Sodium bicarbonate ingestion improves time-to-exhaustion cycling performance and alters estimated energy system contribution: a dose-response investigation

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Abstract

This study investigated the effects of two sodium bicarbonate ($NaHCO_3$) doses on estimated energy system contribution and performance during an intermittent high-intensity cycling test (HICT), and time-to-exhaustion (TTE) exercise. Twelve healthy males (stature: 1.75 ± 0.08 m; body mass: 67.5 ± 6.3 kg; age: 21.0 ± 1.4 years; maximal oxygen consumption: 45.1 ± 7.0 $ml.kg.min^{-1}$) attended four separate laboratory visits. Maximal aerobic power (MAP) was identified from an incremental exercise test. During the three experimental visits, participants ingested either 0.2 $g.kg^{-1}$ BM $NaHCO_3$ (SBC2), 0.3 $g.kg^{-1}$ BM $NaHCO_3$ (SBC3), or 0.07 $g.kg^{-1}$ BM sodium chloride (placebo; PLA) at 60 minutes pre-exercise. The HICT involved 3 x 60 s cycling bouts (90%, 95%, 100% MAP) interspersed with 90 s recovery, followed by TTE cycling at 105% MAP. Blood lactate was measured after each cycling bout to calculate estimates for glycolytic contribution to exercise. Gastrointestinal (GI) upset was quantified at baseline, 30 minutes and 60 minutes post-ingestion, and 5 minutes post-exercise. Cycling TTE increased for SBC2 (+20.2 s; p =0.045) and SBC3 (+31.9 s; p =0.004) compared to PLA. Glycolytic contribution increased, albeit non-significantly, during the TTE protocol for SBC2 (+7.77 kJ; p =0.10) and SBC3 (+7.95 kJ; p =0.07) compared to PLA. GI upset was exacerbated post-exercise after SBC3 for nausea compared to SBC2 and PLA (p <0.05), whilst SBC2 was not significantly different to PLA for any symptom (p >0.05). Both $NaHCO_3$ doses enhanced cycling performance and glycolytic contribution, however, higher doses may maximise ergogenic benefits.

Keywords: anaerobic; ergogenic aid; high-intensity exercise; alkalosis; fatigue; extracellular buffer

Introduction

High-intensity interval training (HIIT) involves near maximal exercise bouts (>80-100% maximum heart rate) separated by brief recovery periods (Islam et al., 2017). The high anaerobic demand associated with maximal efforts results in the accumulation of hydrogen cations (H^+) within the cytosol (Allen et al., 2008). Whilst these are mostly removed by intramuscular and/or extracellular buffering mechanics, production overwhelms neutralisation and this contributes towards a reduced intramuscular pH (Sahlin, 2014), causing exercise-induced acidosis. Such a biochemical state has been suggested to reduce glycolytic energy production and may disrupt calcium ion cross-bridge formation (Fitts, 2016). A common strategy to mitigate these deleterious effects of exercise is to enhance circulating level of extracellular blood bicarbonate (HCO_3^-), which subsequently allows for sustained efflux of H^+ from intramuscular environments during high-intensity exercise (Siegler et al., 2016). Increases in [HCO_3^-] of ~5.0-6.0 mmol.l^{-1} are suggested to be ergogenic and can be achieved via the ingestion of extracellular buffers, such as sodium bicarbonate ($NaHCO_3$) in doses of 0.2-0.3 g.kg^{-1} BM, respectively (Carr et al., 2011; Jones et al., 2016).

