

# Effect of Warm-Up and Sodium Bicarbonate Ingestion on 4-km Cycling Time-Trial Performance

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Purpose: To examine whether an ecologically valid, intermittent, sprint-based warm-up strategy impacted the ergogenic capacity of individualized sodium bicarbonate (NaHCO<sub>3</sub>) ingestion on 4-km cycling time-trial (TT) performance. *Methods*: A total of 8 male cyclists attended 6 laboratory visits for familiarization, determination of time to peak blood bicarbonate, and 4×4-km cycling TTs. Experimental beverages were administered doubleblind. Treatments were conducted in a block-randomized, crossover order: intermittent warm-up + NaHCO<sub>3</sub> (IWSB), intermittent warm-up + placebo, control warm-up + NaHCO<sub>3</sub> (CWSB), and control warm-up+placebo (CWP). The intermittent warm-up comprised exercise corresponding to lactate threshold (5 min at 50%, 2 min at 60%, 2 min at 80%, 1 min at 100%, and 2 min at 50%) and  $3 \times 10$ -second maximal sprints. The control warm-up comprised 16.5 minutes cycling at 150 W. Participants ingested 0.3 g·kg body mass<sup>-1</sup> NaHCO<sub>3</sub> or 0.03 g·kg body mass<sup>-1</sup> sodium chloride (placebo) in 5 mL·kg body mass<sup>-1</sup> fluid (3:2, water and sugar-free orange squash). Paired t tests were conducted for TT performance. Hematological data (blood bicarbonate and blood lactate) and gastrointestinal discomfort were analyzed using repeated-measures analysis of variance. Results: Performance was faster for CWSB versus IWSB (5.0 [6.1] s; P = .052) and CWP (5.8 [6.0] s; P = .03). Pre-TT bicarbonate concentration was elevated for CWSB versus IWSB  $(+9.3 \text{ mmol}\cdot\text{L}^{-1}; P < .001)$  and CWP  $(+7.1 \text{ mmol}\cdot\text{L}^{-1}; P < .001)$ . Post-TT blood lactate concentration was elevated for CWSB versus CWP ( $\pm 2.52$  mmol·L<sup>-1</sup>; P = .022). Belching was exacerbated pre-warm-up for IWSB versus intermittent warm-up + placebo (P = .046) and CWP (P = .027). Conclusion: An intermittent, sprint-based warm-up mitigated the ergogenic benefits of NaHCO<sub>3</sub> ingestion on 4-km cycling TT performance.

Keywords: buffering, alkalosis, metabolic perturbation, sprints, ergogenic aid

Competitive cycling time-trial (TT) events such as the individual pursuit require athletes to almost maximally exert themselves for short durations (~5 min). The substantial anaerobic energy demand results in the accumulation of metabolites including inorganic phosphate, hydrogen ions (H<sup>+</sup>), and lactate. Extracellular buffering mechanisms act to remove these H<sup>+</sup> from the skeletal muscle cell, but once production rates overwhelm neutralization reactions, the excess H<sup>+</sup> contribute toward decreasing intramuscular pH.<sup>2</sup> Exercise-induced acidosis inhibits glycolytic energy production and disturbs calcium ion cross-bridge formation, high-intensity exercise. Strategies that protect against these biochemical disturbances could therefore be vital to optimizing exercise performance.

Various extracellular buffering agents exist that elicit a metabolic alkalosis which improves the capacity to buffer H<sup>+</sup> during high-intensity exercise. Perhaps the most well-established and extensively researched is sodium bicarbonate (NaHCO<sub>3</sub>).<sup>4</sup> This nutritional supplement enhances the extracellular buffering response by elevating circulating blood bicarbonate (HCO<sub>3</sub><sup>-</sup>) ~5 to 6 mmol·L<sup>-1</sup> above baseline,<sup>4</sup> which promotes greater efflux of H<sup>+</sup> from the muscle, in turn protecting against declining intramuscular pH.<sup>5</sup> NaHCO<sub>3</sub> ingestion also elevates strong ion difference (SID) by ~15%,<sup>6,7</sup> subsequently allowing for sustained muscle

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excitability during strenuous exercise. Since there is no singular explanation for performance-enhancing effects, authors should adopt a multifaceted perspective when examining physiological mechanisms associated with NaHCO<sub>3</sub> ingestion.

