Influence of Sodium Bicarbonate on Performance and Hydration in Lightweight Rowing

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Purpose: The effect of sodium bicarbonate (NaHCO₃) ingestion on prerace hydration status and on 2000 m ergometer performance in elite lightweight rowers was examined using a randomized, cross-over, double-blinded design. Methods: To simulate body mass (BM) management strategies common to lightweight rowing, oarsmen reduced BM by approx. 4% in the 24 h preceding the trials, and, in the 2 h before performance, undertook nutritional recovery consisting of mean 43.2 kJ/kg, 2.2 g of CHO per kilogram, 31.8 mg of Na⁺ per kilogram, 24.3 mL of H₂O per kilogram, and NaHCO₃ (0.3 g of NaHCO₃ per kilogram BM) or placebo (PL; 0.15 g of corn flour per kilogram BM) at 70 to 90 min before racing. Results: At 25 min before performance, NaHCO₃ had increased blood pH (7.48 ± 0.02 vs PL: 7.41 ± 0.03, P = .005) and bicarbonate concentrations (29.1 ± 1.8 vs PL: 23.9 ± 1.6 mmol/L, P < .001), whereas BM, urine specific gravity, and plasma volume changes were similar between trials. Rowing ergometer times were similar between trials (NaHCO₃: 397.8 ± 12.6; PL: 398.6 ± 13.8 s, P = .417), whereas posttest bicarbonate (11.6 ± 2.3 vs 9.4 ± 1.8 mmol/L, P = .003) and lactate concentration increases (13.4 ± 1.7 vs 11.9 ± 1.9 mmol/L, P = .001) were greater with NaHCO₃. Conclusion: Sodium bicarbonate did not further enhance rehydration or performance in lightweight rowers when undertaking recommended post-weigh-in nutritional recovery strategies.

Keywords: ergogenic, buffering, acidosis, rehydration, nutrition

Sodium bicarbonate (NaHCO₃) ingestion is proposed to increase the capacity of blood and muscle to buffer lactate (La⁻) and hydrogen (H⁺) ions that are formed during high-intensity activity¹–³ and that may limit exercise performance. Despite this, there is no consistency in the literature regarding the ergogenic benefits of exogenous NaHCO₃ across a variety of sports, with reports of performance gains⁴–⁸ as well as no such improvements.⁹–¹² However, there does appear to be more consistent ergogenic benefits in efforts of 3 to 7 min in duration, especially when NaHCO₃ is ingested at a dosage of 0.3 g of NaHCO₃ per kilogram body mass (BM).¹,² In elite rowing, where races last approximately 6 to 7 min, NaHCO₃ loading is commonly used, but the effectiveness of oral NaHCO₃ ingestion on rowing performance is limited to two published studies.⁵,¹⁰ When comparing NaHCO₃ and placebo trials, McNaughton and Cedaro⁵ reported improvement in 6 min ergometer performance with NaHCO₃ ingestion, whereas Brien and McKenzie¹⁰ reported no such benefit. However, McNaughton and Cedaro⁵ provided NaHCO₃ at 90 min preexercise whereas Brien and McKenzie¹⁰ had participants commence ingestion at 180 min before testing. These differences in the timing sequence of a NaHCO₃ concentration of 0.3 g per kilogram BM might help explain the differences in the effectiveness of the supplementation for single-bout exercise performance,¹³ particularly given that extracellular buffering peaks 60–90 min postingestion.¹⁴–¹⁶ Moreover, it has been suggested that a threshold of elevation in extracellular alkalosis postingestion might exist for NaHCO₃ to influence performance.¹⁷ Interestingly, the improved rowing performance with NaHCO₃ was accompanied by substantially greater elevations in extracellular pH than exhibited in the study of Brien and McKenzie.¹⁰

