Title: Post-exercise supplementation of sodium bicarbonate improves acid base balance recovery and subsequent high-intensity boxing specific performance

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Abstract

The aim of this study was to assess the effects of post-exercise sodium bicarbonate ($NaHCO_3$) ingestion (0.3 $g.kg^{-1}$ body mass) on the recovery of acid-base balance (pH, HCO_3^-, and the SID) and subsequent exercise performance in elite boxers. Seven elite male professional boxers performed an initial bout of exhaustive exercise comprising of a boxing specific high-intensity interval running (HIIR) protocol, followed by a high-intensity run to volitional exhaustion (T_{LIM1}). A 75 min passive recovery then ensued, whereby after 10 min recovery, participants ingested either 0.3 $g.kg^{-1}$ body mass $NaHCO_3$, or 0.1 $g.kg^{-1}$ body mass sodium chloride (PLA). Solutions were taste matched and administered double-blind. Participants then completed a boxing specific punch combination protocol, followed by a second high-intensity run to volitional exhaustion (T_{LIM2}). Both initial bouts of T_{LIM1} were well matched between PLA and $NaHCO_3$ (ICC; $r = 0.94$, $p = 0.002$). The change in performance from T_{LIM1} to T_{LIM2} was greater following $NaHCO_3$ compared to PLA (+164 ± 90 vs. +73 ± 78 sec; $p = 0.02$, CI = 45.1, 428.8, $g = 1.0$). Following ingestion of $NaHCO_3$, pH was greater prior to T_{LIM2} by 0.11 ± 0.02 units (1.4%) ($p < 0.001$, CI = 0.09, 0.13, $g = 3.4$), whilst HCO_3^- was greater by 8.8 ± 1.5 $mmol.l^{-1}$ (26.3%) compared to PLA ($p < 0.001$, CI = 7.3, 10.2, $g = 5.1$). The current study suggests that these significant increases in acid base balance during post-exercise recovery facilitated the improvement in the subsequent bout of exercise. Future research should continue to explore the role of $NaHCO_3$ supplementation as a recovery aid in boxing and other combat sports.

Key Words:

Buffering, alkalosis, acid base balance, combat sports, recovery, nutrition, training

Introduction

High levels of glycolytic flux are essential to maintain the required physiological output during combat exercise (Franchini et al., 2011), although a concomitant fall in both muscle and blood pH and bicarbonate ion concentration ([HCO_3^-]) eventually occurs (Fitts, 1994). This is due to the increases in hydrogen ion (H^+) accumulation, which in turn, disturb the state of equilibrium between acidity and alkalinity of body fluids (i.e. acid base balance). Such an alteration is known as metabolic acidosis and has been associated with fatigue by reducing or impairing the release of calcium ions (Ca^{2+}) from the sarcoplasmic reticulum (Messonier, Kristensen, Juel & Denis, 2007), impeding glycolytic enzyme activity (Atherton, 2003), and altering the strong ion difference leading to reduced action potentials and muscle excitability (Sostaric et al., 2006). The typical daily regimen for a competitive boxer often consists of two sessions comprised of an initial high-intensity intermittent running session followed by a boxing-specific session that mimics the demands of competition, interspersed within a short recovery period (Morton, Robertson, Sutton and Maclaren, 2010). Subsequently a large degree of metabolic acidosis is likely evident in the subsequent bout of exercise, therefore mitigation of the deleterious effects between sessions are prudent to investigate.

Pre-exercise ingestion of 0.3 $g.kg^{-1}$ body mass (BM) sodium bicarbonate ($NaHCO_3$) can lead to an approximate increase in pH (+0.07 ± 0.01) and HCO_3^- from baseline (+3.9 ± 0.9 $mmol \cdot l^{-1}$), eliciting a state of metabolic alkalosis (Carr, Hopkins and Gore, 2011). Ergogenic effects have been reported in combat sports including boxing (Siegler and Hirscher, 2010) and judo (Artioli et al., 2007; Felippe, Lopes-Silva, Bertuzzi, McGinley & Lima-Silva, 2016) by either increasing punches landed or total work done (TWD). Whilst the effects of pre-exercise $NaHCO_3$ ingestion has been well researched (for review see McNaughton et al., 2016), the effects of post-exercise ingestion between two bouts of exercise to promote recovery has received minimal attention. The use of this alternative method might permit a greater observed improvement in acid base balance during the recovery period, whilst the enhanced level of acid base balance would not have been utilised within the initial bout of exercise. These factors combined might therefore increase performance during the subsequent bout of exercise compared to pre-exercise $NaHCO_3$ ingestion. Indeed, Gough et al. (2017) reported that 0.3 $g.kg^{-1}$ $NaHCO_3$ ingested 30 mins into a 90 min post-exercise recovery period improved subsequent cycling time to volitional exhaustion by 33 secs (~14%) in recreationally active individuals. It is likely that an enhanced level of acid base balance was the primary mechanism for such an improvement, as the authors reported marked increases in pH and HCO_3^- prior to

the second bout of exercise compared to the placebo (pH = +0.07, effect size (ES) = 2.6, HCO_3^- = +7 $mmol \cdot l^{-1}$, ES =3.4). It is unknown, however, if these positive findings translate to other exercise modalities such as boxing, and individuals of a higher training status.