Sodium bicarbonate has historically been administered as a 0.3-g·kg body mass<sup>-1</sup> (BM) dose at 60 to 90 minutes preexercise, which may elicit moderate improvements to high-intensity exercise performance.<sup>4</sup> Some authors have reported no effect (≤0.5%) of NaHCO<sub>3</sub> on 4-km cycling TT performance, 8,9 although this was attributed to their failure to account for interindividual variability in HCO<sub>3</sub><sup>-</sup> absorption rates. Athletes are recommended to align NaHCO<sub>3</sub> timing with individualized time-to-peak HCO<sub>3</sub> kinetics, ensuring that peak changes in HCO<sub>3</sub><sup>-</sup> occur immediately preexercise, 10,11 thus maximizing HCO<sub>3</sub> buffering capacity. Individualized NaHCO3 ingestion has previously increased work during repeated sprints (+10.7%)<sup>11</sup> and improved 4-km TT completion times (~8 s).<sup>10,12</sup> Considering that time-to-peak HCO<sub>3</sub><sup>-</sup> varies considerably between athletes, ranging from 40 to 120 minutes depending on administration method (solution vs capsule), 10,13 research should opt for individualized NaHCO3 ingestion to maximize ergogenic potential.

Most studies examining the effect of NaHCO<sub>3</sub> on high-intensity cycling performance provided participants with steady-state warm-ups<sup>9–12</sup> that are unlikely to have replicated metabolic perturbation experienced during warm-up strategies preceding competition. Kilding et al<sup>14</sup> suggested that an intermittent cycling warm-up (20 min at 60%–65% maximal aerobic power; 5×20-s sprints) decreased HCO<sub>3</sub><sup>-</sup> by ~5 mmol·L<sup>-1</sup> from baseline in the placebo trial with only a small increase (+3.7 mmol·L<sup>-1</sup>) reported pre-TT after NaHCO<sub>3</sub> ingestion. Other authors employing sport-specific warm-up strategies observed no effect of NaHCO<sub>3</sub> on

sprint time during water polo (+0.4%; P=.51) and rugby (P>.05) tests. <sup>15,16</sup> As these studies failed to examine differences in acidbase balance between pre-warm-up and post-warm-up, it is difficult to determine the extent to which warm-up strategy impacted upon  $HCO_3^-$  response or may have altered ergogenic capacity. Further investigation is warranted to compare the effect of different warm-up strategies on changes in acid-base balance and performance benefits.

Elite cyclists complete intermittent warm-ups, including bouts of sustained high-intensity and maximal sprints.<sup>17</sup> These exercise bouts result in the accumulation of H<sup>+</sup> within the muscle,<sup>1,3</sup> potentially utilizing the enhanced buffering response prior to competition. To date, no research has investigated whether these metabolic perturbations negatively impact the efficacy of NaHCO<sub>3</sub> ingestion. Therefore, the aim of this study was to examine the effect of an ecologically valid, intermittent, sprint-based warm-up, and individualized NaHCO<sub>3</sub> ingestion on 4-km cycling TT performance in cyclists.

#### Methods

#### **Participants**

A total of 10 club-level male cyclists (1.82 [0.05] m; 73.3 [6.6] kg; 54.8 [5.1] mL·kg·min<sup>-1</sup>; 23 [7] y) volunteered for this study (due to global pandemic only 8 completed). All participants were categorized as either recreationally trained or trained cyclists and performed >4 hours of cycling-based training per week, had cycled for >2 years, and had not ingested buffering agents in the previous 6 months. Ethical approval was gained from the human ethics committee at Nottingham Trent University. Participants signed informed consent prior to data collection with research conducted in accordance with the Revised Declaration of Helsinki 2013.

#### **Experimental Design**

A block randomized, double-blind, placebo-controlled, crossover experimental design was employed for this study. Participants attended 6 separate laboratory visits to perform a graded exercise test and protocol familiarization, determination of time-to-peak HCO<sub>3</sub><sup>-</sup>, and 4×4-km cycling TTs. Participants performed testing at the same time of day (±2 h) and in a 2-hour postprandial state to minimize the confounding effects of circadian rhythms<sup>19</sup> and nutrition on exercise performance. Vigorous exercise and the consumption of alcohol were prohibited for 24 hours prior to all visits. Pretrial nutrition and exercise were replicated for 24 hours prior to experimental trials (checked via visual logs). Participants completed profile of mood states<sup>20</sup> and Pittsburgh Sleep Qual<sup>21</sup> questionnaires to calculate total mood disturbance and global sleep quality index.

