown in acute moderate (2000 to 3000 m) hypoxia are attributed to the reduction in the partial pressure of oxygen (PO$_2$), which hampers O$_2$ delivery and supply to the active musculature (Bassett and Howley, 2000). This reduction in convective O$_2$ transport in hypoxia places a greater reliance on non-oxidative energy pathways, owing to the higher relative intensity required compared to a given absolute workload in normoxia (Wolfel et al., 1991, Romer et al., 2007). Such greater reliance on non-oxidative energy systems increases metabolic perturbation, which subsequently increases the peripheral drive of fatigue compared to normoxia (Amann et al., 2007, Romer et al., 2007). This includes an exacerbated impairment of calcium ion (Ca$^{2+}$) release from the sarcoplasmic reticulum (SR) (Duhamel et al., 2004), more rapid accumulation of energy metabolites (i.e. hydrogen ions (H$^+$) and
inorganic phosphate (Pi)) (Adam and Welch, 1980; Hogan et al., 1999), and greater decrements
in the strong ion difference (SID) compared to the equivalent normoxic exercise (Lühker et al.,
2017). All of these factors are implicated as the source of fatigue during high-intensity exercise
(Allen et al., 2008; Cairns and Lindinger, 2008). Adams and Welch (1980) for instance,
reported a reduction in pH, an increased H⁺ production, and a 3 min shorter cycling time to
exhaustion at 90% VO₂max at acute hypoxia. These biochemical changes show H⁺ accumulation
is more rapid in hypoxia. The impact of such changes on fatigue during exercise are
controversial however (Fitts, 2016, Westerblad, 2016), therefore investigating strategies to
mitigate such acid base balance perturbation may offer greater insight into the determinants of
fatigue in acute hypoxia.

Sodium bicarbonate (NaHCO₃) is one alkalotic supplement that may help alleviate the
heightened acid base balance perturbation in hypoxic conditions, by significantly increasing
blood pH and increasing the availability of bicarbonate (HCO₃⁻) ions. This facilitates an
increased efflux of H⁺ from intramuscular to extracellular compartments for a single bout of
high-intensity exercise, thereby protecting intramuscular acid base balance (Bishop et al.,
2004). Whilst in recovery, NaHCO₃ ingestion has been shown to accelerate the rate of pH and
HCO₃⁻ recovery in multiple studies (Gough et al., 2017; Pruscino et al., 2008), which
subsequently improved performance. An alternative mechanism is the increase in the strong
ion difference (SID) following NaHCO₃ ingestion, as this may increase muscle excitability and
action potentials and therefore improve exercise performance (Allen et al., 2008, Gehlert,
Bloch and Suhr, 2015). Indeed, Sostaric et al. (2006) reported an increase in the SID by 25%
prior to exercise following ingestion of 0.3 g·kg⁻¹ BM NaHCO₃ and in improvement in finger
flexion exercise to exhaustion. This exercise however, does not confirm if this mechanism
would be evident during dynamic whole body exercise. Equally, this study did not assess the
recovery of the SID following such exercise, which may be an important mechanism for a subsequent bout of exercise. Further research is therefore required to determine the relevance of the increase in the SID to exercise performance/fatigue following NaHCO₃ supplementation.

Recently, Gough et al. (2017a, 2018) showed that 0.2 g·kg⁻¹ BM NaHCO₃ was suitable to obtain ergogenic effects by reporting no dose-dependent differences in 4 km TT cycling performance compared to 0.3 g·kg⁻¹ BM NaHCO₃ in normoxia. Importantly, NaHCO₃ was ingested at a pre-determined individual time to peak HCO₃⁻, and this methodological change may explain why ergogenic effects were observed from the lower dose (Jones et al., 2016, Gough et al., 2017b). These findings therefore suggest that 0.2 g·kg⁻¹ BM NaHCO₃ may be sufficient to improve exercise performance, which is particularly important to mitigate the common gastrointestinal (GI) discomfort issues with larger doses (Saunders et al., 2014). Nonetheless, the use of a smaller 0.2 g·kg⁻¹ BM NaHCO₃ dose has been untested in acute hypoxic conditions however, and given the heightened acidic stress in these conditions, this stimulus is an appropriate model to assess the suitability of a lower dose of NaHCO₃. Comparison of the two doses of NaHCO₃ will also allow the potential ergogenic effects in a hypoxic environment to be assessed, and therefore evaluate the suitability of NaHCO₃ to support hypoxic training schedules. The purpose of this study therefore, was to investigate the effects of two separate doses of NaHCO₃ on 4 km TT performance in acute moderate hypoxic conditions.