The mechanisms to explain the performance improvement following $NaHCO_3$ supplementation is not unique to changes in pH and HCO_3^-. Specifically, marked ionic shifts are suggested to contribute to muscle fatigue by impeding maximal Na^+, K^+-ATPase activity, subsequently impairing cell membrane excitability (Fitts, 1994; Stephens, McKenna, Canny, Snow & McConell, 2002; Sostaric et al., 2006). Indeed, both large effluxes of extracellular K^+ concomitant with reductions in Na^+ have been suggested to exacerbate the K^+ induced decline in force production (Bouclin et al., 1995). Pre-exercise ingestion of $NaHCO_3$ has been shown to reduce K^+ and increase Na^+ prior to the onset of exercise (Sostaric et al., 2006; Siegler and Hirscher, 2010; Stephens et al., 2002; Jones et al., 2016). Indeed, Siegler and Hirscher (2010) reported $NaHCO_3$ supplementation prior to a simulated boxing protocol lowered K^+ compared to the placebo condition (4.0 ± 0.1 $mEq.l^{-1}$ vs. 5.3 ± 0.4 $mEq.l^{-1}$, respectively) and subsequently speculated that this reduction might have facilitated the resulting performance improvement. It is widely argued however, that electrolyte balance should be assessed by the collective analysis of the strong ion difference (SID), which is the balance of the fully dissociated cations and anions in intracellular and extracellular fluid (Stewart, 1983). Synergistic changes in electrolytes are suggested to allow for deeper assessment of fatigue mechanisms, as opposed to reporting changes within a single electrolyte. In the only study to date, Gough et al. (2018a) reported a significant increase in the SID following $NaHCO_3$ supplementation and an improvement in 2 x 4 km time trial cycling bouts interspersed by 40 min recovery, although this study was conducted in a normobaric hypoxic environment. The purpose of this study therefore was to investigate the effects of post-exercise ingestion of $NaHCO_3$ on acid base balance recovery, the SID and subsequent boxing performance.

Materials and methods

Seven male elite professional boxers (age: 27.1 ± 5.1 years, stature: 175.8 ± 5.7 cm, body mass: 72.2 ± 10.3 kg, relative peak oxygen uptake ($\dot{V}O_{2peak}$): 55.8 ± 11.4 $ml \cdot kg \cdot min^{-1}$) from various boxing weight classifications including flyweight, lightweight, junior welterweight (WBO/IBF) super lightweight (WBA/WBC), middleweight & super middleweight completed this study. Participants were considered elite standard boxers and

were at least Commonwealth (British Empire), English, International Masters, British Masters, or Midlands Area title holders, with an average of 4.1 ± 3.6 years professional boxing experience. At the time of data collection, all participants were in pre-competition training. The study received institutional ethics committee approval (University of Derby, UK) prior to any testing, and participants were informed of the details of the study, both verbally and in writing, prior to providing written informed consent in accordance with the Declaration of Helsinki. Physical Activity Readiness Questionnaire (PAR-Q) and blood analysis questionnaires were completed prior to each bout of exercise.

Preliminary procedures

Prior to each trial, participants were requested to avoid strenuous exercise and to abstain from caffeine and alcohol ingestion for at least 24 hours. Participants were also encouraged to adopt the same mixed balanced diet with adherence monitored through a food diary, which participants recorded 24 hours prior to testing. A photocopy of the food diary was given to each participant to facilitate dietary replication prior to each experimental trial with 100% adherence achieved. Finally, participants were verbally screened to ensure they had refrained from ingestion of ergogenic buffers such as sodium citrate and β-alanine for 6 months prior to beginning the study.

Participant's body composition was assessed using Dual Energy X-ray Absorptiometry (Lunar iDXA, GE Healthcare, Hertfordshire, UK) 7-10 days prior to the experimental trials for analysis of body mass (kg). During the same visit, following 3 hours of fasting, participants completed an incremental exercise test on a motorised treadmill (Desmo, Woodway, Germany) to assess peak oxygen uptake ($\dot{V}O_{2peak}$). Initially participants warmed up for 5 min at 8 $km \cdot h^{-1}$ with a 0% gradient. The test began with a 3 minute stage at 10 $km \cdot h^{-1}$, subsequently the speed increased by 2 $km \cdot h^{-1}$ every three min until it reached the 16 $km \cdot h^{-1}$ stage. From this point the gradient was increased by 2% every 2 min until volitional exhaustion. Throughout the test expired gas samples were collected via an online breath by breath system (Cortex MetaLyzer II, Biophysik, Leipzig, Germany) which was calibrated before each test as per the manufacturer's guidelines. Expired gas samples were analysed for oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$) and respiratory exchange ratio (RER). The highest value of $\dot{V}O_2$ obtained in any 30 second period was used to calculate $\dot{V}O_{2peak}$.

Familiarisation

During the second laboratory visit participants were familiarised with the high intensity interval run (HIIR) protocol (Table 1), and the punch type techniques and combinations (Table 2), that would be utilised during experimental trials. In the HIIR, emphasis was placed upon exercising at a percentage of running velocity at $\dot{V}O_{2peak}$ during each differing work interval as opposed to heart rate ensuring the total time at each workload was readily matched. Finally, participants ran at a velocity that elicited 90% $\dot{V}O_{2peak}$ to volitional exhaustion (T_{LIM}) as a measure of high-intensity endurance capacity.