#### **Graded Exercise Test and Familiarization**

Participants completed a graded exercise test on their own bike mounted to an online cycling system (Cyclus2; RBM elektronikautomation GmbH, Leipzig, Germany). Baseline capillary blood samples were collected into 20-µL sodium-heparized capillary tubes and analyzed for blood lactate (BLa¯) using the Biosen C-Line ((EKF Diagnostic GmbH, Barleben, Germany). The protocol commenced at 95 W and increased by 35 W every 3 minutes. Heart rate and blood samples were taken at the end of each stage until [BLa¯] exceeded 4.0 mmol·L¯¹, at which point only heart rate was recorded until volitional exhaustion. This was classified by the failure to maintain

self-selected cadence (80 [7] rev·min $^{-1}$ ) despite strong verbal encouragement. Gaseous exchange was collected throughout using a breath-by-breath metabolic analyzer (Vyntus CPX; CareFusion GmbH, Hoechberg, Germany). The power output at lactate threshold (4.0 mmol·L $^{-1}$ ) was used to prescribe the intermittent warm-up strategy.

Participants were familiarized to exercise protocols with 10-minute complete rest seated on a chair between the intermittent warm-up and 4-km cycling TT. This reflects real-life time lapse in elite competition (personal experience of S.H.F.). The intermittent warm-up comprised exercise corresponding to lactate threshold (5 min at 50%, 2 min at 60%, 2 min at 80%, 1 min at 100%, and 2 min at 50%) and 3 × 10-second maximal sprints interspersed with 90-second recovery. All exercise was completed on the participants' own bike. Participants selected frame geometry and gear ratios, which were replicated during experimental trials. Participants were provided with feedback on distance covered and cadence, but elapsed time was blinded.

### Determination of Time-to-Peak HCO<sub>3</sub><sup>-</sup>

The second laboratory visit was conducted to identify time-to-peak HCO<sub>3</sub><sup>-</sup> following the ingestion of 0.3 g·kg BM<sup>-1</sup> NaHCO<sub>3</sub>. Beverages were administered in 5 mL·kg BM<sup>-1</sup> fluid (3:2, water and sugar-free orange squash) and consumed within 5 minutes. Capillary blood samples were taken prior to NaHCO<sub>3</sub> ingestion and collected into 70-μL heparin-coated capillary tubes for analysis of HCO<sub>3</sub><sup>-</sup> using a blood gas analyzer (ABL90 FLEX; Radiometer Medical Ltd, Copenhagen, Denmark). Blood samples were taken every 20 minutes until 60 minutes postingestion, and every 10 minutes between 60 and 120 minutes to determine time to peak HCO<sub>3</sub><sup>-</sup>.

#### **Experimental Trials**

Participants attended 4 laboratory visits performing the intermittent or a control warm-up prior to 4-km cycling TTs. The control warmup comprised cycling at 150 W for 16.5 minutes (matched duration to intermittent warm-up). NaHCO<sub>3</sub> or 0.03 g kg<sup>-1</sup> sodium chloride (placebo) were administered doubleblind in 5 mL·kg BM<sup>-1</sup> fluid (3:2, water and sugar-free orange squash). Experimental trials were conducted in a randomized order: intermittent warm-up + NaHCO<sub>3</sub> (IWSB), intermittent warm-up + placebo (IWP), control warm-up + NaHCO<sub>3</sub> (CWSB), and control warm-up + placebo (CWP). Supplement belief questionnaires were completed postingestion to assess perception of experimental beverages. 10,22 Capillary blood samples were taken at baseline, pre-warm-up, pre-TT, post-TT, and 5 minutes post for the analysis of HCO<sub>3</sub><sup>-</sup>, pH, BLa<sup>-</sup>, and electrolytes including sodium (Na<sup>+</sup>), potassium (K<sup>+</sup>), chloride (Cl<sup>-</sup>), and calcium (Ca<sup>2+</sup>). These were inputted into a freely available spreadsheet to calculate apparent SID:  $[K^+] + [Na^+] + [Ca^{2+}] + [Mg^{2+}] -$ [Cl] - [BLa].<sup>23</sup> Visual analog scales (0 mm = "no symptom"; 100 mm = "severest symptom") were completed at baseline, prewarm-up, pre-TT, and post-TT to measure gastrointestinal (GI) discomfort.<sup>22</sup> The start time of the warm-up varied to ensure TTs commenced at the point coinciding with time-to-peak HCO<sub>3</sub><sup>-</sup>. Participants were instructed to complete each 4-km TT as fast as possible.