**Methods**

**Participants**

Fourteen club-level cyclists (13 male, 1 female, age 28 ± 10 years, body mass 78 ± 12 kg, hypoxic maximal rate of oxygen consumption (VO₂max) 50 ± 6 ml·kg⁻¹·min⁻¹, hypoxic peak
power output 321 ± 39 W) volunteered for this study. Ethical approval was granted from the University Research Ethics Committee (URESC16-LG01) and all participants provided written informed consent.

Experimental design and pre-experimental procedures

After an initial VO$_{2\text{max}}$ in hypoxia, participants visited the laboratory on a further seven occasions in a block randomised, crossover, and double blind designed study (2 x identification of peak blood HCO$_3^-$, 5 x cycling TT’s). Each trial was conducted at a similar time of day (± 1 hour) and participants arrived in a four hour fasted state to limit any confounding effect of nutritional intake (Reilly, 1990). Trials were separated by a minimum of three days, and maximum of seven days to limit the effects of training adaptations (Drust et al., 2005). Participants were specifically asked to avoid participation in any strenuous activity and consumption of alcohol 24 hours prior to any trial. Nutritional intake was replicated 24 hours prior to each trial and monitored using nutritional logs. Participants were also encouraged to maintain nutritional intake across the study duration. Verbal screening was also conducted to ensure participants had not ingested beta alanine in the previous 12 weeks, to account for the long washout period of carnosine (Baguet et al., 2009).

Experimental procedures

In the second and third visit time to peak HCO$_3^-$ was identified, using a previously defined method (Gough et al., 2018). Subsequently, the following five visits, including an initial familiarisation visit, entailed completion of a 4 km TT on a reliable and valid cycle ergometer (Velotron, RacerMate Inc., USA), interfaced with 3D visual Velotron coaching software (RacerMate Inc., USA) (Abbiss et al., 2009, Sporer and McKenzie, 2007). This exercise protocol was chosen as this is a reliable protocol (Stone et al. 2011), and would elicit
significant perturbation to acid base balance (Gough et al., 2018), therefore providing a suitable

test of the acting mechanisms of NaHCO₃. Each TT was completed using an identical method

previously described (Gough et al., 2018), with the exception all were completed in a

normobaric hypoxic chamber (TISS, UK) set at 14.5% fraction of inspired oxygen (FiO₂) to

replicate approximately 3000 m altitude. Consistent temperature (20°C) and humidity (40%)

controls were also put in place. In addition, saturation of oxygen (SpO₂) was recorded

throughout each TT using fingertip pulse oximetry (Nissei, BO-600, Japan). Blood samples

were collected in a 100μl sodium heparin-coated glass clinitube for analysis of pH, HCO₃⁻, and
electrolytes including potassium (K⁺), sodium (Na⁺), calcium (Ca²⁺) and chloride (Cl⁻) using a

blood gas analyser (ABL800 Basic, Radiometer Medical Ltd., Denmark), with an additional

5μl sample for analysis of blood lactate (Lactate Pro 2, Arkray, Japan). This data was then used
to calculate the apparent SID by the following: [K⁺] + [Na⁺] + [Ca²⁺] + [Na⁺] – [Cl⁻] – [Lac⁻]

using a freely available spreadsheet (Lloyd, 2004). These blood measures were taken at rest, at
time to peak HCO₃⁻, pre and post warm-up, and immediately post exercise. This procedure was
repeated for each TT, apart from participants either ingested 0.2 g·kg⁻¹ BM NaHCO₃ (SBC2),
0.3 g·kg⁻¹ BM NaHCO₃ (SBC3), or a taste-matched placebo (PLA) containing 0.07 g·kg⁻¹ BM
sodium chloride, as per previous research (Gough et al., 2018). A control trial was also

