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 $g{\cdot}kg^{-1}$ BM; SBC2, or 0.3 $g{\cdot}kg^{-1}$ BM; SBC3), a taste-matched placebo (0.07 $g{\cdot}kg^{-1}$ 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% VO_{2max} 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 ($NaHCO_3$) 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 (HCO_3^-) 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, $NaHCO_3$ ingestion has been shown to accelerate the rate of pH and HCO_3^- 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 $NaHCO_3$ 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 $g \cdot kg^{-1}$ BM $NaHCO_3$ 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 $NaHCO_3$ supplementation.

Recently, Gough et al. (2017a, 2018) showed that 0.2 $g \cdot kg^{-1}$ BM $NaHCO_3$ was suitable to obtain ergogenic effects by reporting no dose-dependent differences in 4 km TT cycling performance compared to 0.3 $g \cdot kg^{-1}$ BM $NaHCO_3$ in normoxia. Importantly, $NaHCO_3$ was ingested at a pre-determined individual time to peak HCO_3^-, 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 $g \cdot kg^{-1}$ BM $NaHCO_3$ 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 $g \cdot kg^{-1}$ BM $NaHCO_3$ 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 $NaHCO_3$. Comparison of the two doses of $NaHCO_3$ will also allow the potential ergogenic effects in a hypoxic environment to be assessed, and therefore evaluate the suitability of $NaHCO_3$ to support hypoxic training schedules. The purpose of this study therefore, was to investigate the effects of two separate doses of $NaHCO_3$ 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 (VO_{2max}) 50 ± 6 $ml \cdot kg \cdot min^{-1}$, 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_{2max} 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 $NaHCO_3$. 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 (FiO_2) to replicate approximately 3000 m altitude. Consistent temperature (20˚C) and humidity (40%) controls were also put in place. In addition, saturation of oxygen (SpO_2) 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, HCO_3^-, and electrolytes including potassium (K^+), sodium (Na^+), calcium (Ca^{2+}) and chloride (Cl^-) using a blood gas analyser (ABL800 Basic, Radiometer Medical Ltd., Denmark), with an additional 5ul sample for analysis of blood lactate (Lactate Pro 2, Arkray, Japan). This data was then used to calculate the apparent SID by the following: $[K^+] + [Na^+] + [Ca^{2+}] + [Na^+] – [Cl^-] – [Lac^-]$ using a freely available spreadsheet (Lloyd, 2004). These blood measures were taken at rest, at time to peak HCO_3^-, pre and post warm-up, and immediately post exercise. This procedure was repeated for each TT, apart from participants either ingested 0.2 $g{\cdot}kg^{-1}$ BM $NaHCO_3$ (SBC2), 0.3 $g{\cdot}kg^{-1}$ BM $NaHCO_3$ (SBC3), or a taste-matched placebo (PLA) containing 0.07 $g{\cdot}kg^{-1}$ 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 HCO_3^- every 10 min following $NaHCO_3$ ingestion, as per previous research (Gough et al., 2018, 2017a). In addition, a supplement belief questionnaire was administered at time to peak HCO_3^- 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, SpO_2, 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 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 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 ($P\eta^2$), 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 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).

Figure 1 near here

Figure 2 near here

Table 1 near here

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).

Figure 3 near here

Post-$NaHCO_3$ supplementation, the SID was greater in SBC2 compared to PLA (+4 $meq.l^{-1}$; p <0.001; CI = 1.7, 6.3, *g* = 1.5) and CON (+4 $meq.l^{-1}$; p <0.001; 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; CI = 3.1, 7.9; *g* = 3.7) and CON (+6 $meq.l^{-1}$ vs. PLA p <0.001; 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, 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 $NaHCO_3$ compared to PLA and CON (Figures 3 and 4).

Figure 4 near here

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

No effect of $NaHCO_3$ was observed on RPE_O ($P\eta^2$ = 0.04, p = 0.66) or RPE_L ($P\eta^2$ = 0.04, p = 0.47) during the TT. Similarly, HR ($P\eta^2$ = 0.08, p = 0.31) and SpO_2 ($P\eta^2$ = 0.03, p = 0.79) were unaffected by $NaHCO_3$ 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 HCO_3^- (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 $NaHCO_3$ 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.

Figure 5 near here

Table 2 near here

Discussion

The aim of this study was to investigate the effects of two separate doses of $NaHCO_3$ 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 $NaHCO_3$ 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 $g \cdot kg^{-1}$ BM dose of $NaHCO_3$ 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 $NaHCO_3$ supplementation may improve subsequent exercise performance, which future research should address.

The current study findings contrast with previous investigations reporting no effect of $NaHCO_3$ 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 $g \cdot kg^{-1}$ 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 $g \cdot kg^{-1}$

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 $NaHCO_3$'s ergogenic effects, rather than the traditional pH and HCO_3^- mechanisms often discussed, yet contested. The present study findings agree with Sostaric et al. (2006), who demonstrated that $NaHCO_3$ 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 $NaHCO_3$ 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 $NaHCO_3$ 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 $NaHCO_3$ 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 $NaHCO_3$ ingestion in hypobaric hypoxic environments.

Both $NaHCO_3$ doses achieved the full recovery of pH, HCO_3^- 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 $NaHCO_3$ doses displayed similar recovery kinetics, suggesting that a lower $NaHCO_3$ 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 $NaHCO_3$ (0.2 $g \cdot kg^{-1}$ BM) and sodium citrate (0.2 $g \cdot kg^{-1}$) 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 $NaHCO_3$ doses can improve subsequent exercise performance.

Conclusion

The present study shows that $NaHCO_3$ supplementation at a pre-determined individual time to peak HCO_3^- improves 4 km TT cycling performance in acute moderate normobaric hypoxic conditions. The individual responses between $NaHCO_3$ 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.

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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.

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