#### Statistical Analysis

Normality and sphericity were assessed using Shapiro-Wilk and Mauchly tests, respectively. Reproducibility of pretrial nutrition,

total mood disturbance, and global sleep quality index were categorized using intraclass correlation coefficients as poor  $(r \le$ .40), fair (r = .40 - .59), good (r = .60 - .74), or excellent  $(r \ge .74)$ . Paired t tests were conducted on TT performance to assess the effect of NaHCO<sub>3</sub> (CWSB vs CWP), the effect of intermittent warm-up (CWSB vs IWSB), and the combined effects (ΔCWSB/ CWP vs \( \Delta \text{IWSB/IWP} \)). This statistical approach was considered more appropriate than 1-way repeated-measures analysis of variance (ANOVA) due to our a priori hypothesis (ie, no interest in comparing IWSB vs CWP or IWP vs CWSB). Bonferroni corrections were performed to minimize the risk of bias due to type I error following multiple tests.<sup>25</sup> The smallest worthwhile change in 4-km TT performance (4.4 s) was calculated as 0.3 × the interindividual SD for 4-km TT completion time during familiarization.<sup>26</sup> Between-treatment effect sizes were calculated by dividing mean differences by pooled SD, before applying Hedges g bias correction.<sup>27</sup> These were interpreted as trivial (<0.20), small (0.20– 0.49), moderate (0.50–0.79), or large ( $\geq$ 0.80). Hematological data were analyzed using 2-way (treatment × time) repeated-measures ANOVA. Significant interactions were explored by performing 1-way repeated-measures ANOVA across treatments at each time point with Bonferroni correction factors applied. Friedman 2-way ANOVA was conducted for GI discomfort data. Post hoc Wilcoxon matched-pair signed-rank tests were performed when significance was observed, with median and z score reported. Data are presented as mean (SD) and 95% confidence intervals (CIs) reported for differences in performance. Statistical significance was set at P < .05 (adjusted to P < .017 for TT performance following Bonferroni correction) and data analyzed using SPSS (version 26; SPSS Inc, Chicago, IL).

#### Results

#### **Preexperimental Phase**

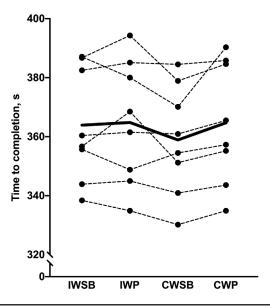
Nutritional intake prior to experimental trials displayed excellent reproducibility for calories (r=.94; P<.001), carbohydrate (r=.94; P<.001), fat (r=.96; P<.001), and protein (r=.98; P<.001). Excellent reproducibility was reported for total mood disturbance (r=.94; P<.001) and global sleep quality index (r=.95 P<.001) across experimental trials.

#### **TT Performance**

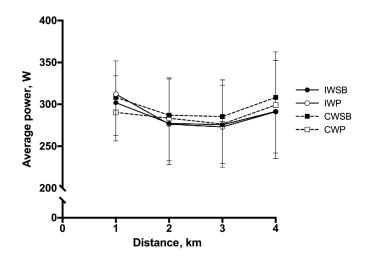
Mean and interindividual variation for 4-km TT completion times are displayed in Figure 1. Completion time was 5.8 seconds faster for CSWB versus CWP (95% CI, 0.7–10.8; P = .03) and displayed a large effect size (g = 0.89). Completion time was 5.0 seconds faster for CWSB versus IWSB (95% CI, 0.7–10.1; P = .052) and displayed a moderate effect size (g = 0.76). Six participants reported their fastest 4-km completion time for CWSB. Four participants improved above the smallest worthwhile change (>4.4 s) for CWSB compared with both CWP and IWSB. A small effect size was displayed for 4-km completion time for ΔCWSB/CWP versus  $\Delta$ IWSB/IWP (-4.9 s; g = 0.42; P = .233). Large effect sizes were reported for completion time during the first kilometer segment for CWSB versus CWP (-2.2 s; g = 0.92; P = .025), during the second kilometer segment for CWSB versus IWSB (-1.5 s; g =0.81; P = .042), and during the final kilometer segment for CWSB versus IWSB (-1.9 s; g = 1.03; P = .018).

Average power across the 4-km distance increased +9.9 W for CWSB versus CWP (95% CI, 0.1–19.8; P = .049) and displayed a

moderate effect size (g=0.77). Average power across the 4-km distance increased +10.7 W for CWSB versus IWSB (95% CI, 2.7–18.6; P=.016) and displayed a large effect size (g=1.04). In total, 6 participants reported their greatest average power for CWSB. A small effect size was displayed for average power across the 4-km distance for  $\Delta$ CWSB/CWP versus  $\Delta$ IWSB/IWP (+11.5 W; g=0.47; P=.192). Moderate effect sizes were reported for average power for CWSB versus IWSB during the second kilometer (+9.7 W; g=0.79; P=.046) and the third kilometer segments (+16.8 W; g=0.70; P=.029). The pacing per kilometer data for average power during the 4-km TTs are displayed in Figure 2.