Common practice is to ingest 0.3 g.kg^{-1} BM $NaHCO_3$ at 60-90 minutes prior to exercise, which is based on historical research showing time to peak pH or HCO_3^- occurs at this time point at the group mean level (Carr et al., 2011; Hadzic et al., 2019). It is, however, likely that through following this strategy the dissociation of $NaHCO_3$ within stomach acid will cause gastrointestinal (GI) upset (Heibel et al., 2018), which may impair performance or dissuade athletes from using $NaHCO_3$ (Cameron et al., 2010, Saunders et al., 2014). Whilst, some authors have observed ergogenic benefits despite moderate GI upset (Gough et al., 2018, Miller et al., 2016), in some cases the upset has been severe or the participant has not been able to continue with the study procedures (Gough et al., 2017; Kahle et al., 2013). The administration of smaller $NaHCO_3$ doses (0.2 g.kg^{-1} BM) might therefore be preferable, as it

can mitigate GI upset and also reduce the sodium load per dose which might alleviate the health risks of ingesting this supplement; although these risks are more associated with long term use of $NaHCO_3$ (Gough et al., 2018; Graudal et al., 2012). McNaughton (1992) reported exacerbated GI upset following higher $NaHCO_3$ doses, while Gough et al. (2018) observed reduced occurrence of bowel urgency and bloating for 0.2 $g.kg^{-1}$ compared to 0.3 $g.kg^{-1}$ BM $NaHCO_3$. Reducing the dose is a simple strategy that might remove some of the negative connotations of ingesting this supplement, whilst it is far more cost effective than some of the recent strategies employed to reduce the GI upset following $NaHCO_3$ ingestion, such as in enteric-coated capsules (Hilton et al., 2019; Hilton et al., 2020).

Contemporary research has administered $NaHCO_3$ using an individualised time-to-peak pH or HCO_3^- approach, which is in response to studies showing that time-to-peak pH or HCO_3^- can vary between 10 and 180 min within individuals, regardless of the ingestion method (i.e. capsule vs. fluid) (Gough et al., 2017; Gough et al., 2018, Jones et al., 2016, Miller et al., 2016). In using the individual time-to-peak approach, this ensures that peak $[HCO_3^-]$ is achieved immediately before exercise, which does seem to lead to a more consistent ergogenic response (Gough et al., 2017; Gough et al., 2018). The identification of this time-to-peak HCO_3^- response presents a logistical challenge to athletes however, as the financial cost is high and requires specialist equipment and staff. It is plausible to suggest further research is therefore required to simplify this strategy, and to assess whether ergogenic benefits still exist for smaller $NaHCO_3$ doses following administration at a standardised time point. This, in turn, could increase the practical application of this supplement, whilst also potentially limiting GI upset.

The ergogenic benefits associated with $NaHCO_3$ ingestion are somewhat related to the increased activation of glycolytic energy pathways (da Silva et al., 2019, Lopez-Silva et al., 2018). Whilst this is debated (Westerblad, 2016), $NaHCO_3$ ingestion attenuates muscle

acidosis during exercise thus preventing the allosteric inhibition of glycogen phosphorlyase and phosphofructokinase (Siegler et al., 2016). This has been shown to increase estimated glycolytic contribution during HIIT protocols (da Silva et al., 2019), while there is robust evidence suggesting enhanced glycolytic flux within the muscle (Hollidge-Horvat et al., 2000). Strategies that elevate glycolytic energy system contribution may enhance exercise capacity during HIIT, however, research is yet to determine whether smaller $NaHCO_3$ doses elicit a similar physiological response.

The purpose of this study therefore was to investigate the effect of 0.2 $g.kg^{-1}$ and 0.3 $g.kg^{-1}$ BM $NaHCO_3$ ingested at 60 minutes pre-exercise on estimated energy contribution during a high-intensity, interval cycling test (HICT), and time-to-exhaustion (TTE) cycling performance.

Materials and Methods

Experimental approach to the problem

A block randomised, across subjects counterbalanced, single-blind, placebo-controlled, crossover experimental design was implemented for this study. Participants visited the laboratory on four separate occasions to complete an incremental exercise test, familiarisation and three experimental trials. All testing was conducted at the same time of day (± 2 hours) to minimise the confounding effects of circadian rhythms on exercise performance (Reilly, 1990). Participants arrived at the laboratory in a 3-hour post-prandial state, having refrained from alcohol ingestion and vigorous exercise for 24 hours prior. Maximal aerobic power (MAP) was determined from the incremental exercise test and used to prescribe the exercise intensities for the HICT and TTE cycling protocols (described below). Participants completed these exercise procedures for three experimental treatment arms: (a) 0.2 $g.kg^{-1}$ BM $NaHCO_3$ (SBC2), (b) 0.3 $g.kg^{-1}$ BM $NaHCO_3$ (SBC3), or (c) 0.07 $g.kg^{-1}$ BM sodium chloride to ensure

