## The effects of sodium bicarbonate supplementation at individual time-to-peak blood bicarbonate on 4-km cycling time trial performance in the heat

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#### ABSTRACT

The purpose of this study was to explore the effect of individualised sodium bicarbonate (NaHCO<sub>3</sub>) supplementation according to a pre-established individual time-to-peak (TTP) blood bicarbonate  $(HCO_{3})$  on 4-km cycling time trial (TT) performance in the heat. Eleven recreationally trained male cyclists (age:  $28 \pm 6$  years, height:  $180 \pm 6$  cm, body mass:  $80.5 \pm 8.4$  kg) volunteered for this study in a randomised, crossover, triple-blind, placebo-controlled design. An initial visit was conducted to determine TTP HCO<sub>3</sub> following 0.2 g.kg<sup>-1</sup> body mass (BM) NaHCO<sub>3</sub> ingestion. Subsequently, on three separate occasions, participants completed a 4-km cycling TT in the heat (30 degrees centigrade; °C) (relative humidity ~40%) following ingestion of either NaHCO<sub>3</sub> (0.2 g.kg<sup>-1</sup> body mass), a sodium chloride placebo (0.2 g.kg<sup>-1</sup> BM; PLA) at the predetermined individual TTP  $HCO_{3}^{-}$ , or no supplementation (control; CON). Absolute peak [ $HCO_{3}^{-}$ ] prior to the 4-km cycling TT's was elevated for NaHCO<sub>3</sub> compared to PLA (+2.8 mmol.l<sup>-1</sup>; p = 0.002; g = 2.2) and CON (+2.5 mmol.l<sup>-1</sup>; p < 0.001; g = 2.1). Completion time following NaHCO<sub>3</sub> was 5.6 ± 3.2 s faster than PLA (1.6%; Cl: 2.8, 8.3; p = 0.001; q = 0.2) and  $4.7 \pm 2.8$  s faster than CON (1.3%; Cl: 2.3, 7.1; p = 0.001; g = 0.2). These results demonstrate that NaHCO<sub>3</sub> ingestion at a pre-established individual TTP HCO<sub>3</sub> improves 4-km cycling TT performance in the heat, likely through enhancing buffering capacity.

#### **Highlights**

- This is the first time NaHCO<sub>3</sub> ingestion has been shown to improve 4-km cycling TT performance in conditions of high ambient heat.
- A smaller dose of NaHCO<sub>3</sub> (0.2 g.kg<sup>-1</sup> BM) is ergogenic in the heat, which is smaller than the dose typically ingested for sports performance (0.3 g.kg<sup>-1</sup> BM). This is important, as gastrointestinal discomfort is typically lower as the dose reduces.
- This study suggests that the individualised time-to-peak HCO<sub>3</sub> ingestion strategy with lower doses of NaHCO<sub>3</sub> is an ergogenic strategy in conditions of high ambient heat.

## Introduction

Athletes who compete indoors or internationally are frequently exposed to warm-hot ambient environments, as such events are commonly held in these climates (e.g. Summer Olympic Games). Equally, many athletes worldwide are required to perform in warm-hot environments during the warmest season, or at the hottest part of the day. This is apparent during indoor track cycling events where temperatures are approximately 28–34°C (depending on the location and ventilation guality); whilst in most cases air conditioning is not available. Indeed, at the most recent Olympic games (Tokyo, 2020) daily temperatures exceeding 30°C were common (Costa, Gaskell, McCubbin, & Snipe, 2020), which creates a greater physiological challenge for the athlete compared to competing in a thermoneutral

environment. Specifically, exercise in warm-hot environments causes alterations to muscle energy metabolism, such that in the scenario of high-intensity exercise over 1 min, there is a significant shift in contribution of adenosine triphosphate (ATP) supply from aerobic to anaerobic energetic systems, such as anaerobic glycolysis (Febbraio, 2001; Febbraio, Snow, Stathis, Hargreaves, & Carey, 1994; Fink, Costill, & Van Handel, 1975). Combine this with greater level of extracellular lactate during exercise in the heat versus thermoneutral conditions for the same given exercise, this suggests a greater level of acidosis is present (Robergs, Ghiasvand, & Parker, 2004). Unsurprisingly, Mitchell, Rogers, Basset, and Hubing (2014) reported a significant reduction in exercise capacity (time to exhaustion) in hot environments (37°C) compared to cool environments (10°C) at both 80% and 100% VO<sub>2max</sub>. It is intuitive to suggest

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therefore that nutritional interventions that could support ATP production via anaerobic energy pathways and reduce acidic stress may mitigate the deleterious physiological changes caused by exercising in the heat.