conducted which entailed no supplementation. Treatments were block randomised and
administered double-blind. Gastrointestinal (GI) discomfort was recorded from rest until time
to individual peak HCO₃⁻ every 10 min following NaHCO₃ ingestion, as per previous research
(Gough et al., 2018, 2017a). In addition, a supplement belief questionnaire was administered
at time to peak HCO₃⁻ to assess whether the placebo was appropriately taste matched.

Following completion of the TT, participants sat quietly for a 40 min recovery in the hypoxic
chamber, where measures for heart rate, SpO₂, GI discomfort and the aforementioned blood
variables were taken. Only water was permitted to be ingested during all experimental trials, with the volume replicated in each trial.

Statistical analysis

No violation of normality (Shapiro-Wilk) or sphericity (Mauchly) within the assessed variables was observed. Therefore, a paired t test was used to compare both the differences in blood responses (time to peak and absolute changes in pH and $\text{HCO}_3^-$), and GI discomfort (severity and aggregate score) between SBC treatments. Performance data (time to complete the TT, mean power, and mean speed) and blood data (change in both pH and $\text{HCO}_3^-$ during exercise, and the absolute changes in these analytes from post-exercise to 40 min recovery) were assessed for differences using a repeated measures ANOVA. In addition, performance data was analysed using magnitude based inferences (MBI), which were calculated (with 90% CI) using the recommended thresholds within a freely available spreadsheet (Batterham and Hopkins, 2006). The threshold value to determine a benefit or harm was determined by the 3.3 second typical error (TE) of the 4 km TT. This procedure was conducted as the Cohen $d$ small effect size threshold (0.2) is often too small to display meaningful performance improvements. Otherwise, a two-way [treatment x time] repeated measures ANOVA was employed, and where a significant main effect was observed, the Bonferroni post-hoc pairwise comparison was applied. The effect size of the interactions/main effects are reported as the partial eta squared ($\eta^2$), and for between treatment analysis, Hedge’s $g$ effect sizes ($g$) are calculated and interpreted in accordance with conventional Cohen’s $d$ interpretations (Cohen, 1988). Confidence intervals (CI) are reported (± 95%) between experimental treatments for significant effects only. Two-way mixed effects model Intraclass correlation coefficients (ICC) were used with both the point value ($r$) and significance reported; to assess the reproducibility of the absolute changes in pH and $\text{HCO}_3^-$ between the initial
identification of time to peak blood HCO$_3^-$ trial, and the subsequent cycling trials. Data is reported as mean ± standard deviation (SD) unless otherwise stated, with statistical significance set at $p < 0.05$. Data were analysed using a statistical software package, SPSS (V.22, SPSS Inc., Chicago, IL, USA).

Results

Preliminary trials to determine time to peak blood bicarbonate

Time to peak HCO$_3^-$ occurred between 30 and 110 min in SBC2 (mean: $69 ± 22$ min; median: 60; CV: 32%), and between 50 to 100 min in SBC3 (mean: $72 ± 17$ min; median: 70; CV: 24%; vs. SBC2 $p = 0.91$). The absolute change in HCO$_3^-$ from baseline to peak was greater in SBC3 by $1.2$ mmol.l$^{-1}$ compared to SBC2 ($6.9 ± 1.2$ vs. $5.7 ± 0.9$ mmol.l$^{-1}$; $p < 0.05$).

The reproducibility of the absolute change from baseline to peak in HCO$_3^-$ was good in SBC2 ($r = 0.66$, $p = 0.04$) and excellent in SBC3 ($r = 0.76$, $p = 0.01$).

