Effects of Sodium Bicarbonate, Caffeine, and Their Combination on Repeated 200-m Freestyle Performance

Cathryn L. Pruscino, Megan L.R. Ross, John R. Gregory, Bernard Savage, and Troy R. Flanagan

The purpose of this study was to investigate the effects of sodium bicarbonate (NaHCO$_3$), caffeine, and their combination on repeated 200-m freestyle performance. Six elite male freestyle swimmers ingested NaHCO$_3$ (0.3 g/kg; B), caffeine (6.2 ± 0.3 mg/kg; C), a combination of both (B+C), and placebo (P) on 4 separate occasions before completing 2 maximal 200-m freestyle time trials (TT1 and TT2) separated by 30 min. No significant differences ($p = .06$) were observed for performance in TT1 (B 2:03.01 ± 0:03.68 min, C 2:02.42 ± 0:03.17 min, B+C 2:01.69 ± 0:03.19 min, P 2:03.77 ± 0:03.21 min) or TT2 (B 2:02.62 ± 0:04.16 min, C 2:03.90 ± 0:03.58 min, B+C 2:01.70 ± 0:02.84 min, P 2:04.22 ± 0:03.75 min). The drop-off in performance time from TT1 to TT2, however, was significantly greater when C was ingested than with B (–1.5%, $p = .002$) or B+C (–1.2%, $p = .024$). This is likely because of the lower blood pH and slower recovery of blood HCO$_3$ post-TT1 after C ingestion. These findings suggest that the ergogenic benefit of taking C alone for repeated 200-m swimming performance appears limited. When combined with NaHCO$_3$, however, its negative impact on repeated maximal exercise performance is reversed.

**Keywords**: ergogenic aid, blood pH, blood lactate, bicarbonate buffering, swimming

Repeated short-term, maximal sprint efforts are common during many sports. In the case of swimming, the ability of an athlete to perform repeated maximal efforts when heats or finals must be performed within a short time frame largely depends on the individual’s ability to rapidly restore muscle acid-base balance to perform optimally in the subsequent event. During short-term maximal exercise, the accumulation of hydrogen ions (H$^+$) in the working muscle is thought to inhibit the process of anaerobic glycolysis (Sutton, Jones, & Toews, 1981), bring about a reduction in force production (Fabiato & Fabiato, 1978; Mainwood & Cechetto, ____________

Pruscino, Savage, and Flanagan are with the Dept. of Sport Science, Victorian Inst. of Sport, Melbourne, Victoria 8006 Australia. Ross is with the Dept. of Physiology, Australian Inst. of Sport, Belconnen, ACT 2617 Australia. Gregory is with the Dept. of Sport Science, Tasmanian Inst. of Sport, Prospect, Tasmania 7250 Australia.
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1980), and contribute to the onset of fatigue (Costill, Verstappen, Kuipers, Janssen, & Fink, 1984).

For repeated exercise performance, the recovery from fatigue is accelerated by an increase in extracellular pH (Bishop, Edge, Davis, & Goodman, 2004; Mainwood & Cechetto, 1980). Ingestion of sodium bicarbonate (\(\text{NaHCO}_3\)) before short-term, maximal exercise has previously been found to enhance performance in repeated exercise bouts (Bishop et al.; Costill et al., 1984; Sutton et al., 1981). This is achieved by the creation of a metabolic alkalosis in the exercising tissues, providing an enhanced bicarbonate-buffering capacity (Costill et al.; Katz, Costill, King, Hargreaves, & Fink, 1984; Mero, Keskinen, Malvela, & Sallinen, 2004). During high-intensity exercise, the intracellular lactic-acid production resulting from glycolysis, coupled with an increase in extracellular bicarbonate concentration, creates an increased pH gradient across the cell membrane (Costill et al.). This in turn promotes the efflux of H\(^+\) out of the muscle cell (Costill et al.). By enhancing the removal of H\(^+\) from the cell and buffering its buildup in the blood, the fatigue-related changes in intracellular pH are delayed, allowing more work to be performed before fatigue sets in (Bishop et al.; Costill et al.; Sutton et al.; Wilkes, Gledhill, & Smyth, 1983).