While exogenous HCO₃⁻ is proposed to enhance performance by influencing buffering capacity, it has been suggested that the coingested Na⁺ offers ergogenic potential by acutely expanding plasma volume.¹⁸,¹⁹ Plasma volume expansion is particularly relevant to rowers in the lightweight division who often use aggressive BM loss strategies, including fluid restriction, to “make weight” before racing.²⁰ However, lightweight rowers often self-select rehydration strategies of low Na⁺ content²⁰ and, thus, NaHCO₃ ingestion in capsule form...
may offer a palatable means of increasing Na⁺ intake to promote effective rehydration and associated plasma volume expansion while also enhancing blood buffering capacity. The coingestion of 1500 mL of fluid, which has been shown to be well tolerated following weigh-in,₂¹ delivers a sodium load of approximately 170 mmol of Na⁺·L⁻¹, which easily achieves the recommended intake of sodium for aggressive rehydration.₂¹⁻²³ In previous exercise studies investigating acute plasma volume expansion, similar sodium loads have also been tolerated.₁⁸,₁⁹ Therefore, the aim of this study was to assess the effect of oral NaHCO₃ ingestion on prerace hydration status and 2000 m ergometer performance in elite lightweight rowers adopting typical precompetition acute BM loss and nutritional recovery.

Methods

Subjects

Despite the population of elite lightweight rowers being limited in number and availability, seven highly trained, nationally competitive, male lightweight rowers (age = 22.0 ± 1.9 y; pretest BM = 75.9 ± 3.8 kg), who all competed for national team selection in the year of testing and were experienced with acute BM loss strategies to “make weight” for the lightweight division, volunteered for the study. Participants provided informed consent, with the study approved by the Ethics Committee of the Australian Catholic University. Two weeks before the study, participants performed a familiarization 2000 m time trial with no BM restrictions. The average power output in this trial was used to determine workloads for the warm-up on experimental trial days.

Design

To investigate the effect of NaHCO₃ ingestion on rowing performance, participants completed two 2000 m ergometer time trials separated by four days. The study was designed in a randomized, cross-over, double-blinded fashion, where participants were administered either NaHCO₃ or placebo (PL) supplementation. To replicate common pre-regatta BM management strategies, participants were required to reduce their BM by approximately 4% in the 24 h preceding both trials. A 2 h recovery period was provided between weigh-in and the ergometer time trial.₂⁴

Methodology

Participants presented at the testing location 24 h before each ergometer time trial (day prior). For assessment of hydration status, participants provided a urine sample from the first void of that day for measurement of urine specific gravity (Uₜₘ) using a hand-held optical refractometer (Atago, URC-Ne, Japan). Participants were then required to lie supine for 15 min before collection of a capillary blood sample for plasma volume assessment (ie, measures of hemoglobin concentration, [Hb], and hematocrit, [Hct]). This procedure was repeated for any blood sampling for plasma volume assessment. Changes in plasma volume following acute BM loss and rehydration were assessed using the equation of Dill and Costill,₂⁰ which was originally derived from venous blood samples. Given good agreement for [Hb] and [Hct] between venous and capillary sampling, twenty-seven capillary samples provide valid assumptions of plasma volume changes with only systematic differences resulting. For all blood collection, capillary sampling by skin puncture using aseptic procedures was performed. Blood pH, blood bicarbonate concentration, [HCO₃⁻] (mmol·L⁻¹); base excess (BE) (mmol·L⁻¹); [Hb] (g·L⁻¹); and Hct were assessed using the i-STAT portable blood analyzer (Abbott Point of Care Inc., East Windsor, USA), with samples (∼200 µL) collected in sodium heparinized capillary tubes and immediately delivered to i-STAT CG8+ and CG4+ cartridges for analysis. The i-STAT provides reliable measures of blood pH,₂₈,₂₉ blood [HCO₃⁻] and BE,₂⁹ and [Hb] and Hct.³⁰,³¹ The Lactate Pro analyzer (Arkay KDK Corporation, Shiga, Japan) was used to assess blood [La⁻], with a ∼5 µL sample placed on a Lactate Pro Test Strip (Arkay Inc., Kyoto, Japan). Bladder-voided BM was then recorded using calibrated digital scales accurate to 0.1 kg (Wedderburn, HD-316, Tanita Corporation, Japan) with participants wearing shorts only.