**Figure 1** — Mean differences (heavily bolded line) and interindividual variation for 4-km cycling TT performance. CWP indicates control warm-up+placebo; CWSB, control warm-up+sodium bicarbonate; IWP, intermittent warm-up+placebo; IWSB, intermittent warm-up+sodium bicarbonate; TT, time trial.



**Figure 2** — Pacing per kilometer for average power during the 4-km cycling TTs. Data are presented as mean (SD). Some error bars removed for clarity. CWP indicates control warm-up+placebo; CWSB, control warm-up+sodium bicarbonate; IWP, intermittent warm-up+placebo; IWSB, intermittent warm-up+sodium bicarbonate; TT, time trial.

#### **Hematological Data**

Significant treatment×time interactions were observed for [HCO<sub>3</sub><sup>-</sup>] (P<.001;  $\eta_p^2$  = .877) and blood pH (P<.001;  $\eta_p^2$  = .774). Pre-warm-up [HCO<sub>3</sub><sup>-</sup>] and blood pH were elevated for NaHCO<sub>3</sub> versus placebo conditions (~6.2 mmol·L<sup>-1</sup>; ~0.060 AU). Participants only entered the TT in an alkalotic state following CWSB with pre-TT [HCO<sub>3</sub><sup>-</sup>] and blood pH greater for CWSB versus IWSB (+9.3 mmol·L<sup>-1</sup>; +0.060 AU), IWP (+15.3 mmol·L<sup>-1</sup>; +0.064 AU), and CWP (+7.1 mmol·L<sup>-1</sup>; +0.056 AU). Absolute decline in [HCO<sub>3</sub><sup>-</sup>] and blood pH during the TT were greater for CWSB versus IWSB (-9.6 mmol·L<sup>-1</sup>; -0.108 AU). Post-TT [HCO<sub>3</sub><sup>-</sup>] and blood pH were elevated for NaHCO<sub>3</sub> versus placebo conditions (~2.9 mmol·L<sup>-1</sup>; ~0.067 AU). At 5 minutes post, [HCO<sub>3</sub><sup>-</sup>] and blood pH were elevated for NaHCO<sub>3</sub> versus placebo conditions (~3.3 mmol·L<sup>-1</sup>; ~0.078 AU). Mean (SD) for [HCO<sub>3</sub><sup>-</sup>] and blood pH response are displayed in Figure 3A and 3B.

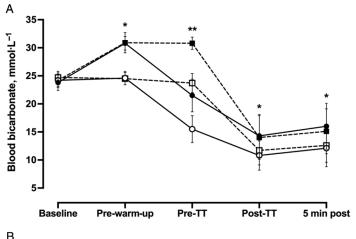
Significant treatment x time interactions were observed for [BLa]  $(P < .001; \eta_p^2 = .877)$  and SID  $(P < .001; \eta_p^2 = .826)$ . Pre-warm-up SID was elevated for NaHCO3 versus placebo conditions ( $\sim$ 7.0 meq·L<sup>-1</sup>). The intermittent warm-ups elicited a rise in [BLa<sup>-</sup>] compared with the control warm-ups ( $\sim$ 6.75 mmol·L<sup>-1</sup>). Extracellular ionic disturbances were only present prior to the TT following CWSB with pre-TT SID greater for CWSB versus IWSB  $(+7.0 \text{ meg} \cdot \text{L}^{-1})$ , IWP  $(+14.0 \text{ meg} \cdot \text{L}^{-1})$ , and CWP  $(+7.9 \text{ meg} \cdot \text{L}^{-1})$ . Absolute increase in [BLa] during the TT was greater for CWSB versus IWSB (+7.05 mmol·L<sup>-1</sup>). Post-TT [BLa<sup>-</sup>] was greater for CWSB versus CWP (+2.52 mmol·L<sup>-1</sup>). Post-TT SID was elevated for NaHCO<sub>3</sub> versus placebo conditions (~4.8 meq·L<sup>-1</sup>). At 5 minutes post, [BLa] was greater for CWSB versus IWP  $(+1.80 \text{ mmol} \cdot \text{L}^{-1})$  and CWP  $(+2.39 \text{ mmol} \cdot \text{L}^{-1})$ . At 5 minutes post, SID was elevated for NaHCO3 versus placebo conditions (~4.9 meq·L<sup>-1</sup>). Mean (SD) for [BLa<sup>-</sup>] and SID response are displayed in Figure 4A and 4B.