The benefit to short-term (up to 2 min) high-intensity repeated exercise performance derived from caffeine ingestion appears varied, with some previous research revealing its effectiveness (Anselme, Collomp, Mercier, Ahmaidi, & Prefaut, 1992), others reporting its efficacy only in trained participants (Collomp, Ahmaidi, Chatard, Audran, & Prefaut, 1992), and others proving it ineffective (Greer, McLean, & Graham, 1998). Similarly, improved performance of a single short-term high-intensity exercise bout with caffeine ingestion has been demonstrated by some (Collomp et al.; Doherty, Smith, Hughes, & Davison, 2004) but rejected by others (Williams, Signorile, Barnes, & Henrich, 1988). By operating as an adenosine-receptor antagonist (Biaggioni, Paul, Puckett, & Arzubiaga, 1991), caffeine increases neurotransmitter release, augmenting motor-unit recruitment and enhancing skeletal-muscle contractility (Williams, 1991). Furthermore, it stimulates the cerebral cortex, preventing drowsiness and improving mental alertness (Williams). It has been suggested that the increase in postexercise blood lactate concentrations after caffeine ingestion might be the result of the liberation of calcium, which in turn increases glycolysis, generating greater anaerobic metabolism and improvements in total work output (Collomp et al.; Jackman, Wendling, Friars, & Graham, 1996).

When considering the combined effects of caffeine and NaHCO\(_3\) ingestion, the combination of central nervous system stimulation, greater anaerobic metabolism, and the buffering of H\(^+\) ions is potentially a beneficial recipe for performance enhancement. NaHCO\(_3\) and caffeine are commonly used ergogenic aids, but there appears to have been no research investigating their combined effects on repeated high-intensity exercise performance.

Therefore, the purpose of this study was to investigate the effects of ingesting NaHCO\(_3\), caffeine, and a combination of the two to determine whether any would bring about an improvement in repeated maximal swimming performance via increased anaerobic metabolism or improved muscle buffering. This was measured on repeated 200-m freestyle swimming separated by 30 min, on four separate
occasions, similar to swimming an individual and relay swim in international competition.

## Methods

### Participants

Six highly trained elite male freestyle swimmers volunteered to participate in the investigation ($N = 6$). Only male participants were included in the study because of the effects of the female menstrual cycle and the oral contraceptive pill on caffeine elimination (Kalmar & Cafarelli, 1999).

Before the study began, all participants were informed of the requirements, risks, and benefits of the investigation, and written informed consent was obtained from each. Approval for the procedures of the study was granted by the Australian Institute of Sport Ethics Committee.

### Procedures

Data collection was conducted over a 3-week period, during which time participants swam in four trials on four separate testing days separated by at least 3 days. In the 48 hr before each trial, participants were asked to abstain from caffeine intake. Food diaries were kept by each athlete in the 24 hr before the first trial. This food-intake regimen was then replicated before each subsequent trial. In the 24 hr before each trial, participants were restricted from performing high-intensity exercise. Before the commencement of each trial, the body mass (BM) of each participant was recorded to ensure accurate determination of supplement dosage. For the accurate measurement of BM, participants were required to weigh in dressed only in their swimsuits.

On arrival at the testing venue, each participant had 100 µl of blood collected from the fingertip into heparinized capillary tubes to measure baseline blood pH and blood HCO$_3^-$ Further blood samples were collected and analyzed for blood lactate, pH, and blood HCO$_3^-$ at the following 12 time points throughout each trial: 5 min before TT1 (pre-TT1); 0, 3, 5, 10, 15, and 20 min after TT1; 5 min before TT2 (pre-TT2); and 0, 3, 5, and 10 min after TT2. Blood lactate was determined using a portable lactate analyzer (Lactate Pro, Arkray, Japan). Blood pH and blood HCO$_3^-$ analysis was performed using a portable analyzer (i-STAT 1 analyzer Immuno-ready, Abbot, USA). Previous research has reported the validity of the i-Stat portable analyzer for point-of-care testing across a range of variables with acceptable results (Connelly, Magee, & Kiessling, 1996; Jacobs, Vadasdi, Sarkozi, & Colman, 1993; Mock, Morrison, & Yatscoff, 1995; Sedianne, Zerah-Lancner, d’Ortho, Adnot, & Harf, 1999). With regard to the variables measured in the current study, results have been validated against standard laboratory blood analyzers (Radiometer analyzers ABL 700 and 520, Radiometer, Villeurbanne, France) with correlations for pH reported at $r = .978$ and HCO$_3^-$ at $r = .920$ (Verwaerde, Malet, Lagente, de la Farge, & Braun, 2002).