Participants were required to reduce BM by approximately 4% over the next 22 h (ie, preceding each ergometer time trial), so as to weigh in at this target weight 2 h before the ergometer time trial. This degree of BM loss and the time over which it occurred is similar to previous rowing-specific, acute BM loss studies, and reflects the approach typically adopted by lightweight rowers when making weight for competition. Body mass loss was induced via a combination of additional training plus food and fluid restriction, with the diet and exercise strategies used by each participant documented and replicated for both trials. In addition to acute food and fluid restriction, participants used a combination of rowing, running, swimming, and cycling exercise on the day before testing, with four of the seven participants using identical exercise regimens (120 min on-water row and 40 min swim, with 40–60 min walk if necessary). On the day of testing, participants rowed on an ergometer in sweat gear to reach target BM before the commencement of the session.

On the day of each ergometer time trial, participants provided an upon-waking urine sample for assessment of hydration. A capillary blood sample was taken for the assessment of plasma volume change, blood pH, blood [HCO₃⁻] and BE 120 min before exercise (weigh-in), before recording bladder-voided BM.

After weigh-in, participants were provided with a nutritional recovery formula that was ingested over 90 min. The recovery formula (mass 24.4 g·kg⁻¹, 43.2 kJ·kg⁻¹, 2.2 g carbohydrate·kg⁻¹, 31.8 mg Na⁺·kg⁻¹, 22.2...
mL water·kg⁻¹) included 1000 mL of Gastrolyte, 600 mL of Gatorade, a 65 g sports bar, 41 g of carbohydrate gel, and two standard slices of toasted white bread with ~10 g of Vegemite spread. The NaHCO₃ (0.3 g·kg⁻¹ BM) or PL (corn flour, 0.15 g·kg⁻¹ BM) supplementation was also provided, with dosage mass determined using an Ainsworth AC-400DR top-loading balance (Denver Instruments, Denver), accurate to 0.001 g. Both NaHCO₃ and PL were administered in identical gelatin capsules to blind for appearance and taste. Each participant received a total of 17 to 19 capsules (depending on BM), delivered at three time points (90, 80, and 70 min before the trial). The timing of ingestion was implemented in an attempt to achieve peak [HCO₃⁻] in the blood at the time of ergometer testing.¹⁴⁻¹⁸ and the ingestion protocol was also consistent with previous studies.⁵⁷ Participants drank a further 150 mL of solution in the 17 min before the ergometer time trial; the experimental (ie, NaHCO₃) trial was provided Gatorade (~9 g of CHO), while the PL trial received water that was colored to match the appearance of Gatorade. Ingestion of carbohydrate drinks when warming up in a fed state does not affect glucoregulation during exercise³³ or power output in high-intensity exercise of relevant duration.³⁴ This ensured that the total amount of carbohydrate was equivalent between trials, because ~9 g of CHO was provided in the corn flour capsules in the PL trial.

Twenty-five minutes before the trial (pre warm-up), participants gave another capillary blood sample to assess acid–base and plasma volume changes in response to NaHCO₃ and PL supplementation, as well as assessing resting blood [La⁻]. At this time, a urine sample (for Uₖg), bladder-voided BM, and completion of a 16-item gastrointestinal discomfort questionnaire were collected. The questionnaire (modification of questionnaire³⁵) asked participants to rate, “Which of the following are you experiencing at this point in time?” on a 10-point scale (eg, “no problem at all” = 0 points; “moderate problems” = 4 points; “severe” = 6 points; “the worst it has ever been” = 9 points). The symptoms to rate were reflux, heartburn, bloating, vomiting, nausea, upper abdominal cramps, left abdominal pain, right abdominal pain, intestinal cramp, flatulence, urge to urinate, urge to defecate, loose stool, dizziness, headache, and muscle cramp. Through the entire period from weigh-in to pretest, all excretions were collected in polyethylene containers and volumes determined with a measuring cylinder.

A standardized warm-up commenced 17 min before the ergometer trial, with participants completing two 4-min submaximal workloads (at 50% and 65% of average power output of the familiarization trial), separated by 1 min of passive recovery. After 2 min of passive recovery, participants performed 10 maximal strokes to simulate the start of a race. This procedure was repeated 2 min later. This warm-up replicated that used by Slater et al.²¹ Immediately before the time trial, participants provided another capillary blood sample (pretest), as well as using a Likert scale (1 = very poor; 2 = below average; 3 = average; 4 = above average; 5 = excellent) to indicate their motivation and performance expectation for the trial.