*** Tables 1 and 2 about here ***

Experimental Design and Protocol

Experimental trials were conducted using a repeated measures, partially counterbalanced (due to odd number sample size), double-blind, and placebo controlled design, each separated by seven days. Participants reported for each trial three hours postprandial and at the same time of day to avoid any circadian rhythm effects on performance (Forbes-Robertson, Dudley, Vadgama, Cook, Drawer & Kilduff, 2012). Body mass was measured and recorded at the start of each laboratory visit (Seca 761 weight scales, Birmingham, UK), to monitor possible fluctuations between experimental trials due to the participants being in pre-competition training stages. The following baseline measures were obtained after 5 min seated rest: heart rate (HR; (Polar, FT40, Finland), blood lactate concentration ($[Bla^-]$), base excess (BE), bicarbonate ion concentration ($[HCO_3^-]$) and a range of electrolytes (sodium $[Na^+]$, potassium $[K^+]$, calcium $[Ca^{2+}]$ and chloride $[Cl^-]$). The electrolyte data was used to calculate the apparent SID using an online spreadsheet (Lloyd, 2004) based on the following formula: $[K^+] + [Na^+] + [Ca^{2+}] + [Na^+] – [Cl^-] – [Bla^-]$. Blood variables were collected via a finger prick capillary blood sample and analysed with a blood gas analyser (ABL90 Flex, Radiometer, West Sussex, UK). Perceived readiness to exercise (PRE) was then recorded against an 11 point (0-10) scale with 0 representing ‘not at all ready to exercise’ and 10 representing ‘completely ready to exercise’ (Higgins et al., 2013).

Exercise trials commenced with a 5 minute treadmill run at a velocity eliciting ~60% $\dot{V}O_{2PEAK}$ (warm-up) immediately followed by the HIIR protocol (Table 1) which was repeated three times to imitate the demands of 3x4 minute boxing rounds, each separated by 60 sec

active recovery. A self-selected active recovery was recorded and replicated for each recovery interval in both experimental trials. Subsequently, a fourth and final bout was performed on a treadmill at a running velocity eliciting ~90% $\dot{V}O_{2PEAK}$ to volitional exhaustion (T_{LIM1}) with participants blinded from distance and time completed. Overall (i.e. related to cardiovascular strain) ratings of perceived exertion (RPE_O) (Borg scale 6-20; 1982) were recorded within the final 5 sec of each round. Immediately post-exercise HR and RPE_O were recorded. Five min post-exercise HR and blood metabolite/electrolyte data was collected as previously described.

Participants then recovered passively for 75 min prior to undertaking subsequent boxing performance. This was selected due to previous data showing this time period is approximately when acid base balance returns to baseline following high-intensity exercise (Gough et al., 2017). Ten minutes into recovery, participants consumed either 0.3 $g.kg^{-1}$ body mass of $NaHCO_3$ or 0.1 $g.kg^{-1}$ body mass of sodium chloride (placebo; PLA) within a standardised five minute period. This time period was selected due to the fear of vomiting if ingestion began immediately post-exercise, whilst a longer time period was not used as this may have allowed acid base balance to recover back to baseline values prior to ingestion (Gough et al., 2017). Both drinks were mixed in 4 $ml \cdot kg^{-1}$ body mass tap water and 1 $ml.kg^{-1}$ body mass of double strength no added sugar orange squash (Sainsbury's, London, U.K) (Higgins et al., 2013). Thirty min post exercise abdominal discomfort (AD) and gut fullness (GF) were recorded using an 11 point (0-10) scale, with 0 representing 'empty' and 'completely comfortable', and 10 representing 'bloated' and 'unbearable pain' respectively (Higgins et al., 2013). Water was consumed *ad libitum* during recovery (mean 582 ± 40 ml).

At the end of the 75 minute recovery, HR, PRE, blood metabolites/electrolytes, AD and GF were all recorded prior to participants performing a 5 min standardised dynamic warm up. Participants then completed the boxing specific protocol (Table 2) whereby they were required to strike the focus pads (Serious, Rapid Fire Punch Mitts, London, UK), which were worn by the same researcher for all trials. Each complete cycle consisted of 21 punches with participants instructed to stay in their preferred boxing stance (orthodox or southpaw) throughout. The punch combination cycle was performed repeatedly for 3x3 minute rounds, each separated by 60 sec passive recovery. Participants were all given the same boxing gloves (10 oz, Adidas, Hi-Tech Multi-Boxing Glove, Germany) for both experimental trials. An audio and visual boxing gym timer (Title Boxing, De luxe gym timer, USA) kept timing of rounds. Immediately at the end of each round participants HR, RPE_O and ratings of perceived exertion localised to the arms (RPE_A; Borg scale 6-20) were recorded. Upon completion of the 3 boxing specific

rounds AD and GF were also recorded. Following a 60 second rest period, participants then performed a final high intensity treadmill run corresponding to a speed that elicited ~90% $\dot{V}O_{2peak}$ to volitional exhaustion (T_{LIM2}). Immediately post exercise HR, $RPE_{O,}$ AD and GF were recorded, and five min post-exercise HR, blood metabolite/electrolytes, AD and GF were recorded.