Each swim was performed in a 50-m indoor swimming pool using a dive start from starting blocks with start gun and electronic touch-pad timing system (Omega
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ARES, Swiss Timing, Switzerland ISO 9001 approved). Participants were required to perform two maximal 200-m time trials separated by 30 min. Participants arrived at the testing venue at the same time on each of the four testing days. Participants were randomly assigned to four treatments over each of the four testing days using a double-blind randomized research design. The four treatments were administered in relation to BM: 0.3 g/kg NaHCO$_3$ (B), 6.2 ± 0.3 mg/kg caffeine (C), 0.3 g/kg NaHCO$_3$ with 6.2 ± 0.3 mg/kg caffeine (B+C), and glucose placebo (P).

A dose of 0.3 g/kg BM of NaHCO$_3$ has previously been found to enhance performance (Bishop et al., 2004; Sutton et al., 1981; Wilkes et al., 1983), whereas smaller doses have been found to be less effective (Horswill et al., 1988; Pierce, Eastman, Hammer, & Lynn, 1992). A caffeine dose of 6 mg/kg BM was chosen because the most conclusive evidence in the literature supported enhanced performance using this dose (Bruce et al., 2000; Cox et al., 2002; Jackman et al., 1996).

For treatments containing NaHCO$_3$, gelatin capsules each containing 840 mg of NaHCO$_3$ (Sodibic, Aspen Pharmacare Australia Pty. Ltd.) were administered. Placebo was administered using gelatin capsules containing 550 mg glucose polymer (Polyjoule, Nutricia Australasia Pty. Ltd.). The treatments containing caffeine used tablets containing 100 mg caffeine (No Doz Awakeners, Key Pharmaceuticals Pty. Ltd.).

NaHCO$_3$ supplementation was administered in seven doses over a 90-min ingestion period. Two hours before the first time trial, the first dose was administered with a normalized snack (1 g CHO/kg BM) to minimize gastric discomfort. Equal numbers of Sodibic or Polyjoule capsules (depending on the treatment) were ingested with a standardized volume of water (20 ml/kg BM), which was also ingested over the 90-min period to reduce the possible negative effect on gastrointestinal function (Figure 1). The timing of the NaHCO$_3$ dose and the normalized food and fluid protocol were developed and refined during previous field-based experimentation on reducing the gastric discomfort experienced by elite swimmers after NaHCO$_3$ supplementation.

When caffeine was administered it was ingested in a single dose 45 min before TT1. Placebo capsules replaced caffeine in the noncaffeine trials so that equal numbers of supplements were given on each occasion.

After the ingestion period, participants immediately commenced a standardized warm-up equivalent to normal practices of current elite, Olympic-standard swimmers. Including a warm-up ensured that swimmers achieved maximal performance during the subsequent trials. The standardized 1,200-m warm-up lasted for approximately 20 min and consisted of a 500-m easy swim of choice, 5 × 100-m individual medley, and 2 × 100 m of drills and variable sprints. After this, participants left the pool and had 10 min to prepare for TT1.

After completing TT1 and after the 5-min post-TT1 blood collection, participants commenced an 800-m standardized low-intensity recovery swim that is equivalent to current normal practices of elite swimmers. Blood samples were collected throughout this recovery swim at the specified times. At the end of the recovery swim, participants left the pool and had 10 min to prepare for TT2. Blood pH and blood lactate concentrations have been shown to return to near preexercise values by 30 min postexercise (Costill et al., 1984; Katz et al., 1984), so this time period was chosen for the recovery between time trials.
Figure 1 — Supplementation strategy and associated timeline.
After the entire testing period, participants were given a questionnaire to evaluate whether they could guess the order of treatments they had been given. They were also asked to report any gastrointestinal disturbance they had experienced.