The 2000 m time trials were performed on a rowing ergometer (Model D, Concept 2, Morrisville, USA), with ergometers set to a drag factor of 120.²¹ Participants did not receive any instruction about how to undertake the time trial, but were reminded that the trial was to be of maximal effort. Ergometer monitor screens were partially covered, thereby only providing information to participants about distance remaining, stroke rating, and 500 m average split times. All trials were performed alongside another rower to provide competition. Verbal encouragement was provided during the trial. Heart rate (Polar S210, Polar Electro Oy, Finland) was recorded upon completion of every 250 m segment of the trial. Participants were not shown the time taken to complete the trial, to eliminate any influence on the subsequent trial. Upon completion of the trial, a rating of perceived exertion (RPE) score (15-point scale³⁶) was obtained, as well as average power output, stroke rating, and 250 m split and total times. A posttest capillary blood sample was collected 4 min after exercise.³⁷

**Statistical Analysis**

A power analysis of the only previous study of similar design² indicated that seven participants was a sufficient sample size to exhibit any significant response in the experimental trial, based on a power of 80%. Data were tested for normality using the Shapiro-Wilk test. Data were deemed to be normally distributed if Shapiro-Wilk values were P ≥ 0.05, and skewness and kurtosis values were within the range of −1 to 1. However, owing to the lack of power of skewness and kurtosis with a small sample size,³⁸ histograms, normal probability plots, and de-trended normal plots were also generated so that the distributions of parameters could be evaluated and verified visually.³⁹ If all data for a parameter were normally distributed, a two-way, two-factor, repeated-measures ANOVA was used to determine statistically significant effects of the trial and time points. Student’s paired t test was used to determine differences between trials at specific time points, between time points within a trial, or to detect any initial baseline differences. The level of significance was taken at P < .05. Data not normally distributed were log transformed and retested for normality.³⁹ If the log-transformed data were deemed to be normally distributed, an ANOVA and Student paired t tests were performed using the log-transformed data; otherwise, t tests were performed using the nonparametric Wilcoxon signed-rank test. No correlations (r > .8) between measures were found to appropriately include as covariates. All data are presented as mean ± SD; however, data not normally distributed are also reported as median and range. Effect size (ES) was used as a standardized measure of the magnitude of an observed effect, with Cohen’s d used when reporting ES.⁴⁰
Results

Hydration Status

Markers of hydration status were collected at three time points (day prior, weigh-in, pre-warm-up) and are shown in Table 1. Body mass loss from day before weigh-in was $4.5 \pm 0.9\%$ for NaHCO$_3$ trial ($P < .001$, ES = 0.95) and $4.1 \pm 1.6\%$ for PL trial ($P = .001$, ES = 0.75), and was not different between conditions ($P = .283$, ES = 0.33). Plasma volume decreased by $6.1 \pm 3.0\%$ and $3.3 \pm 5.7\%$ in the NaHCO$_3$ and PL trials, respectively, owing to acute BM loss (NaHCO$_3$: $P = .027$, ES = 3.93; PL: $P = .116$, ES = 0.84), and between conditions these changes were not different ($P = .346$, ES = 0.59). Values for $U_{\text{sg}}$ increased over the 22 h of acute BM loss (NaHCO$_3$: $P = .056$, ES = 0.86; PL: $P = .011$, ES = 0.92).

After consuming the recovery formula, participants restored $1.6 \pm 0.2$ kg (2.2 ± 0.2%) BM in the NaHCO$_3$ trial and $1.6 \pm 0.1$ kg (2.1 ± 0.1%) in the PL trial. Plasma volume was also partially restored and was not different between trials ($P = .916$, ES = 0.03), whereas $U_{\text{sg}}$ fell in both trials (NaHCO$_3$: $P = .025$, ES = 1.23; PL: $P = .036$, ES = 0.91) and was restored to day-prior values. Urinary losses during the recovery period between weigh-in and pre-warm-up (NaHCO$_3$: 102 ± 65 mL; PL: 182 ± 152 mL) were not different between trials ($P = .268$, ES = 0.67); however, an ES of this magnitude is considered a “medium” effect,$^{41}$ suggesting that the smaller losses with NaHCO$_3$ ingestion might have assisted rehydration$^{23}$ more than in the PL condition. Importantly, there were no differences between NaHCO$_3$ and PL conditions for BM, plasma volume, or $U_{\text{sg}}$ at any time points from day before pre-warm-up (see Table 1), but with the small sample size and reported variability in these data, some caution should be taken with their interpretation.