Sodium bicarbonate (NaHCO<sub>3</sub>) supplementation is a well-known alkalotic buffer, which can increase circulating blood bicarbonate  $(HCO_3^-)$  within the extracellular compartments (typically around 5–6 mmol.l<sup>-1</sup>). Typical dosage is between 0.2 and 0.4 g.kg<sup>-1</sup> body mass (BM), although a 0.3 g.kg<sup>-1</sup> BM dose is most widely accepted as being optimal for performance enhancement (McNaughton, Gough, Deb, Bentley, & Sparks, 2016). Whilst some authors suggest acidosis is not a contributor to fatigue (see Westerblad, 2016), ingestion of NaHCO<sub>3</sub> is suggested to increase extracellular buffering capacity and therefore improve intramuscular hydrogen cation (H<sup>+</sup>) handling. Specifically, upregulation of the lactate-H<sup>+</sup> co-transporter to efflux H<sup>+</sup> from the active musculature into circulation may occur, which subsequently maintains the acid base balance of the intracellular (muscle) compartments (Bishop, Edge, Davis, & Goodman, 2004; Mainwood & Worsley-Brown, 1975). Moreover, as H<sup>+</sup> provides competition at the troponinbinding site, the greater efflux of H<sup>+</sup> from the muscle cytosol caused by NaHCO<sub>3</sub> supplementation could facilitate improved cross-bridge cycling (; Knuth, Dave, Peters, & Fitts, 2006). This, in theory, should allow for sustained muscle contractions during high-intensity exercise. Based on these mechanisms, the supplementation of NaHCO<sub>3</sub> could support the increased demand for anaerobic energy production that is evident under thermal stress, and in turn, improve exercise performance. Despite this, only Mündel (2018) has investigated NaHCO<sub>3</sub> ingestion (0.5 g.kg<sup>-1</sup> BM) in conditions of ambient heat stress (30°C) and reported that total work done (kJ) was improved in the second bout of two 30 s Wingate tests compared to a placebo (effect size = 0.35; p < 0.05). Based on the encouraging findings, it is plausible that the use of NaHCO<sub>3</sub> could be ergogenic for other types of performance including, but not limited to, track cycling events.

To date, several studies have reported ergogenic effects of NaHCO<sub>3</sub> during a 4-km cycling time trial (TT) when ingestion was aligned with an individualised time-to-peak (TTP) HCO<sub>3</sub><sup>-</sup> (Gough, Brown, Deb, Sparks, & McNaughton, 2018; Gough, Deb, Brown, Sparks, & McNaughton, 2019; Gough, Deb, Sparks, & McNaughton, 2018; Gurton, Faulkner, & James, 2021; Hilton, Leach, Hilton, Sparks, & McNaughton, 2020). Some of these studies have also examined the use of 0.2 g.kg<sup>-1</sup> BM NaHCO<sub>3</sub>, which elicited almost identical ergogenic benefits compared to the traditionally administered 0.3 g.kg<sup>-1</sup> BM NaHCO<sub>3</sub> dose (Gough et al., 2019;

Gough, Brown, et al., 2018; Gough, Deb, et al., 2018). Indeed, Gough, Deb, et al. (2018) reported no difference in time to complete a 4-km cycling TT between these two doses (Hege's g effect size (q) = 0.02, p = 0.870), whilst both were significantly faster than the placebo  $(g \ 0.2 \ \text{g.kg}^{-1} \ \text{BM} = 0.64, \ g \ 0.3 \ \text{g.kg}^{-1} \ \text{BM} = 0.66; \ \text{both} \ p$ < 0.05). Considering that smaller doses of NaHCO<sub>3</sub> lead to lower gastrointestinal (GI) discomfort and sodium load (Gough, Deb, Sparks, & McNaughton, 2017; Gurton, Gough, Sparks, Faghy, & Reed, 2020), and that track cyclists are likely to repeatedly supplement NaHCO<sub>3</sub> over a few events within 48–72 hours, the smaller dose may be a more attractive option. The aim of this study therefore, was to investigate  $0.2 \text{ g.kg}^{-1}$ BM NaHCO<sub>3</sub> ingestion supplemented at each individuals peak  $HCO_3^-$  on 4-km cycling TT performance in conditions of high ambient heat. The hypothesis of this study was that NaHCO<sub>3</sub> ingestion would improve 4-km  $\Pi$  cycling performance in the heat compared to the placebo.

#### Methods

#### **Participants**

Eleven male recreationally trained cyclists (de Pauw et al., 2013) ( $180 \pm 6 \text{ cm}$ ;  $80.5 \pm 8.4 \text{ kg}$ ;  $28 \pm 6 \text{ years}$ ) volunteered for this study. Whilst a *priori* power calculation was not conducted, our sample size was identical to previous research highlighting n = 11 would be required to satisfy statistical power for a 4-km cycling TT, although this study was conducted in thermoneutral conditions (Gough et al., 2019). All protocols were submitted to, and approved by, an institutional review board for testing of human subjects (Gough/3647/Mod/2019/Sep /HELSFAEC), and all participants provided written informed consent prior to any experimental procedure.