Data Analysis

All values are reported as $M \pm SD$. A two-way analysis of variance with repeated measures was used to determine differences between the four treatments with regard to blood and performance parameters. A Tukey’s honestly significant difference post hoc test was used to establish where any differences occurred, and statistical significance is reported when $p < .05$. Statistical analysis was carried out using SPSS for Windows (Release 11.0.0, Standard Version). Swimming time was used to track percent changes in performance. Because these changes are not uniformly proportional, the data were log-transformed for all analyses. Uncertainty in population values of statistics was expressed as the 95% confidence interval and as a likelihood that the true value of the effect represents substantial change (slower or faster). The magnitude of change for the smallest worthwhile improvement in elite swimmers has previously been established as 0.4% (Pyne, Trewin, & Hopkins, 2004; Trewin, Hopkins, & Pyne, 2004). Magnitude of the effect size was qualified by conventional threshold values as 0.0–0.2 being a trivial effect, 0.2–0.5 a small effect, 0.5–0.8 a moderate effect, and 0.8 or greater a large effect size.

Results

Performance

There was no significant difference observed between treatments for absolute performance time in TT1 or TT2 ($p = .06$; Table 1). Despite no statistical difference in mean scores, 3 out of 6 participants recorded their fastest times in TT1 after ingesting B+C, and this number increased to 4 out of 6 in TT2 for the same treatment. The remaining participants recorded their fastest swim times after ingesting C (2 participants in TT1 and 1 in TT2) or B (1 participant in TT1 and TT2). No participants performed their fastest swim for TT1 or TT2 under placebo conditions (P).

Table 1  Mean Performance Time (min:s.ms) in Repeat 200-m Freestyle Performance After Supplementation in Elite Male Swimmers ($N = 6$), $M (SD)$

<table>
<thead>
<tr>
<th>Supplement</th>
<th>Time trial 1</th>
<th>Time trial 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bicarbonate</td>
<td>2:03.01 (3.68)</td>
<td>2:02.62 (4.16)</td>
</tr>
<tr>
<td>Caffeine</td>
<td>2:02.42 (3.17)</td>
<td>2:03.90 (3.58)</td>
</tr>
<tr>
<td>Bicarbonate + caffeine</td>
<td>2:01.69 (3.19)</td>
<td>2:01.70 (2.84)</td>
</tr>
<tr>
<td>Placebo</td>
<td>2:03.77 (3.21)</td>
<td>2:04.22 (3.75)</td>
</tr>
</tbody>
</table>

Note. No significant treatment effect observed ($p = .060$).
With regard to performance time in TT2 compared with TT1, participants swam on average 1.2% ± 0.7% slower in TT2 than in TT1 after ingesting C and 0.4% ± 0.7% slower after ingesting P. In the B trial, however, participants swam on average 0.3% ± 0.7% faster in TT2 than in TT1, and in the B+C trial the performance time between the two trials was maintained (0.0% ± 1.1%). As such, the reduction in performance in TT2 compared with TT1 was significantly greater when C was ingested than when B (p < .01) or B+C (p < .05) was ingested, but the change in the P trial was not significantly different from any other treatment. Pairwise comparisons of the treatments are fully outlined in Table 2. Supplement comparisons for a one-off 200-m time-trial performance (TT1) are outlined in Table 3.

### Blood HCO<sub>3</sub>

Baseline blood HCO<sub>3</sub> was not different across treatments (p > .05, Figure 2). After NaHCO<sub>3</sub> ingestion, pre-TT1 blood HCO<sub>3</sub> was significantly higher than baseline for B (p < .01) and B+C (p < .05). The decline in blood HCO<sub>3</sub> during TT1 was significantly greater (p < .01) in B+C (–14.7 ± 2.3 mmol/L) than in P (–10.1 ± 2 mmol/L) and C (–11.1 ± 2.5 mmol/L), as well as in B compared with P (–13.5 ± 1.9 vs. –10.1 ± 2.0 mmol/L, respectively; p < .05). During TT1 there was no statistical difference in the decline of blood HCO<sub>3</sub> for B versus B+C. However, 25 min after TT1, at the pre-TT2 time point, blood HCO<sub>3</sub> remained higher than

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**Table 2  Pairwise Comparison of Supplements on Repeat 200-m Freestyle Performance, M ± SD, N = 6**