Performance

Time to complete the TT following SBC2 was $1.1 ± 1.0\%$ faster compared to CON ($p = 0.009$; CI = 8.1, 1.0; $g = 0.20$) and $0.9 ± 1.1\%$ faster compared to PLA ($p = 0.04$; CI = 6.8, 0.3; $g = 0.16$). The performance effect was more pronounced in SBC3 however, reporting a $1.6 ± 1.3\%$ improvement compared to CON ($p = 0.002$; CI = 11.1, 1.9; $g = 0.28$) and $1.4 ± 1.0\%$ improvement compared to PLA ($p = 0.005$; CI = 9.9, 1.1; $g = 0.24$; Figure 1). Using an MBI approach, a very likely beneficial effect was determined for both SBC2 and SBC3 compared to PLA (Table 1). There was no significant difference between SBC3 and SBC2 ($p = 0.13$; $g = 0.10$; Figure 2), however a mean $2$ s ($0.5 ± 0.8\%$) improvement was observed in the SBC3 treatment, which was determined as a likely benefit in MBI analysis (Table 1).
Blood responses

During experimental trials a [treatment x time] interaction was observed for pH ($P_{\eta^2} = 0.34, p <0.001$), such that pH was greater post-supplementation of NaHCO$_3$ in SBC2 compared to PLA (+0.06; $p <0.001$; CI = 0.6, 0.8, $g = 3.7$) and CON (+0.06; $p <0.001$; CI = 0.5, 0.9, $g = 3.7$). The largest increases were observed in SBC3 (vs. SBC2 +0.02; $p <0.005$; CI = 0.1, 0.3; $g = 1.9$; vs. PLA and CON; $p <0.001$; Figure 3). Similarly, higher pH values were observed in both SBC treatments post-warm up and post-TT compared to PLA and CON ($p <0.005$; Figure 3), although SBC3 was significantly greater (+0.02) than SBC2 post-warm up ($p = 0.04$; CI = 0.01, 0.4, $g = 0.7$). A [treatment x time] interaction was also observed for HCO$_3^-$ ($P_{\eta^2} = 0.60; p <0.001$; Figure 3), as SBC3 elicited the greatest change in HCO$_3^-$ from baseline to post-supplement (+7 mmol.l$^{-1}$) compared to SBC2 (+5.8 mmol.l$^{-1}$, $p = 0.01$; CI = 0.3, 2.3; $g = 1.4$) and both PLA and CON ($p <0.001$). This was also evident post warm-up, where SBC3 was 1.8 mmol.l$^{-1}$ greater than SBC2 ($p = 0.02$; CI = 0.3, 3.2; $g = 1.0$); however no differences between these two treatments were seen post-TT ($p = 0.35$). Both treatments were also greater than both PLA and CON at the post warm-up and post-TT stages ($p <0.005$). A [treatment] effect was observed for the change in HCO$_3^-$ during the TT ($P_{\eta^2} = 0.70; p <0.001$) where there were marginal differences between SBC2 and SBC3 (10.6 ± 3.0 vs. 11.4 ± 2.7 mmol.l$^{-1}$; $p = 0.72; g = 0.3$), however significantly greater changes compared to PLA and CON (8.0 ± 2.4 and 8.1 ± 2.2 mmol.l$^{-1}$; both $p <0.001$). Blood lactate was greater post-TT in both SBC treatments compared to both PLA and CON (both $p <0.002$), with no differences between SBC treatments ($p >0.05$; Figure 3).
Post-NaHCO₃ supplementation, the SID was greater in SBC2 compared to PLA (+4 meq.l⁻¹; \( p < 0.001; \text{CI} = 1.7, 6.3, g = 1.5 \)) and CON (+4 meq.l⁻¹; \( p < 0.001; \text{CI} = 2.0, 5.2, g = 1.5 \)). Similarly, the SID was greater in SBC3 compared to PLA (+6 meq.l⁻¹ vs. CON \( p < 0.001; \text{CI} = 3.1, 7.9, g = 3.7 \)) and CON (+6 meq.l⁻¹ vs. PLA \( p < 0.001; \text{CI} = 3.9, 8.1, g = 4.1 \); Figure 4). There was no difference between SBC conditions (\( p > 0.05 \)). Post-warm up, the SID was significantly greater to all other treatments in SBC3 (all \( p < 0.05 \)). Whereas, SBC2 was only significantly greater compared to PLA (+3 meq.l⁻¹, \( p = 0.02, \text{CI} = 0.3, 6.2, g = 1.0 \)), although did reveal a large effect size compared to CON (\( g = 0.97; p = 0.63 \)). Post-TT there was no difference in the SID between any treatment (\( p > 0.05 \)). Post-exercise recovery of acid base balance was accelerated with both doses of NaHCO₃ compared to PLA and CON (Figures 3 and 4).