Statistical analysis

Data was firstly checked for normality via a Shaprio-Wilk test, followed by a Mauchly test for homogeneity of variance/sphericity. A paired t test was used for some performance (T_{LIM1} and T_{LIM2}) and blood/perceptual data (change in HCO_3^- during T_{LIM2}, change in HCO_3^- during recovery, and aggregated GI discomfort). A two-way [treatment × time] repeated measures ANOVA was conducted with a Bonferroni correction for changes in blood variables (pH, HCO_3^- and lactate). Effect size (ES) for interactions from the ANOVA are reported as partial eta squared ($P\eta^2$), whilst between treatment ES are reported as Hedge's g effect sizes (g) (interpreted as per conventional thresholds described by Cohen 1988). If $p < 0.05$ then 95% CI are reported, where changes that do not cross the zero boundary treated as significant. A Friedman test was used for non-normally distributed data (AD, GF), and where the a priori alpha value was observed (i.e. $p<0.05$) a post hoc Wilcoxon signed rank-test was conducted with median, z score, and p value reported. For non-normally distributed data the ES is calculated by $Z/\sqrt{n}$ with 0.10, 0.24 and 0.37 considered as small, medium and large effects, respectively (Ivarsson et al., 2013). Reproducibility of the performance in T_{LIM1} was assessed using intraclass correlation coefficients (ICC), with the *r* value and significance reported. Additional statistics such as confidence intervals and effect sizes were used due to the small sample size in the study, which might not be suited to statistical procedures such as t test and ANOVA in isolation. Data were analysed using a statistical software package, SPSS (V.24, IBM Inc., Chicago, IL, USA).

Results

Performance

Both initial bouts for T_{LIM1} were well matched between PLA and $NaHCO_3$ (328 ± 155 vs 307 ± 142 s; ICC: $r = 0.94$, $p = 0.002$; t test, $p = 0.526$), showing that participants were at a similar level of fatigue at the start of the recovery period. Performance in T_{LIM2} was greater by

70 ± 90 sec (28%) following $NaHCO_3$ compared to PLA (p = 0.084, CI = -153.8, 12.9; Figure 1a), with a moderate effect size (g = 0.41). The change in performance from T_{LIM1} to T_{LIM2} was greater following $NaHCO_3$ compared to PLA (+164 ± 90 vs. +73 ± 78 sec; p = 0.02, CI = 45.1, 428.8, g = 1.0; Figure 1b). One participant displayed an ergolytic effect following $NaHCO_3$ ingestion, such that T_{LIM2} decreased by 13% compared to PLA (545 vs. 623 sec). This participant also suffered from moderate to severe GI discomfort.

Blood variables

No differences in pH between PLA and $NaHCO_3$ were observed at baseline (7.43 ± 0.04 vs. 7.42 ± 0.02; p = 0.233), or post T_{LIM1} (7.31 ± 0.04 vs. 7.31 ± 0.04; p = 0.696). Following the recovery period, and the ingestion of $NaHCO_3$, pH was greater prior to T_{LIM2} by 0.11 ± 0.02 units (1.4%) (p <0.001, CI = 0.09, 0.13, g = 3.4). Post T_{LIM2}, no difference between treatments was observed for pH (7.31 ± 0.06 vs. 7.33 ± 0.08; p = 0.271; Figure 2a). There were no differences in HCO_3^- between PLA and $NaHCO_3$ at baseline (25.9 ± 1.5 vs. 26.0 ± 1.6 $mmol.l^{-1}$; p = 0.750), post T_{LIM1} (16.6 ± 2.2 vs. 16.8 ± 2.2 $mmol.l^{-1}$; p = 0.723), or post T_{LIM2} (17.7 ± 3.1 vs. 19.0 ± 3.4 $mmol.l^{-1}$; p = 0.196). Following recovery however, HCO_3^- was greater by 8.8 ± 1.5 $mmol.l^{-1}$ (26.3%) post-$NaHCO_3$ supplementation compared to PLA (p <0.001, CI = 7.3, 10.2, g = 5.1; Figure 2b). The change in HCO_3^- during recovery (post T_{LIM1} to pre T_{LIM2}) was greater following $NaHCO_3$ ingestion compared to PLA (16.6 ± 1.4 vs. 8.0 ± 2.1 $mmol.l^{-1}$; p <0.001; CI = 6.5, 10.7, g = 4.5). During T_{LIM2}, the change in HCO_3^- during exercise was greater for $NaHCO_3$ compared to PLA (14.3 ± 2.9 vs. 6.9 ± 2.5 $mmol.l^{-1}$ p <0.001, 10.3, 4.5, g = 2.5). Post T_{LIM2}, BLa^- was 5.2 ± 2.6 $mmol.l^{-1}$ (39.5%) greater following $NaHCO_3$ (p = 0.002, CI = 2.6, 7.3, g = 2.0), with no difference at any other time point (p >0.05; Figure 2c).

Ingestion of $NaHCO_3$ caused marked changes in Na^+, K^+, Ca^{2+} and Cl^- (Figure 3). A time*treatment interaction was observed for the SID (p = 0.023, $P\eta^2$ = 0.576), such that a 10% increase in the SID was observed post recovery following $NaHCO_3$ ingestion compared to PLA (46 ± 1 vs. 36 ± 4 meq/l; p <0.001, CI = 6.3, 13.7, g = 3.2; Figure 4).

Heart rate and perceptual measures

Post-exercise ingestion of $NaHCO_3$ increased HR in rounds 2 and 3 compared to PLA (p <0.05), whilst no effect was observed on RPE_O or RPE_A (p >0.05) during any round of the HIIR. No effect on post T_{LIM2} HR (p = 0.217, g = 0.46) was observed following $NaHCO_3$.