#### **Experimental overview**

Participants visited the laboratory on five separate occasions in a block randomised, crossover, tripleblind, placebo-controlled study. Each participant was required to record nutritional intake 24 hours prior to laboratory visits and encouraged to maintain identical nutritional ingestion throughout the study. During the initial laboratory visit, a 24-hour dietary recall was conducted, and participants were asked to replicate their intake before subsequent visits to the laboratory. Participants arrived 3 hours post-prandial to each trial, which were conducted at a similar time of day (±1 hour) to manage the effects of circadian rhythms (Reilly, 1990). Trials were separated by a minimum of three days and maximum of five days to reduce the influence of training adaptations (Drust, Waterhouse, Atkinson, Edwards, & Reilly, 2005). Participants were not ingesting any supplements that could interfere with the results during the study, and none had ingested beta alanine previously.

### **Pre-experimental procedures**

The initial visit involved ingestion of 0.2 g.kg<sup>-1</sup> BM NaHCO<sub>3</sub> (Bicarbonate of Soda, Dr Oatker, United Kingdom) to determine TTP HCO<sub>3</sub><sup>-.</sup> Following ingestion of all vegetarian capsules (size 00, Bulk Powders, UK) within a 5 min period, a stopwatch was started, and participants remained seated for 120 min. Finger prick blood samples were collected at baseline in 70µl heparin-coated capillary tubes, following 30 min and subsequently every 15 min using a blood gas analyser (ABL9, Radiometer, Denmark) to analyse blood HCO<sub>3</sub>. TTP  $HCO_3^-$  was determined by the highest  $HCO_3^-$  value (absolute-peak) and this established the ingestion timing for each participant during experimental trials. Using a visual analogue scale (Miller et al., 2016), GI discomfort was recorded at baseline and every 15 min until participants reached their respective TTP HCO<sub>3</sub>. The scale was a 20 cm horizontal line that was anchored with "no symptom" to the left and "severe discomfort" to the right.

#### **Experimental procedures**

The following four separate visits involved the completion of a 4-km cycling TT, with one of those visits including a familiarisation trial on a Wattbike cycle ergometer (Nottingham, UK). All trials were conducted in an environmental chamber (Altitude Centre, UK) maintained at 30°C (~40% relative humidity). These conditions were selected, as these were the most commonly reported temperatures upon consultation with track cyclists known to the lead author during pilot work. Participants selected their preferred positions on the Wattbike (i.e. saddle and handlebar) and remained seated (10 min) to accustom to the conditions. Following an individualised warm-up that participants selected based on the knowledge they were preparing for a 4-km TT, participants then passively rested for 5 min and then performed the 4-km cycling TT as quickly as possible. The individualised warm-up was replicated for each experimental trial. This protocol was selected because of its ecological validity and reproducibility (Stone, Thomas, Wilkinson, Gibson, & Thompson, 2011), but also as it would cause sufficient disturbances in acid base balance (Gough, Deb, et al.,

2018), thus providing an appropriate protocol to examine the operating mechanisms of  $NaHCO_3$ .

During each trial, the Wattbike monitor was blinded from the participant, however, verbal encouragement and distance covered was provided. Time to complete (s), mean power (W), and speed  $(km.h^{-1})$  were recorded for the total 4-km distance. Heart rate (HR) (Polar Electro, Finland) and whole-body rating of perceived exertion (RPE) were recorded at rest and every 1 km (scale, 6-20; Borg, 1982). Tympanic temperature was recorded using an infrared tympanic thermometer (Braun ThermoScan<sup>®</sup> 7 IRT 6520, Braun GmbH, Kornberg, Germany) outside and 10 min in the chamber along with post warm-up and post exercise. Blood measures for acidbase balance (HCO<sub>3</sub>, and pH) were recorded at baseline, absolute-peak and post-exercise using a blood gas analyser (ABL9, Radiometer Medical Ltd., Denmark). Blood lactate (BLa<sup>-</sup>) was also measured (0.3 µl sample) using a portable lactate analyser (Lactate Pro 2, Arkray, Japan) at the same time points. Participants repeated this experimental trial three occasions apart from ingesting either: 0.2 g.kg<sup>-1</sup> BM NaHCO<sub>3</sub>, 0.2 g.kg<sup>-1</sup> BM sodium chloride (placebo; PLA) in vegetarian capsules (size 00, Bulk Powders, UK) or no supplementation (control; CON). Capsules were identical in appearance and the volume of water was recorded and replicated for any subsequent trial (mean =  $452 \pm 34$  ml). A laboratory technician that was not involved in the research had manually prepared and administered the supplementation of NaHCO<sub>3</sub> in a block-randomised order to ensure the study remained blinded. Participants remained seated following NaHCO<sub>3</sub> or PLA ingestion until their respective absolute-peak, with only water permitted *ab-libitum*. Once this was achieved, participants began their individualised warm-up. Lastly, GI discomfort was recorded every 15 min, as per the previously defined method. At the end of each experimental trial, a supplement belief questionnaire was used to assess the blinding efficacy, as per previous research (Gough, Brown, et al., 2018; Gough, Deb, et al., 2018; Gurton, Macrae, Gough, & King, 2021).