**Rating of perceived exertion, heart rate, oxygen saturation, and gastrointestinal discomfort**

No effect of NaHCO₃ was observed on RPE₀ (\( \text{Pƞ}^2 = 0.04, p = 0.66 \)) or RPEₐ (\( \text{Pƞ}^2 = 0.04, p = 0.47 \)) during the TT. Similarly, HR (\( \text{Pƞ}^2 = 0.08, p = 0.31 \)) and SpO₂ (\( \text{Pƞ}^2 = 0.03, p = 0.79 \)) were unaffected by NaHCO₃ at any 500 m segment of the TT or during recovery (Table 2). More participants suffered from GI discomfort following SBC3 compared to SBC2 from ingestion to time to peak HCO₃⁻ (11/14 SBC3, 7/14 SBC2). Both the severity and aggregate score of GI discomfort was greater in SBC3 compared to SBC2 (severity: 7.6 ± 2.0 vs. 5.3 ± 2.4; \( p = 0.002; g = 1.0 \)) (aggregated score: 20 ± 14 vs. 9 ± 6; \( p = 0.005; g = 1.0 \); Figure 5).
There was a significant positive correlation for the absolute amount of NaHCO₃ ingested and the resulting aggregated score of GI discomfort, however only following SBC3 ($r^2 = 0.57$; $p < 0.03$). No GI discomfort was reported in the recovery period. The supplement was correctly identified by the participant on 4/42 occasions.

Discussion

The aim of this study was to investigate the effects of two separate doses of NaHCO₃ on 4 km TT cycling performance and post-exercise acid base balance recovery in acute moderate hypoxic conditions. Both SBC2 and SBC3 improved performance compared to PLA, revealing a ‘very likely’ beneficial effect; therefore, athletes can supplement NaHCO₃ to support maintenance of performance during normobaric hypoxic training schedules. Nonetheless, SBC3 displayed a greater magnitude of performance improvement compared to SBC2, showing a ‘likely’ beneficial effect. Due the inter-individual performance between responses and the lack of significance in parametric testing however, both should be trialled to determine the most optimal dose for performance benefits. This evidence also suggests that a 0.2 g·kg⁻¹·BM dose of NaHCO₃ is physiological optimal for some, despite the additional acidic stress cause by the hypoxic stimulus. Moreover, both SBC doses displayed a greater recovery of acid base balance following the TT compared to PLA suggesting NaHCO₃ supplementation may improve subsequent exercise performance, which future research should address.

The current study findings contrast with previous investigations reporting no effect of NaHCO₃ ingestion on performance in moderate normobaric hypoxic conditions equivalent
to 3000 m (Saunders et al., 2014, Flinn et al., 2014). The current study findings instead support recent investigations by Deb et al. (2017) who reported 0.3 g kg\(^{-1}\) BM NaHCO\(_3\) improved performance during a 3 min all-out, and intermittent high-intensity exercise to exhaustion (Deb et al., 2018b); at 3000 m acute hypoxia. Both Saunders et al. (2014) and Flinn et al. (2014) employed a set period for NaHCO\(_3\) ingestion prior to exercise (240 and 90 min, respectively) which fails to account for the high inter-individual variation to achieve peak alkalosis (Jones et al., 2016, Gough et al., 2017b). This suggests buffering capacity may not have been maximised in some individuals, thus leading to a reduced effect of NaHCO\(_3\) supplementation (Jones et al., 2016, Gough et al., 2017b). In contrast, both the present study and the investigations by Deb et al. (2017, 2018b) accounted for such inter-individual variation by supplementing NaHCO\(_3\) at either a pre-determined individual time to peak pH or HCO\(_3^-\), which may explain the more pronounced effect on performance. Identification of individual time to peak HCO\(_3^-\) following NaHCO\(_3\) ingestion is therefore important to heighten the ergogenic effects.