taste-matching (placebo; PLA) (Gough et al., 2018). Participants were instructed to maintain activity levels and dietary intake throughout the study, which were assessed via written logs. All experimental trials were separated by seven days.

Participants

Twelve healthy males (stature: 1.75 ± 0.08 m; body mass: 67.5 ± 6.3 kg; age: 21.0 ± 1.4 years; maximal oxygen consumption: 45.1 ± 7.0 ml.kg.min^{-1}) volunteered for this study. All participants were recreationally active and completed at least 60 minutes of vigorous exercise per week. Participants were excluded if they had any history of hypertension (>140/80 mmHg), were currently taking any medication/sports supplements, or had ingested intra- or extracellular buffering agents within the previous 6 months. The study was approved by the institutional departmental review board. Each participant was informed of the benefits and risks of the investigation prior to signing informed consent to participate in the study. Procedures were conducted in accordance with the World Medical Association's Declaration of Helsinki.

Procedures

On the initial visit, participants performed an incremental exercise test on a cycle ergometer (Excalibur Sport, Lode, Netherlands) to determine MAP. Gaseous exchange was collected using a breath-by-breath metabolic cart (Oxycon Pro, Jaeger, Hoechberg, Germany) to determine maximal rate of oxygen consumption (VO_{2max}). To determine VO_{2max}, the highest 30 s rolling average was calculated. Following a 5-minute warm-up (70 W; 70-90 rev.min^{-1}), increments of 20 W.min^{-1} were applied until volitional exhaustion. This was deemed as the failure to maintain cycling cadence >60 rev.min^{-1} despite verbal encouragement. Maximal anaerobic power was calculated as the fraction of time in the final stage divided by test

154 increment, added to completed power (Pinot and Grappe, 2014). Familiarisation to exercise
155 procedures (HICT and TTE cycling) was completed after 30 minutes of passive recovery.
156 This involved three bouts of 60 s cycling (90%, 95% and 100% MAP), interspersed with 90 s
157 of active recovery (100 W) and TTE cycling at 105% MAP. These were completed on the
158 cycle ergometer, with handle bar and seat height position adjusted according to preference,
159 which was subsequently replicated for all experimental trials. The TTE cycling protocol was
160 terminated when cadence dropped 10 rev.min^{-1} below the preferred cadence, and when
161 participants were unable to re-establish preferred cadence (range of selected cadence = 70-90
162 rev.min^{-1}). Participants were encouraged to exercise until volitional exhaustion, but total
163 exercise time was not revealed.

164 During experimental trial visits, participants completed visual analogue scales (VAS)
165 were used for baseline GI upset (0 mm = "no symptom"; 100 mm = "severest symptom") that
166 quantified the severity of nausea, flatulence, abdominal discomfort (AD), gut fullness (GF),
167 bowel urgency rating (BUR), diarrhoea, vomiting and belching (Gough et al., 2018).
168 Participants then consumed one of three experimental beverages (SBC2, SBC3 or PLA)
169 across a 5-minute period 60 minutes prior to exercise. Ingestion time was chosen in-line with
170 previous work that showed the absorption kinetics between these doses are not significantly
171 different up to this time point (Gough et al. 2017), and is the most practiced ingestion timing
172 (Carr et al., 2011; Hadzic et al., 2019). These were served as a chilled aqueous solution of 4
173 ml.kg^{-1} BM water and 1 ml.kg^{-1} BM squash (double strength orange squash, Tesco, UK) to
174 increase the palatability and taste-match each beverage (Higgins et al., 2013). A supplement
175 belief questionnaire was completed post-ingestion to assess the efficacy of the single-blind
176 design, and to ensure that no psychological bias regarding the impact of $NaHCO_3$ ingestion
177 was transferred onto participants (Gough et al., 2019). Symptoms of GI upset were repeated
178 at 30- and 60-minutes post-ingestion. Pre-exercise capillary blood samples were collected