Likewise, no difference in HR between $NaHCO_3$ and PLA were observed at any time point during T_{LIM2} (all p >0.05). Similarly, $NaHCO_3$ supplementation had no effect on post T_{LIM2} RPE_O (Z = 1.47, p = 0.383), with no difference observed between treatments at any time point (p >0.05).

Abdominal discomfort was greater following $NaHCO_3$ ingestion at 30 min recovery, displaying a moderate effect size (3.6 ± 3.0 vs. 1.6 ± 2.3; Z = 1.76, p = 0.07, g = 0.7). At the end of recovery, abdominal discomfort had generally reduced, although $NaHCO_3$ was still greater (1.7 ± 1.7 vs. 0.7 ± 1.3; Z = -1.89, p = 0.06, g = 0.6). No time*treatment interaction was observed for gut fullness (p = 0.219, η^2 = 0.213). Aggregated GI discomfort was not significantly different between $NaHCO_3$ ingestion and PLA (19 ± 13 vs. 13 ± 15; p = 0.175, -14.1, 3.2), although it was associated with a moderate effect size (g = 0.40; Figure 5).

Discussion

This study investigated the effects of post-exercise $NaHCO_3$ ingestion on subsequent high-intensity boxing performance. Following $NaHCO_3$ ingestion, acid base balance was increased prior to T_{LIM2} compared to PLA which subsequently improved subsequent boxing specific exercise performance. Athletes and coaches can therefore implement this strategy to support training at times when multiple bouts of exercise are carried out with limited recovery interspersed.

The findings of the current study show that $NaHCO_3$ ingestion improved subsequent boxing specific performance, by markedly reducing the decline from T_{LIM1} to T_{LIM2}. This adds to previous work evaluating post-exercise $NaHCO_3$ supplementation as a recovery supplement (Gough et al., 2017). Indeed, Gough et al. (2017) showed that $NaHCO_3$ ingestion 30 min into a 90 min recovery period improved subsequent cycling capacity, such that a moderate effect size (g = 0.5) was observed versus the placebo within a group of recreationally trained males. The current study adds however, similar ergogenic effects can be achieved with post-exercise $NaHCO_3$ ingestion within a shorter recovery time, individuals of a higher training status, and combat exercise. In addition, these findings also support previous literature showing $NaHCO_3$ ingestion is an effective supplement to improve combat performance when ingested prior to exercise (Siegler and Hirscher, 2010; Lopes-Silva et al., 2018). Future research could consider the impact of $NaHCO_3$ ingestion to enhance subsequent performance in other combat sports.

Based on the observed improvements it can be speculated that if $NaHCO_3$ supplementation could be adapted into a chronic weekly supplementation strategy, this might lead to greater adaptation to training. Previous work by Percival et al. (2015) has shown mRNA expression of PGC-1a, a known mechanism for mitochondrial adaptation, was increased 3 hours following a high intensity training session with acute $NaHCO_3$ ingestion compared to a placebo. Based on this evidence it is plausible that this may aid training adaptation in boxing, however, the study by Percival et al. (2015) was in cycling and in lesser trained individuals to the current study (healthy men vs. elite boxers). In addition, other studies investigating the effects of $NaHCO_3$ ingestion to support training adaptations are equivocal within trained individuals. Indeed, Edge, Bishop and Goodman (2006) reported chronic $NaHCO_3$ ingestion significantly increased lactate threshold by 11% and time to fatigue (100% VO_{2peak}) by 41% compared to a placebo following 8 weeks of cycling interval training. Both Driller et al. (2013) and Siegler et al. (2018) however, have shown no greater training adaptations following $NaHCO_3$ ingestion within rowing and resistance exercise modalities across 4 weeks and 10 weeks of training, respectively. Considering positive findings have been reported in combat exercise following $NaHCO_3$ ingestion (Siegler et al., 2010; Lopez-Silva et al., 2018), further research could explore if greater training adaptations occur with chronic $NaHCO_3$ ingestion.

In the present study, the likely mechanism to explain the improvement in subsequent performance is the changes in blood acid base balance between bouts, such that pH, HCO_3^- and the SID were significantly higher at 75 min recovery following $NaHCO_3$ ingestion. Full recovery of pH, HCO_3^-, and the SID was achieved in approximately 30-35 min. This is in contrast to the placebo condition, which failed to recover any of these blood analytes to baseline within 75 min of recovery. As a result, $NaHCO_3$ ingestion mitigated the disturbance to acid base balance during T_{LIM2}, which subsequently may explain the performance improvement. Such a greater state of metabolic alkalosis has been shown to increase buffering capacity by facilitating efflux of H^+ from the active muscle by enhanced circulating HCO_3^-, and thus, increasing the glycolytic energy contribution to high-intensity exercise (Bishop et al., 2004; Lopes-Silva et al., 2018). The current study supports these mechanisms, reporting a two-fold increase in the HCO_3^- change during T_{LIM2}, and a marked increase in lactate post-T_{LIM2} following $NaHCO_3$ ingestion. Indeed, Lopes-Silva et al. (2018) showed similar changes in post-exercise lactate following $NaHCO_3$ ingestion, but also reported a significant 31% increase in estimated glycolytic activity during simulated taekwondo combat. It is important to note however, the link between metabolic acidosis and fatigue has been widely criticised, suggesting

at physiologically valid muscle temperatures, accumulation of H^+ has limited effects on muscle contractile ability (Westerblad, 2016). As the current study did not assess either temperature or metabolite accumulation in muscle, we cannot confirm that acidosis has a direct impact on fatigue and performance.