<table>
<thead>
<tr>
<th>Supplement comparison</th>
<th>Difference in change in mean, TT2 – TT1 (%)</th>
<th>p</th>
<th>Effect size (95% CL)</th>
<th>Effect magnitude/Practical outcome (effect of taking 1st listed supplement over the 2nd)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C vs. B</td>
<td>–1.5 ± 0.7</td>
<td>.002*</td>
<td>–0.58 ± 0.26</td>
<td>Small harm to large harm (slower)</td>
</tr>
<tr>
<td>C vs. B+C</td>
<td>–1.2 ± 1.0</td>
<td>.024*</td>
<td>–0.48 ± 0.38</td>
<td>Large harm (slower) to trivial effect</td>
</tr>
<tr>
<td>C vs. P</td>
<td>–0.9 ± 1.1</td>
<td>.094</td>
<td>–0.34 ± 0.42</td>
<td>Moderate harm (slower) to trivial effect</td>
</tr>
<tr>
<td>B vs. B+C</td>
<td>0.3 ± 1.3</td>
<td>.535</td>
<td>0.12 ± 0.48</td>
<td>Small harm (slower) to moderate benefit (faster)</td>
</tr>
<tr>
<td>B vs. P</td>
<td>0.7 ± 0.7</td>
<td>.052</td>
<td>0.25 ± 0.26</td>
<td>Trivial effect to moderate benefit (faster)</td>
</tr>
<tr>
<td>B+C vs. P</td>
<td>0.3 ± 1.5</td>
<td>.568</td>
<td>0.13 ± 0.55</td>
<td>Small harm (slower) to moderate benefit (faster)</td>
</tr>
</tbody>
</table>

*Note. CL = confidence limits; C = caffeine; B = bicarbonate; B+C = bicarbonate + caffeine; P = placebo.

*Significant difference observed in taking one supplement over the other.
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baseline levels only in the B trial \((p < .05)\). Conversely, in the C trial, pre-TT2 blood \(\text{HCO}_3\) was significantly lower than baseline values \((20.3 \pm 1.5 \text{ vs. } 25.7 \pm 1.4 \text{ mmol/L, respectively; } p < .01)\). There was no change in blood \(\text{HCO}_3\) between baseline, pre-TT1, and pre-TT2 for P.

During TT2, the decline in blood \(\text{HCO}_3\) was significantly \((p < .01)\) greater in B \(\left(-13.7 \pm 0.9 \text{ mmol/L}\right)\) than with P \(\left(-8.4 \pm 1.2 \text{ mmol/L}\right)\) and C \(\left(-7.5 \pm 2.1 \text{ mmol/L}\right)\). The decline in blood \(\text{HCO}_3\) in B during TT2 was also higher \((p < .05)\) than that observed in B+C \(\left(-10.4 \pm 2.4 \text{ mmol/L}\right)\).

Blood pH

There was no difference between the initial baseline values for blood pH across treatments \((p > .05, \text{Figure } 2)\). After ingestion of B and B+C, pre-TT1 pH values were significantly higher \((p < .01)\) than baseline values. The decline in blood pH during TT1 was significantly greater in B+C than in B \(\left(0.274 \pm 0.025 \text{ vs. } 0.199 \pm 0.039; \ p < .01\right)\) and in C than in B \(\left(0.253 \pm 0.051 \text{ vs. } 0.199 \pm 0.039; \ p < .05\right)\). As such, 25 min later at the pre-TT2 time point, pH values remained higher than baseline values only in the B trial \(\left(7.492 \pm 0.037 \text{ vs. } 7.368 \pm 0.038; \ p < .01\right)\).

In the post-TT1 period, blood pH was higher in Trial B than in Trial C at 3 min \(\left(7.288 \pm 0.048 \text{ vs. } 7.116 \pm 0.043; \ p < .05\right)\), 5 min \(\left(7.284 \pm 0.073 \text{ vs. } 7.114 \pm 0.051; \ p < .05\right)\), and 15 min post-TT1 \(\left(7.426 \pm 0.092 \text{ vs. } 7.258 \pm 0.076; \ p < .05\right)\). There were no differences in blood pH between the treatments in the post-TT2 period \((p > .05)\).