into 20 µL end-to-end sodium heparised capillary tubes (EKF Diagnostic GmbH, Germany) and analysed for blood lactate concentration ([BLa^-]) using the Biosen C-Line (EKF Diagnostic GmbH, Germany). Participants rested for 5 minutes to determine baseline oxygen consumption and respiratory exchange ratio (RER), before completing the HICT and TTE protocols, during which gaseous exchange was measured throughout, and blood samples were taken after each cycling bout. Additional visual analogue scales were completed immediately post-exercise for GI upset. An overview of experimental trials is displayed in **Figure 1**.

[INSERT **Figure 1** near here]

Estimated energy system contribution calculations

Absolute energy demand and energy contribution from the oxidative and glycolytic energetic systems were estimated via non-invasive technique. The oxidative phosphorylation pathway (W_{AER}) was determined by subtracting resting oxygen consumption (i.e. the mean VO_2 value during the final 30 s of baseline) from the area under the oxygen consumption curve for each of the three 60 s bouts (90%, 95% and 100% MAP) during the HICT (di Prampero and Ferretti, 1999). Area under the curve was calculated using the trapezoidal method. This approach has recently been shown to provide reliable and valid estimations for W_{AER} during intermittent exercise (da Silva et al., 2019; Milioni et al., 2017). The glycolytic pathway ($W_{[LA]}$) was calculated from the assumption that a difference of 1 $mmol.l^{-1}$ of BLa^- obtained by subtracting baseline [BLa^-] from peak [BLa^-] (i.e. delta [BLa^-]) corresponded to 3 $ml.kg^{-1}$ BM of O_2 (Beneke et al., 2002; Brisola et al., 2015; da Silva et al., 2019; Milioni et al., 2017; Zagatto et al., 2016). Therefore, delta [BLa^-] for each of the three 60 s bouts and during TTE cycling (i.e. difference from pre to post) was multiplied by 3 and the participants' body mass

to calculate $W_{[LA]}$. The caloric quotient of 20.92 kJ was used to convert between absolute energy demand (in L of O_2) and energy contribution (in kJ) for both energetic systems.

Statistical analysis

Normality and sphericity were assessed using Shapiro-Wilk and Mauchly tests, before correcting for any violations (Greenhouse Geisser). One-way repeated measures analysis of variance (ANOVA) were conducted for cycling TTE performance and total energy demand and contribution from W_{AER} and $W_{[LA]}$ during exercise protocols. The smallest worthwhile change (SWC) in performance (9.1 s) was calculated as 0.3 x the between-individual SD for cycling TTE during familiarisation (Hopkins, 2004). This was then used as a threshold for interpreting individual differences and in an attempt to identify a true change in exercise performance between the $NaHCO_3$ and the placebo conditions. Two-factor (treatment x time) repeated measures ANOVA's were performed for $[BLa^-]$, RER, W_{AER} and $W_{[LA]}$ for each of the three 60 sec bouts during the HICT. When significant interactions were observed, pairwise comparisons using the bonferroni correction factor were performed. Friedman's two-way ANOVA's were conducted for GI upset. Post-hoc Wilcoxon matched-pair signed rank tests were performed when significance was observed, with median, *Z* score and significance reported. Fisher's exact test was used to assess the efficacy of the single-blind design. For ANOVA interactions, effect sizes were presented as partial eta-squared (η_p^2) (Olejnik and Algina, 2003). Between treatment effect sizes were calculated by dividing the difference in means by the pooled SD (Nakagawa et al., 2007), before applying a Hedges g (*g*) bias correction to account for the small sample size (Lakens, 2013). These were interpreted as trivial (<0.20), small (0.20–0.49), moderate (0.50–0.79), or large (≥0.80) (Cohen, 1988). Data are presented as mean ± SD and 95% confidence intervals (CI) reported for mean differences.