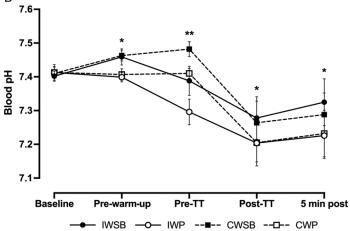
#### **Perceptual Responses**

Four participants identified all experimental beverages, whereas 4 were unable to consistently distinguish between NaHCO<sub>3</sub> and placebo. A total o participants experienced some GI discomfort, with the most severe symptoms experienced by each participant during each experimental trial shown in Table 1. No treatment effects were observed for GI discomfort at baseline, pre-TT, or post-TT (all Ps > .05). Pre-warm-up belching was exacerbated for IWSB versus IWP (3.5 vs 0 mm; z = -1.997; P = .046) and CWP (3.5 vs 0 mm; z = -2.207; P = .027). Aggregate GI discomfort scores revealed mild symptom severity pre-warm-up for IWSB (39 [49] mm) and CWSB (16 [30] mm), but not at pre-TT. All participants reported that GI discomfort did not negatively impact their performance.

# **Discussion**

This study was the first to examine the effect of an ecologically valid, intermittent, sprint-based warm-up, and individualized NaHCO $_3$  ingestion on 4-km cycling TT performance in trained cyclists. Our novel findings were that time to completion and average power displayed moderate-to-large improvements for CWSB only with almost no change in performance for IWSB. The small-to-moderate combined effects on performance in favor of NaHCO $_3$  ingestion ( $\Delta$ CWSB/CWP vs  $\Delta$ IWSB/IWP) suggest that the intermittent warm-up dampened the ergogenic capacity of



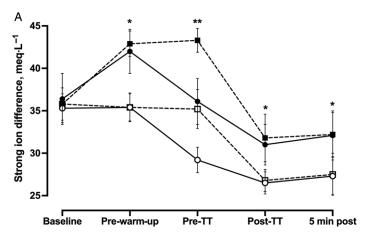


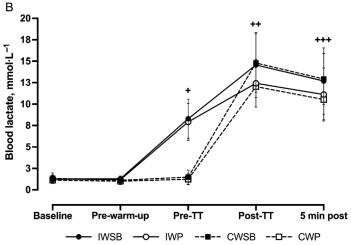
**Figure 3** — Mean (SD) for blood bicarbonate (A) and pH (B) response from baseline to 5 minutes post. Symbols denote significant difference (*P* < .05): \*IWSB and CWSB versus IWP and CWP; \*\*CWSB versus IWSB, IWP, and CWP. CWP indicates control warm-up+placebo; CWSB, control warm-up+sodium bicarbonate; IWP, intermittent warm-up+placebo; IWSB, intermittent warm-up+sodium bicarbonate; TT, time trial.

NaHCO<sub>3</sub> ingestion. Elevated acid–base balance (HCO<sub>3</sub><sup>-</sup>, pH), increased preexercise SID, and greater postexercise BLa<sup>-</sup> offer explanations for performance benefits. NaHCO<sub>3</sub> ingestion resulted in mild GI discomfort pre-warm-up; however, these symptoms were typically reduced prior to the 4-km cycling TT.

Improvements in time to completion (5.8 s) and average power (9.9 W) for 4-km cycling TTs were observed during CWSB compared with CWP. These results are consistent with previous findings reporting improved 4-km cycling TT performance following 0.3 g·kg BM<sup>-1</sup> NaHCO<sub>3</sub> compared with placebo conditions. One was some variation in performance responses with 4 participants improving above the smallest worthwhile change (>4.4 s), whereas 3 participants only experienced trivial improvements (<3.0 s). Several studies have reported no mean differences in 4-km cycling TT performance following NaHCO<sub>3</sub> ingestion, of although benefits might not occur consistently unless absolute change in HCO<sub>3</sub><sup>-</sup> reaches a 6.0-mmol·L<sup>-1</sup> "zone of erogeneity" threshold. These authors of failure to adopt a time-to-peak HCO<sub>3</sub><sup>-</sup> strategy likely prevented participants from achieving peak alkalosis immediately pre-TT, theoretically

dampening the ergogenic potential. This concept has recently been challenged with de Oliveira et al<sup>28</sup> claiming that a long-lasting window of ergogenic potential (+6.0 mmol·L<sup>-1</sup>; 90–225 min) exists following capsule NaHCO<sub>3</sub> ingestion. An individualized





**Figure 4** — Mean (SD) for blood lactate (B) and strong ion difference (A) response from baseline to 5 minutes post. Symbols denote difference (*P* < .05): \*IWSB and CWSB versus IWP and CWP; \*\*CWSB versus IWSB, IWP, and CWP; <sup>+</sup>IWSB and IWP versus CWSB and CWP; <sup>++</sup>CWSB versus CWP; and CWP; CWSB versus IWP and CWP. CWP indicates control warm-up + placebo; CWSB, control warm-up + sodium bicarbonate; IWP, intermittent warm-up + placebo; IWSB, intermittent warm-up + sodium bicarbonate; TT, time trial.