#### Statistical analysis

All data were assessed for normality using both Shapiro-Wilks tests and graphical methods including skewness and kurtosis. Homogeneity of variance/sphericity were analysed using Mauchly tests and any violations were corrected via Greenhouse-Geisser adjustments. The reproducibility of peak change in HCO<sub>3</sub><sup>-</sup> (TTP visit vs. NaHCO<sub>3</sub> treatment) and 4-km cycling TT performance (PLA vs CON treatments) was determined using Intraclass Correlation Coefficients (ICC's) and categorised as

poor ( $r = \le 0.40$ ), fair (r = 0.40-0.59), good (r = 0.60-0.74) or excellent ( $r = \ge 0.74$ ). One-way repeated measures ANOVA were conducted on 4-km cycling TT performance (completion time, mean power, mean speed). To determine individual changes in time to complete the smallest worthwhile change (SWC) statistic was used (0.2 \* between-subject standard deviation; SD) (Paton & Hopkins, 2006). Two-way repeated measures ANOVA were used to analyse blood metabolite responses (HCO<sub>3</sub><sup>-</sup>, pH, H<sup>+</sup> and BLa<sup>-</sup>), HR, tympanic temperature and RPE. Significant two-way interactions (treatment x time) were explored further by conducting post hoc testing with bonferroni correction factors. The assumption of normal distribution was violated for GI discomfort, therefore Wilcoxon matched-paired signed-rank tests were conducted as the non-parametric alternative, with median and z values reported. For ANOVA interactions and main effects, the effect size is reported as partial eta squared ( $\eta_p^2$ ). Between treatment effect sizes (g) were calculated by dividing mean difference by the pooled SD (Nakagawa & Cuthill, 2007) and applying Hedge's q bias correction to account for the small sample size (Lakens, 2013). The interpretations of these effect sizes were categorised as trivial ( $\leq 0.2$ ), small (0.2–0.49), moderate (0.5–0.79) or large (≥0.8) (Cohen, 1988). Effect sizes for non-normally distributed data (r) were calculated from  $z/\sqrt{n}$ , with 0.10, 0.24 and 0.37 considered as small, medium and large, respectively (Ivarsson, Andersen, Johnson, & Lindwall, 2013). Data are presented as Mean ± SD (unless stated otherwise) and 95% confidence intervals (CI) reported for differences in performance, with variances that do not cross the zero-boundary treated as significant (Gardner & Altman, 1986). Statistical significance was set at p < p0.05 and all statistical data were analysed using SPSS software version 26 (IBM, Chicago, IL, USA).

## Results

#### 4-km Cycling time trial performance

No trial order effect was reported from the performance data (completion time) (p = 0.972,  $\eta_p^2 = 0.003$ ). Completion time following NaHCO<sub>3</sub> was  $5.6 \pm 3.2$  s faster than PLA (1.6%; Cl: 2.8, 8.3; p = 0.001; g = 0.2) and  $4.7 \pm 2.8$  s faster than CON (1.3%; Cl: 2.3, 7.1; p = 0.001; g = 0.2). In total, six out of eleven participants reported "true" improvements following NaHCO<sub>3</sub> ingestion (i.e. change in performance vs. either CON or PLA above the SWC). Excellent reproducibility was observed for completion time between PLA and CON treatments (ICC = 0.998; p < 0.001). Mean and inter-individual variation for 4-km completion time is displayed in Figure 1



**Figure 1.** Mean  $\pm$  SD and inter-individual variation (dashed lines) for 4-km cycling TT completion times in the heat. \*\* denotes NaHCO<sub>3</sub> significantly faster (p < 0.05) compared to placebo (PLA) and control (CON).

and Table 1. Only two participants were able to identify the NaHCO<sub>3</sub> treatment correctly, with all other participants stating they "don't know".