Statistical significance was set at $p < 0.05$ and data were analysed using SPSS v25 (SPSS Inc., IBM, USA).

Results

Performance was greater for SBC2 (136.4 ± 43.5 s) and SBC3 (158.7 ± 63.3 s) compared to PLA (116.2 ± 46.6 s) (**Figure 2**). These increases were significant for SBC2 (+20.2 s; CI: 0.4, 39.9; $p = 0.045$; $g = 0.77$) and SBC3 (+31.9 s; CI: 10.8, 53.1; $p = 0.004$; $g = 1.13$). A total of 8 out of 12 participants improved their performance above the SWC following SBC2, whilst 11 participants (out of 12) improved above this threshold following SBC3 (**Figure 3**). There was an 11.7 s mean difference in favour of SBC3 vs. SBC2, but this increase was not significant ($p = 0.303$; $g = 0.48$). Nonetheless, seven of the participants (out of 12) improved their performance above the SWC for SBC3 vs. SBC2, whilst this was only in favour of SBC2 for a single participant.

[INSERT **Figure 2-3** near here]

Grouped mean ± SD data for [BLa^-] and RER are presented in **Table 1**. No significant differences were displayed during the HICT protocol ($p > 0.05$). Post-TTE [BLa^-] was elevated for SBC2 (+2.35 $mmol.l^{-1}$; CI: 0.06, 4.64; $p = 0.04$; $g = 0.77$) and SBC3 (+3.13 $mmol.l^{-1}$; CI: 1.44, 4.82; $p = 0.001$; $g = 1.40$) compared to PLA. There was a small effect size for SBC3 vs. SBC2 (+0.78 $mmol.l^{-1}$; $p = 0.34$; $g = 0.46$). Peak RER was also increased for SBC2 (+0.09 AU; CI: 0.03, 0.15; $p = 0.005$; $g = 1.14$) and SBC3 (+0.11 AU; CI: 0.03, 0.19; $p = 0.011$; $g = 0.98$) compared to PLA.

[INSERT **Table 1** near here]

Total energy demand and contribution of the oxidative and glycolytic energetic systems during the HICT are presented in **Table 2**. No significant differences were displayed for energy demand or contribution from W_{AER} or $W_{[LA]}$ ($p > 0.05$), although $W_{[LA]}$ contribution was moderately increased for SBC2 (+3.71 kJ; $p = 0.09$; $g = 0.66$) and SBC3 (+7.12 kJ; $p = 0.14$; $g = 0.60$) compared to PLA (23.40 ± 8.93 kJ). There was a small effect size for $W_{[LA]}$ contribution when comparing SBC3 vs. SBC2 (+3.41 kJ; $p = 0.99$; $g = 0.27$). Energy contribution from W_{AER} was greater during the second 60 s bout for PLA vs. SBC2 (+4.16 kJ; CI: 0.50, 7.81; $p = 0.03$; $g = 0.86$). No significant differences were observed for energy contribution from W_{AER} or $W_{[LA]}$ during TTE cycling ($p > 0.05$; **Figure 4 A-B**), although $W_{[LA]}$ was moderately increased for SBC2 (+7.77 kJ; $p = 0.10$; $g = 0.65$) and SBC3 (+7.95 kJ; $p = 0.07$; $g = 0.70$) compared to PLA (15.62 ± 9.27 kJ). No difference was reported for $W_{[LA]}$ when comparing SBC3 vs. SBC2 (+0.18 kJ; $p = 1.00$; $g = 0.01$).