Blood Lactate

Pre-TT1 blood lactate concentration was not different across treatments \((p > .05, \text{Figure } 2)\). In the post-TT1 period, blood lactate was significantly higher for B+C than for P at 5 min \(\left(15.0 \pm 1.9 \text{ vs. } 10.7 \pm 1.9 \text{ mmol/L; } p < .05\right)\) and 10 min (12.5

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**Table 3  Supplement Comparison for a One-Off 200-m Freestyle Performance, \(M \pm SD, N = 6\)**

<table>
<thead>
<tr>
<th>Supplement comparison</th>
<th>Effect (%), intervention TT1 – placebo TT1</th>
<th>(p)</th>
<th>Effect size (95% CL)</th>
<th>Effect magnitude/Practical outcome (effect of using a placebo over the 2nd listed supplement)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C vs. P</td>
<td>1.1 ± 0.9</td>
<td>.027*</td>
<td>0.42 ± 0.35</td>
<td>Moderate benefit (faster) to trivial effect</td>
</tr>
<tr>
<td>B vs. P</td>
<td>0.6 ± 2.1</td>
<td>.484</td>
<td>0.24 ± 0.83</td>
<td>Moderate benefit (faster) to small harm (slower)</td>
</tr>
<tr>
<td>B+C vs. P</td>
<td>−1.0 ± 1.0</td>
<td>.547</td>
<td>−0.18 ± 0.73</td>
<td>Small benefit (faster) to moderate harm (slower)</td>
</tr>
</tbody>
</table>

*Note. CL = confidence limits; C = caffeine; P = placebo; B = bicarbonate; B+C = bicarbonate + caffeine.*

*Significant difference observed in taking one supplement over the other.*
Figure 2 — Whole-blood HCO₃, pH, and lactate concentration after 200-m repeat freestyle performance following bicarbonate (B), caffeine (C), bicarbonate + caffeine (B+C), or placebo (P) ingestion in elite male swimmers (N = 6). α = baseline significantly different than pre-TT1 and pre-TT2 for B (p < .01) and pre-TT1 for B+C (p < .05). β = B significantly different than C and P (p < .05). γ = B+C significantly different than C (p < .05). δ = baseline significantly different than pre-TT2 for B (p < .01). *B significantly different than C (p < .05). #Baseline significantly different than pre-TT1 for B and B+C (p < .01). ε = B+C significantly different than P (p < .05). Solid black bar represents each 200-m swimming time trial. Data presented as M ± SD.
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± 3.3 vs. 8.4 ± 2.8 mmol/L; p < .05). At the pre-TT2 time point, 25 min after TT1, blood lactate concentrations for all treatments had returned to near baseline levels. Pre-TT2 blood lactate concentration was not different across treatments (p > .05). After TT2, however, B+C ingestion resulted in significantly higher blood lactate concentrations than P at 0 min (15.3 ± 1.9 vs. 10.9 ± 2.1 mmol/L; p < .05), 3 min (15.8 ± 1.6 vs. 11.2 ± 2.3 mmol/L; p < .05), 5 min (14.9 ± 2.9 vs. 10.8 ± 2.2 mmol/L; p < .05), and 10 min post-TT2 (13.4 ± 2.2 vs. 9.3 ± 2.6 mmol/L; p < .05).

Questionnaire

At the end of the study, 4 of the 6 participants were able to successfully determine the order in which their supplements had been administered. The ability of some participants to successfully determine treatment order during caffeine trials has been previously reported (Doherty et al., 2004; Graham, Hibbert, & Sathasivam, 1998; Jackman et al., 1996). The reasons for this are reportedly the jittery and energetic sensations experienced by participants after caffeine ingestion (Graham et al.). Some participants in the current study also reported discomfort (stomach cramp, diarrhea, or dizziness) resulting from bicarbonate or caffeine ingestion.

Discussion

The current study is the first to compare the effects of ingesting NaHCO₃, caffeine, and their combination on repeated 200-m freestyle performance. The findings reveal that although there were no significant differences between treatments for performance time in either TT1 or TT2, ingesting C alone resulted in a significantly slower 200-m performance time in TT2 compared with TT1 than when NaHCO₃ was ingested with or without C. It appears that this is because of lower blood pH and blood HCO₃⁻ in TT1 after C ingestion.