Gastrointestinal Complaints

Responses to the gastrointestinal discomfort questionnaire indicated there to be no noteworthy difficulties resulting from NaHCO$_3$ compared with PL ingestion. Specifically, using a 10-point (0 to 9) rating scale for the 16 questionnaire items, total participant ratings in the NaHCO$_3$ condition ranged from 0 to 12 from a total 144 points, while in the PL condition total scores ranged from 0 to 14 points. The most reported symptoms were similar in both conditions, being bloating, reflux, urge to defecate, and urge to urinate, with only one of the seven participants reporting a “serious problem” (ie, bloating) following ingestion of NaHCO$_3$ with the nutritional formula, and only one participant reporting a “serious problem” in 2 (ie, bloating and reflux) of the 16 questionnaire items following ingestion of PL with the nutritional formula.

Performance Measures

Subjective measures of “performance expectation” (NaHCO$_3$: 3.0 ± 0.0, PL: 2.8 ± 0.8; $P = .482$, ES = 0.41) and “motivation” (NaHCO$_3$: median 3.0, range 2.0, PL: 2.5, 3.0; $P = .059$, ES = 0.58) assessed immediately before each trial were not different between trials. Sodium bicarbonate ingestion had no effect on the time to complete 2000 m when compared with the PL trial (397.8 ± 12.6 vs 398.6 ± 13.8 s, respectively; $P = .417$, ES = 0.06). Similarly, average power output was not different between trials (NaHCO$_3$: 358 ± 32 W; PL: 356 ± 35 W; $P = .413$, ES = 0.05). These trials did not differ from the familiarization trial (time: 398.7 ± 16.6 s; power output: 357 ± 41 W), suggesting that acute BM reduction and the associated recovery strategy did not affect expected performance. Moreover, Figure 1 shows individual performance times in response to NaHCO$_3$ relative to PL supplementation, demonstrating that NaHCO$_3$ produced no consistent ergogenic effect, nor was a phenomenon of “responders” and “nonresponders” to NaHCO$_3$ supplementation observed. The largest performance gain (1.2%) was associated with NaHCO$_3$ ingestion.

At the completion of each 2000 m performance, there were no differences in RPE (NaHCO$_3$: 18.9 ± 0.9; PL: 19.0 ± 0.8; $P = .356$, ES = 0.17), average heart rate (NaHCO$_3$: 177 ± 12; PL: 179 ± 9 bpm; $P = .602$, ES = 0.12), or peak heart rate (NaHCO$_3$: 187 ± 6; PL: 185 ± 6 bpm; $P = .384$, ES = 0.25) between trials.

Table 1 Markers of hydration before and after ingestion of a nutritional recovery formula with NaHCO$_3$/placebo supplementation