[INSERT **Table 2.** near here]

[INSERT **Figure 4 A-B** near here]

Treatments were successfully single-blinded and taste-matched (Fisher's exact test, $p = 0.28$). One subject identified all three beverages, eight only correctly perceived one of the three beverages, and the remaining three were unsure on all treatments. Eight participants reported their severest symptom after either SBC2 (4/12) or SBC3 (4/12), although some reported no difference between treatments (3/12), whereas one experienced the severest symptom following PLA (**Table 3**). No intervention or time interaction was observed at 30- or 60-minutes post-ingestion for any GI symptom ($p > 0.05$), or at post-exercise for vomiting, flatulence, GF, BUR or diarrhoea ($p > 0.05$). Nonetheless, symptom severity was increased

post-exercise following SBC3 compared to PLA for nausea (10.0 mm vs. 1.0 mm; Z = -2.197; p = 0.028) and belching (8.0 mm vs. 1.0 mm; Z = -2.371; p = 0.018), but not for SBC2 compared to PLA (p > 0.05). Increases in the severity of nausea post-exercise was also observed following SBC3 compared to SBC2 (Z = 2.366; p = 0.018; **Figure 5A**), but not belching (Z = 1.352; p = 0.176; **Figure 5B**). There was no difference between aggregate GI upset between SBC2 and SBC3 at any time point (all p > 0.05).

[INSERT **Table 2.** and **Figure 5 A-B** near here]

Discussion

This study is the first to explore the dose-response effects of $NaHCO_3$ ingestion when administered at a standardised time point on estimated energy system contribution and performance during intermittent cycling exercise. Both 0.2 $g.kg^{-1}$ and 0.3 $g.kg^{-1}$ BM $NaHCO_3$ improved cycling TTE and estimated glycolytic contribution during HICT, therefore both doses can be employed as an ergogenic strategy. Only minimal dose-dependent differences in GI upset were observed, although the smaller dose mitigated severity of post-exercise nausea and belching. The key finding of this study therefore is that 0.2 $g.kg^{-1}$ BM of $NaHCO_3$ can increase estimated glycolytic system contribution and be ergogenic for intermittent exercise performance.

Improvements in cycling TTE were observed for SBC2 and SBC3, with the moderate-to-large effect sizes reflective of previous findings employing a similar TTE protocol (Higgins et al., 2013). The present study adds to previous work (McKenzie et al., 1986; McNaughton, 1992), however, that ergogenic benefits can also be observed with a lower dose of $NaHCO_3$. Importantly, however, more participants improved over the SWC for SBC3 vs. SBC2, and a small effect size between treatments was observed in favour of SBC3 at the

group level. This contradicts findings by McKenzie et al. (1986) that displayed a 4 s difference in TTE for 0.15 $g.kg^{-1}$ and 0.3 $g.kg^{-1}$ BM $NaHCO_3$, and Gough et al. (2018) where only a 0.1% variation in 4-km cycling time trial performance was present for 0.2 $g.kg^{-1}$ and 0.3 $g.kg^{-1}$ BM doses. This discrepancy could be explained by differences in administration approach (standardised time point vs. time-to-peak), or the high-degree of inter-individual variation present in acid base balance following $NaHCO_3$ ingestion. Nonetheless, based on seven participants improving their performance following SBC3 vs. SBC2 (based on SWC), it is likely the athlete will secure the largest benefit from this higher dose. These dose-dependent differences in performance could also be attributed to the timing of exercise protocols. The cycling TTE protocol commenced ~75 minutes after $NaHCO_3$ ingestion accounting for both the warm-up and HICT, however it is expected that [HCO_3^-] will continue to rise until ~80 minutes post-ingestion for SBC3, by which point [HCO_3^-] will have started to decline for SBC2 in most individuals (Gough et al., 2017, Gough et al., 2018). Nonetheless, athletes unable to pre-determine their time-to-peak HCO_3^- can still employ either dosing strategy of the present study to obtain performance benefits during high-intensity cycling exercise.