Mean power across the 4-km distance following NaHCO<sub>3</sub> increased by  $10.0 \pm 7.1$  W compared to PLA (+3.6%; Cl: 3.9, 16.1; p = 0.003; g = 0.2) and by 6.7 ± 8.7 W compared to CON (+2.4%; Cl: -0.8, 14.3; p = 0.085; g = 0.1). Mean speed following NaHCO<sub>3</sub> was 0.6  $\pm 0.4$  km.h<sup>-1</sup> higher than PLA (+1.5%; Cl: 0.3, 1.0; p = 0.001; g = 0.2) and  $0.5 \pm 0.5$  km.h<sup>-1</sup> higher than CON (+1.1%; Cl: 0.1, 0.9; p = 0.025; g = 0.2).

**Table 1.** Inter-individual differences for changes in 4-km cycling TT performance and absolute increase in  $[HCO_3^-]$  from baseline to absolute peak.

	4-km TT performance (s)		Change in [HCO <sub>3</sub> ] (mmol.l <sup>-1</sup> )	
Participant	NaHCO₃ vs. PLA	NaHCO₃ vs. CON	NaHCO₃ vs. PLA	NaHCO₃ vs. CON
1	-6.7	-2.8	5.7	3.6
2	-11.0	-6.7	0.5	1.1
3	-4.2	-3.3	0.5	0.2
4	-7.3	-6.6	2.7	2.2
5	-10.7	-9.7	1.7	1.7
6	-5.8	-7.8	1.5	0.5
7	-4.0	-0.6	2.1	1.0
8	-3.5	-3.4	3.9	4.1
9	-0.5	-1.6	2.5	2.3
10	-3.4	-4.5	4.8	4.3
11	-4.2	-4.7	2.8	2.4

Abbreviations:  $HCO_3^-$ , blood bicarbonate; TT, time trial;  $NaHCO_3$ , sodium bicarbonate; *PLA*, placebo; *CON*, control. Changes in 4-km TT performance highlighted as bold text represent improvements that achieved the smallest worthwhile change (-4.7 s).

#### Blood metabolite response

During the preliminary TTP visit, absolute change in  $[HCO_3^-]$  was  $2.6 \pm 0.9 \text{ mmol.I}^{-1}$  (range,  $1.5-4.4 \text{ mmol.I}^{-1}$ ) and occurred between 30 and 105 min ( $62.7 \pm 19.0 \text{ min}$ ; CV = 30%). Absolute change in blood pH was  $0.04 \pm 0.02 \text{ AU}$  (range, 0.02-0.07 AU) and occurred between 30 and 105 min ( $69.5 \pm 21.5 \text{ min}$ ; CV = 31%). These changes in  $[HCO_3^-]$  from baseline to peak value displayed excellent reproducibility when compared to absolute change following NaHCO<sub>3</sub> during cycling trials (+2.6 mmol.I<sup>-1</sup>; ICC = 0.976; p < 0.001).

Significant treatment × time interactions were observed during the cycling trials for  $[HCO_3^-]$  (p < 0.001;  $\eta_p^2 = 0.628$ ), blood pH (p = 0.018;  $\eta_p^2 = 0.359$ ), [H<sup>+</sup>]  $(p = 0.022; \eta_p^2 = 0.350)$  and  $[BLa^-]$   $(p < 0.001; \eta_p^2 =$ 0.708). Absolute peak  $[HCO_3]$  prior to the 4-km cycling TT's was elevated for NaHCO<sub>3</sub> compared to PLA  $(+2.8 \text{ mmol.l}^{-1}; p = 0.002; q = 2.2)$ and CON (+2.5 mmol.l<sup>-1</sup>; p < 0.001; g = 2.1). Peak blood pH was also higher for NaHCO<sub>3</sub> compared to PLA (+0.048 AU; p = 0.007; q = 1.9) and CON (+0.061 AU; p = 0.055; q =1.1). Prior to the 4-km cycling TT's, [H<sup>+</sup>] was lower following NaHCO<sub>3</sub> compared to PLA (-4.5 nmol.l<sup>-1</sup>; p = 0.008; q = 1.8) and CON (-6.3 nmol.l<sup>-1</sup>; p = 0.099; q = 1.0). Inter-individual differences for the change in [HCO3-] from baseline to peak are displayed in Table 1. Absolute decline in  $[HCO_3^-]$  during the 4-km cycling TT's for NaHCO<sub>3</sub> was  $4.4 \pm 3.4$  mmol.l<sup>-1</sup> greater than PLA (p =0.005; g = 1.8) and  $4.1 \pm 2.1$  mmol.l<sup>-1</sup> greater than CON (p < 0.001; q = 2.2). The total H<sup>+</sup> efflux during the 4-km cycling TT's for NaHCO<sub>3</sub> was  $7.3 \pm 6.7$  nmol.l<sup>-1</sup> higher than PLA (p = 0.014; g = 0.8) and  $12.8 \pm 14.6$  nmol.l<sup>-1</sup> higher than CON (p = 0.047; q = 1.0). Post-exercise [HCO<sub>3</sub>] was lower following NaHCO<sub>3</sub> compared to PLA  $(-1.5 \text{ mmol.l}^{-1}; p = 0.046;$ *g* = 0.7) and CON  $(-1.6 \text{ mmol.I}^{-1}; p = 0.01; q = 0.9)$ . Peak [BLa<sup>-</sup>] after exercise was elevated following NaHCO<sub>3</sub> compared to PLA  $(+2.76 \text{ mmol.I}^{-1}; p = 0.001; g = 1.2)$ and CON  $(+2.53 \text{ mmol.}\text{I}^{-1}; p = 0.002; q = 1.1)$ . There were no differences at any time point between PLA and CON treatments (all p > 0.05). Mean ± SD for blood metabolite response is displayed in Figure 2 (A-D).