<table>
<thead>
<tr>
<th>Time</th>
<th>BM (kg)</th>
<th>$U_{\text{sg}}$ (g·mL$^{-1}$)</th>
<th>Plasma Volume (% Change)$^9$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day prior</td>
<td>NaHCO$_3$</td>
<td>Placebo</td>
<td>NaHCO$_3$</td>
</tr>
<tr>
<td></td>
<td>76.4 ± 3.7</td>
<td>75.5 ± 4.2</td>
<td>1.020 ± 0.009</td>
</tr>
<tr>
<td></td>
<td>(75.6, 14.0)</td>
<td>(75.6, 14.0)</td>
<td></td>
</tr>
<tr>
<td>Weigh-in</td>
<td>NaHCO$_3$</td>
<td>Placebo</td>
<td>NaHCO$_3$</td>
</tr>
<tr>
<td></td>
<td>72.9 ± 3.2$^a$</td>
<td>72.3 ± 3.3$^a$</td>
<td>1.028 ± 0.004</td>
</tr>
<tr>
<td></td>
<td>(74.4, 8.9)</td>
<td>(72.6, 9.8)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-warm-up</td>
<td>NaHCO$_3$</td>
<td>Placebo</td>
<td>NaHCO$_3$</td>
</tr>
<tr>
<td></td>
<td>74.5 ± 3.3$^a$</td>
<td>73.9 ± 3.3$^a$</td>
<td>1.019 ± 0.008$^a$</td>
</tr>
<tr>
<td></td>
<td>(75.9, 9.3)</td>
<td>(75.9, 9.3)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. Data are mean ± SD (median, range); $n = 7$ (except for $n = 6$, where outlier data of one participant were removed because it was greater than 2 SD from the mean). Plasma volume percentage changes are relative to day-prior values. Significant difference from previous time period at $^*P < .05$, $^{1*}P < .01$, $^{2*}P < .001$. $^a$Log-transformed data were used.
Acid–Base Balance

The effect of NaHCO₃ ingestion on measures of acid–base balance in blood is shown in Table 2. In the NaHCO₃ trial, blood pH rose following ingestion (after weigh-in), continued to increase during the warm-up period, and was greater at pretest ($P = .003$, ES = 1.12) when compared with preingestion (ie, weigh-in) values. Accordingly, at the pre-warm-up and pretest time points, blood pH was greater in the NaHCO₃ trial compared with the PL trial (pre-warm-up: $P = .004$, ES = 1.07; pretest: $P = .005$, ES = 1.59). Blood [HCO₃⁻] also increased after NaHCO₃ ingestion and remained elevated after the standardized warm-up period ($P = 0.003$, ES = 1.58). Consequently, at all time points after NaHCO₃ ingestion, blood [HCO₃⁻] was greater in the NaHCO₃ compared with the PL trial (pre-warm-up: $P = 0.002$, ES = 1.45; pretest: $P < 0.001$, ES = 1.64; posttest: $P = 0.009$, ES = 0.93). Base excess demonstrated a similar response to that observed for blood [HCO₃⁻], increasing after NaHCO₃ supplementation and remaining elevated following the warm-up period ($P = .004$, ES = 1.87), resulting in values greater than PL values at all time points after NaHCO₃ ingestion (pre-warm-up: $P = 0.001$, ES = 1.44; pretest: $P < 0.001$, ES = 1.67; posttest: $P = 0.020$, ES = 0.84).

After time trials, regardless of supplementation, pH values decreased, with no difference in pH values between trials ($P = .077$, ES = 0.60). In Table 2, it is shown that blood [HCO₃⁻] also fell during the time trials (NaHCO₃: $P < .001$, ES = 1.88; PL: $P < .001$, ES = 1.88), with [HCO₃⁻] at posttest remaining greater in the NaHCO₃ trial compared with the PL trial ($P = .009$, ES = 0.93). Base excess also fell during both time trials ($P < .001$, ES = 18.99), with BE being greater posttest in the NaHCO₃ trial compared with the PL trial ($P = .020$, ES = 0.84).

Blood [La⁻] responses for the performance-related time periods are shown in Table 3. Immediately before the warm-up, [La⁻] was not different between groups ($P = .764$, ES = 0.25). However, after the standardized warm-up, [La⁻] had increased in the NaHCO₃ trial and was greater than the PL trial ($P = .010$, ES = 0.95).
Table 2  Blood acid–base parameters for NaHCO₃ and placebo trials

<table>
<thead>
<tr>
<th>Time</th>
<th>pH</th>
<th>HCO₃⁻ (mmol-L⁻¹)</th>
<th>Base Excess (mmol-L⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NaHCO₃</td>
<td>Placebo</td>
<td>NaHCO₃</td>
</tr>
<tr>
<td>Weigh-in</td>
<td>7.45 ± 0.03ᵃ</td>
<td>7.44 ± 0.02</td>
<td>26.0 ± 2.0ᵇ</td>
</tr>
<tr>
<td>Pre-warm-up</td>
<td>7.46 ± 0.03ᶜ</td>
<td>7.43 ± 0.03</td>
<td>29.8 ± 2.4ᶜ</td>
</tr>
<tr>
<td>Pretest</td>
<td>7.48 ± 0.03ᶜ</td>
<td>7.41 ± 0.03</td>
<td>29.1 ± 1.8ᶜ</td>
</tr>
<tr>
<td>Posttest</td>
<td>7.18 ± 0.08ᶜ</td>
<td>7.13 ± 0.09ᶜ</td>
<td>11.6 ± 2.3ᵃ</td>
</tr>
</tbody>
</table>