Moderate, albeit non-significant, increases were observed for $W_{[LA]}$ during the HICT without altering energy demand or contribution from W_{AER}, which is in agreement to findings from recent studies (Brisola et al., 2015; da Silva et al., 2019; Lopes-Silva et al., 2018). Despite not achieving statistical significance, these increases were considered substantial for both SBC2 (+15.8%) and SBC3 (+30.3%) when compared to PLA, with the relatively small absolute changes in $W_{[LA]}$ attributed to the controlled total mechanical work during the HICT (da Silva et al., 2019). The most novel finding, however, was that there may be a dose-response effect of $NaHCO_3$ ingestion on changes in energy system contributions, with a small effect size present for $W_{[LA]}$ in favour of SBC3. Considering that enhanced HCO_3^- buffering

capacity is responsible for elevating glycolytic contribution, one explanation for these dose-dependent results could relate to the total amount of H^+ that can be neutralised. Assuming that total blood volume is ~5 L and that [HCO_3^-] was as small as ~1.0 mmol.l^{-1} higher for SBC3 vs. SBC2, then the higher dose could have allowed the neutralisation of an extra ~5 mmoles of H^+ (based on the 1:1 stoichiometry of HCO_3^- and H^+ reaction), in theory eliciting a greater up-regulation of glycolytic contribution (da Silva et al., 2019). It is important to note, however, that as the current methodology only indirectly assesses glycolytic flux (i.e. from changes in [BLa^-]), these increases in $W_{[LA]}$ contribution may overestimate glycolytic activation, instead reflecting greater lactate efflux from working muscles (Siegler et al., 2016). Nonetheless, previous research has corroborated the findings of the present study following $NaHCO_3$ ingestion (Hollidge-Horvat et al., 2000), therefore it seems plausible that both dosing strategies partially up-regulate glycolytic activation during high-intensity cycling.

The ingestion of $NaHCO_3$ resulted in mild-to-moderate GI symptoms, although both doses were well tolerated, which agrees with previous research (Gough et al., 2017). Minimal dose-dependent differences were observed for GI upset, though the reduced post-exercise nausea and belching for SBC2 agrees with Gough et al. (2018) where belching was exacerbated for the higher dose. The reduced severity of GI upset from this study could be attributed to the body mass of the participants in the present study (mean = 68 ± 6 kg) compared to those that have reported greater severity of GI upset in healthy males (Kahle et al., 2013) and trained rugby players (Cameron et al., 2013) (90 ± 6 and 95 ± 13 kg). Relative dosing protocols were derived during early laboratory studies to normalise post-exercise base deficit (Singer et al., 1955), and therefore fail to account for physiological differences such as body mass and the total absolute $NaHCO_3$ dose. Athletes with high body mass administer a greater absolute $NaHCO_3$ dose despite minimal differences in gut absorption rates,

particularly for the first 60 min post-ingestion (Gough et al. 2017), which most likely exacerbates GI upset. There might be an upper threshold for absolute $NaHCO_3$ doses, with doses above this exacerbating GI upset. At present, 0.2 $g.kg^{-1}$ BM $NaHCO_3$ is a suitable strategy for mitigating GI upset; however, future research could examine the effect of absolute dosage on symptom severity and exercise performance.

There are methodological limitations in the present study that future research should address. Firstly, the single-blind design of this study is a limitation that is important to note. Important methodological choices were adopted, however, to mitigate any potential impact of this design. This included the standardised verbal encouragement during exercise, and the use of a supplement belief questionnaire, as per previous research (Gough et al., 2018). The findings from the latter methodological decision suggested that the supplement was blinded from the participants and therefore the single-blind design has no impact on the efficacy of $NaHCO_3$ ingestion. Moreover, our inability to quantify changes in absolute demand and contribution from the ATP-PCr energetic system is a limitation. This was due to the relatively short recovery period (90 s) between each bout of the HICT that did not allow a clear EPOC curve to form and therefore, it was decided that the ATP-PCr energy contribution calculations should be excluded from our analysis. Lastly, it was not possible to measure changes in $[HCO_3^-]$ following $NaHCO_3$ ingestion in the present study. Evidence suggests, however, that the HCO_3^- response is similar for 0.2 $g.kg^{-1}$ and 0.3 $g.kg^{-1}$ BM $NaHCO_3$ doses within ~60 mins, therefore participants were likely at a similar level of alkalosis irrespective of dose (Gough et al., 2017; Gough et al., 2018). This timing of $NaHCO_3$ ingestion employed in this study was selected to assess of the potential ergogenic effects for athletes unable to adopt an individualised time-to-peak HCO_3^- approach, or access a blood gas analyser. Based on the observed ergogenic benefits for both doses vs. PLA, it should further enhance the practical application of $NaHCO_3$ supplementation to the athlete with limited funding.