NaHCO₃ supplementation elevates extracellular blood HCO₃⁻, increasing the pH gradient across the cell membrane. This promotes the efflux of H⁺ and lactate out of the muscle cell during exercise, thereby preserving intracellular pH levels (Sutton et al., 1981) and delaying the onset of fatigue (Bishop et al., 2004; Costill et al., 1984). Conversely, a low extracellular HCO₃⁻ might limit the muscle’s glycolytic capacity (Sutton et al., 1981) and increase the time course of recovery of maximal force after fatigue (Mainwood & Cechetto, 1980). In the current study, blood HCO₃⁻ was significantly reduced after TT1 after the ingestion of C and remained significantly lower than baseline levels at pre-TT2, 25 min after the first 200-m effort. A large decline in blood HCO₃⁻ was also observed after TT1 in the B and B+C trials, but because of preexercise NaHCO₃ ingestion pre-TT1 HCO₃⁻ for both of these trials was significantly elevated compared with baseline. The recovery of blood HCO₃⁻ in the post-TT1 period appears to have been superior in the B trial, because only in this trial was pre-TT2 blood HCO₃⁻ still significantly higher than baseline.

These results suggest that caffeine supplementation is associated with a greater need for H⁺ buffering and therefore is associated with a slower recovery of blood HCO₃⁻ in the 25-min postexercise period. This is further demonstrated by the dissociation of the pH curve from the lactate curve in the post-TT1 period and clearly highlights the independent effects of caffeine and bicarbonate ingestion on postexercise acid-base balance. Post-TT1 lactate concentrations were highest
in the C-containing treatments, suggesting that caffeine might have facilitated a greater anaerobic contribution to performance. In contrast, the B-containing treatments resulted in the highest post-TT1 pH concentrations, indicating a more alkaline environment, because of higher preexercise blood HCO$_3^-$ and the additional buffering capacity that this produced. Given that this buffering capacity is absent in C, the trend toward greater post-TT1 acidosis might be explained by C’s capacity to increase glycogenolysis, generating greater anaerobic metabolism. The C-containing supplements produced a greater drop in blood pH after TT1, but only in the C trial was blood pH significantly lower than B in the post-TT1 period. Therefore, mean swim times in TT2 compared with TT1 were slower for C than for B and B+C. Similar to previous findings on repeated exercise (Bishop et al., 2004; Costill et al., 1984; Sutton et al., 1981), NaHCO$_3$ ingestion enabled participants to either maintain or improve their repeated exercise performance whether taken alone (B) or in combination with caffeine (B+C), although it is difficult to determine whether there is any benefit of taking one supplement over the other. On the other hand, there appears a clear benefit in taking either of these supplements over C alone. The drop-off in performance time from TT1 to TT2 was significantly greater when C was ingested than with B, B+C, and possibly even P, but given the uncertainty of the performance outcome, further investigation comparing C and P is required.

The negative impact of ingesting caffeine on repeated short-term high-intensity exercise performance has previously been reported. Greer et al. (1998) found that caffeine had no effect on power output in the first two sprints and had a negative effect compared with placebo in the last two of four 30-s Wingate sprints separated by 4 min of rest. They attributed their results to the use of recreationally active participants, rather than highly trained anaerobic athletes, and the possibility that caffeine’s ergogenic effect is elicited primarily in oxidative muscle fibers. The former is consistent with previous findings (Collomp et al., 1992), but the latter is debatable given that other researchers have shown an ergogenic effect of caffeine during short-term high-intensity exercise predominantly involving fast-twitch muscle fibers (Anselme et al., 1992; Collomp et al.). Notably, caffeine appeared only to maintain mean swimming velocity in the second 100-m swim in the former study (Collomp et al.), and in the latter study it appeared to increase pedal frequency during four 6-s sprint efforts, thereby improving work output, although the highest workload achieved was the same with or without caffeine (Anselme et al.). Therefore, the real benefit to repetitive high-intensity-exercise performance remains unclear. In the current study, even though statistical significance was not achieved, the mean performance times for TT1 indicate that C was second-fastest only to B+C, and mean swim times under placebo conditions were fourth-fastest of all four supplements tested. This suggests that caffeine provides some benefit for a one-off 200-m performance (Table 3) and might have elicited some ergogenic benefit in TT1, but the cost of the initial effort seems to have hindered the repeat 200-m performance.