An alternative mechanism to explain the performance improvement might be the increases in the SID following $NaHCO_3$ ingestion. Reductions in K^+ and Cl^- were observed, whilst Na^+ was increased in the recovery period, which lead to an overall increase in the SID. This could lead to an increase in electrical excitation, membrane potentials and muscle action potentials, which in turn, could support maximal Na^+, K^+-ATPase activity (Fitts, 1994; Cairns and Lindinger, 2008). Previous research, however, has suggested the most important electrolyte change is K^+, by demonstrating that raised extracellular concentration depresses muscle excitability (Cairns and Lindinger, 2008). This suggests that the important changes that $NaHCO_3$ supplementation elicits is in K^+. Nonetheless, shifts in Cl^- similar to those observed in the present study have been suggested to drive K^+ back to the muscle fibre through inward rectifier channels, which assist in returning the cell back to resting membrane potential (Lindinger and Heigenhauser, 1991). A well-designed study by Bouclin et al. (1995) also showed that when an increased K^+ and reduced Na^+ were altered in combination, the effects on twitch and tetanic contractions were greater than the changes in these ions in isolation. It is more likely therefore, that collective changes in electrolyte regulation explain the ergogenic mechanism of $NaHCO_3$ supplementation. Further research should therefore continue to explore the effects of $NaHCO_3$ supplementation on the SID and exercise performance.

One individual presented moderate to high GI discomfort following $NaHCO_3$ ingestion and displayed an ergolytic effect on performance. These findings agree with prior investigations suggesting GI discomfort might be a factor that negates the performance improvement from $NaHCO_3$ (Saunders et al., 2014; Cameron et al., 2010; Deb et al., 2018). Indeed, Saunders et al. (2014) reported upon removing participants who suffered GI discomfort following $NaHCO_3$ ingestion, only then did total work done (TWD) improve ($p = 0.01$, $d = 0.25$) compared to when all participants were included ($p = 0.16$, $d = 0.14$). However, performance benefits in combination with the onset of GI discomfort have occurred previously, whilst there is a lack of a direct link between GI discomfort and exercise performance following $NaHCO_3$ ingestion (Higgins et al., 2013; Gough et al., 2018b). Individuals that suffer from severe GI discomfort could benefit from a lower dose of $NaHCO_3$, as 0.2 $g.kg^{-1}$ BM $NaHCO_3$ has been shown to produce similar ergogenic responses whilst significantly reducing GI

discomfort (Gough et al., 2018b). Alternatively, the athlete could consider gastric bypass methods of delivery (i.e. enteric coated capsules), as novel data has suggested this may be suitable to reduce GI discomfort but still achieve the required increase in acid base balance (Oliveira et al., 2018; Hilton et el., 2019); although the performance responses are currently unclear. Further research should explore both lower doses of $NaHCO_3$ and the use of gastric bypass methods of delivery to understand the link between GI discomfort and performance following $NaHCO_3$ ingestion.

A limitation of this study is the small sample size, meaning further work is required to establish the impact of manipulating post-exercise acid base balance on performance and recovery. Despite this, the participant cohort were of an elite standard which are typically difficult to access. The current study findings therefore still have high practical application in sports performance, although further research with larger sample sizes are required. These findings compliment previous research investigating $NaHCO_3$ supplementation and exercise performance within lesser-trained combat athletes (Artioli et al., 2007; Tobias et al., 2013; Lopes-Silva et al., 2017) and support the use of $NaHCO_3$ supplementation to promote superior recovery.

Conclusion

The use of $NaHCO_3$ is a suitable ergogenic aid to achieve a greater magnitude of acid base balance recovery and improve subsequent boxing performance within elite level boxers. Being the first study to assess this within an elite participant cohort, the results of this study are of significance to athletes and coaches in an applied setting. Boxers within the elite category could therefore implement this strategy to augment training performance and potentially the subsequent adaptations. One participant did present ergolytic effects following $NaHCO_3$ ingestion however, which seemed to be due to high GI discomfort. Athletes should therefore trial $NaHCO_3$ ingestion to assess individual tolerability. Future research should implement similar recovery interventions within a larger sample of elite athletes to explore the effectiveness of $NaHCO_3$ supplementation as a recovery strategy.

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Author contributions statement

This study was conceived by MFH and designed by MFH and SR; data were collected by SR and analysed by LAG and MFH; data interpretation and manuscript preparation were undertaken by LAG, MFH, LRM, and AS. All authors approved the final version of the paper.

Conflict of interest statement and funding disclosure

None of the authors have any financial interest or benefit arising from the direct applications of this research and there is no conflict of interest. No funding was provided for this study.

List of figures

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References

Artioli, G. G., Gualano, B., Coelho, D. F., Benatti, F. B., Gailey, A. W., and Lancha Jr, A. H. (2007). Does sodium-bicarbonate ingestion improve simulated judo performance?. Int. J. Sport. Nutr. Exerc. Metab. 17(2), 206-217.

Atherton, J.C. (2003). Acid-base balance: maintenance of plasma pH. Anaesth. Intensive. Care. 4 (12), pp. 419–422.