Conclusion

Ingestion of 0.2 $g.kg^{-1}$ and 0.3 $g.kg^{-1}$ BM elevated glycolytic contribution to high intensity exercise and are ergogenic strategies to improve exercise performance. It is likely that athletes will gain increased benefit from SBC3, despite the occurrence of higher GI upset. Nonetheless, some athletes may still opt for the lower dose if this displays greater tolerability, whilst still securing an ergogenic benefit. The present study also shows that the contemporary time to peak alkalosis strategy might not be required when ingested 60 min prior to exercise, however direct comparisons between these two methods of ingestion are required.

Acknowledgements and conflicts of interest

We would like to thank all the participants for their time and efforts in this study. All authors have no conflict of interests to declare.

Author contributions

KR, WG and LG designed the study. WG completed the data collection, whilst WG, LG completed the majority of the manuscript, MF, AS, KR also contributed. All authors reviewed the paper and provided feedback. LG and WG completed the preparation of the manuscript.

Contribution to the field statement

Recently a contemporary approach to sodium bicarbonate supplementation has been mooted as the optimal way to obtain improvements in exercise performance. This approach requires complex, expensive kit and specialist knowledge of blood biochemistry, however, and so it is unlikely to be available to many athletes. The purpose of this study therefore was to re-

explore traditional options to assess if athletes could gain the improvements in performance without this outlay. Equally, lower doses of sodium bicarbonate have been shown to provide benefits to exercise performance to a similar extent than higher doses of the supplement. This is important as lower doses typically lead to less negative side effects (e.g. stomach ache, vomiting), and have a lower total sodium load per dose. The present study showed that both doses of sodium bicarbonate lead to improvements in performance, although it is likely that the higher dose will offer a greater improvement. Nonetheless, we have shown that athletes and coaches can use this strategy instead of opting for the more scientific approach, yet still achieve improvements to performance. This may subsequently improve the use of sodium bicarbonate supplementation in a practical setting, and make this more attractive to the athlete that has limited funding.

Figure legends

Figure 1. Schematic overviewing procedures during experimental visits; MAP – maximal aerobic power; TTE – time to exhaustion.

Figure 2. Mean differences and inter-individual variation for TTE cycling performance; SBC2 – 0.2 $g.kg^{-1}$ BM $NaHCO_3$; SBC3 – 0.3 $g.kg^{-1}$ BM $NaHCO_3$; PLA – sodium chloride (placebo); * sig difference compared to PLA trial ($p < 0.05$).

Figure 3. Individual changes (with mean; clear bar) in TTE duration compared to PLA condition; SBC2 – 0.2 $g.kg^{-1}$ BM $NaHCO_3$; SBC3 – 0.3 $g.kg^{-1}$ BM $NaHCO_3$; PLA – sodium chloride (placebo); dashed horizontal line depicts SWC in performance (9.1 s).

Figure 4 A-B. Mean ± SD for W_{AER} (A) and $W_{[LA]}$ (B) contribution during TTE cycling; SBC2 – 0.2 $g.kg^{-1}$ BM $NaHCO_3$; SBC3 – 0.3 $g.kg^{-1}$ BM $NaHCO_3$; PLA – sodium chloride (placebo).

Figure 5 A-B. Inter-individual variations in post-exercise nausea and belching; self-reported symptoms via visual analogue scales (out of 100 mm); SBC2 – 0.2 $g.kg^{-1}$ BM $NaHCO_3$; SBC3 – 0.3 $g.kg^{-1}$ BM $NaHCO_3$; PLA – sodium chloride (placebo).

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