approach is most important for solution administration as a large proportion of  $HCO_3^-$  is lost from the neutralization of gastric acid<sup>13</sup> and for smaller doses that display shorter peak ergogenic potential.<sup>4</sup> Future research should refine practical application of NaHCO $_3$  supplementation by comparing ergogenic benefits between ingestion strategies (solution vs capsule) and timing protocols (standardized vs time to peak), and examining factors that may account for interindividual variation in performance responses.

The most practically significant finding of our study was that warm-up strategy impacts the efficacy of individualized NaHCO<sub>3</sub> ingestion. Improvements in 4-km time to completion (5.0 s) and average power (10.7 W) for CWSB compared with IWSB displayed moderate-to-large effect sizes. Moreover, the small-tomoderate combined effects on performance in favor of NaHCO<sub>3</sub> ingestion (ΔCWSB/CWP vs ΔIWSB/IWP) confirm that the intermittent, sprint-based warm-up mitigated the ergogenic effect of NaHCO<sub>3</sub> ingestion. These results are similar to studies employing sport-specific warm-up strategies<sup>8,14–16</sup> and can be attributed to differences in pre-TT metabolic perturbation, primarily as the sprint efforts during the intermittent warm-up would have resulted in greater accumulation of H<sup>+</sup> within the muscle.<sup>1,3</sup> Considering that HCO<sub>3</sub><sup>-</sup> buffering mechanisms are partly responsible for the removal of these H<sup>+</sup> into extracellular compartments,<sup>5</sup> the enhanced buffering response will have been partially utilized pre-TT, thus dampening the ergogenic potential of NaHCO<sub>3</sub> ingestion. From an applied standpoint, these results advocate that practitioners adapt their warm-up regimes to ensure ergogenic benefits following NaHCO3 supplementation. Since the intermittent, sprint-based warm-up alone had no effect on 4-km cycling TT performance, it is recommended that practitioners adopt evidencebased nutritional practices when designing prerace strategies, as these may prove more beneficial to overall performance. The current intermittent warm-up reflects prerace programs for individual pursuit events; however, cyclists competing in maximal, shorter sprint races (~1 min) may adopt warm-up strategies that further exacerbate precompetition metabolic perturbation. Additional work is required to examine the impact of these sprint warmup strategies on the efficacy of NaHCO<sub>3</sub> during "all-out" sprint exercise.

Disturbances in acid–base balance ( $HCO_3^-$ , pH) and increased postexercise  $BLa^-$  offer mechanistic insight to explain improved TT performance. Absolute change in  $HCO_3^-$  from baseline to pre-TT (+6.6 mmol· $L^{-1}$ ) was above the suggested 6.0 mmol· $L^{-1}$  threshold<sup>4</sup> and greater than increases from previous studies (~3.0–5.0 mmol· $L^{-1}$ ) reporting no performance benefits.<sup>8,9</sup> Participants only entered the TT in an alkalotic state during CWSB, but

Table 1 Severest GI Symptoms Experienced by Each Participant During Each Experimental Trial

Participant	IWSB	IWP	CWSB	CWP
1	<b>AD</b> (21)	Flatulence (3)	Nausea (11)	GF (3)
2	GF (5)	Flatulence (2)	Diarrhea (45)	GF (2)
3	Belching (25)	Nausea (4)	Belching (5)	Nil (0)
4	Nausea (22)	Nausea (20)	Belching (23)	Nausea (24)
5	GF (3)	<b>AD</b> (5)	GF (3)	Belching (1)
6	Vomiting (21)	Nil (0)	Nil (0)	Nil (0)
7	<b>BUR</b> (67)	Nil (0)	Diarrhea (21)	Nil (0)
8	Nil (0)	Nil (0)	Nil (0)	Nil (0)

Abbreviations: AD, abdominal discomfort; BUR, bowel urgency rating; CWP, control warm-up+placebo; CWSB, control warm-up+sodium bicarbonate; GF, gut fullness; IWP, intermittent warm-up+placebo; IWSB, intermittent warm-up+sodium bicarbonate. Note: Severest symptom for each participant is highlighted in bold; symptom severity score (on a scale of 0–100) is displayed in parenthesis.