Bishop, D., Edge, J., Davis, C., and Goodman, C. (2004). Induced metabolic alkalosis affects muscle metabolism and repeated-sprint ability. Med. Sci. Sports. Exerc. 36(5), 807-813.

Borg, G. A. (1982). Psychophysical bases of perceived exertion. Med. Sci. Sports. Exerc. 14(5), 377-381.

Bouclin, R., Charbonneau, E., and RenauD, J.M., (1995). Na+ and K+ effect on contractility of frog sartorius muscle: implication for the mechanism of fatigue. American Journal of Physiology-Cell Physiology. 268 (6), pp. 1528–1536.

Cairns, S. P., and Lindinger, M. I. (2008). Do multiple ionic interactions contribute to skeletal muscle fatigue?. J. Physiol. 586(17), 4039-4054.

Cameron, S. L., McLay-Cooke, R. T., Brown, R. C., Gray, A. R., and Fairbairn, K. A. (2010). Increased blood pH but not performance with sodium bicarbonate supplementation in elite rugby union players. Int. J. Sport. Nutr. Exerc. Metab. 20(4), 307-321.

Carr, A. J., Hopkins, W. G., and Gore, C. J. (2011). Effects of acute alkalosis and acidosis on performance. Sports. Med. 41(10), 801-814.

Cohen, J. (1988). Statistical power analysis for the behavioural sciences. L. Erlbaum Associates.

Deb, S. K., Gough, L. A., Sparks, S. A., and McNaughton, L. R. (2018). Sodium bicarbonate supplementation improves severe-intensity intermittent exercise under moderate acute hypoxic conditions. Eur. J. Appl. Physiol. 118(3), 607-615.

Driller, M. W., Gregory, J. R., Williams, A. D., and Fell, J. W. (2013). The effects of chronic sodium bicarbonate ingestion and interval training in highly trained rowers. Int. J. Sport. Nutr. Exerc. Metab. 23(1), 40-47.

Edge, J., Bishop, D., and Goodman, C. (2006). The effects of training intensity on muscle buffer capacity in females. Eur. J. Appl. Physiol. 96(1), 97-105.

Felippe, L. C., Lopes-Silva, J. P., Bertuzzi, R., McGinley, C., and Lima-Silva, A. E. (2016). Separate and combined effects of caffeine and sodium-bicarbonate intake on judo performance. Int. J. Sports. Physiol. Perform. 11(2), 221-226.

Fitts, R.H. (1994). Cellular mechanisms of muscle fatigue. Physiological Reviews. 74 (1), pp. 49–94.

Forbes-Robertson, S., Dudley, E., Vadgama, P., Cook, C., Drawer, S., and Kilduff, L. (2012). Circadian disruption and remedial interventions. Sports. Med. 42(3), 185-208.

Gough, L. A., Rimmer, S., Osler, C. J., and Higgins, M. F. (2017). Ingestion of Sodium Bicarbonate (NaHCO3) Following a Fatiguing Bout of Exercise Accelerates Postexercise Acid-Base Balance Recovery and Improves Subsequent High-Intensity Cycling Time to Exhaustion. Int. J. Sport. Nutr. Exerc. Metab. 27(5), 429-438.

Gough, L. A., Brown, D., Deb, S. K., Sparks, S. A., and McNaughton, L. R. (2018a). The influence of alkalosis on repeated high-intensity exercise performance and acid–base balance recovery in acute moderate hypoxic conditions. Eur. J. Appl. Physiol. 118(12), 2489-2498.

Gough, L. A., Deb, S. K., Sparks, S. A., and McNaughton, L. R. (2018b). Sodium bicarbonate improves 4 km time trial cycling performance when individualised to time to peak blood bicarbonate in trained male cyclists. J. Sports. Sci. 36(15), 1705-1712.

Higgins, M.F., James, R.S., and Price, M.J., (2013). The effects of sodium bicarbonate ($NaHCO_3$) ingestion on high intensity cycling capacity. J. Sports. Sci. 31 (9), pp. 972–81.

Hilton, N. P., Leach, N. K., Sparks, S. A., Gough, L. A., Craig, M. M., Deb, S. K., and McNaughton, L. R. (2019). A novel ingestion strategy for sodium bicarbonate supplementation in a delayed-release form: a randomised crossover study in trained males. Sports medicine-open, *5*(1), 4.

Ivarsson, A., Andersen, M. B., Johnson, U., and Lindwall, M. (2013). To adjust or not adjust: nonparametric effect sizes, confidence intervals, and real-world meaning. Psychol. Sport. Exerc. 14(1), 97-102.

Jones, R. L., Stellingwerff, T., Artioli, G. G., Saunders, B., Cooper, S., and Sale, C. (2016). Dose-response of sodium bicarbonate ingestion highlights individuality in time course of blood analyte responses. Int. J. Sport. Nutr. Exerc. Metab. 26(5), 445-453.

Lloyd, P., (2004). Strong ion calculator - a practical bedside application of modern quantitative acid-base physiology. Crit. Care. Resusc. 6 (4), pp. 285–94.

Lindinger, M. I., and Heigenhauser, G. J. (1991). The roles of ion fluxes in skeletal muscle fatigue. Can. J. Physiol. Pharmacol. 69(2), 246-253.