An interesting finding of this study was that SBC3 was ‘likely’ beneficial to performance compared to SBC2 in magnitude based inferences analysis. In the current study, the TE between the familiarisation and the CON trial was 3.3 seconds, and in using this cut off, three participants displayed improvements in SBC3 versus SBC2. This more pronounced effect in hypoxia from SBC3 versus SBC2 may be explained by the exacerbated acidic stress in hypoxic conditions, as a normoxic study displayed minimal differences when employing both the same dose and exercise protocol (Gough et al., 2018). Nonetheless, eleven participants displayed minimal differences between SBC2 and SBC3 (<3.3 seconds), and SBC2 still significantly improved performance compared to PLA. This suggests there is large inter-individual responses to the NaHCO\(_3\) dose in acute hypoxic conditions, and for most, 0.2 g kg\(^{-1}\)
BM NaHCO$_3$

BM NaHCO$_3$ may be physiologically optimal. Individuals should therefore trial both SBC2 and SBC3 to identify which is the most ergogenic and opt for SBC3 only if this provides an additive ergogenic effect.

The changes in both blood acid base balance and lactate in the present study offer mechanistic insight to explain the enhanced performance following NaHCO$_3$ ingestion. Indeed, the change in HCO$_3^-$ during exercise was enhanced by 25% in SBC2 and 30% in SBC3 compared to PLA, whilst greater blood lactate post-exercise was also observed in both SBC conditions (SBC +22%, SBC3 +23% vs. PLA). These changes infer a greater amount of extracellular H$^+$ buffering occurred during exercise following NaHCO$_3$, thereby protecting the pH gradient between the intramuscular and extracellular compartments. Alternatively, the post-exercise increase in lactate following NaHCO$_3$ ingestion may lead to upregulation of glycolytic flux and utilisation by preventing the inhibition of key glycolytic enzymes (i.e. phosphorylase and phosphofructokinase) (Hollidge-Horvat et al., 2000, Percival et al., 2015). These indirect markers cited in the present study are contested in literature however suggesting acidosis does not hinder anaerobic exercise performance, and that increases in post-exercise lactate actually infer a reduction of lactate uptake into inactive muscle tissue (Granier et al. 1996; Westerblad, 2016). Nonetheless, a recent study reported a 34% significantly greater estimated glycolytic energy contribution during taekwondo exercise following NaHCO$_3$ ingestion (Lopes-silva et al., 2018). Therefore, the findings of the current study support that the mechanism of action following NaHCO$_3$ ingestion to be augmented glycolytic contribution.

The SID was significantly enhanced following NaHCO$_3$ prior to exercise in the current study, primarily due to increases in Na$^+$, and reductions in Cl$^-$ from baseline to pre-exercise. These changes suggest action potentials within the T-system were better protected by
eliciting a greater ionic charge and thus, sustaining muscle excitability (Allen et al., 2008, Gehlert, Bloch and Suhr, 2015). Subsequently, these changes offer an alternative site of action for NaHCO₃'s ergogenic effects, rather than the traditional pH and HCO₃⁻ mechanisms often discussed, yet contested. The present study findings agree with Sostaric et al. (2006), who demonstrated that NaHCO₃ ingestion increased the SID and improved performance in finger flexion exercise to exhaustion. By eliciting ionic fluxes consistent with dynamic whole-body exercise however, the current study findings are more pertinent to support this mechanism of action. Such findings are restricted to extracellular ionic fluxes nonetheless, and therefore further work is required to investigate the intracellular ionic charges following NaHCO₃ ingestion to obtain a more valid measure of the effects of muscle ionic movements during fatiguing exercise.