# *Heart rate, tympanic temperature and perceived exertion*

Significant treatment × time interactions were observed for HR (p = 0.036;  $\eta_p^2 = 0.196$ ), but not tympanic temperature (p = 0.892;  $\eta_p^2 = 0.036$ ) or RPE (p = 0.066;  $\eta_p^2 = 0.199$ ). At the 3-km segment, HR was elevated following NaHCO<sub>3</sub> compared to PLA (+5 b.min<sup>-1</sup>; p = 0.008; g =0.6) and CON (+4 b.min<sup>-1</sup>; p = 0.053; g = 0.5). Maximum HR at the end of the 4-km cycling TT was elevated following NaHCO<sub>3</sub> compared to PLA (+5 b.min<sup>-1</sup>; p = 0.027; g = 0.7) and CON (+5 b.min<sup>-1</sup>; p = 0.063; g = 0.5).

#### Gastrointestinal discomfort

Aggregate GI discomfort was exacerbated for NaHCO<sub>3</sub> compared to PLA (median, 16 vs. 2 cm) and displayed a large effect size (z = -2.807; p = 0.005; r = 0.8). Severity score for peak GI symptom was higher for NaHCO<sub>3</sub> compared to PLA (median, 5 vs. 1 cm) and displayed a large effect size (z = -2.670; p = 0.008; r = 0.8; Table 2).

#### Discussion

This study aimed to investigate the effects of NaHCO<sub>3</sub> ingestion on 4-km cycling TT performance in conditions of high ambient heat. In agreement with the hypothesis, this study reports, for the first time, that NaHCO<sub>3</sub> ingestion improves 4-km cycling TT performance in the heat. The current study findings are therefore more applicable to real-world track cycling, which are typically performed in higher temperatures than the thermoneutral conditions used in previous laboratory research. These findings also suggest that exercise of a similar intensity and duration might also benefit from the use of NaHCO<sub>3</sub> as an ergogenic strategy (e.g. track and field). Moreover, the current study is the first to demonstrate that an acute, lower dose of NaHCO<sub>3</sub> (0.2 g.kg<sup>-1</sup> BM) is a viable strategy to improve performance in high ambient heat conditions; therefore, corroborating previous findings in thermoneutral conditions (Gough, Brown, et al., 2018; Gough, Deb, et al., 2018). Based on GI discomfort typically being reduced from lower doses of NaHCO<sub>3</sub> (i.e. 0.2 g.kg<sup>-1</sup>) this might be a more attractive option in a practical setting compared to larger doses (i.e.  $0.3 \text{ g.kg}^{-1}$ ) (Gough et al., 2017; McNaughton, 1992). Further research is warranted investigating the use of NaHCO<sub>3</sub> ingestion during other exercise modalities that are often conducted in the heat (e.g. Olympic sports).

The current study supports previous research investigating NaHCO<sub>3</sub> ingestion in high ambient heat, whereby improvements in performance were observed during the second bout of repeated Wingate tests ( $2 \times 30$  s interspersed with 5 min active recovery) following chronic ingestion of 0.5 g.kg<sup>-1</sup> BM NaHCO<sub>3</sub> ingestion split into three equal doses across an eight hour period (Mündel, 2018). Whilst multiple differences in experimental approach between the authors and the current study exist (e.g. ingestion dose and timing, exercise mode and duration), the present study adds novel findings such that NaHCO<sub>3</sub> is ergogenic for longer



**Figure 2.** (A-D) Mean  $\pm$  SD blood metabolite response (A: Blood bicarbonate, HCO<sub>3</sub><sup>-</sup>; B: blood pH; C: Hydrogen cations, H<sup>+</sup>; D: Blood lactate, BLa<sup>-</sup>) from baseline to post-exercise. Some error bars have been removed for clarity. Symbols denote significant difference (p < 0.05): \* NaHCO<sub>3</sub> vs. placebo (PLA); \*\* NaHCO<sub>3</sub> vs. placebo (PLA) and control (CON).