Caffeine ingestion before repeated high-intensity exercise has previously been reported to produce elevated postexercise blood lactate concentrations, generated by greater anaerobic metabolism and improved total work output (Anselme et al., 1992; Collomp et al., 1992). In the current study, and at least one other (Greer et al., 1998), ingesting C alone did not result in significantly higher postexercise blood
lactate concentrations or improved work output during repeated maximal exercise. Jackman et al. (1996) demonstrated that caffeine ingestion increased muscle lactate during two 2-min bouts of maximal cycling, but this was not evident in the blood at the end of the exercise period. This might be because of differences in the blood pH and HCO$_3^-$ of the extracellular fluid, which influence the rates at which lactate leaves the muscle cell (Sutton et al., 1981). A 2-min cycling bout (Jackman et al.) or a 200-m swim as in the present protocol might produce a greater extracellular acidosis, leading to lower extracellular HCO$_3^-$ than that observed over shorter swim distances (Collomp et al.), thereby limiting lactate removal from the muscle (Sutton et al.) and the subsequent rise in blood lactate concentrations. The greater extracellular acidosis created after ingestion of C in the current study, coupled with the attenuated increase in blood lactate concentration, supports the theory that the rate of lactate removal from the muscle might be impaired during acidosis (McCartney, Heigenhauser, & Jones, 1983; Sutton et al.). In contrast, when C is ingested in combination with an induced metabolic alkalosis as in B+C, there was a marked increase in extracellular blood lactate in the post-time-trial periods. Furthermore, given that blood pH for B and B+C were both elevated before TT1, the difference in blood pH restoration before TT2 suggests that there was a greater efflux of H$^+$ out of the muscles in the post-TT1 period for B+C. This indicates that B+C might have generated greater production of intracellular lactic acid, likely because of the caffeine component.

A greater intracellular lactic-acid production in B+C might indicate a greater anaerobic energy contribution (Collomp et al., 1992; Nevill, Boobis, Brooks, & Williams, 1989), but for B+C this did not translate into significantly faster performance times in TT1 or TT2 ($p = .06$). It is interesting to note, however, that most freestyle swimmers recorded their fastest performance times in TT1 (3 out of 6) and TT2 (4 out of 6), and 1 participant recorded his personal-best time when supplemented with B+C. Supplementation using B+C resulted in unique and considerable changes to the underlying physiology of the participants, but these changes did not translate into substantial differences in 200-m swimming performance. This might be the result of the small participant number, which is a limitation of the current study. A larger sample size might have provided the statistical power to achieve significant differences in performance time at $p < .05$. Alternatively, it might suggest there are other factors, apart from acidosis, contributing to the onset of fatigue during maximal exercise. Nielsen, de Paoli, and Overgaard (2001) reported that the exercise-induced increase in K$^+$ was responsible for depressing the production of force in rat muscles while the subsequent acidosis actually acted as a protective mechanism. As such, it is possible that the changes in intracellular and extracellular K$^+$ coupled with the decline in Na$^+$-K$^+$-ATPase activity during maximal exercise might influence muscle function, contributing to fatigue (Leppik et al., 2004). These factors must be considered with regard to the onset of fatigue in 200-m swimming performance, but their specific involvement is beyond the scope of this study.

Although previous research has investigated the rate of appearance of caffeine in the blood after caffeine supplementation (Cox et al., 2002), the interaction between NaHCO$_3$ supplementation, caffeine supplementation, and a standardized warm-up on the rate of appearance of caffeine and HCO$_3^-$ in the blood is unknown and should also be considered when examining possible explanations for the variance in performance.
In summary, although C alone might be beneficial for a one-off 200-m performance, its efficacy for multiple performances close together appears limited. Ingesting C alone significantly slowed swimmers in their second 200-m swim compared with ingesting B, B+C, and potentially even P. The findings of this study demonstrate there is a clear benefit on repeated short-term high-intensity performance after taking NaHCO$_3$, with or without C, over C alone. The ergogenic advantage of caffeine supplementation on repeated exercise performance seems limited, and as such, its benefit to sports of a high-intensity repetitive or even intermittent nature might be questionable. It is well known that NaHCO$_3$ supplementation enhances repeated exercise performance, but further research is required to elucidate whether the ingestion of B+C provides any advantage over the ingestion of B alone for this type of exercise.

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**References**