A practical finding of this study is that individuals can implement NaHCO₃ supplementation to increase performance during a single bout high-intensity exercise in a hypoxic environment. This may be important to sustain overall training intensity and volume during intermittent hypoxic training schedules such as ‘live-low, train-high’, and therefore partly mitigate the fear of a detraining effect. Future research may wish to investigate if chronic NaHCO₃ ingestion during an intermittent hypoxic training schedule leads to greater adaptation of muscle buffering capacity and therefore performance, as the role of manipulating acid base balance on performance in this environment is unknown. Debate exists however, whether a normobaric replicates a hypobaric hypoxic environment, pointing to clinically significant differences in ventilation, fluid balance and acute mountain sickness (AMS) (Mounier and Brugniaux, 2012). This subsequently questions the application of the results of the current study. Nonetheless, athletes in training commonly employ a normobaric hypoxic stimulus (Millet et al., 2010), and therefore the results of this study may help such individuals to
maximise the output of their training. Future research may wish to address the effects of NaHCO₃ ingestion in hypobaric hypoxic environments.

Both NaHCO₃ doses achieved the full recovery of pH, HCO₃⁻ and the SID to baseline at a faster rate compared to PLA and CON (20 to 40 min vs. >40 min). These changes suggest a subsequent exercise could be improved, as the perturbation of acid base balance that typically occurs following high-intensity exercise would be alleviated. Of interest, both NaHCO₃ doses displayed similar recovery kinetics, suggesting that a lower NaHCO₃ dose may be suitable for recovery, particularly if <40 min is available between exercise bouts. These findings are in agreement with Robergs et al. (2005), who reported a similar accelerated recovery of acid base balance following combined NaHCO₃ (0.2 g kg⁻¹ BM) and sodium citrate (0.2 g kg⁻¹) ingestion in hypoxic conditions. Nonetheless, the current study adds that such accelerated recovery can be achieved at a higher level of hypoxia, and one that is more applicable to intermittent hypoxic training schedules (3000m vs. 1570m). Neither the current study, nor Robergs et al. (2005) included a subsequent bout of exercise however, therefore further research is required to establish if both NaHCO₃ doses can improve subsequent exercise performance.

Conclusion

The present study shows that NaHCO₃ supplementation at a pre-determined individual time to peak HCO₃⁻ improves 4 km TT cycling performance in acute moderate normobaric hypoxic conditions. The individual responses between NaHCO₃ doses were varied however, and individuals should therefore trial both amounts to assess which is the most ergogenic. The selection of the dose may be dependent on the GI discomfort responses, as SBC3 displayed significantly greater severity and instances of GI discomfort compared to
SBC2. Lastly, both SBC treatments displayed similar recovery of acid base balance back to baseline, which was also greater than PLA and CON. This suggests that both SBC treatments may improve subsequent exercise performance, which may support individuals obtaining sufficient training volume and intensity during intermittent hypoxic training schedules.

Disclosure statement

The authors declare that they have no conflict of interest.

References


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**Figure 1.** Mean (± SD) and individual (solid horizontal lines) time to TT completion following each treatment. ** denotes significantly improved compared to PLA and CON (p < 0.05).

**Figure 2.** Individual responses following SBC2 and SBC3. No significant difference observed between treatments (p >0.05).

**Figure 3.** Mean (± SD) blood pH (A), bicarbonate (HCO$_3^-$) (B) and lactate (C) following NaHCO$_3$. ** SBC3 greater than (p <0.05) PLA and CON, ## SBC2 greater than PLA and CON, † SBC3 greater than SBC2. Horizontal dotted lines represent baseline levels.
**Figure 4.** Mean (± SD) strong ion difference (SID) responses over time. SBC3 greater than (p <0.05) CON (*) and PLA (**), SBC2 greater than CON (#) and PLA (##), † SBC3 greater than SBC2. Horizontal dotted lines represent baseline levels.

**Figure 5.** Aggregated (± SD) gastrointestinal (GI) discomfort following SBC2 and SBC3 from ingestion to individual time to peak bicarbonate (HCO₃⁻). * SBC3 significantly greater than SBC2.

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**Table 1.** Magnitude based inferences (MBI’s) overview of performance data.

**Table 2.** Heart rate (HR), saturation of oxygen (SpO₂) and perceptual data overview during the 4 km time trial (TT).