differences in absolute decline from pre-TT to post-TT are equally significant, as these infer whether an enhanced buffering response was present during exercise. The absolute decline in HCO<sub>3</sub> and pH was substantially higher for CWSB versus IWSB, confirming that enhanced buffering capacity was utilized during the intermittent, sprint-based warm-up. The induced alkalosis also likely prevented the allosteric inhibition of phosphofructokinase and glycogen phosphorylase, in turn upregulating glycolytic activation.<sup>29</sup> Post-TT BLa was elevated by ~20% following NaHCO<sub>3</sub> ingestion, which was similar to previous studies. 6,10,11 The absolute increase from pre-TT to post-TT was much higher for CWSB versus IWSB, thus further explaining differences in performance. These changes in BLa might reflect greater efflux rates from the muscle and not only increased glycolytic energy production,<sup>29</sup> although it is likely that, when combined with the alkalosis, this partially accounted for the ergogenic benefits.

The NaHCO<sub>3</sub> ingestion elevated SID above baseline levels, primarily attributable to increased Na<sup>+</sup> and reduced Cl<sup>-</sup>. The intermittent warm-up mitigated these increases in SID, which expands upon our mechanistic explanation for the differences in performance between NaHCO3 trials. Preexercise changes in SID were consistent with previous findings,6 although SID remained elevated by ~19% following NaHCO<sub>3</sub> post-TT. This discrepancy can be explained by greater metabolic perturbation during the hypoxic conditions employed previously. These ionic changes reflect greater protection of action potentials within T-systems, allowing for sustained excitation of working muscles. <sup>1,7</sup> The results cited in the present study only reveal changes occurring within extracellular compartments<sup>6</sup> and do not infer whether alkalosis markedly increased ionic disturbances within contracting muscle. Moreover, these changes in SID may have been exacerbated by differences in the molecular composition of Na<sup>+</sup> between the 2 experimental beverages (ie, greater Na<sup>+</sup> content for 0.3 g·kg BM<sup>-1</sup> NaHCO<sub>3</sub>). Pilot testing revealed it was not possible to taste match an equimolar sodium chloride dose (0.21 g·kg BM<sup>-1</sup>); therefore, future research should administer NaHCO3 via capsules to determine whether similar extracellular ionic changes are observed.

Our findings are consistent with previous studies reporting mild GI discomfort following NaHCO<sub>3</sub> ingestion, 6,10,22 although these symptoms were reduced pre-TT and did not impair performance. There was also a large degree of interindividual and intraindividual variation, with 2 participants reporting severe diarrhea and bowel urgency pre-warm-up for one NaHCO<sub>3</sub> trial, but not the other. The severity of symptoms might have been reduced following capsule administration, or by coingesting a high-carbohydrate meal, 13 but the latter was not feasible with typically only a short window (~45 min) between ingestion and warm-up. Further investigation is warranted to better understand variability in GI discomfort, with athletes recommended to trial NaHCO<sub>3</sub> ingestion during training to inform decisions regarding practical application of the supplement.

# **Practical Applications**

An intermittent, sprint-based warm-up strategy mitigates the ergogenic potential of NaHCO<sub>3</sub> ingestion by utilizing the enhanced extracellular buffering capacity prior to commencing the 4-km cycling TT. Improvements in performance were only observed when individualized NaHCO<sub>3</sub> ingestion was combined with a steady-state, control warm-up. There was a large degree of variation in performance responses and GI discomfort following NaHCO<sub>3</sub> ingestion; therefore, athletes are recommended to trial

the supplement during training before use in competition. Practitioners and athletes should opt for an individualized time-to-peak  $HCO_3^-$  ingestion strategy and alter warm-up strategies to maximize the performance benefits of NaHCO<sub>3</sub> ingestion.

## Conclusion

This study was the first to demonstrate that an ecologically valid, intermittent, sprint-based warm-up reduces the ergogenic capacity of individualized NaHCO3 ingestion on 4-km cycling TT performance in cyclists. Metabolic perturbation associated with the intermittent warm-up dampened the ergogenic potential of NaHCO<sub>3</sub>. Improvements in 4-km TT completion time and average power following NaHCO<sub>3</sub> ingestion and the control warm-up were attributed to enhanced HCO<sub>3</sub><sup>-</sup> buffering response, upregulation of glycolytic activation, and sustained excitation of contracting muscles. The NaHCO3 ingestion resulted in mild GI discomfort, although this did not impact performance and displayed a large degree of interindividual and intraindividual variation. Our results provide practitioners with evidence-based practice advocating the inclusion of individualized NaHCO<sub>3</sub> supplementation within prerace regimes, as this proves more beneficial for improving 4-km cycling TT performance than an intermittent, sprint-based warm-up strategy.

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