Lopes-Silva, J. P., Da Silva Santos, J. F., Artioli, G. G., Loturco, I., Abbiss, C., and Franchini, E. (2018). Sodium bicarbonate ingestion increases glycolytic contribution and improves performance during simulated taekwondo combat. Eur. J. Sport. Sci. 18(3), 431-440.

McNaughton, L. R., Gough, L., Deb, S., Bentley, D., and Sparks, S. A. (2016). Recent developments in the use of sodium bicarbonate as an ergogenic aid. Curr. Sports. Med. Rep**.** 15(4), 233-244.

Mero, A. A., Hirvonen, P., Saarela, J., Hulmi, J. J., Hoffman, J. R., and Stout, J. R. (2013). Effect of sodium bicarbonate and beta-alanine supplementation on maximal sprint swimming. J. Int. Soc. Sports. Nutr. 10(1), 52.

Messonnier, L., Kristensen, M., Juel, C., and Denis, C. (2007). Importance of pH regulation and lactate/H+ transport capacity for work production during supramaximal exercise in humans. J. Appl. Physiol. 102 (5), pp. 1936–44.

Morton, J. P., Robertson, C., Sutton, L., and Maclaren, D.P.M. (2010). Making the weight: a case study from professional boxing. Int. J. Sport. Nutr. Exerc. Metab. 20(1), 80-85.

de Oliveira, L. F., Saunders, B., & Artioli, G. G. (2018). Is by-passing the stomach a means to optimise sodium bicarbonate supplementation? a case-study with a post-bariatric surgery individual. Int. J. Sport. Nutr. Exerc. Metab. 1-15.

Peart, D. J., Siegler, J. C., and Vince, R. V. (2012). Practical recommendations for coaches and athletes: a meta-analysis of sodium bicarbonate use for athletic performance. J. Strength. Cond. Res. 26(7), 1975-1983.

Percival, M. E., Martin, B. J., Gillen, J. B., Skelly, L. E., MacInnis, M. J., Green, A. E., ... and Gibala, M. J. (2015). Sodium bicarbonate ingestion augments the increase in PGC-1α mRNA expression during recovery from intense interval exercise in human skeletal muscle. J. Appl. Physiol. 119(11), 1303-1312.

Pruscino, C. L., Ross, M. L., Gregory, J. R., Savage, B., and Flanagan, T. R. (2008). Effects of sodium bicarbonate, caffeine, and their combination on repeated 200-m freestyle performance. Int. J. Sport. Nutr. Exerc. Metab. 18(2), 116-130.

Saunders, B., Sale, C., Harris, R. C., and Sunderland, C. (2014). Sodium bicarbonate and high-intensity-cycling capacity: variability in responses. Int. J. Sports. Physiol. Perform. 9(4), 627-632.

Siegler, J. C., and Hirscher, K. (2010). Sodium bicarbonate ingestion and boxing performance. J. Strength. Cond. Res. 24(1), 103-108.

Siegler, J. C., Marshall, P. W., Finn, H., Cross, R., and Mudie, K. (2018). Acute attenuation of fatigue after sodium bicarbonate supplementation does not manifest into greater training adaptations after 10-weeks of resistance training exercise. PloS one, 13(5), e0196677.

Stephens, T. J., McKenna, M. J., Canny, B. J., Snow, R. J., and McConell, G. K. (2002). Effect of sodium bicarbonate on muscle metabolism during intense endurance cycling. Med. Sci. Sports. Exerc. 34(4), 614-621.

Sostaric, S. M., Skinner, S. L., Brown, M. J., Sangkabutra, T., Medved, I., Medley, T., ... and McKenna, M. J. (2006). Alkalosis increases muscle K+ release, but lowers plasma [K+] and delays fatigue during dynamic forearm exercise. J. Physiol. 570(1), 185-205.

Westerblad, H. (2016). Acidosis is not a significant cause of skeletal muscle fatigue. Med. Sci. Sports. Exerc. 48(11), 2339-2342.

Zabala, M., Requena, B., Sánchez-Muñoz, C., González-Badillo, J. J., García, I., Ööpik, V., and Pääsuke, M. (2008). Effects of sodium bicarbonate ingestion on performance and perceptual responses in a laboratory-simulated BMX cycling qualification series. J. Strength. Cond. Res. 22(5), 1645-1653.

Zabala, M., Peinado, A. B., Calderón, F. J., Sampedro, J., Castillo, M. J., and Benito, P. J. (2011). Bicarbonate ingestion has no ergogenic effect on consecutive all out sprint tests in BMX elite cyclists. Eur. J. Appl. Physiol. 111(12), 3127-3134.

Table 1

Exercise duration (sec)	~%V̇O2PEAK	Intensity level
30	90	High
30	75	Moderate
30	90	High
30	75	Moderate
30	90	High
30	75	Moderate
30	90	High
30	75	Moderate
60 AR*	SS**	Low

Key: AR* = Active recovery; SS** = self-selected during familiarisation

557 **Table 2**

Phase 1	Phase 2 (combinations)	Phase 3
MIR	J-	MOR
MIR	J-BH	MOR
MIR	J-BH-LU	MOR
MIR	J-BH-LU-BU	MOR
MIR	J-BH-LU-BU-LH	MOR
MIR	J-BH-LU-BU-LH- BHH	MOR

Key: MIR: move in range, MOR: move out of range, J: Jab, BH: backhand, LU: lead uppercut, BU: backhand uppercut, LH: lead hook, BHH: Backhand hook

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