duration exercise in the heat, and arguably within a more ecologically valid protocol (4-km cycling TT vs Wingate). The ergogenic effect in the current study was also more profound compared to the findings of (Mündel, 2018), which may be due to the sub-maximal intensity of the 4-km cycling TT compared to that of the supramaximal intensity Wingate protocol of Mündel (2018). This is due to the potential for supramaximal intensity exercise to cause a rapid decline in pH that

Table 2. Peak gastrointestinal (GI) discomfort severity score experienced by each participant during each experimental trial.

Participant	NaHCO <sub>3</sub>	PLA	CON
1	Belching (1)	Bloating (1)	Nil <i>(0)</i>
2	BUR (3)	AD (2)	Nil <i>(0)</i>
3	Belching (8)	Nil <i>(0)</i>	Nil <i>(0)</i>
4	Belching (7)	Nil <i>(0)</i>	Nil <i>(0)</i>
5	Vomiting (5)	Nausea (2)	Nil <i>(0)</i>
6	Nil (0)	Nil (0)	Nil <i>(0)</i>
7	Belching (10)	Vomiting (9)	Nil <i>(0)</i>
8	Belching (13)	AD (7)	Nil <i>(0)</i>
9	Diarrhoea (18)	AD (2)	AD (2)
10	Belching (4)	Nil (0)	Nil <i>(0)</i>
11	AD (4)	Nil (0)	Nil <i>(0)</i>

Abbreviations: *NaHCO*<sub>3</sub>, sodium bicarbonate; *PLA*, placebo; *CON*, control; *BUR*, bowel urgency rating; *AD*, abdominal discomfort. Symptom severity score (on a scale of 0–20) is displayed in parenthesis.

completely saturates the monocarboxylate transporter with H<sup>+</sup> and therefore leaving no capacity for NaHCO<sub>3</sub> to cause ergogenic effects (Messonnier, Kristensen, Juel, & Denis, 2007). Moreover, the current study offers novelty as a smaller, acute dose of  $0.2 \text{ g.kg}^{-1}$  BM NaHCO<sub>3</sub> was ergogenic despite mild GI discomfort. This corroborates with previous research reporting similar ergogenic effects of smaller NaHCO<sub>3</sub> doses during an identical exercise protocol in thermoneutral and hypoxic conditions (Gough et al., 2017; Gough et al., 2019; Gough, Brown, et al., 2018). It is likely that the use of the individualised time-to-peak HCO<sub>3</sub> strategy ensured the NaHCO<sub>3</sub> dose was ergogenic, as previous research using a standardised time point of ingestion (i.e. 60 min prior to exercise for all participants) has shown the 0.2 g.kg<sup>-1</sup> BM dose was not effective (McNaughton, 1992). Future studies should compare various ingestion strategies and doses to continue optimising the effectiveness of NaHCO<sub>3</sub> in environments of high ambient heat, whilst also striving to balance the side effects and performance benefits.

The ergogenic effect of NaHCO<sub>3</sub> ingestion on performance in high ambient heat corroborates with findings in thermoneutral environments (Gough, Deb,

et al., 2018), which is surprising given the additional anaerobic demand in the heat that would potentially suggest this supplement would be more ergogenic compared to thermoneutral conditions. Other research supplementing NaHCO<sub>3</sub> in hypoxia also display similar findings (Deb, Gough, Sparks, & McNaughton, 2017; Gough et al., 2019; Gough, Brown, et al., 2018), all of which are also more demanding of anaerobic energy production. It is also suggested that pre-exercise increases in plasma volume could be ergogenic in conditions of high ambient heat (Sims, Rehrer, Bell, & Cotter, 2007; Sims, van Vliet, Cotter, & Rehrer, 2007), to which this mechanism should be evident following alkalising agents (Vaher, Timpmann, Aedma, & Ööpik, 2015). Indeed, Vaher et al. (2015) reported following ingestion of sodium citrate (500 mg.kg<sup>-1</sup>) 120 min prior to exercise, plasma volume increased by around 4%. It might have been the short duration of the current studies exercise protocol that nullified any impact of plasma volume, however, as the current study and other published studies to date (Mündel, 2018) employing NaHCO<sub>3</sub> supplementation in the heat have ranged from between 30 s to 7 min, whereas this mechanism is more likely relevant to longer duration exercise. Future research could therefore investigate the effectiveness of NaHCO<sub>3</sub> ingestion in conditions of heat versus temperate conditions, whilst also using longer duration exercise protocols. This would help identify how far-reaching the ergogenic mechanisms of NaHCO<sub>3</sub> ingestion could be in conditions of high ambient heat.