Note. Data are mean ± SD (median, range); n = 7. Significant difference from previous time period at ⁿ>P < .05, ᵉ>P < .01, ᵇ>P < .001. Significant difference from the placebo at the same time point at ᵌ>P < .05, ᵙ>P < .01, ᵛ>P < .001. *Log transformed data were used.

Table 3  Blood [La⁻] responses (in mmol-L⁻¹) for NaHCO₃ and placebo supplementation before and following a 2000 m ergometer time trial

<table>
<thead>
<tr>
<th>Time</th>
<th>NaHCO₃</th>
<th>Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-warm-up</td>
<td>2.6 ± 0.6</td>
<td>2.5 ± 0.7ᵃ</td>
</tr>
<tr>
<td>Pretest</td>
<td>3.3 ± 0.8ᵇ</td>
<td>2.6 ± 0.5 (2.4, 1.5)</td>
</tr>
<tr>
<td>Posttest</td>
<td>16.7 ± 1.2ᵃ</td>
<td>14.4 ± 1.5ᵉ (14.7, 4.6)</td>
</tr>
<tr>
<td>Change pre- to posttest</td>
<td>13.4 ± 1.7ᶜ</td>
<td>11.9 ± 1.9</td>
</tr>
</tbody>
</table>

Note. Data are mean ± SD (median, range); n = 7, except for n = 5. Significant difference from previous time period at ⁿ>P < .05, ᵉ>P < .01. Significant difference from placebo at ᵀ>P < .001. *Log transformed data were used.

As expected, [La⁻] increased during the time trials, with both the absolute [La⁻] value posttest (P = .001, ES = 1.28) and the change in [La⁻] from pre- to posttest (P = .003, ES = 0.81) being greater in the NaHCO₃ compared with the PL trial.

**Discussion**

This is the first study to investigate the effectiveness of oral NaHCO₃ supplementation on performance and rehydration in elite lightweight rowers undertaking typical acute BM loss (−4%) to make weight before competition. The results indicate that NaHCO₃ ingestion provides no enhancements to 2000 m ergometer performance or to the recovery of body mass or hydration status when added to recommended nutritional strategies⁵¹ following acute body mass loss.

Previous work investigating the effect of oral NaHCO₃ supplementation on rowing performance in elite-standard oarsmen⁴₅,¹⁰ is equivocal, similar to that reported in other sports/activities (see reviews¹²,²²,⁴³). The findings of this study are in agreement with Brien and McKenzie¹⁰ who reported no difference in 6 min ergometer performance between NaHCO₃ and placebo trials, while contrasting the work of McNaughton and Cedaro⁵ who reported significant improvements in 6 min ergometer performance with NaHCO₃ supplementation. Similar to Brien and McKenzie,¹⁰ increases in blood pH and [HCO₃⁻] following NaHCO₃ ingestion in our study were substantially less than those reported by McNaughton and Cedaro,⁵ adding support to the proposition that a threshold of elevation in extracellular alkalosis might be necessary for performance benefits.¹⁷ Taking this further, a novel aspect of our work was the ingestion of NaHCO₃ alongside an aggressive post-weigh-in nutritional regimen. With work showing absorption rates of dietary supplements being slowed when coingested with other food,⁴⁴ it is possible that, in our study, the concurrent nutritional intake slowed HCO₃⁻ appearance in the blood, thereby resulting in lesser increases in extracellular pH and [HCO₃⁻] at the time of exercise than observed in previous rowing studies, ultimately contributing to an absence of performance gains. If this is indeed the case, consideration should be given to adjusting the timeline for bicarbonate ingestion to ensure that peak extracellular alkalosis is achieved at the time of performance.

It has been suggested that the contribution of sodium and/or fluid associated with NaHCO₃ ingestion might be implicated in its reported benefits on exercise performance. Mitchell et al.⁵ in an infusion study, reported that both NaHCO₃ and placebo supplementation improved high-intensity, endurance cycling performance when sodium content (154 mmol-L⁻¹) and fluid volume (∼1500 mL) were controlled. As part of the nutritional recovery provided in our study, fluid ingestion was equivalent in the two trials (1764 mL in 120 min) and sodium loads were 193–211 mmol Na⁺-L⁻¹ (depending on body mass) in the NaHCO₃ trial and 57 mmol Na⁺-L⁻¹ in the PL trial. Maughan and Leiper⁵² have shown there to be no differences in plasma volume expansion following ingestion of solutions of 52 and 100 mmol Na⁺-L⁻¹. Thus, taken together, our data are in agreement with those of Mitchell et al.,⁵³ proposing that NaHCO₃ supplementation offers little ergogenic benefit to anaerobic, endurance performance when concomitant ingestion of sodium and fluid are of substantial magnitude. Specifically, the nutritional recovery formula (57 mmol Na⁺-L⁻¹; 1764 mL of fluid)
ingested by rowers in the present study was sufficient enough such that NaHCO₃ supplementation provided no additional benefit to the reestablishment of hydration status and no subsequent improvement in ergometer performance. In practice, for individuals who have palatability concerns with high Na⁺ solutions, the ingestion of sodium in capsule form may offer a palatable option for effective rehydration.

In agreement with previous work, greater blood [La⁻] was observed posttest in the NaHCO₃ (16.7 ± 1.2 mmol·L⁻¹) when compared with PL (14.4 ± 1.5 mmol·L⁻¹) trial, suggesting that NaHCO₃ supplementation assists lactate efflux from muscle. This would also explain the significant post-warm-up increase in blood [La⁻] observed in the NaHCO₃ trial. An increased blood [HCO₃⁻] and the concomitant enhancement in blood buffering capacity would, in turn, help maintain the pH gradient between cell and blood. This would lead to enhanced H⁺ efflux from the cell (supported by our data), with the rise in blood [La⁻] being a consequence of greater H⁺/La⁻ cotransport.

In conclusion, even though the ingestion of 0.3 g of NaHCO₃ per kilogram BM is effective for eliciting alkalosis in the blood, the present study indicates that NaHCO₃ ingestion when accompanied by appropriate nutritional strategies provides no ergogenic benefit for 2000 m rowing ergometer performance in highly trained lightweight rowers. Equally, the ingestion of NaHCO₃ in conjunction with recommended prerace nutrition does not compromise rowing performance either.

**Practical Applications and Conclusions**

This work is particularly relevant to sports comprising weight divisions (eg, lightweight rowing, boxing, wrestling) in which athletes routinely rapidly reduce body mass to make weight before competition. In these sports, there is often a time period after weigh-in during which athletes undertake nutritional recovery. The findings of this study show that NaHCO₃ ingestion during this time period offers little performance or rehydration benefit if accompanied by aggressive nutritional strategies that deliver substantial sodium and fluid volume. However, there is evidence indicating that athletes (eg, lightweight rowers) normally self-select low-Na⁺ nutritional strategies following weigh-in. Thus, there is the potential that oral NaHCO₃ ingestion in capsule form or via other means may offer a palatable alternative for increasing Na⁺ intake for athletes whose post-weigh-in nutrition does not comply with current guidelines. In addition, if athletes use NaHCO₃ in capsule form, there may not be a need for the ingestion of high Na⁺-containing foods and fluids that may enhance the palatability of post-weigh-in recovery meals. In such situations, athletes may need to reconsider the timing of HCO₃⁻ if indeed coingestion with food influences its rate of appearance in blood and, thus, the potential for enhancing blood buffering capacity. The influence of coingestion of NaHCO₃ with food on the time course of enhanced buffering capacity warrants further investigation.

**Acknowledgments**

The authors thank the Australian Institute of Sport for providing the recovery formula for this research.

**References**