The current study findings support previous work on cycling time trials performed in thermoneutral conditions that report ergogenic effects following NaHCO<sub>3</sub> ingestion (Gough, Brown, et al., 2018; Gough, Deb, et al., 2018; Gurton, Faulkner, et al., 2021; Hilton et al., 2020). The rise in extracellular acid base balance likely explains the improved performance following NaHCO<sub>3</sub> ingestion, as such increases are suggested to increase  $H^+$  efflux due to the upregulation of the lactate/ $H^+$ cotransporter, and subsequently more H<sup>+</sup> is buffered (Marx et al., 2002). These biochemical changes are suggested to offer protective effects to cross-bridge binding and Ca<sup>2+</sup> handling (Fitts, 2008, 2016). Support for this can be found from both the significantly increased absolute decline in HCO<sub>3</sub> during the 4-km cycling TT (i.e. greater amount of extracellular buffering) and the elevated post-exercise lactate for the NaHCO<sub>3</sub> versus PLA treatment, which are both indicative of a higher exercise intensity following NaHCO<sub>3</sub> ingestion. Equally, the  $H^+$  analysis in the current study corroborates this notion, as this metabolite was lower pre-exercise in the NaHCO<sub>3</sub> condition vs. PLA/ CON, but was the same at the post-exercise time point,

thus inferring a greater  $H^+$  efflux from muscle during the 4-km cycling TT. These findings of the current study must be interpreted with caution as the measurements were only extracellular and therefore, we cannot account for changes at the muscular level. Nonetheless, NaHCO<sub>3</sub> ingestion likely improves buffering capacity and will lead to improved exercise performance in most participants.

The increase in  $HCO_3^-$  from baseline to absolute peak NaHCO<sub>3</sub> ingestion were low  $(2.6 \pm$ following  $0.9 \text{ mmol.l}^{-1}$ ) in comparison to the typical average increase in previous research (Gough et al., 2017; Jones et al., 2016; Renfree, 2007; Siegler, Midgley, Polman, & Lever, 2010). Indeed, Gough et al. (2017) reported the mean increase of  $HCO_3^-$  following 0.2 g.kg<sup>-1</sup> BM NaHCO<sub>3</sub> was 5.7  $\pm$  0.9 and 5.6  $\pm$  1.1 mmol.l<sup>-1</sup> in repeated trials, with 13 out of 15 participants achieving >5 mmol.l<sup>-1</sup>. In fact, in the current study not a single participant reached the 5 mmol.l<sup>-1</sup> increase from baseline to peak  $HCO_3^-$  that is a purported threshold to ensure the ergogenic effect from NaHCO<sub>3</sub> ingestion (Carr, Hopkins, & Gore, 2011). Despite this, a significant improvement in performance was reported in this study, and in particular, three participants displayed increases in  $HCO_3^-$  of  $\leq 2 \text{ mmol.I}^{-1}$ , however, improved their performance above the SWC of the test protocol. Similarly, one participant reported a 4.5 mmol. $I^{-1}$ increase in HCO<sub>3</sub>, however did not improve their performance above the SWC. These findings subsequently question the requirement to ingest such large doses of NaHCO<sub>3</sub> (Gough et al., 2019), as reaching a threshold increase in  $HCO_3^-$  does not seem as important as previous research suggests (Jones et al., 2016; Farias de Oliveira et al., 2020). The reason why such low increases in  $HCO_3^-$  were observed might be due to the known inter-individual variation in HCO<sub>3</sub><sup>-</sup> changes following NaHCO<sub>3</sub> supplementation, and that the current study employed a lower dose of NaHCO<sub>3</sub> (Gough et al., 2017; Jones et al., 2016). Future research should attempt to explore reasons why such a large inter-individual variation in bicarbonate uptake occurs (i.e. preexercise procedures, training status, genetics), and how this uptake relates to the resulting performance effect.

## Conclusion

The ingestion of  $0.2 \text{ g.kg}^{-1}$  BM NaHCO<sub>3</sub> is a suitable ergogenic aid for improving 4-km TT cycling performance in ambient heat. Athletes and coaches can therefore employ this strategy with a degree of confidence that an ergogenic effect will be observed. Lower doses of NaHCO<sub>3</sub> may also be attractive due to the lower sodium load and the potential to reduce GI discomfort,

compared to the most commonly ingested  $0.3 \text{ g.kg}^{-1}$ BM NaHCO<sub>3</sub> dose. Future research should explore the use of NaHCO<sub>3</sub> in high ambient heat conditions in a range of other competitive events and/or training bouts.

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